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UNITED STATES ARMY
IN WORLD WAR II

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MEDICAL DEPARTMENT, UNITED STATES ARMY

PREVENTIVE MEDICINE IN WORLD WAR II

Volume IV

COMMUNICABLE DISEASES

Transmitted Chiefly Through

RESPIRATORY AND ALIMENTARY TRACTS

Prepared and published under the direction of

Major General S. B. HAYS

The Surgeon General, United States Army

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Volume IV

COMMUNICABLE DISEASES

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MEDICAL DEPARTMENT, UNITED STATES ARMY

The volumes comprising the official history of the Medical Department of the United States Army in World War II are prepared by The Historical Unit, United States Army Medical Service, and published under the direction of The Surgeon General, United States Army. These volumes are divided into two series: (1) The administrative or operational series; and (2) the professional, or clinical and technical, series. This is one of the volumes published in the latter series.

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Foreword

It is well known from histories of wars that diseases caused by infectious and parasitic agents the communicable diseases of men and animals have inflicted great losses upon armies and related groups essential to a military effort. At times, these diseases have impeded campaigns or have contributed to the defeat of armed forces. At other times, they have been milder deterrents. In all instances, however, either as threats or as actual attackers, they have presented large and difficult problems. Military leaders and their medical supporters have always been aware of the handicaps imposed by diseases among troops and of the need to protect troops against disability of sickness, or possibly loss by death, resulting from infectious diseases. These lessons from the past, with almost appalling projections into the imminent future, imparted both urgency and comprehensiveness to the Medical Department's program for the prevention and control of communicable diseases in the Army during World War II. Through its actions in this field, the Medical Department functioned as a part of the administration of the whole Army.

During World War II, the problems of the prevention and control of communicable diseases in the Army, and in the populations with which the Army was in contact, were of unprecedented complexity and magnitude. A story of the activities of the Medical Department in dealing with these problems, together with some account of deficiencies and successes, will occupy at least three volumes in the preventive medicine series of the history of the Medical Department in World War II. This foreword to volume IV is, therefore, the prologue to the presentation that will fill several volumes.

The prevention and control of communicable diseases constituted the major segment of the general mission of the Medical Department in securing and maintaining the health of the Army. The understanding of why this was so is readily grasped from apprehending that the segment included a myriad of diseases caused by bacteria, spirochetes, rickettsiae, viruses, fungi, protozoa, helminths, ectoparasites, and toxins, transmitted from man to man and from animals to man by a large variety of means and vectors, under such variations of global epidemiology as made some familiar diseases seem almost as exotic as the newly encountered diseases of foreign lands.

The mission was carried out through many organizations and devices. Among these were the Preventive Medicine Service in the Office of the Surgeon General; the Preventive Medicine Divisions in the Offices of the Chief Surgeons of theaters of operations; the medical components of combat units in the theaters; units of civil affairs and military government; and appropriate units in the Zone of Interior in the continental United States and in the overseas Departments. Governmental agencies, particularly the United States Public

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Health Service and the entomological divisions of the Department of Agriculture, numerous civilian foundations and organizations in the health field, and research advisory bodies such as the National Research Council's Division of Medical Sciences and the Committee on Medical Research of the Office of Scientific Research and Development, and hundreds of individual civilian consultants were drawn into a concerted attack on these problems. In addition, two new organizations were created within the Army and War Department to reinforce this attack; namely, the Army Epidemiological Board and the United States of America Typhus Commission. The power thus assembled was equal to the national, international, and global task of operating military preventive medicine in an army spread over the world.

The work was done in the tradition established by Surgeon General George M. Sternberg, "father of American bacteriology," who, during his long term of office from 1893 to 1902, initiated the modern extensive program of military preventive medicine that was so effectively applied to the prevention and control of communicable diseases in World War II. This program included research, the application of the latest scientific methods for disease control based on knowledge of the infectious agents, modes of transmission, biology of vectors, susceptibility of human and animal hosts, epidemiological data, environmental factors, and collaboration between the Army Medical Department and all appropriate governmental and civilian agencies.

The success achieved during World War II is indicated by the reduction of deaths from disease to a number below that of deaths from battle. In the Spanish-American War, approximately 5 American soliders died of disease to 1 killed in battle. In World War I, the ratio was about even 1:1. In World War II, the ratio was 0.07:1.

The prevention and control of malaria and typhus by the use of the new insecticides developed during the war deserve to be ranked among the greatest scientific achievements of the time. Demonstrations in important military and civilian situations showed how these scourges may be eradicated.

The results of operational military preventive medicine in the control of communicable diseases in the Army during World War II, set forth in this and subsequent volumes, confirm the conviction that "a strong program of preventive medicine is a basic requirement for the successful conduct of modern war." They inure also to the benefit of civil public health.

S. B. Hays,
Major General,
The Surgeon General.

Preface

Although military preventive medicine has greatly broadened its scope, the control of communicable diseases continues to be the central activity. In the present volume and two to follow, the primary objectives are to indicate the magnitude of the communicable disease factor in United States Army operations in World War II and to define and characterize the problems of communicable disease in military practice as distinct from those of civilian life.

There has been a notable decline in death rates from communicable diseases in the United States and in many other countries. In World War II, for the first time in the history of United States Army wartime military operations, the number of deaths from disease in the Army was less than the number of deaths from battle casualties. This fact is encouraging and reflects great credit upon the Medical Department and those individuals who were primarily concerned with the health and welfare of the troops. The death rates do not, however, tell the whole story. During World War II, disease ranked first among the three major categories of military casualties (disease, battle casualty, and nonbattle casualty) as a cause of disability in the Army and was the main component in the noneffective rates. Disease was therefore a significant drain on the operating efficiency of the Army.

There are many reasons why the communicable diseases must continue to be a major concern of military preventive medicine. The prime military consideration is the maintenance of a maximally effective fighting force. Experiences of World War II demonstrated that this must be achieved for troops operating in all parts of the world, under a wide variety of climatic, sanitary, and epidemiologic environments. Rates and ranges of movement and concentration and other circumstances of troops, as well as local populations and prisoners of war, expose military personnel to hazards of communicable disease not present in peacetime civilian life. Such conditions as the diarrheas and dysenteries, epidemic hepatitis, common upper respiratory infections, fungus infections, the exanthemata of childhood, and many others present special problems in wartime military practice. Vigilance, planning, and foresight and the proper indoctrination and education of commanders and troops at all echelons are needed to control these conditions as well as to minimize the likelihood of epidemics of internationally quarantinable diseases such as smallpox and plague.

The authors of chapters in this volume were chosen, as were authors of the other volumes of this series, by the Advisory Editorial Board for the preventive medicine volumes of the official history of the Medical Department in World War II. These authors have been selected because of their experience and distinction in their special fields. They are all very busy people, and it has therefore been the more gratifying that they have so willingly and generously given of their time to this project. Sincere appreciation is due to members of the

Advisory Editorial Board for their over-all planning and supervision as well as for help and advice, as a group and individually, in reviewing manuscripts and making suggestions in numerous detailed matters.

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Through the generous cooperation of the President, Chancellor, Administrative Officers, and Staff of the Medical College of Virginia, the work of the Editorial Office, located at the Medical College, has been facilitated and aided. Sincere thanks are expressed to President R. Blackwell Smith, Jr., Chancellor William T. Sanger, and Maj. Gen. William F. Tompkins, Comptroller. Their continuing support and encouragement is deeply appreciated. Thanks are accorded to Miss Margaret McCluer, Librarian of the Tompkins-McCaw Library of the Medical College of Virginia, and her staff for their friendly interest and assistance. Sincere thanks are also expressed to the following secretaries who worked on these manuscripts in the Editorial Office: Mrs. Jacqueline Pate, Mrs. Geraldine Glick, Mrs. Jeannette Martin, and Mrs. Virginia Wilson. Their painstaking attention to the technical details of manuscript preparation has greatly expedited this project. Dr. Audrey A. Bill served as a member of the Editorial Staff for several years and has contributed to the editorial preparation of several of the chapters of this volume. Her cooperation is gratefully acknowledged.

The relevancy of statistics in a volume on communicable diseases is self-evident. The authors and editors have relied greatly upon the services of the Medical Statistics Division. Mr. E. L. Hamilton, Chief, Mr. A. J. McDowell, Assistant Chief, and Mr. M. C. Rossoff, Assistant Chief, Statistical Analyses Branch, have not only provided essential data but also checked and reviewed all statistical information in this volume. We are greatly indebted to them. The Scientific Illustration Division, Medical Illustration Service, Armed Forces Institute of Pathology, under the direction of Mr. Herman Van Cott, prepared the illustrations for this volume. We are happy to express to the various branches of the Historical Unit appreciation of their work: The Research and Archives Branch, devotedly led by Mrs. Josephine P. Kyle, provided essential source material and spent many hours locating and verifying references; the Administrative Branch, first under direction of Mrs. Catherine Marshall and later of Mrs. Hazel Hine, gave administrative support and typed the final copies of these chapters.

EBBE CURTIS HOFF, Ph. D., M. D.

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Part I

INTRODUCTION

CHAPTER I

General Considerations of Modes of Transmission

John E. Gordon, M. D.

The control of communicable disease remains the basic activity of military preventive medicine. Preventive medicine had its origin in the control of communicable disease and for many years was largely restricted to this field. As reasonable and useful additions were made to the program for prevention, the infections came to have relatively less emphasis, but the total effort expended in their control is still much the same. The typical organization of preventive services at the combat level in World War II consisted of a section each on epidemiology, venereal disease control, nutrition, and sanitation. Two of the four sections were wholly concerned with the control of communicable diseases, and the other two had much to do with limiting the spread of infection. Comprehensive programs were planned by the Office of the Surgeon General and the theater commands, but the outstanding activity was still the control of communicable disease.

Programs of prevention have been broadened materially in the course of years to include battle casualties and nonbattle injuries as well as disease—the three recognized classes of death and disability in military practice. A refined knowledge of wound ballistics has led to major developments in body armor and other protective devices, along with changes in schemes for evacuation of the wounded. Nonbattle injury has superseded disease, including communicable disease, in importance as a cause of death in the Armed Forces in both peace and war, with the result that traumatic injury of accidental origin and poisoning due to alcohol and to other toxic agents take a rightful place in preventive programs.

The concept of prevention of disease itself also has enlarged. Military medicine has had a leading part in the development of the science of mental health. Nutrition is recognized as a primary feature of preventive medicine. Occupational medicine originally attracted attention when it was restricted to industry, in which the Armed Forces have many interests related to the production of armaments; now it has become a matter of concern with respect to the health hazards which arise through the specific occupation of a soldier. Noninfective mass disease is not neglected, although conditions of this class have less significance in the Armed Forces than in a general population because the bulk of a military population is in the younger age groups.

The continuing emphasis on communicable disease in the organization of military preventive services and in the underlying policies of military physicians holds in spite of a generally appreciated and well-documented decrease in deaths from communicable disease in the United States and in many other countries. Progress is most definite in nations of the Western World, but even there a measurable irregularity exists with respect to the various specific infectious processes. The development of antibiotics and the expansion of chemotherapy are a main influence in the lowering of the fatality rate; another influence is the lesser frequency of cases and hence the lowering of morbidity rates. The opinion is increasingly heard in general medical circles that the problems of communicable diseases are largely of a bygone day and that infections are now so adequately controlled that eradication in many instances is just around the corner. The military appraisal that communicable disease is far from being conquered presumably has justification. Military decisions of strategic nature rarely are made without a sound marshaling of facts.

COMMUNICABLE DISEASE IN MILITARY PRACTICE

A possible explanation of the emphasis on the communicable diseases in military preventive medicine is that military operations provide a situation different from that of civilian existence. Certain communicable diseases are in fact recognized as peculiarly military diseases, the diarrheas and the dysenteries and epidemic hepatitis being typical examples. Hemolytic streptococcal infections are frequently exaggerated in populations of young adults, such as an army. The common communicable diseases of childhood—mumps, measles, and the rest—always have been more of a problem under military conditions than in civilian life.

The potentiality of epidemics for a variety of diseases conceivably may be greater under military conditions, or the risk of epidemics may center peculiarly in those infections collectively grouped as the internationally quarantinable diseases (smallpox, plague) which flare up into world disaster because of the conditions of war.

The nature of an army population may be a decisive factor; military populations are young populations reasonably envisaged as having more than their share of infectious disease as compared with a general population.

The military physician, in developing his opinion of communicable disease, also may employ criteria other than those usual in civilian health practice, or he may have information not ordinarily available. For years, the costs of the communicable diseases have been measured in terms of the deaths they cause, largely by necessity, for the information on deaths is more complete and more reliable than that for incidence, length of disability, or residual defect. Morbidity reporting has never reached a satisfactory performance in any country, with the possible exception of Denmark. There is, furthermore, no need to deny the common clinical as well as lay opinion that so long as a disease does not kill it is hardly worthy of note. Nevertheless, many diseases, such

as the common cold and numerous helminthic infections, which have a far-reaching effect socially, economically, and medically, have little or no fatality rating. And, for those infectious diseases which are frequently fatal, morbidity rates supplement the knowledge derived from death rates. The practitioner of military preventive medicine has at his disposal information on morbidity of a quality not available in any other social organization; records of admission to hospital and quarters are a part of the military system. These data permit sound evaluation of noneffectiveness and of the reasons for discharge for medical disability. In military practice, both considerations are important, but no more so than in civilian practice.

The daily noneffective rate, that is, the number of men absent from duty, is an index of practical usefulness to physician and commander alike, for it measures, in part, the fighting efficiency of an army. It is a measure of disability; it is a measure of morbidity as contrasted to mortality; and, since an account is required of each man on the roster, it has unusual precision.

From a military standpoint, the loss of a man through discharge for permanent disability is equally as significant as a loss by death. In both instances, the Armed Forces are permanently depleted. The records of these losses are likewise complete. The costs of communicable disease are thus determined in terms of death, disability, and defect; that is, in terms of total cost. To evaluate the total cost of communicable disease is an endeavor incapable of practical fulfillment under conditions other than those of military practice. In the Army, judgment of a communicable disease and of communicable diseases in general thus rests on a broader base. A part of the optimism about control of the communicable diseases seemingly depends on opinion derived mainly from the number of resultant deaths, too little weight being placed on disability and permanent defect resulting from these diseases.

Newer and improved methods of mass control and clinical management have led to gains in the control of communicable diseases which are more or less evident everywhere. These gains are far from being evenly distributed, however. Within countries and between countries, economic and social conditions have limited the application of the newer and improved methods; a deficiency in medical facilities and professional training is sometimes a factor. The environment of Tropics and Arctics often introduces factors not present in the Temperate Zone where these newer methods have been standardized, with the result that procedure becomes more complicated or suffers in efficiency. At any rate, in large areas of the world containing the greater part of the human population, the communicable diseases are still the main health problem. Modern war calls for troops to operate in these regions. The different environment and the greater seeding of infection may well lead to a poorer result in the control of communicable disease, even when Americans are managed under American methods.

For these and other reasons, the Army experience, in which more than 10 million Americans served under conditions foreign to their usual environment

and mode of life, affords an opportunity to measure more accurately the extent of the progress that has been made in the control of communicable disease.

To look upon the communicable diseases as being now under control is at best provincial. Proof that they are under control scarcely exists so far as the United States is concerned, although the gains in the direction of control have been great. Communicable diseases are certainly not under control on a world-wide scale; and, in peace as well as in war, the world tends increasingly to become a single epidemiologic universe. Opinion concerning progress in the control of communicable diseases is too frequently based on deaths alone, an incomplete measure. Disability and residual defect are other primary considerations; good examples of diseases in which these two considerations are particularly important are acute upper respiratory infections and modern scarlet fever, respectively. That progress has been made in the control of communicable diseases is evident; conclusions on the extent of that progress are too often extrapolated beyond the existing evidence.

The record of the behavior of the communicable diseases in World War II is presented in this and other volumes of the preventive medicine series of the history with the primary purpose of demonstrating how much of a factor those diseases were in the conduct of military operations at that time. A secondary aim is to define those particular features which characterize the communicable diseases in military practice as contrasted with usual civilian conditions. The experience of this war was comprehensive, without parallel in the history of warfare. Similar methods and the same general policies were employed under a wide variety of environmental situations, from the Tropics to the Arctic. Conceivably, this experience may lead to an improved evaluation of the place of the communicable diseases in the public health practice of today. The first consideration is the system of classification of military casualties.

Classes of Military Casualties

Military casualties are divided into the following three categories: Battle casualties, nonbattle injuries, and those the result of disease. Disease is thus set apart from injury as a source of disability. Injury is further broken down into injuries of battle origin and those of noncombat origin. The classification of disabilities is generally clear cut but is sometimes arbitrarily made. For example, trenchfoot contracted in the line is classified as a nonbattle injury. Furthermore, similar disabilities may be classified as battle or noncombat in origin, depending on the circumstances under which the injury was sustained. A gunshot wound of the hand incurred accidentally in a training area or self-inflicted anywhere is a nonbattle injury and is distinct from the battle injury in which the same kind of wound results from contact with the enemy. The definition of terms that follows is taken from War Department Army Regulations No. 40-1080, dated 28 August 1945.

Patients are classified according to the primary cause of initial admission, and their cases are reported in one of the following three categories: Disease, nonbattle injury, or battle casualty. In instances of patients suffering from

both disease and injury at the time of initial admission, the most serious condition present is taken as the primary cause of initial admission and determines the classification. Patients admitted for a battle casualty and a disease or injury are classed as battle casualties. Thus, both primary and secondary diagnoses must be taken into account in determining the frequency of any particular condition, such as one of the communicable diseases.

All cases other than those due to injury or battle casualty are classed as disease. Included among the disease cases are those of patients suffering from reactions to medication other than acute poisoning, those of patients admitted for the sequela of an injury they had incurred before entering service, and those of patients readmitted for the results of a traumatism (battle or nonbattle) incurred during service.

Nonbattle injury includes traumatisms other than those defined as battle casualties. Traumatism refers to morbid conditions due to external causes. The term is applied to acute poisoning, except food poisoning, and to the results of exposure to heat, cold, and light as well as to various types of wounds.

A battle casualty is a traumatism (wound or injury) which either is incurred as a direct result of enemy action during combat or otherwise or is sustained by an individual while he is immediately engaged in, going to or returning from, a combat mission. It does not include traumatisms occurring on purely training flights or missions. Psychiatric cases occurring in combat are not reported as battle casualties.

The measurement of losses from whatever cause is accomplished by computation of rates that relate to three principal demographic characteristics. The first of these, mortality rate, is an expression of the number of deaths from a particular cause that occur per unit of population and time, a frequent unit of population in military practice being 1,000 men and the interval of time 1 year. Mortality rates in this discussion are usually on the more practical basis of 100,000 average strength per year. The rates for shorter periods are based on the assumption that the observed frequency would have continued over a year. The mortality rate represents a definite and certain military loss, irrespective of cause, of time, or of nature, and is one of the absolute indexes of the cost of war.

The morbidity rate, when expressed as an admission rate, refers to the number of persons affected by a given condition as determined by patients admitted to hospital or quarters and regularly is computed in terms of 1,000 per annum per average strength. Morbidity rates as so defined represent not all persons affected but only (1) those persons admitted to hospital or quarters (those seriously enough involved to be absent from duty), and (2), for most diseases, certain patients who are treated while they remain in a duty status and whose cases are carded for record only. Nevertheless, these indexes of illness as employed in military practice are more satisfactorily indicative of the existing situation than is usual in public health or preventive medicine, because reporting is particularly good. The significance of any particular morbidity rate as an influence on tactical and strategical operations depends, in the first

instance, on the duration of the disability ordinarily associated with the condition; secondly, on the expected fatality; and, finally, on the prospect for complete recovery and return to duty.

The daily noneffective rate represents the number of men absent from duty by reason of disease or injury for each 1,000 troop strength per day. The complementary value shows the proportion available for duty at any prescribed time. Persons suffering permanent disability either partial or complete as a result of a disease or injury of sufficient seriousness to interfere with ordinary duties are discharged from military service. The designation of separation for disability is thus a measure of permanent defect, and numbers are listed by cause in relation to population concerned.

Most of the data on cases of disease in this and succeeding volumes are preliminary data based on sample tabulations of individual medical records. They include both primary and secondary diagnoses. As such, they are designated incidence. Admission rates refer only to primary diagnoses. Morbidity thus may be expressed in either term. Incidence is usual. The final tabulations will introduce some changes but presumably not enough to affect the interpretations made. In some instances, information is from weekly summary reports, field records which lack the reliability of analysis from individual medical records.

Data on deaths are on the same basis as data for cases, except that total deaths of the war (table 1) are from The Adjutant General's final report. Examination of individual case records will result in some rearrangement of the data of table 1 but not in number of deaths.

TABLE 1.—*Admissions and deaths, by classification, U. S. Army, 1942-45*

Classification	Admissions		Deaths	
	Number	Percent of total	Number	Percent of total
Disease	[1] 14,969,028	85.2	15,779	5.1
Battle casualties	[2] 592,170	3.4	[3] 234,874	75.2
Nonbattle injury	1,995,398	11.4	61,640	19.7
Total	17,556,596	100.0	312,293	100.0

[1] Includes certain cases (mostly venereal disease cases) treated while the patient remained in duty status.

[2] Includes 556 admissions during December 1941 and 4 during 1946.

[3] This total is comprised of 192,798 killed in action, 26,762 dead of wounds and injuries, 6,058 declared dead, and 9,256 dead from nonbattle causes while captured or missing in action. Deaths for December 1941 are included.

Source: Battle admission data and all deaths, *Army Battle Casualties and Nonbattle Deaths in World War II*, Final Report, 7 Dec. 1941-31 Dec. 1945, The Adjutant General's Office. Nonbattle admissions, preliminary data based on sample tabulations of individual medical records.

The communicable diseases are listed within a number of categories in the official tabulations. The most significant medically and from a military standpoint are the infectious and parasitic diseases, which represent those communi-

cable processes of specifically determined etiology; they have an estimated incidence of 126.4 cases per annum per 1,000 average strength. The list is essentially that of the Office of Vital Statistics, United States Public Health Service, except that influenza, bacterial pneumonia, and rheumatic fever are included. The largest block of communicable disease is the common respiratory diseases with an annual rate for the total Army of 159.3 per 1,000. The diarrheas, excluding the dysenteries, are another appreciable source of morbidity; incidence 29.1 per 1,000 per year. Pneumonias other than specific bacterial, incidence 10.1, and fever of undertermined origin, incidence 5.7, are to be added. Miscellaneous items of lesser number, corresponding to those listed in Control of Communicable Disease in Man (American Public Health Association, 1955) and not included among the categories given above contribute a further incidence of 12.7 per annum per 1,000; the main items are dermatophytosis, impetigo contagiosum, and trachoma. The greater proportion of the conditions listed are infectious and reasonably to be added to the specific infectious and parasitic diseases in accumulating a total of communicable disease. Rates of incidence as here cited are in part estimates, particularly for cases reported as secondary diagnoses during 1942 and 1943. The incidence for all communicable disease on this basis is 343.3 per annum per 1,000.

The information on admissions (primary diagnoses) is almost complete. The rate for disease for the total Army during 1942–45 was 587.5 per annum per 1,000 average strength. Similar data for the communicable diseases as just presented give an admission rate of 310.4. Thus, in the total Army during the war, more than one-half of all reported disease was of a communicable nature.

The aim now is to determine the place of communicable disease among military casualties, and the influence of these diseases on military operations, in this war and in relation to other wars and other years. The practical approach initially is through disease of all forms principally, because the communicable diseases were more than one-half of total disease in World War II, because the exact numbers of infections remain indeterminate by reason of indefinite clinical identification within several large groups, and because in records of earlier years the separation of communicable from other disease processes was still looser.

DISEASE AS A FACTOR IN MILITARY OPERATIONS

The ratio of deaths from disease to deaths from battle casualties for the wars of the 18th and 19th centuries was sometimes as great as 12 to 1. A generally accepted ratio was 4 to 1; as for example, in the Russo-Turkish War of 1877–78 where deaths from disease numbered approximately 80,000 and those from battle casualties 20,000. The ratio during the campaign in the Crimea was even greater, with some 70,000 deaths from disease and 7,500 from battle casualties among the French forces. Approximately three-fifths of the disease

and battle deaths that occurred in the Union Army during the American Civil War were deaths from disease, which marked an improvement over the Mexican War when deaths from disease outnumbered those from battle casualties in the proportion of 6 to 1. The ratio during the Spanish-American War was lower, with an excess of deaths from disease over losses in battle in the proportion of about 5 to 1.

Fewer deaths from disease than from battle casualties were noted for the first time in the War of 1864 which Denmark waged against Austria and Prussia (table 2). Both opponents established a ratio of 1 death from disease to 2 for casualties of battle. Although the number of men engaged in that war was small, communications between the armies and home countries were good and environmental conditions were favorable. The War of 1864 was nevertheless a remarkable event—a turning point in the history of wars. In the Franco-Prussian War of 1870–71, the first major war to see the new ratio maintained, the German Army had a proportion of 0.86 deaths from disease for each battle casualty. The health record of the German Army has been consistently good, for of five wars, dating from the Danish action of 1864 and including World War I, deaths from disease have been less than those from battle casualties with the single exception of the War of 1866 and that was close to parity. The Russo-Japanese War of 1904, the next great conflict after the Franco-Prussian War, gave the Japanese forces an opportunity to set a new ratio of 0.37 deaths from disease per battle casualty.

World War I was the first United States experience in which deaths from disease were fewer than deaths from battle casualties, and then this was true only in relation to troops in the active European campaign of 1918 (table 2). For the United States Army as a whole and for all men under arms, the rate was still slightly greater for disease. World War II brought a complete departure from previous experience and a health record never approached previously in any war. The ratio of deaths from disease to those from battle casualties was 0.07:1.

The gains which have been made in recent times are primarily due to improved control of acute infectious disease. Not only are deaths from this cause far less frequent in proportion to those at risk, but the case incidence of communicable disease is decidedly less. This has brought significant changes in the qualitative character of the losses that still result from disease as distinguished from injury and battle casualties. Noncommunicable disease has become increasingly important, especially psychiatric disorders, aside from considerations of loss by death.

Of all deaths for all United States troops under arms in World War II, 75.2 percent were due to battle casualties, 19.7 percent to nonbattle injury, and 5.1 percent to disease (table 1). The established excess of deaths from disease over deaths from battle injury was strongly reversed in World War II. Even nonbattle injuries became a more important source of fatal casualties. Deaths, however, are not the sole measure of costs of disease and injury, nor are they

TABLE 2.—*Deaths from disease and battle deaths in principal wars, foreign armies and U. S. Army, 1846-1945*

War	Date	Deaths from—		Ratio of deaths from disease to deaths from battle injuries and wounds
		Disease	Battle injuries and wounds [1]	
Mexican War (United States) [2]	25 Apr. 1846–5 July 1848.	11, 155	1, 721	6. 48:1
Crimean War (French)	1854–56	70, 000	7, 500	9. 33:1
Civil War (North)	15 Apr. 1861–1 Aug. 1865.	[3] 199, 720	138, 154	1. 45:1
Danish War	1864			
German		310	738	. 42:1
Danish		820	1, 446	. 57:1
German War (German)	1866	5, 219	4, 008	1. 30:1
Franco-Prussian War (German)	1870–71	14, 904	17, 225	. 86:1
Russo-Turkish War	1877–78	80, 000	20, 000	4. 00:1
Sino-Japanese War (Japanese)	1894–95	15, 850	1, 311	12. 09:1
Spanish-American War	1 May 1898–31 Aug. 1898.	1, 939	369	5. 25:1
Philippine Insurrection	February 1899–December 1902.	4, 356	1, 061	4. 11:1
Boer War (British)	1899–1901	11, 377	6, 425	1. 77:1
War in Southwest Africa (German)	1904–7	689	802	. 86:1
Russo-Japanese War	1904–5			
Japanese		21, 802	58, 257	. 37:1
Russian, less Port Arthur		18, 830	23, 008	. 82:1
World War I	1 Apr. 1917–31 Dec. 1918.			
Total United States Army		51, 447	[4] 50, 510	1. 02:1
American Expeditionary Forces.		16, 951	[4] 50, 105	. 34:1
World War II	7 Dec. 1941–31 Dec. 1945.			
Total United States Army		15, 779	234, 874	. 07:1
United States Army in Europe		1, 779	135, 576	. 01:1

[1] Includes deaths due to disease or nonbattle injury while captured or missing in action.

[2] Data are derived in part from Historical Register and Dictionary of the United States Army, 1789-1903, vol. 2, Francis B. Heitman, Government Printing Office, 1903, p. 282, and are somewhat understated.

[3] Includes disease deaths among the relatively small number of volunteers remaining in Federal service subsequent to 1 Aug. 1865.

[4] Includes gas casualties.

Source: (1) Data for Crimean, Danish, German, Franco-Prussian, Russo-Turkish, Sino-Japanese, Boer, Southwest Africa, and Russo-Japanese Wars: Gordon, J. E., and Aycock, W. L.: Preventive Medicine and Epidemiology. Am. J. M. Sc. 215: 311-326, January-June 1948. (2) Data for Civil War: Statistical Exhibit of Deaths in United States Army During the Late War. The Adjutant General, Washington, 1885. (3) Data for Spanish-American War and Philippine Insurrection: Annual reports of The Adjutant General, 1899 and 1899-1904, respectively. (4) Data for World War I: Annual Report of The Surgeon General, 1918-19; The Medical Department of the United States Army in the World War. Statistics. Washington: Government Printing Office, 1925, vol. XV, pt. 2; and Annual Report of The Adjutant General, 1926. (5) Data for World War II: Army Battle Casualties and Nonbattle Deaths in World War II, Final Report, 7 Dec. 1941-31 Dec. 1945, The Adjutant General's Office.

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always the most indicative. The intent now is to examine disease in World War II in relation to the two classes of injuries, battle and nonbattle, and then to relate the specific infectious and parasitic diseases to disease as a whole.

Mortality From Disease

Disease in World War II ranked third among major classes of deaths (table 1). The contrast with other wars is extreme, where deaths from diseases outnumbered the total of all others and sometimes in a proportion inversely as great as that presented here. On the basis of deaths, disease clearly has decreased significantly as a factor in military operations.

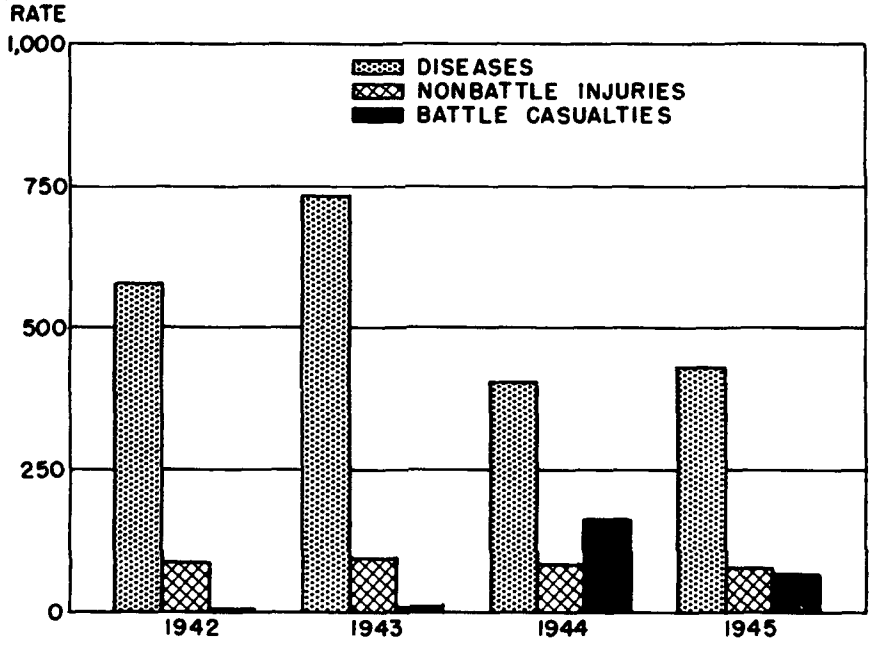
Disease Morbidity

As a cause of disability in World War II, disease ranked first among the three major categories of military casualties (table 1); in fact, the number of admissions for disease was more than five times as great as the number of admissions for battle casualties and nonbattle injuries. Somewhat more than 85 percent of all admissions to hospital and quarters were because of conditions

CHART 1.—*Admissions to hospitals and quarters for diseases, nonbattle injuries, and battle casualties, ETOUSA, 1942–45* [1]

[Preliminary data based on tabulations of individual medical records and summaries of statistical health reports]

[Rate expressed as number of admissions per annum per 1,000 average strength]



[1] These data may include certain cases carded for record only and treated while the patient was in a duty status.

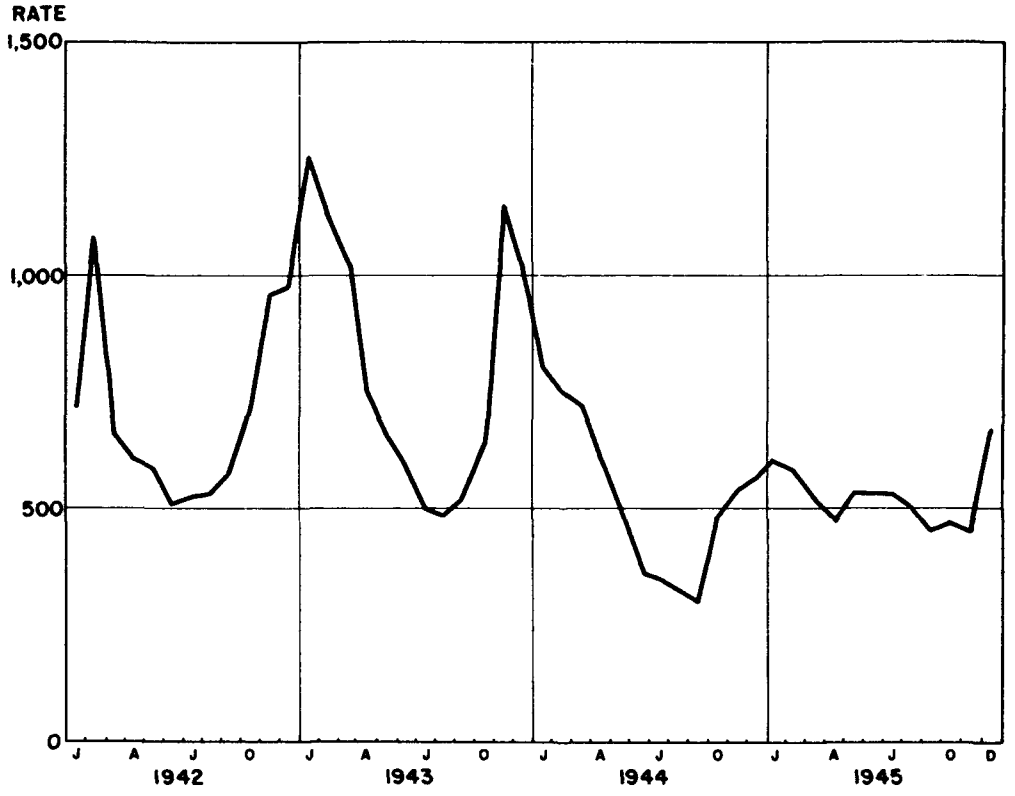
classed as disease. The experience of ETOUSA (European Theater of Operations, United States Army) is cited as an example since that theater was the largest and since environmental conditions there were those of a temperate region.

For each of the 4 years that the European theater was in existence, disease was the most frequent cause of admission to hospital or quarters (chart 1). The highest admission rate was in 1943, the widespread epidemic of influenza in the autumn of that year being a contributing factor. The rates from year to year showed little variation, irrespective of whether the battle was fast or slow. There was no direct correlation between the activity of military operations and the frequency of disease. The numbers of persons affected were regularly great, since each year about one out of every two soldiers tended to suffer some disability from disease of sufficient degree to interfere with military duties. The regularly occurring annual peak of incidence in late autumn or early winter (chart 2) shows common respiratory infection to be the dominant factor in frequency of disability due to disease.

CHART 2.— *Admissions to hospitals and quarters for all diseases, ETOUSA, by month, 1942–45* [1]

[Preliminary data from statistical health reports]

[Rate expressed as number of admissions per annum per 1,000 average strength]



[1] These data may include some readmissions.

No particular significance attaches to the experience of the first 2 years in the European theater. The morbidity rates for disease were in all respects satisfactory and the health record good. The striking feature is in respect to the last 2 years. During the height of the campaign in continental Europe which started in the middle of 1944 and ended in late spring of 1945, the morbidity rates for disease as judged by admission to hospital and quarters were at a lower level (chart 2) than at any other time during the war. Proverbially and throughout the history of wars, this is the time when losses have been great. Granted that many soldiers will not report sick during the height of military operations, and particularly in time of advance, nevertheless, the fact that so few were disabled by disease is perhaps the clearest evidence of the progress made in environmental sanitation and in the practice of preventive measures.

Noneffective Rates for Disease

The proportion of a command absent from duty on a particular day is a reflection of current morbidity and of the deaths that occur. The extent of noneffectiveness is also an index of the kinds of disability, for those of disease in the age groups that characterize an army are commonly short while those of injury whether of battle or nonbattle origin tend to be longer. Disease is the main component of noneffective rates. For the Army as a whole and over the war period, the preliminary estimates per day per 1,000 average strength are 6.38 for nonbattle injury, 7.73 for battle casualty, and 30.22 for disease.

The significance of the noneffective rate and the drain on operating efficiency of an army is expressed in simpler terms by consideration of the number of man-days lost. Estimates in round numbers show a total of 72 million days lost because of battle casualties for all the Army troops, including the Air Force, during World War II. This exceeds the corresponding figure of 59,863,000 for nonbattle injuries, but scarcely approaches the loss of 285,918,000 days attributed to disease (table 3). A comparable estimate for days lost because of infectious and parasitic disease is 55,688,000 days of the total 285,918,000. The experience of ETOUSA again is drawn upon to indicate

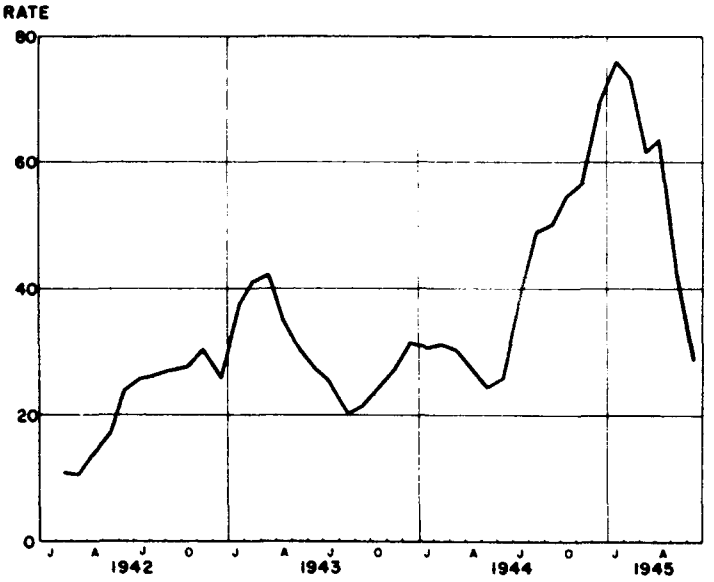
TABLE 3.—*Number and percentage of man-days lost, by classification of casualties, U. S. Army, 1942–45*

[Preliminary estimates based on sample tabulations of individual medical records]

Classification of casualties	Number of man-days lost	Percent of total
Disease	285, 918, 000	68. 5
Battle casualties	72, 000, 000	17. 2
Nonbattle injury	59, 863, 000	14. 3
Total	417, 781, 000	100. 0

CHART 3.—*Average daily noneffective rates, ETOUSA, by months, February 1942 to June 1945, inclusive*

[Rate expressed as average daily number noneffective per 1,000 average strength]



Source: Weekly summary reports, ETOUSA.

variations in noneffectiveness over time (chart 3). The data are from field records but are sufficiently reliable to establish relationships and trends.

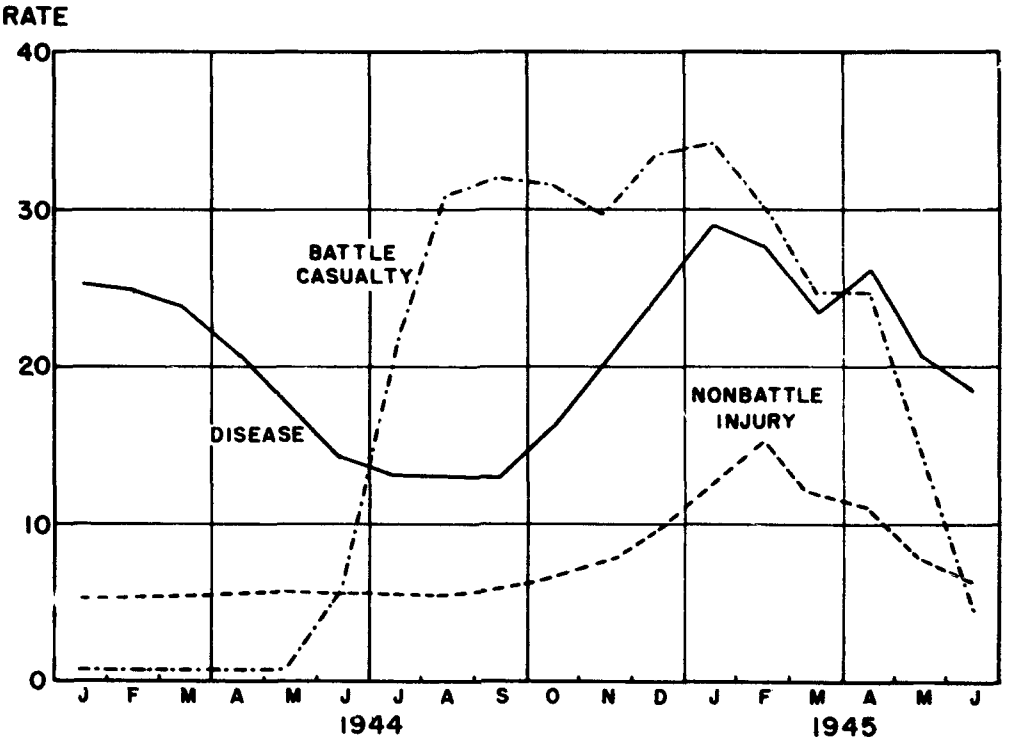
Discounting the early part of 1942 when the small troop strength of the European theater accounted for irregularities in the demonstrated pattern, each year of the 4-year period of World War II saw the high point of noneffectiveness centered about the early months of the calendar year and minimal values during the summer. The seasonal incidence of upper respiratory infections was the main influence on this fluctuation. Variations from year to year were not great until the latter part of 1944 when the values for all months increased precipitately over the established norm. This was coincident with the beginning of active operations in Continental Europe.

The division of this particular combat experience into the three components which make up the total noneffective rate (chart 4) gives ready demonstration of the factors involved. The noneffectiveness related to disease continued according to established pattern, with rates in 1945 almost identical with those that characterized 1944, the year just preceding the campaign. A significant part of the excess noneffectiveness came about through a greater frequency of nonbattle injuries, principally cold injury. The most important variable was that of battle casualties, with the data of chart 4 demonstrating clearly the high noneffectiveness of the campaign period as due to that cause.

The generalizations to be drawn from this experience are that year in and year out the principal cause of noneffectiveness of troops is disease. The losses from nonbattle injuries are ordinarily much less, about one-fifth of those from

CHART 4.—*Average daily noneffective rates for disease, nonbattle injury, and battle casualty, ETOUSA, January 1944 to June 1945 inclusive*

[Rate expressed as average daily number noneffective per 1,000 average strength]



Source: Weekly summary reports, ETOUSA.

disease. The noneffectiveness that comes from battle casualties is subject to great variation wholly related to the nature of operations. The impression is not to be left that the cost of battle casualties is unpredictable, for the expected losses in a major operation can be estimated with an exactness rivaling those of disease and injury.

All three classes of casualties give rise to irregular fluctuations in morbidity and in noneffectiveness which can be related with much certainty to environmental, seasonal, or other ecologic factors. The peaks of excess incidence that mark the behavior of battle casualties and nonbattle injuries may be as outstanding as any introduced into the general curve through action of an epidemic of disease.

Discharge for Permanent Disability

The final consideration of noneffectiveness is that of separation from the military service by reason of physical or other disability, variously due to residual effect of battle casualty, nonbattle injury, or disease. Such losses in World War II greatly exceeded the losses from death; death accounted for 312,293 absolute losses, separation because of significantly impaired usefulness the much greater number of 956,232. The distribution according to class of

casualty is shown in table 4, the data being for enlisted men only. Battle casualties were a greater factor than nonbattle injuries, but disease was almost eight times the sum of the other two.

TABLE 4.—*Separation from service, by cause, enlisted men only, U. S. Army, 1942–45*

[Data based on individual reports of separation received from The Adjutant General]

Cause of separation	Number of separations	Percent of total
Disease:		
Infectious and parasitic diseases	25, 115	2. 6
Other diseases	821, 038	85. 9
Total	846, 153	88. 5
Traumatism:		
Nonbattle injuries	(1)	[2] 3. 2
Battle casualties	(1)	[2] 8. 3
Total	110, 079	11. 5
Grand total	956, 232	100. 0

[1] Data are not presently available.
[2] Based on 90,794 disability separations due to results of traumatisms during 1942 and 1945.

The endeavor now is to identify the contribution of the communicable diseases to these several rates and indexes. The main reliance is on those conditions collectively grouped as the infectious and parasitic diseases, a category used in common in both military and civilian vital statistics although with minor variation in the diseases included. The group is not synonymous with the communicable diseases. Many diarrheas are surely dysentery, to such an extent that the two conditions are considered jointly in a subsequent chapter. They cannot be adequately separated. An interpretation based solely on confirmed bacillary dysentery, even with clinically recognized dysentery added, is not representative. The common respiratory diseases presumably include a goodly proportion of the influenzas; this group is excluded and so is a part of the pneumonias. Large numbers of fever of undetermined origin in malarial zones are actually malaria. The infectious and parasitic diseases (as listed in table 10, p. 26) are however the main problem and in large measure determine the military significance of the communicable diseases.

THE SPECIFIC INFECTIOUS AND PARASITIC DISEASES

The long-term behavior of the infectious and parasitic diseases in respect to the general population of the United States is to be ascertained through comparison of deaths from these diseases with deaths from all causes; it is a

useful control in judgment of the military experience. Deaths are the necessary criteria because reporting of cases in the general population is incomplete for all of the communicable diseases, and a number are not reported at all. Selected years indicate a consistent and impressive downward trend (table 5). Infectious and parasitic diseases, as listed by the Bureau of the Census in Vital Statistics of the United States, do not include pneumonia and influenza, whereas United States Army tabulations for World War II include bacterial pneumonia and influenza.

TABLE 5.—*Deaths, all causes, and deaths from infectious and parasitic diseases, total United States, for 1900, 1925, and 1950*

Year	Total deaths, all causes	Deaths, infectious and parasitic diseases [1]	
		Number	Percent of total deaths, all causes
1900	539,939	113,059	20.9
1925	1,219,019	143,958	11.8
1950	1,452,454	51,650	3.6

[1] Does not include pneumonia and influenza.

Source: (1) U. S. Department of Commerce, Bureau of the Census, Mortality Statistics, 1900–4, pp. LXVIII, LXIX. (2) U. S. Department of Commerce, Bureau of the Census, Mortality Statistics, 1925, pt. I, pp. 137–140. (3) U. S. Department of Commerce, Bureau of the Census, Vital Statistics of the United States, 1950, vol. III, p. 76.

Death rates for infectious and parasitic diseases show a material variation (table 6). Rates increase progressively with age, tuberculosis being a major influence. The proportion of deaths from infectious and parasitic diseases to deaths from all causes is materially greater at the younger ages. One reason for the emphasis on communicable diseases in military practice is thus evident. Military populations have a strong bias in ages in which the infectious and parasitic diseases account for the largest proportion of deaths and also a bias in males, among whom death rates exceed those for females.

The greater problem presented by the infectious and parasitic diseases in the general as contrasted with the military population of the United States is illustrated in chart 5 where the rates for the two populations are compared by 5-year periods from 1900 to 1950. The list of infectious and parasitic diseases is again that of the Office of Vital Statistics, pneumonia and influenza being deleted from the Army data. The frequency is regularly less in the military population (chart 5) which is primarily of young adults. The more valid comparison is with males aged from 20 to 29 years of the general population; the advantage still holds.

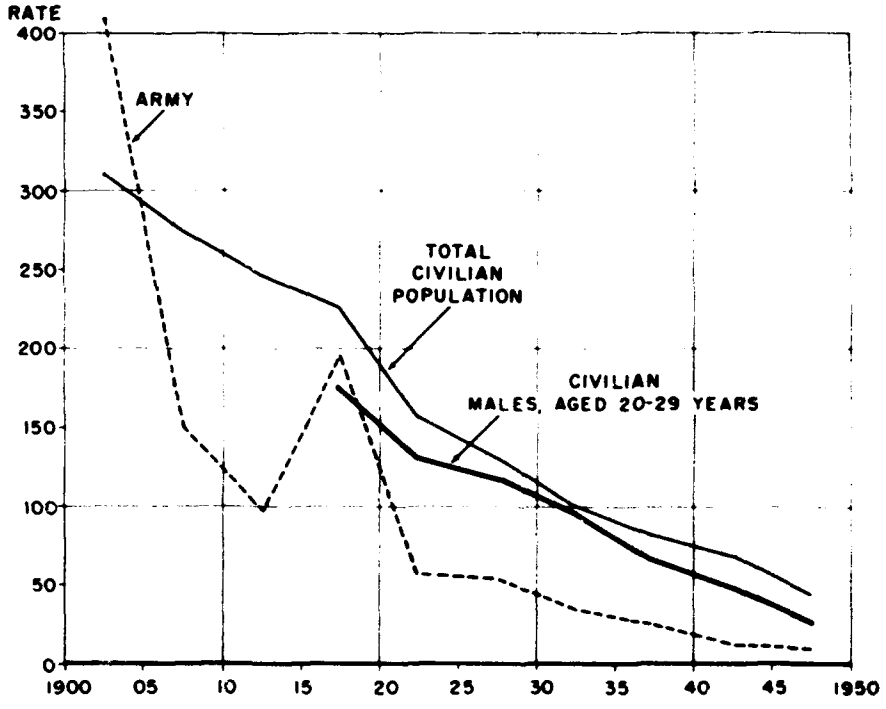
TABLE 6. *Average annual deaths, all causes, and deaths from infectious and parasitic diseases, total United States, by age groups, 1948–52*

Age in years	Total deaths, all causes, 5-year average	Deaths from infectious and parasitic diseases		
		Number, 5-year average	Rate	Percent of total deaths from all causes
Under 10	136,377	7,212	24.5	5.3
10 to 19	18,686	2,091	9.6	11.2
20 to 29	36,711	5,118	21.5	13.9
30 to 39	55,611	6,484	28.5	11.7
40 to 49	108,014	8,097	42.0	7.5
50 to 59	201,822	9,495	61.2	4.7
60 or over	905,400	16,199	88.4	1.8
Total	[2] 1,463,867	[3] 54,736	36.3	3.7

[1] Expressed as number per annum per 100,000 population.
[2] Includes 1,246 deaths reported as "age not stated."
[3] Includes 40 deaths reported as "age not stated."
Source: U. S. Department of Commerce, Bureau of the Census, Vital Statistics of the United States, 1948–52

CHART 5.—*Deaths from infectious and parasitic diseases (with pneumonia and influenza excluded from Army data), 1900–50*

[Rate expressed as deaths per annum per 100,000 population]



Source: U. S. Department of Commerce, Bureau of the Census, Vital Statistics of the United States, 1900–50; Annual Reports of The Surgeon General, 1900–41 (1941 unpublished); and preliminary tabulations of individual medical records, 1942–50.

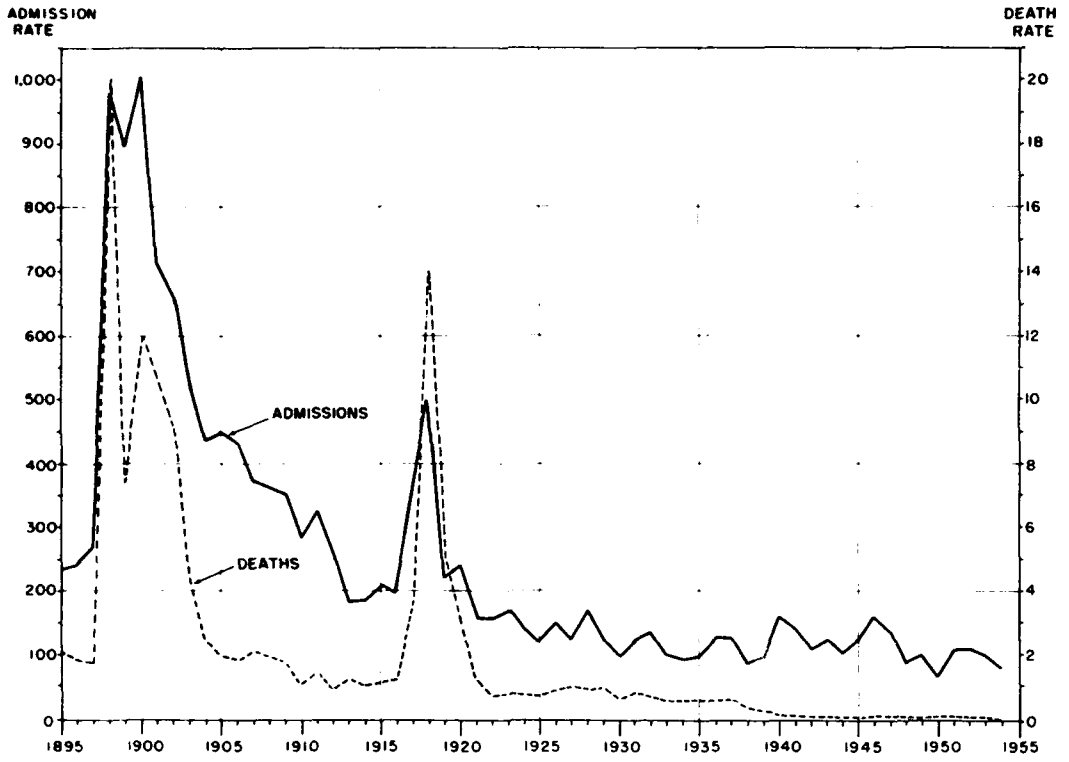
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Army Experience Over the Years

The extent of the problem provided by the infectious and parasitic diseases is measurable for the Army in terms of both morbidity and mortality, something which is not possible for the civilian population. Admissions to hospital and quarters represent, within limits of human fallibility, all illnesses due to specific infections where specific infection was the primary diagnosis. Because of coding practices which prevailed from 1924 through 1943, data for that period as well as for the war years 1942–45 may be considered for all practical purposes as representing incidence; namely, both primary and secondary diagnoses. Data from 1946 are admissions, primary diagnosis, only. The series of data in chart 6 includes the last five wars of this country. They mark the coming and going of epidemics and the intervening endemic periods but, withal, the continuing downward trend in morbidity and mortality. The high point in cases and deaths is that of the Spanish-American War, 1898, with an admission rate somewhat less than 1,000 per annum per 1,000 average strength and a death rate of 21 per 1,000. The influenza epidemic of

CHART 6. *Admission*[1] *and death rates for infectious and parasitic diseases, in the U. S. Army, 1895–1954*

[Rate expressed as number of admissions or deaths per annum per 1,000 average strength]



[1] Data for 1924 through 1945 also include secondary diagnoses of most of the diseases classified as infectious and parasitic.

Source: Annual Reports of The Surgeon General of the Army, 1895–1941 (1941 unpublished) and 1953–54, and preliminary tabulations of individual medical records, 1942–52.

1918 and the lesser event of 1920 are readily identified. Mobilization years of 1917 and 1940 produced higher rates as recruits were brought together in large numbers. The fluctuations from year to year were sometimes great, but the outstanding observation is that deaths from this group of diseases showed almost no departure during World War II from the established trend; this was despite potential hazards never before faced by our military forces.

Previous Wars

The changing behavior of the communicable diseases is strikingly brought out by comparison of morbidity and mortality rates for infectious and parasitic diseases in the several wars of United States history for which records are reasonably reliable (table 7). Deaths per 1,000 average strength have dropped

TABLE 7.—*Admissions and deaths from infectious and parasitic diseases, U. S. Army, in 4 major wars, 1861–1945*

[Admission rate and death rate expressed as number per annum per 1,000 average strength]

War	Admission rate	Death rate
Civil War (white Union troops) (1861–65)	[1] 1,030.34	[1] 34.77
Spanish-American War (1898)	[1] 986.89	[1] 20.81
World War I (1 Apr. 1917–31 Dec. 1919)	[2] 427.03	[2] 10.43
World War II (1942–45)	112.46	.15

[1] Includes all types of pneumonia.
[2] Excludes rheumatic fever.

Source: (1) The Medical and Surgical History of the War of the Rebellion. Medical History. Washington: Government Printing Office, 1870, vol. I, pt. I, pp. 636–641. (2) Annual Report of The Surgeon General, 1900, p. 402. (3) The Medical Department of the United States Army in the World War. Statistics. Washington: Government Printing Office, 1925, vol. XV, pt. 2, pp. 86–108, 134–148. (4) World War II data from preliminary tabulations of individual medical records.

from a rate of 34.77 for white Union troops of the Civil War (1861–65) to a rate of 0.15 in World War II (1942–45). Morbidity has by no means decreased proportionately, but the admission rate of 112.46 in World War II is wholly satisfactory in relation to the admission rate of 1,030.34 of the Civil War. Each successive war showed definite and progressive improvement over its predecessor; the proportionate gain in World War II over World War I far outdistanced all others.

World War II

About 20 percent of all reported disease in the Army for the war years of 1942–45 was in that group classed as infectious and parasitic diseases, excluding rheumatic fever, the number of cases being nearly 3,200,000 to give an incidence

rate of 124 per annum per 1,000 average strength (table 8). Numbers of cases were about equal for troops stationed in the United States and overseas, but the rates were materially greater for overseas troops. Troops stationed in continental United States had a rate of 107; the rate for those serving abroad was 148. The proportion of infectious and parasitic diseases to all disease was of similar order, for 18 percent of reported cases at home were of this nature; the frequency for troops serving in theaters of operations was 26 percent.

TABLE 8.—*Admissions for all disease and incidence of infectious and parasitic diseases in the U. S. Army, by theater or area of admission, 1942–45* [1]

[Preliminary data based on sample tabulations of individual medical records]

Theater or area	Admissions for disease	Incidence of infectious and parasitic diseases [2]		
		Cases (number)	Rate [3]	Ratio to all diseases [4]
Continental United States	8, 818, 758	1, 574, 613	106. 80	17. 9
Overseas:				
Europe	1, 946, 168	467, 364	106. 24	24. 0
Mediterranean [5]	1, 108, 207	329, 166	221. 95	29. 7
Middle East	122, 804	33, 341	228. 06	27. 1
China-Burma-India	349, 056	108. 298	246. 99	31. 0
Southwest Pacific	1, 273, 413	356, 721	194. 24	28. 0
Central and South Pacific	761, 306	183, 866	146. 28	24. 2
North America [6]	240, 344	26, 250	53. 31	10. 9
Latin America	234, 530	59, 588	156. 24	25. 4
Total overseas [7]	6, 150, 270	1, 592, 713	148. 35	25. 9
Total Army [8]	14, 969, 028	3, 167, 326	124. 31	21. 2

[1] Includes certain cases treated on a duty basis, most of which were venereal disease cases.
[2] Excludes rhematic fever. (The rheumatic fever rate in the total Army was 0.80.)
[3] Expressed as number of cases per annum per 1,000 average strength.
[4] Number of cases per 100 disease admissions.
[5] Includes North Africa.
[6] Includes Alaska and Iceland.
[7] Includes admissions on board transports.
[8] Does not include 31,850 secondary cases estimated for 1942 and 1943 for those diseases identified by footnote 4 in table 10.

Parallel relationships held for deaths from specific communicable disease (table 9). The proportion of deaths from infections to deaths from disease of all forms in the Army as a whole was measurably greater, however, than the similar ratio for cases: 25 percent for deaths and 20 percent for cases. For troops stationed in the United States, some 20 percent of all deaths from disease were due to infections; the proportion overseas was just about three-fifths greater, or 32 percent. The spread between absolute death rates from infectious disease at home and abroad was also strikingly different—11 per annum

per 100,000 average strength in the United States and 20 overseas. Stated in other fashion, the risk of contracting an infectious disease during service overseas was greater than at home and the risk of death from such disease was still greater.

A possible explanation of this situation is that the communicable diseases of overseas areas included infectious processes characterized by higher fatality than existed in the United States. There is the second possibility that the kinds of infectious disease were much the same but with enhanced fatality related to environmental differences. A third consideration is that the decisive factor may be the stress and strain of combat, a mode of life in the field contrasted with that in training and in barracks and with attendant difficulties in providing an equal quality of medical care. The same considerations enter into explanation of the attack rates abroad, which are appreciably greater than under home conditions but less so than the observed differences in death rates.

TABLE 9.—*Deaths from all disease and from infectious and parasitic diseases in the U. S. Army, by theater or area of admission, 1942–45*

[Preliminary data based on tabulations of individual medical records]

Theater or area	All disease deaths	Deaths, infectious and parasitic diseases [1]		
		Number	Rate [2]	Percent of all disease deaths
Continental United States	8,061	1,572	10.66	19.5
Overseas:				
Europe	2,314	532	12.09	23.0
Mediterranean [3]	926	290	19.55	31.3
Middle East	109	42	28.73	38.5
China-Burma-India	454	234	53.37	51.5
Southwest Pacific	1,639	755	41.11	46.1
Central and South Pacific	780	176	14.00	22.6
North America [4]	254	48	9.75	18.9
Latin America	210	47	12.32	22.4
Total overseas [5]	6,844	2,170	20.21	31.7
Total Army	14,905	3,742	14.69	25.1

[1] Excludes rheumatic fever deaths. (Total rheumatic fever death rate was 0.11.)
[2] Expressed as number of deaths per annum per 100,000 average strength.
[3] Includes North Africa.
[4] Includes Alaska and Iceland.
[5] Includes deaths among transport admissions.

The contribution of the infectious and parasitic diseases to the total amount of disease resulting in separation from the service because of disability was insignificant. Among enlisted men only 2.6 percent of total separations

were for these diseases, as opposed to 88.5 percent for disease of all forms. The total number of separations because of the aftereffects of infectious and parasitic disease is, however, impressive; in all, 25,115 among enlisted personnel.

The distributions of infectious disease by theaters of operations, by the several years of military operations, and by the kinds of communicable disease that characterized each may now be examined to advantage. To do this, it is necessary to know the size and nature of the problem in its entirety; that is, to know the kinds of infectious and parasitic disease, with the number of admissions or cases and of deaths for each, experienced by the United States Army in World War II (table 10).

Distribution by Areas and Theaters of Operations

Incidence of infectious and parasitic disease varied greatly from one theater to another, from a low rate of 53 per annum per 1,000 average strength in the North American area (table 8) to a high of 247 in the China-Burma-India theater. Other theaters and areas with high incidence were the Middle East (rate 228), Mediterranean (rate 222), and the Southwest Pacific (rate 194). A scant familiarity with these regions is enough to bring out the low levels of environmental sanitation, the greater frequency of infection among peoples resident there, and the common presence of some kinds of communicable disease scarcely known in temperate regions of the Western World.

High rates for communicable disease among foreign troops operating in such regions would be anticipated, because of the strange environment and because of a susceptibility presumably greater than that of persons indigenous to the region. It is a matter of satisfaction, however, that when environmental conditions were similar and risks comparable, the morbidity rates for communicable disease in soldiers overseas compared favorably with those of troops stationed in continental United States. The North American area and the European theater actually had better rates, although allowance must be made for the problems associated with recruits in the Zone of Interior and the selection and greater resistance of seasoned troops sent overseas.

Morbidity rates for infectious and parasitic diseases in the North American area (53 per annum per 1,000 average strength) were not only the best of any area or theater of operations but they were about one-half those for troops of continental United States. A portion of the territory of this theater was American, with the advantages of close cooperation with civilian health authorities and an organization for health which followed the United States pattern. Troops were isolated from contact with civilian populations more than in many situations, either at home or abroad. Troop strength was small and relatively stable, lacking the continual buildup which characterized so many operational areas. These factors presumably restricted the continuous seeding of a command with newly introduced infection. On the other hand, troops

of this region often were subjected to rigorous cold and a primitive environment which found expression in unusually high rates for nonbattle injury, a type of disability which then and now is a feature of cold climates. This environment might well have been expected to favor occurrence of communicable diseases. The observed rates are wholly commendable.

The European theater, the largest in respect to troop strength and the site of some of the most active combat of the war, had incidence rates for infectious and parasitic diseases which averaged 106 per annum per 1,000 average strength for the 4 war years, a rate about equal to that for continental United States, which was 107. Climate and environmental conditions were much the same as those to which troops were accustomed in the United States. For troops stationed in Britain, a high grade of cooperation existed with civilian health authorities and with an old and well-established health organization.

Two conclusions may be drawn. First, armed warfare does not of itself bring increased hazard of the communicable diseases to troops in areas of active combat. The European theater had rates about equal to those of continental United States which was a training area with no open warfare. Within the European theater itself, communicable disease was of more concern in service troops of the Communications Zone than in combat units in forward areas. Second, excess incidence of infectious disease seems in this experience clearly related to environments conducive to greater risk and to military operations in areas of known high prevalence of these diseases. Another reason for emphasis on communicable disease in military practice thus becomes evident — the need in global warfare to operate in regions where the communicable diseases are the main factor in morbidity and where rates for infectious and parasitic diseases in American troops may be expected to be greater than those prevailing in the home country.

Deaths by areas and theaters of operations

With minor exceptions, death rates from infectious and parasitic diseases arrange themselves in much the same order for the several theaters of operations as do rates for cases (table 9). When incidence is high, mortality is high. The China-Burma-India theater with highest incidence had a mortality rate of 53 per annum per 100,000 average strength and the North American area with the lowest incidence had the lowest death rate, 10 per 100,000. Material differences in rates between theaters are evident.

Death rates from communicable disease in the theaters have a greater spread over death rates of Zone of Interior troops than do rates of incidence in these two elements of command. For example, morbidity rates for these diseases as a whole in the China-Burma-India theater were a trifle more than twice those for continental United States; mortality rates were five times as great. The North American area had less than half the continental United States morbidity rate, but death rates were about equal in the two areas.

TABLE 10.—*Cases and deaths due to certain infectious and parasitic diseases[1] in the U. S. Army,[2] by diagnosis and area of admission, 1942-45*

[Preliminary data based on tabulations of individual medical records]

[Incidence rate expressed as number of cases per annum per 1,000 average strength; death rate expressed as number of deaths per annum per 100,000 average strength]

Diagnostic category and disease	Total Army				Continental United States		Overseas	
	Number of cases	Incidence rate	Number of deaths	Death rate	Incidence rate	Death rate	Incidence rate	Death rate
Diseases due to bacteria:								
Tuberculosis, pulmonary	27,135	1.06	556	2.18	1.33	1.63	0.71	2.93
Tuberculosis, nonpulmonary	4,413	.17	250	.98	.15	1.01	.21	.94
Gonorrhea	881,386	34.59	24	.09	31.52	.14	38.81	.04
Meningitis, meningococcal	12,925	.51	511	2.01	.67	2.58	.28	1.22
Scarlet fever	29,512	1.16	61	.24	1.77	.35	.32	.08
Erysipelas	2,398	.09	2	.01	.11	.01	.07	.01
Streptococcal sore throat [3]	20,471	1.33	12	.08	2.02	.07	.77	.08
Pneumonia, bacterial [4]	50,943	2.00	404	1.59	2.54	1.69	1.26	1.44
Intestinal infection [4]	21,477	.84	15	.06	.84	.03	.85	.10
Typhoid fever	505	.02	36	.14	.01	.02	.04	.31
Paratyphoid fever	839	.03	6	.02	.01	.01	.07	.05
Dysentery, bacillary	25,815	1.01	16	.06	.29	.01	2.00	.13
Diphtheria	5,724	.22	123	.48	.04	.06	.48	1.06
Pertussis	174	.01	0	0	.01	0	.00	0
Chancroid	99,361	3.90	1	.00	1.43	0	7.29	.01
Brucellosis	1,305	.05	2	.01	.06	.01	.03	.01
Tularemia	200	.01	4	.02	.01	.02	.00	.01
Anthrax	12	.00		0	.00	0	.00	0
Tetanus [5]	12	.00	5	.02	.00	.02	.00	.02
Glanders [4]	21	.00		0	.00	0	.00	0
Cholera	10	.00	2	.01	0	0	.00	.02
Plague		0		0	0	0	0	0
Gas bacillus infection [6]	[3] 2,649	[3] .17		0	[3] .01	0	[3] .30	0
Leprosy	26	.00		0	.00	0	.00	0
Other bacterial infection [4]	14,291	.56	110	.43	.59	.42	.52	.45

Diseases due to protozoa:								
Malaria	377,994	14.84	302	1.19	3.01	.07	31.08	2.72
Balantidium infection [4]	36	.00		0	.00	0	.00	0
Giardia infection [4]	1,212	.05		0	.03	0	.07	0
Trichomonas infection [4]	1,016	.04		0	.04	0	.04	0
Trypanosomiasis	14	.00		0	.00	0	.00	0
Leishmaniasis	497	.02		0	.00	0	.04	0
Amebiasis	34,524	1.35	39	.15	.24	.09	2.89	.24
Other protozoal infection [4]	2,913	.11	4	.02	.03	.03	.22	0
Diseases due to spirochetes:								
Neurosyphilis	9,739	.38	24	.09	.51	.12	.21	.06
Syphilis, other	318,380	12.50	46	.18	15.12	.23	8.89	.11
Vincent's infection [4]	100,230	3.93	8	.03	5.13	.01	2.29	.06
Relapsing fever	171	.01		0	.00	0	.01	0
Weil's disease	[3] 60	[3] .00	7	.03	[3] .00	.01	[3] .01	.06
Yaws	32	.00		0	.00	0	.00	0
Other spirochetal [4]	51	.00		0	.00	0	.00	0
Diseases due to viruses:								
Influenza	189,461	7.44	30	.12	9.33	.13	4.83	.10
Measles	60,809	2.39	33	.13	3.69	.19	.60	.05
German measles	135,830	5.33	7	.03	8.51	.04	.96	.01
Mumps	103,055	4.04	5	.02	5.59	.02	1.92	.02
Chickenpox	10,664	.42		0	.58	0	.20	0
Smallpox	117	.00	30	.12	.00	.01	.01	.27
Encephalitis, acute infectious [4]	434	.02	21	.08	.01	.06	.02	.11
Poliomyelitis, acute	1,006	.04	267	1.05	.03	.58	.05	1.69
Choriomeningitis, lymphocytic [4 7]	758	.03	1	.00	.03	0	.04	.01
Yellow fever		0		0	0	0	0	0
Dengue	90,861	3.57	4	.02	.01	0	8.46	.04
Sandfly fever	18,993	.75		0	.00	0	1.77	0
Rabies [5]	9	.00	6	.02	.00	.03	.00	.02
Herpes zoster [4]	10,119	.40		0	.37	0	.44	0
Keratoconjunctivitis, infectious [4 7]	684	.03		0	.04	0	.02	0
Lymphogranuloma venereum	14,999	.59		0	.64	0	.52	0
Infectious hepatitis [4]	[8] 182,383	[8] 7.16	[3] 259	[3] 1.69	[8] 3.17	[3] .26	[8] 12.63	[3] 2.85

See footnotes at end of table.

TABLE 10.—*Cases and deaths due to certain infectious and parasitic diseases*[1] *in the U. S. Army,*[2] *by diagnosis and area of admission, 1942–45*—Continued

[Preliminary data based on tabulations of individual medical records]

[Incidence rate expressed as number of cases per annum per 1,000 average strength; death rate expressed as number of deaths per annum per 100,000 average strength]

Diagnostic category and disease	Total Army				Continental United States		Overseas	
	Number of cases	Incidence rate	Number of deaths	Death rate	Incidence rate	Death rate	Incidence rate	Death rate
Diseases due to viruses—Continued								
Infectious mononucleosis [4]	[8] 18,532	[8] 0.73	[3] 3	[3] 0.02	[8] 0.92	[3] 0	[8] 0.46	[3] 0.04
Other virus diseases [5]	764	.03	1	.00	.04	.01	.01	0
Diseases due to rickettsia:								
Typhus, epidemic	104	.00	------	0	0	0	.01	0
Typhus, endemic	787	.03	15	.06	.03	.01	.03	.13
Scrub typhus	5,441	.21	283	1.11	.00	.01	.51	2.63
Typhus, unqualified [9]	[3] 109	[3] .01	[10] 16	[10] .09	[3] .00	[10] 0	[3] .01	[10] .18
Rocky Mountain Spotted Fever	131	.01	12	.05	.01	.08	0	0
Trench fever	[3] 15	[3] .00	(7)	[7] 0	0	0	[3] .00	[7] 0
Other rickettsial [4]	31	.00	2	.01	.00	0	.00	.02
Diseases due to fungi:								
Actinomycosis [4]	207	.01	4	.02	.01	.03	.01	0
Blastomycosis [4]	88	.00	5	.02	.00	.03	.00	.01
Favus [4] [11]	772	.03	------	0	.03	0	.03	0
Coccidioidomycosis	3,809	.15	39	.15	25	.22	.02	.07
Sporotrichosis [4]	48	.00	------	0	.00	0	.00	0
Torula infections [3]	5	.00	------	0	0	0	.00	0
Other fungus disease [4] [12]	8,729	.34	7	.03	.13	.03	.63	.03
Diseases due to helminths:								
Nematode infection	34,826	1.37	2	.01	.78	.01	2.19	.01
Ankylostomiasis [4]	22,238	.87	------	0	.53	0	1.35	0
Strongyloides infection [4]	1,242	.05	------	0	.03	0	.07	0
Ascaris infection [4]	5,031	.20	------	0	.10	0	.34	0
Enterobius infection [4]	1,272	.05	------	0	.03	0	.08	0

Filariasis [4]	2,483	.10	1	.00	.03	0	.19	.01
Onchocerciasis [4]		0		0	0	0	0	0
Trichinosis [4]	285	.01	1	.00	.01	.01	.01	0
Other nematode [4]	2,275	.09		0	.05	0	.15	0
Cestode infection	2,035	.08	1	.00	.09	.01	.06	0
Echinococcus infection [4]	22	.00	1	.00	.00	.01	.00	0
Cysticercosis	[3] 1	.00		0	[3] 0	0	[3] .00	0
Other cestode [4]	2,013	.08		0	.09	0	.06	0
Trematode infection	2,576	.10	2	.01	.01	.01	.22	.01
Schistosomiasis	2,541	.10	2	.01	.01	.01	.22	.01
Other trematode [4]	35	.00		0	.00	0	.00	0
Arthropod infection:								
Pediculosis [4]	6,673	.26		0	.32	0	.18	0
Scabies [4]	99,669	3.91		0	3.83	0	4.02	0
Other arthropod [4]	70	.00		0	.00	0	.01	0
Other infectious and parasitic diseases:								
Dysentery, unclassified	31,877	1.25	16	.06	.17	.01	2.74	.14
Granuloma inguinale	883	.03		0	.04	0	.02	0
Others [4]	1,707	.07	4	.02	.04	.01	.11	.03
Total [13]	3,167,326	124.31	3,742	14.69	106.80	10.66	148.35	20.21

[1] Excludes rheumatic fever.

[2] Includes data on Air Force personnel.

[3] For 1944–45 only; data are not presently available for 1942 and 1943.

[4] With respect to morbidity, data on this disease are shown in terms of admissions only for the 4-year period; data on secondary diagnosis cases for 1942–43 are not available.

[5] Because of special interest in deaths involving tetanus and rabies, these deaths have been shown even though not always in accordance with the rules of assignment of cause of death as stated in the Manual of Joint Causes of Death (4th ed.). Ordinarily, these deaths would be classified as due to the wounds or injuries which led to tetanus or rabies.

[6] According to the Manual of Joint Causes of Death (4th ed.), deaths in which this condition was present usually were classified to the wounds or injuries which preceded the infection.

[7] For 1943–45 only; data are not presently available for 1942.

[8] Estimated for 1942 and 1943.

[9] Data reported as unqualified typhus fever during 1942 and 1943 were coded as epidemic or endemic typhus, on the basis of geographic area and other information furnished on the medical record.

[10] Does not include 1943; data are not presently available.

[11] A preliminary study, involving a review of a sample of medical records of cases admitted in 1942 and 1943, revealed that approximately 40 percent of the cases shown as favus, actually should have been reported as tinea, other than tinea favosa.

[12] Except dermatophytosis, trichophytosis, etc.

[13] In addition to the data shown above, these totals include (a) some 1944–45 secondary diagnosis cases of those conditions which have been presented in terms of admissions only for the 4-year period (footnote 4), and (b) data which have not been presented for particular categories because they were judged to be understated.

NOTE: Absolute zero is indicated by zero in the units column; 0.0 indicates a rate of more than zero but less than 0.05 and 0.00 a rate of more than zero but less than 0.005.

The European theater had approximately the same incidence rate as continental United States but had a mortality rate of 12 per annum per 100,000 average strength compared with 11 for the continental United States. Broadly viewed, the morbidity rate for infectious and parasitic disease among all troops overseas compared with those at home was greater by about 39 percent. Death rates for these diseases overseas were essentially 100 percent more than in the Zone of Interior.

Possible explanations are that the kinds of infectious disease active in the two situations are different, that diseases present in common occur with greater severity, or that differences in quality and facilities for medical care are a determining factor. The validity of the first two assumptions may be determined from examination of the detailed frequencies of cases and deaths by individual diseases as presented in table 10. The third possibility is difficult to evaluate, involving as it does the balance between preventive and curative services and the lack of factual data capable of quantitative analysis. One conclusion is definite: death rates for infectious and parasitic disease in overseas troops are in this experience proportionately greater in relation to incidence than for Zone of Interior troops, a circumstance which holds whether absolute incidence in the particular theater is high or low.

This analysis of cases and deaths from infectious and parasitic disease in the war years of 1942–45 is now extended from differences according to place (overseas theaters of operations in comparison with the Zone of Interior) to a consideration of time relationships. The suggestion has been raised that the significance attached to communicable disease in military practice may rest in more frequent occurrence of epidemics of the usual fluctuating endemic diseases or in outbreaks or threatened outbreaks of the great pandemic diseases which include the designated internationally quarantinable diseases and influenza. If that be so, then irregularities in cases and deaths should be evident in random fashion from one year to another, affecting parts of the total command, and identifying local epidemics; or a uniform irregularity marking a single year, affecting all theaters, and establishing the presence of a pandemic of the nature of the influenza outbreak of the First World War.

Communicable diseases by years, 1942–45

An outstanding feature of the incidence of infectious and parasitic disease among troops of continental United States (1942–45) was the regularity of occurrence from one year to another (table 11). The rates were in close agreement; the best year was 1944 with a rate of 100 per 1,000 average strength.

No serious deviation in the proportion of cases of infectious and parasitic disease to all disease occurred during the 4-year period, either in the Zone of Interior or overseas; the ratio in both instances was greatest in 1945. Since the two major fractions of the command behaved in similar fashion, it follows that the same pattern held for the total Army.

Rates of incidence for overseas troops were on an average some 39 percent higher than for troops in the Zone of Interior, but the trend in behavior over

TABLE 11.—*Incidence and deaths from infectious and parasitic diseases in the U. S. Army, by area of admission and year, 1942–45* [1]

[Preliminary data based on tabulations of individual medical records]

Area and year	Incidence [2]			Deaths		
	Number of cases	Ratio to all disease admissions [3]	Rate [4]	Number	Percent of all disease deaths	Rate [5]
Continental United States:						
1942	281, 290	15. 9	105. 87	337	18. 6	12. 68
1943	562, 818	16. 1	108. 60	640	21. 7	12. 35
1944	397, 989	18. 6	100. 20	343	18. 4	8. 64
1945	332, 516	23. 4	113. 42	252	17. 7	8. 60
Total	1, 574, 613	17. 9	106. 80	1, 572	19. 5	10. 66
Overseas:						
1942	84, 006	23. 7	143. 41	96	23. 8	16. 39
1943	325, 668	25. 2	192. 94	358	33. 7	21. 21
1944	486, 635	22. 7	127. 42	727	30. 1	19. 04
1945	696, 404	29. 5	149. 98	989	33. 3	21. 30
Total	1, 592, 713	25. 9	148. 35	2, 170	31. 7	20. 21
Total Army:						
1942	365, 296	17. 2	112. 65	433	19. 5	13. 35
1943	888, 486	18. 6	129. 32	998	24. 8	14. 53
1944	884, 624	20. 7	113. 54	1, 070	25. 0	13. 73
1945	1, 028, 920	27. 2	135. 83	1, 241	28. 2	16. 38
Total	3, 167, 326	21. 2	124. 31	3, 742	25. 1	14. 69

[1] Excludes rheumatic fever.

[2] May be somewhat underreported for 1942 and 1943; secondary diagnosis cases for some of the infectious and parasitic diseases (see table 10, diagnoses identified by footnote 4) are not available.

[3] Number of cases per 100 disease admissions.

[4] Expressed as number per annum per 1,000 average strength.

[5] Expressed as number per annum per 100,000 average strength.

the 4 years was much the same for both. The second year of the war, 1943, had increased rates over the first year, appreciably so for troops overseas; the most favorable rates were in 1944, and incidence returned to the higher levels of 1942 and 1943 in the last year of combat.

Death rates for the infectious and parasitic diseases during the 4 years and for the Army as a whole followed the same pattern as the rates for cases except that the rise in 1945 was lacking for ZI troops, the shifting of units and rapid demobilization having an effect on troop strength and therefore on rates.

The ratio of deaths from infectious disease to deaths from all disease for the Army as a whole was fairly fixed throughout the war; the moderate increase in 1945 was related to troops overseas.

These four sets of data, the ratio of cases and of deaths from infectious disease to all disease and the annual rates for cases and deaths due to infectious processes, suggest the general behavior of communicable disease during the war to have been characterized in the first 2 years by a somewhat enhanced endemic level of infection, punctuated by local epidemics. This circumstance is usual for recruits brought together in large numbers as in the continental United States. Rubella and meningococcal meningitis in 1943 were good examples of the kinds of epidemics that occurred.

Troops arriving overseas had, in some instances, the advantage of fairly prolonged seasoning, but often they did not. Irrespective of seasoning, in foreign theaters they commonly faced infections with which they had had no previous experience,such as filariasis in the South Pacific and scrub typhus in the Southwest Pacific Area. Amebiasis, ordinarily an endemic disease, reached epidemic proportions in the China-Burma-India theater and several others. Both continental and overseas troops thus encountered an unfamiliar mode of life, a strange environment, and, for overseas troops, a life in places where risk of contact with infection was notably greater than any to which they had been accustomed.

The rates for infectious and parasitic disease that prevailed in the third year of the war seemingly represent expectancy in terms of a trained functioning army. Case rates and death rates were improved both in the Zone of Interior and overseas.

Both incidence and mortality were greater in 1945 for the Army as a whole. The incidence rate for home troops was in general comparable to that of the recruit years, but the mortality rate was as low as in 1944. For overseas troops, both rates were comparable to those of early adjustment to the new conditions of a theater. The excess was largely related to the period when the war ended and thereafter. Until the war ended, the record for 1945 was much the same as for 1944. In the postwar period, military discipline relaxed, and control measures were more difficult to apply; for some diseases, especially the venereal diseases, rates increased precipitately. Recovered prisoners of war also made their contribution to greater incidence and mortality, particularly in respect to tuberculosis.

Two medical events were of general occurrence, affecting troops in appreciable numbers both in the Zone of Interior and overseas. The first, in 1942, was the manmade epidemic of serum hepatitis; the second, in 1943, was an outbreak of influenza A. The data discount the outstanding prevalence of any one of the great epidemic diseases, the famous six quarantinable diseases.

Interest now turns to the kinds of communicable disease involved in the troubles of recruits in the Zone of Interior and of newcomers to an overseas theater of operations, to identify such local epidemics as occurred under both sets of circumstances, to compare the endemic disease that characterized the various theaters of operations, and especially to view the behavior of what history has long established as the infectious diseases peculiar to military operations.

Continental United States

The problems of specific communicable disease in continental United States centered mainly in the first 2 years of the war in which rapid buildup of the Army was taking place. They were the problems of recruits.

Tuberculosis of all forms was consistently at a low level, 1.48 cases per annum per 1,000 average strength for the 4 years, largely because of the effective screening at the time of induction. The first and final years had a higher incidence than the intervening years, 1.86 and 2.16 per 1,000, respectively. The death rates for tuberculosis decreased from 3.46 per 100,000 average strength in 1942 to 2.15 in 1945, the average for the 4 years being 2.64.

As in theaters of operations, the venereal diseases in the Zone of Interior contributed strongly to the incidence rate of the infectious and parasitic diseases. Indeed, for the war years as a whole, gonorrhea headed the list for reported cases of infectious and parasitic disease both in the continental United States and overseas. The 1942 rate for gonococcal infection in troops of continental United States was 31.44. In 1945, this rate rose to 43.21, a circumstance which held for a number of the communicable diseases as discipline relaxed with the end of the war and an association with civilian populations was greater and easier. Syphilis, excluding neurosyphilis, started with a satisfactory rate of 6.88 in 1942, reached a high point of 19.60 in 1944, and declined thereafter. The incidence of chancroid in Zone of Interior troops was one-fifth that of troops stationed overseas. Rates for gonorrhea also favored troops at home, 31.52 compared with 38.81 per 1,000 for overseas troops as a whole and for the war period. Overseas troops had much the better rates during the first 3 years, but, with cessation of active combat, that advantage was lost in 1945 when gonococcal infection increased to such extent that the year ended with an annual overseas incidence of 55.72. For the war period as a whole, syphilis was far more frequent among Zone of Interior troops than those overseas, the respective rates, excluding neurosyphilis, being 15.12 and 8.89 per annum per 1,000 average strength.

Influenza among troops in continental United States was more or less limited to the first 2 years of the war, as it was in most foreign areas where American troops were stationed. Incidence was somewhat higher, 16.20, in 1942, but the bulk of cases occurred in 1943 during an epidemic of type A. Death rates were low, 0.30 and 0.15 per 100,000, for the 2 years. Cases and deaths from bacterial pneumonia were in the same pattern, with the highest admission rate (3.52 per annum per 1,000 average strength) in 1942.

Meningococcal meningitis was second among causes of death from infectious and parasitic disease with rates of 2.41 per 100,000 strength per year in 1942 and 4.48 in 1943, the 4-year average being 2.58. Meningitis thus holds its place among communicable diseases of military significance, but the epidemic of 1943 (incidence rate 1.23 per 1,000) was a minor event compared with the outbreaks of World War I.

The common communicable diseases of childhood consistently give concern in recruit populations. All were relatively frequent in the first 2 years, with the exception of whooping cough which seems never to give much trouble in armies and chickenpox which regularly is less frequent than mumps, measles, and rubella. In order of frequency, mumps commonly ranks first; measles, somewhat erratic, usually second; and rubella, third. In the World War II experience of troops in the United States, mumps behaved satisfactorily, incidence 5.59 per annum per 1,000 average strength; measles was of lesser consequence, 3.69 per 1,000; while rubella in this instance produced the epidemic with 125,530 cases and an incidence of 8.51 per 1,000. Most cases of rubella (88,775) occurred in 1943 to give an attack rate of 17.13 per 1,000 average strength. Chickenpox as usual was inconsequential, incidence 0.58 cases per 1,000 strength. The frequency of both chickenpox and mumps remained much the same over the 4 years.

Epidemic hepatitis and coccidioidomycosis provided problems of special note among infectious and parasitic diseases of continental United States troops. Hepatitis was largely limited to 1942, with 33,569 admissions, a rate of 12.63 per 1,000; this was primarily serum hepatitis, easily the largest epidemic of this particular form ever recorded. A full account is given in another volume.[1]

Thereafter, infectious hepatitis, a disease justifiably included among infections of military importance, was the prevailing form. The rates for infectious hepatitis were low in 1943 and 1944, but the last year of the war gave an annual admission rate of 2.08 per 1,000 average strength. In the continental United States, the rate never reached the proportions that it did in a number of overseas theaters, especially in the last 2 years of hostilities. Serum hepatitis occurred among overseas troops in 1942, but thereafter it was the directly communicable infectious hepatitis that was responsible for the continued high admission rates among troops overseas—14.79 in 1943, 6.44 in 1944, and 15.16 in 1945.

Coccidioidomycosis, by contrast, was mainly a disease of troops in the United States. Of 3,809 cases for the Army as a whole, 3,626 were in continental United States. For the 4-year period (1942–45), the greatest number of cases, approximately 1,310, occurred during 1943. Deaths totaled 32 in the United States and 7 overseas. The death rate was not great (0.22 per annum per 100,000 average strength in the continental United States), but the behavior of the disease and the problems presented were an outstanding event of World War II medicomilitary history (p. 286).

Among continental United States troops, the chief causes of death due to infectious and parasitic diseases were tuberculosis, 2.64 deaths per annum per 100,000 average strength; meningococcal meningitis, 2.58; bacterial pneumonia, 1.69; poliomyelitis, 0.58; and infectious hepatitis, all forms, 0.55. Deaths from infectious hepatitis may have been underreported.

[1] Medical Department, United States Army. Preventive Medicine in World War II. Volume V. Communicable Diseases Transmitted Through Contact or Unknown Portals of Entry. [In preparation.]

European Theater of Operations

In the European theater, the incidence of infectious and parasitic diseases, next to the lowest among theaters of operation and about the same as that of continental United States troops, suggests little of particular note in a command that at one time involved more than 3,000,000 men and stands as the largest foreign effort of the United States Army in this or any other war.

The influenza epidemic of the early years of World War II was more or less worldwide. Incidence was greater among troops of the European theater, 20 and 18 per 1,000 for the years 1942 and 1943, than for troops at home and indeed was exceeded only by the experience of the Mediterranean theater, 39 and 15 per 1,000 during the same 2 years. The Eastern or Pacific theaters were less affected than those of the West or Atlantic area. As a consequence, over-all rates for troops at home and those abroad favored troops overseas; Zone of Interior troops had incidence rates of 16 in 1942 and 13 in 1943; for all troops overseas, incidence was 12 and 12 per 1,000 in the corresponding years.

Rates for bacterial pneumonia were in those years appreciably higher in continental United States than in overseas areas, even in the European and Mediterranean theaters where influenza was more common. This contrast invites consideration as to the contribution of exposure, which is presumably greater in field operations than in training areas.

The venereal diseases had an erratic history in the European theater. Incidence during the years of active combat was lower than among troops at home, but when the war ended this advantage was quickly lost. The incidence rate for gonorrhea in 1944 was 24.79 per 1,000; in 1945 it increased to 71.71. Chancroid rose from a frequency of less than 2 per 1,000 to 5.72. Syphilis, excluding neurosyphilis, essentially doubled in frequency over the previous year, showing an incidence of 12.15 per 1,000. Comparing overseas troops as a whole with those of the Zone of Interior, gonorrhea was more of a problem overseas (38.81 per 1,000) than at home (31.52); chancroid was overwhelmingly a disease of soldiers stationed abroad, 7.29 against 1.43 per 1,000; while syphilis, excluding neurosyphilis, was more frequently observed among domestic troops, 15.12 per 1,000 average strength for troops at home and 8.89 for those abroad.

Viral hepatitis was a problem in the European theater. Though serum hepatitis occurred rather extensively in 1942, the greatest problem was with infectious hepatitis which occurred mainly in troops operating in continental Europe, in 1944 and 1945. The number of admissions in 1944 was 4,330 (rate 2.58 per 1,000), and 20,575 admissions in 1945, a frequency of 8.67 per 1,000 average strength. For the 2-year period, 1944–45, combined deaths from serum and infectious hepatitis had ranked fifth among deaths from communicable disease in the theater, an average rate of 1.14 per annum per 100,000 average strength.

Data for scarlet fever and the partial information now available for streptococcal sore throat indicate that hemolytic streptococcal infections were

less prevalent among troops of the European theater than in continental United States, both regions being in the north Temperate Zone.

The malaria incidence rate reported in the theater, 4.88 per 1,000 for the 4-year period, was almost wholly relapse of original infections contracted in other theaters by troops transferred to Europe in connection with the major military effort of the final years of the war. There was little indigenous malaria.

The common communicable diseases of childhood recognized in the European theater were only a fraction of those reported for troops in the continental United States. Mumps was the commonest, as it usually is among seasoned troops. The report of more herpes zoster (1,295 admissions) than chickenpox (774 cases) is unusual.

That concern about communicable disease increases as a war ends is a principle of military preventive medicine. The venereal diseases illustrate this well, as do three other infectious diseases of this experience, typhoid fever, paratyphoid fever, and diphtheria. Both typhoid fever and paratyphoid fever were almost nonexistent in the early years of the theater—4 cases of each disease occurred over a 2-year period. In 1944, 25 cases of typhoid and 20 of paratyphoid occurred during active combat and field operations. The main trouble, however, centered about those spring months of 1945 which saw the end of hostilities. For the year as a whole, the number of cases of typhoid and paratyphoid totaled 35 and 60, respectively. The really serious problem, however, was among the prisoners of war (p. 182).

Diphtheria in the European theater had much the same history. The first 2 years showed few cases, 27 and 45. During the third year, cases increased to 245, but 1945 produced an epidemic as troop strength was consolidated in West Germany where the disease was highly prevalent in the civilian population. Admissions for the first 6 months of the year were at the rate of 0.60 per annum per 1,000 average strength; the rates were twice that during the last 6 months, 1.26 per 1,000.

The 10 cases of louseborne typhus fever in 1945 warrant little attention of themselves in the total account of communicable disease. The significance lies in that small number of infections among troops operating in a region where typhus fever was widely dispersed and broadly epidemic. Military preventive medicine has no finer accomplishment.

The principal cause of death from communicable disease in the theater was tuberculosis with a death rate of 4.41 per 100,000, in large part determined by the 6.02 rate of 1945, this in turn related chiefly to recovered prisoners of war. The death rate for tuberculosis was greater in the European theater than in others. For all troops overseas, deaths from tuberculosis were some 47 percent in excess of those for men stationed in continental United States.

Mediterranean Theater of Operations

The environment in which troops of this command were called upon to operate was one of the more difficult among American areas of influence; the

communicable diseases consequently occurred frequently. Incidence and death rates for tuberculosis were less than the average for all overseas troops; for pneumonia, they were somewhat greater. For malaria, the incidence rate was 42.68 per annum per 1,000 average strength, placing the theater fifth among the eight overseas theaters in incidence, and the death rate was 3.84 per 100,000. Incidence was greatest in 1943, when the rate reached 71.84 per 1,000. During the 4 years of war, there were 63,292 cases of malaria in the theater.

Next to the Southwest Pacific areas, epidemic hepatitis was most prevalent in the Mediterranean theater. Data on admissions are available only for the last 2 years of the war: 18.16 per 1,000 average strength in 1944 and 19.88 for 1945. The death rate per 100,000 for the 2 years was 3.39 per annum, about one-half that for the combined Pacific theaters; in 1945 in the Southwest Pacific Area, admission rates reached 34.85 per annum per 1,000 average strength.

The venereal diseases were of more frequent occurrence in this theater than in any other. Rates were consistently higher than in Europe. For gonorrhea, the incidence rate of 66.42 per annum per 1,000 exceeded the rate for the European theater, which was 50.09. As in other theaters, rates rose at the end of the war; the main difference was that in the Mediterranean theater the rise started a year earlier. For chancroid, the 22.25 per 1,000 annual rate over the 4 years exceeded that for any other major command, as did the syphilis rate of 15.22, even though the latter rate was only fractionally greater than in continental United States.

The dysenteries were an outstanding problem. The rate for bacillary and unclassified dysentery combined was 6.43 cases per annum per 1,000 average strength; for amebiasis, the rate was 1.26. These three categories are only part of a larger group of acute intestinal infections described in detail in a subsequent chapter (p. 340).

The Mediterranean theater had a moderate poliomyelitis problem. Other theaters had somewhat greater incidence, notably the China-Burma-India theater with 0.18 per 1,000, compared with 0.09 for the Mediterranean area, and death rates were higher, China-Burma-India being 8.2 per annum per 100,000. In all, this theater had 127 cases of poliomyelitis during the 4 years.

Fungus infections and the intestinal parasites, while of some consequence, were measurably less significant than in the Pacific and China-Burma-India theaters. Sandfly fever was a special problem. Incidence rates were 7.56 per 1,000 strength with a total of 11,206 cases, distributed with comparative regularity over the years of 1943–45. The theater was activated in late 1942, and only 11 cases occurred that year. There were no deaths in the total series of cases. The number of cases in the Mediterranean theater exceeded that in any other, but rates of incidence were only one-fourth those prevailing in the Middle East theater, a smaller command which had 4,399 cases.

The newer methods for control of louseborne typhus fever had their initial test in the Naples epidemic of 1943, and the principle was there estab-

lished that troops could operate with relative safety in typhus areas; the theater had only 16 cases during 1942–45.

The six leading causes of death due to infectious and parasitic disease were in the following order: Malaria with 3.84 deaths per annum per 100,000 average strength; tuberculosis, 3.51; infectious hepatitis, 3.39 (1944–45); poliomyelitis, 2.49; bacterial pneumonia, 1.89; and meningococcal meningitis, 1.35.

Middle East Theater

Tuberculosis was no particular problem in the Middle East theater; in fact, the record achieved there was better than in any other overseas command. No deaths were reported for nonpulmonary tuberculosis, which was unique among the nine major commands. This form of tuberculosis accounted for about a fourth of all tuberculosis deaths overseas and more than a third in continental United States.

The admission rate of bacterial pneumonia was the greatest of any theater of operations; the death rate, 2.05 per 100,000 strength, was second only to that of the China-Burma-India theater. No theater had the admission rate of continental United States for bacterial pneumonia, 2.54 per 1,000 strength. For all troops overseas, the admission rate was 1.26. Death rates for continental United States were 1.69 per annum per 100,000 and for overseas theaters 1.44.

The Middle East is notably a malarial zone, and incidence rates of 65.32 per annum per 1,000 over the 4 years were second only to the rate in the China-Burma-India region.

The dysenteries were extremely prevalent with a combined incidence rate of 21.76 for bacillary and unclassified dysenteric disease. Amebiasis reached the appreciable rate of 8.08 per 1,000, again second only to China-Burma-India. Food poisoning was prevalent in 1944.

The theater record for gonococcal infection was good, but chancroid was at high levels of 19.41 per 1,000; syphilis ranked well up among all major commands of the Army with a rate of 13.48. The incidence of lymphogranuloma venereum (1.94 per 1,000) was higher than in any other theater or area except the China-Burma-India theater.

The area featured an incidence of 30.09 for sandfly or pappataci fever, the highest rate among commands by a large margin. Cases numbered 4,399. An individual theater characteristic was the presence of leishmaniasis with incidence of 1.93 per 1,000 and 282 cases reported. The disease appeared in 6 other theaters, and 22 cases are in the records of continental United States as first recognized there. In all, there were some 497 cases in the entire Army, with half of them in this small theater.

Poliomyelitis cases were 21, for a rate of 0.14 per 1,000, and 8 patients died.

The list of main causes of death from infectious and parasitic diseases departs greatly from any thus far noted. In order, they were malaria (8.21), poliomyelitis (5.47), and smallpox (3.42), followed by tuberculosis, bacterial pneumonia, and diphtheria, each with rates of 2.05 per annum per 100,000.

China-Burma-India Theater

The exigencies of war that required operation of foreign troops in the area included within the China-Burma-India theater brought all the support necessary to the emphasis placed by military medicine on the communicable diseases. To those not previously acquainted with this general region, realization comes quickly that there are parts of this universe in which the communicable diseases certainly are not conquered.

First consideration is given to those two communicable diseases which are responsible the world over for most deaths and disability and which are still to be found in almost any list of 10 leading causes of death. Tuberculosis and pneumonia are well represented in this present experience of World War II in the Far East. The admission rate for bacterial pneumonia was third among theaters of operations, preceded by the Middle East and Mediterranean areas. The death rate, however, was measurably in excess of any other and indeed was close to twice that of continental United States, which, as will be recalled, was itself greater than for the theaters, all troops outside continental United States.

The incidence rate for tuberculosis, all forms, was 0.98 per 1,000 average strength compared with 0.92 for all overseas troops and 1.48 for the United States. Death rates per annum per 100,000 average strength were 3.88, similar to the rate for all units serving abroad (3.87) but higher than in the United States (2.64).

The theater had its difficulties with the venereal diseases. The incidence of gonorrhea was satisfactorily low, well below average for troops at home or abroad. Lymphogranuloma venereum, however, had the highest incidence rate among theaters or the Zone of Interior with a rate of 2.50 per 1,000. Syphilis was well above average for soldiers serving abroad and so was chancroid, each by about one-half.

The theater had the top malaria rate among major commands with an incidence of 86.70 per annum per 1,000 over the war period.

The intestinal infections were uniformly frequent. Typhoid fever totaled 78 cases and paratyphoid 96. The incidence of dysentery, as judged by data for bacillary and unclassified forms but far from a complete rate, was 21.19 per annum per 1,000 strength; the Middle East being the only close rival. Amebiasis had the extreme rate of 23.95 per annum per 1,000; not even the Middle East offered any sort of competition and the Southwest Pacific was in third place with 6.67 per 1,000.

The highest incidence of poliomyelitis among theaters was in China-Burma-India, and the disease in general was more frequent abroad than at home. The general list of parasitic diseases was enhanced in frequency, and no theater had more fungus infections. Complete data for hepatitis are not at hand, but those available indicate the disease occurred at close to average level for troops of all theaters.

The China-Burma-India theater had its individual problems. Sandfly fever was a fairly common disease with 2,941 cases giving a rate of 6.71 per

1,000. Dengue fever was variously epidemic, especially in 1943 and 1944. Cases totaled 8,217, and average annual rates over the 4 years were 18.74 per 1,000. Scrub typhus enters the list of diseases thus far recorded with 804 cases, the incidence rate 1.83 per 1,000. The average annual death rate for scrub typhus in the China-Burma-India theater topped that of any other communicable disease in any theater of World War II.

That the infectious and parasitic diseases need to be judged in terms other than those referable to the United States is well demonstrated by the principal causes of death among diseases of this class as recorded in this Far Eastern experience. First on the list is scrub typhus, 14.60 deaths per 100,000 troop strength. Next in order are malaria, 9.12; poliomyelitis, 8.21; smallpox, 4.56; tuberculosis, 3.88; bacterial pneumonia, 3.19; and meningococcal meningitis, 2.05. The items included and the values themselves are rather startling. For a number of these diseases, sharp irregularities in annual rates of occurrence mark the coming and going of epidemics.

Southwest Pacific Areas

The part of the world that includes the Southwest Pacific areas rivals the China-Burma-India area in frequency of communicable disease; if anything, the variety is greater. This Army area had a part in the influenza of 1942. Tuberculosis and bacterial pneumonia were at average levels for overseas troops. The record in respect to the venereal diseases was good, even for lymphogranuloma venereum and chancroid, which was a creditable achievement in this environment.

The main difficulty was with malaria, for the theater had an average of 57.07 cases per annum per 1,000 average strength during the war period. No less than 104,809 cases are included in the medical records of the command. A proverbially endemic disease, malaria was epidemic in 1943 when the incidence rate for the year reached 209.56 per 1,000 strength. Energetic control measures resulted in a wholly satisfactory record the next year and a low 33.58 in 1945.

Mumps, as would be expected, was first among the common communicable diseases of childhood, but a moderate epidemic of rubella in 1942 was an unusual event which carried into the following year. An epidemic of diphtheria, 505 cases, occurred in 1945. More cases of poliomyelitis (224) were reported than from any other theater, the incidence being 0.12 per 1,000 average strength.

The full data on infectious hepatitis are not available, but the 36,110 admissions in 1945, making a rate of 34.85 per 1,000, was not duplicated in any theater during this or the preceding year. Even in 1944 in this theater there were as many as 4,966 cases, the rate being 9.21. In the Southwest Pacific areas, deaths due to infectious hepatitis averaged 8.44 per annum per 100,000 during 1944–45.

Leishmaniasis with 29 cases, 5 cases of rabies, 70 of arthropodborne encephalitis, and 20 cases of yaws gave variety to the larger events just recorded.

Deaths per annum per 100,000 average strength for the Southwest Pacific Area gave first place among infectious and parasitic diseases to scrub typhus (11.43), followed by infectious hepatitis (8.44 for 1944–45), malaria (6.15), poliomyelitis (3.81), and tuberculosis, all forms (3.71). Bacterial pneumonia (1.52) was not within the first five. The list is as bizarre as that of the China-Burma-India theater to those who are accustomed to western public health problems.

Dengue fever was another prevalent disease of the Southwest Pacific. A total of 50,903 cases occurred; the over-all annual incidence rate for 1942 to 1945 was 27.72 per 1,000. The epidemic year was 1944 with 28,292 cases and an annual rate of 52.47; 1942 had rates even higher, but fewer numbers were involved. Only a favorable situation in 1945 permitted the average rates cited.

The dysenteries were commonly present as would be anticipated. Bacillary and unclassified dysentery together gave a total theater rate of 9.73 per annum per 1,000. Amebiasis had a rate of 6.67 with some 12,244 cases reported; most of them, 11,475, occurred in the final year of 1945, with the evident implication that actually there had been more cases in other years than had been recognized. Typhoid fever accounted for 73 cases, and paratyphoid fever for another 183 cases. Food poisoning was more common than for overseas troops in general.

Scrub typhus was a special feature of communicable disease occurrence in this theater. Of 5,436 cases reported from all theaters of operations, 4,459 were in the Southwest Pacific, average annual rates being 2.43 per 1,000. Fleaborne typhus was present to the extent of 87 cases, but there was no louse-borne typhus.

The parasitic diseases were common, particularly hookworm and ascariasis. There were some 323 admissions for filariasis, 233 of them in 1944; over-all theater rates were 0.18 per 1,000. Schistosomiasis accounted for 1,545 cases, rate 0.84, with 1,460 cases in the single year of 1945. Fungus diseases were as abundant as in the China-Burma-India theater.

Central and South Pacific Area

The health record established by this area in respect to the venereal diseases was outstanding; it was second only to the record in the North American area and was far better than that of continental United States. The incidence rate for gonorrhea was 10.87; for syphilis, 3.64; chancroid, 1.91; and lymphogranuloma venereum, 0.33 per 1,000 average strength; in each instance, with the exception of chancroid, markedly below the average for troops overseas (table 10) or at home.

The incidence rate for tuberculosis was about equal to the average rate for overseas troops, but the rate for bacterial pneumonia was lower than rates for all other overseas areas.

As in the Southwest Pacific Area, malaria was the main problem, and the history of events was much the same in the two theaters. During the first year, the number of cases was small because of the limited time of exposure and the small troop strength, but in 1943 cases numbered 55,050 and the rate per 1,000 was 188.81, not dissimilar to that of Southwest Pacific in the same year. The recovery was more prompt, however, for the incidence rate in 1944 was 24.33 and the last year of the war had the excellent record for that locality of 22.48 per 1,000.

The intestinal infections were the next broad problem. The dysenteries of recognized form, bacillary and unclassified, prevailed in a frequency of 5.58 per 1,000; the main difficulty appeared in the initial year of 1942, and progressive improvement thereafter gave a rate in 1945 of 3.43 per 1,000. Food poisoning was of noteworthy frequency in the years after it was established as a reportable condition; in 1945 there were 1,205 admissions and a rate of 3.21 per 1,000. Amebiasis (rate 2.50) was less commonly recognized than in the Southwest Pacific (rate 6.67 per 1,000). Typhoid and paratyphoid fever fever cases were 28 and 33, respectively.

In the Central and South Pacific area, dengue came close to matching its behavior in the Southwest Pacific, rates for the Central and South Pacific being 23.83, and in the Southwest Pacific 27.72 per 1,000. The disease was epidemic in both 1943 and 1944, a total of 28,092 cases being reported during the 2 years.

Although the incidence of infectious hepatitis was heavy, it was much lower than in the Southwest Pacific. Both parasitic and fungus diseases were prominent. There were 171 cases of scrub typhus, but this disease was of no significance here compared with the other Pacific theater; fleaborne typhus was more frequent than in any other theater, but there were only 123 cases.

This theater was characterized by the preponderance of filariasis; 1,348 admissions were reported, most of them in 1943 and 1944. The Southwest Pacific had far less, but even in the Central and South Pacific the rate was only 1.07 per 1,000 average strength. The 116 cases of schistosomiasis were a minor event in comparison with the observed frequency of that disease in the Southwest Pacific Area. It was in this area that one of the two admissions for glanders in foreign theaters was reported, the other being in the Middle East theater.

Diphtheria appeared in minor epidemic proportions in the last 2 years of the recorded period.

The six leading causes of death due to infectious and parasitic diseases in this theater during 1942–45 were tuberculosis, all forms (3.66), malaria (3.50), infectious hepatitis (1.96 for 1944–45), bacterial pneumonia (1.11), poliomyelitis (0.48), and meningococcal meningitis (0.40). These rates are expressed in terms of number per annum per 100,000 average strength.

Latin American Area

The Latin American area was in a tropical and subtropical region on the other side of the world from the two Pacific theaters. Comparison is scarcely productive for the theater was small and singularly free from combat action and the associated field conditions. The incidence of communicable disease and the deaths from infection are instructive in respect to what can be accomplished with modern preventive services under environmental conditions that provide more than usual risk.

Tuberculosis death rates and the incidence and death rates for bacterial pneumonia were favorable; these two communicable diseases lead almost all other infectious processes in temperate zones as causes of death and rank high almost everywhere.

Incidence of the venereal diseases was high. Gonococcal infection ranked close to the top for all theaters, and occurrence of syphilis was much above the average. Chancroid was at the high level of 12.19 per 1,000 strength, and lymphogranuloma venereum had a rate of 1.60 per 1,000, three times that for theaters in general and more than twice the home incidence. One peculiarity marked this experience in control of the venereal diseases. The usual pattern is one of initial difficulties, subsequent improvement in rates until the war ends, and then an incidence even greater than before. The Latin American area, after having had consistently high rates throughout the war, ended with a marked improvement in 1945 for all of the four diseases mentioned.

Malaria was also prominent among the problems of this theater. The total wartime experience ended with a rate of 41.01 per 1,000 per year, but the first year was responsible for most of this with an annual 1942 rate of 99.78. The improvement that followed was remarkable, and 1945 ended with an incidence rate of 8.17 per 1,000 average strength.

Intestinal infections were at high risk, but the rate for recognized dysenteries was good, 1.72 per 1,000 per year. Parasitic infections were relatively frequent among infections as a whole. Ten cases of typhoid fever occurred and seventy of paratyphoid. Infectious hepatitis was at a low level.

A series of epidemics of rubella was an unusual occurrence, the annual rate for the 4 years being 5.14 per 1,000, with 9.82 in 1945. Only in continental United States was this disease relatively so prevalent; in the Latin American area, rubella exceeded both mumps and measles.

Poliomyelitis was represented in this theater by 13 cases and 3 deaths for the 4 years. The theater had 1 of the 6 cases of rabies in overseas theaters. Sandfly fever provided a minor problem with 35 cases.

The deaths from infectious and parasitic disease in the Latin American area are again a curious collection as judged by conditions in continental United States for they rank in order as follows: Malaria, 4.72 per annum per

100,000 average strength; tuberculosis, 2.63; bacterial pneumonia, 0.79; and poliomyelitis, 0.79. The values themselves are notably low.

North American Area

Of all major commands, this small theater had the best record for death and disability from disease, all forms, and from communicable diseases; indeed, the theater far outdistanced all others. The venereal diseases were at an unbelievably low level, uniformly for all major categories and throughout the 4 years. The usual increase in the last year of the war occurred here but served only to bring the rate for gonococcal infections to 10.43 per annum per 1,000 and for syphilis to 3.97.

The 1942 and 1943 experience with influenza was much the same as in all theaters, but the disease continued through 1945 as the most frequently occurring item of the infectious and parasitic list, in all 6,165 cases. Bacterial pneumonia was infrequent.

A small outbreak of meningococcal meningitis in 1943 was one of the few epidemic events. There is little to note but a good record.

Causes of death from infectious and parasitic disease were tuberculosis, all forms, 3.46 per annum per 100,000 average strength; hepatitis, mainly serum hepatitis of 1942, 2.64 (on the basis of 13 reported deaths, 11 for 1942); meningococcal meningitis, 1.02; bacterial pneumonia, 0.81; and influenza, 0.61.

Distribution by Mode of Transmission

The mechanisms by which an infectious agent is transported from reservoir to susceptible human host are a fundamental factor in designing methods for the control of the communicable diseases. Modes of transmission serve as a logical means for classification of these disease processes. Table 12 shows cases of infectious and parasitic diseases, by mode of transmission.

Among troops in the continental United States the incidence rate for infectious diseases transmitted chiefly through the respiratory tract was more than two and one-half times the rate for all overseas areas. The Far East and Pacific theaters had notably low rates, with other theaters occupying a middle position except for the North American theater which had rates of 26.13 per annum per 1,000 average strength. It is reaffirmed that common respiratory disease is not included.

Intestinal infections showed greater variation between theaters than did the respiratory infections. The rate for Zone of Interior troops was 1.86 per annum per 1,000 compared with 9.47 for all troops overseas. The European and the North American theaters had good rates, equal to or below those of continental United States. This circumstance, along with correspondingly low rates for arthropodborne infection, accounts in large part for the over-all good record of these two theaters. The annual incidence of 0.84 per annum per 1,000 for intestinal infections in the North American theater is in contrast

TABLE 12.—*Incidence rates for infectious and parasitic diseases in the U. S. Army, by mode of transmisson and theater or area of admission, 1942–45* [1]

[Preliminary data based on tabulations of individual medical records]

[Rate expressed as number of cases per annum per 1,000 average strength]

Theater or area	Respiratory diseases	Intestinal diseases	Diseases transmitted by direct or indirect contact		Arthropod borne diseases	Diseases of unknown or uncertain origin	Miscellaneous and other diseases
			Venereal [2] diseases	Other diseases			
Continental United States	41. 11	1. 86	49. 20	5. 00	3. 12	4. 29	2. 22
Overseas:							
Europe	16. 27	1. 86	64. 30	7. 98	4. 99	8. 25	2. 59
Mediterranean [3]	20. 08	9. 45	104. 51	7. 78	50. 58	26. 72	2. 83
Middle East	18. 26	34. 17	62. 44	2. 09	97. 85	8. 11	5. 14
China-Burma-India	11. 28	49. 16	47. 29	4. 92	114. 69	11. 29	8. 36
Southwest Pacific	8. 35	20. 03	37. 02	10. 79	87. 59	25. 73	4. 73
Central and South Pacific	10. 83	10. 70	16. 99	6. 62	86. 83	9. 64	5. 12
North America [4]	26. 13	. 84	9. 80	2. 00	. 39	12. 52	1. 63
Latin America	19. 78	4. 18	63. 82	11. 10	42. 76	7. 06	7. 54
Total overseas [5]	15. 29	9. 47	55. 74	7. 86	42. 14	14. 19	3. 66
Total Army [6]	30. 23	5. 07	51. 96	6. 20	19. 56	8. 46	2. 83

[1] Excludes rheumatic fever.

[2] Excludes granuloma inguinale; this disease is included in the "Miscellaneous and other" category.

[3] Includes North Africa.

[4] Includes Alaska and Iceland.

[5] Includes cases among transports admissions.

[6] Does not include secondary diagnosis cases estimated for 1942 and 1943 for those diseases for which such data are not available (table 10, diagnoses identified by footnote 4).

to the 49.16 of the China-Burma-India theater. Other tropical areas also had high levels of intestinal infection; the rates of the Mediterranean and Central and South Pacific theaters deserve commendation considering the environmental conditions under which troops operated.

The venereal diseases have been compared in discussion of the theaters. Scabies is the main component of contact infections listed as "others."

Malaria mainly determines the extent of the arthropodborne group of diseases; one-fourth of mankind lives in malarial zones and United States Army troops were engaged in most of the places where malaria flourishes. Dengue and sandfly fever were other important elements. Malaria and scrub typhus were the chief contributors to the number of deaths.

Epidemic hepatitis was the important disease in the class of infections where mode of transmission remains uncertain, notably so when serum hepatitis is included as in this instance. In general, the incidence rates, shown in table

12, are representative of infectious hepatitis (including some serum hepatitis), since the incidence of the other three diseases in this category (infectious mononucleosis, acute poliomyelitis, and lymphocytic choriomeningitis) was relatively low.

INTERNATIONALLY QUARANTINABLE DISEASES

Never in the history of warfare has an army traveled so far or so widely as did troops of the United States during World War II. Never before has an army been called upon to take up occupational duties in such farflung parts of the world. No recognized focus of the great pandemic diseases of history was untouched by American interest and influence nor unvisited by American soldiers.

Not only was the amount and extent of travel by United States Army troops greatly increased during the war, but it was potentially more dangerous travel. Compromise of established measures for international quarantine is unavoidable in time of war. Civilian health staffs of countries at war are depleted. Necessary supplies for prevention and control of disease among civilian populations are directed in considerable part to military needs. Newly developed methods are at the first disposal of the military. Port control in invaded countries was invariably taken over by the occupying enemy forces; when these areas were liberated, port sanitation was usually found to be totally disorganized. Many ordinary regulations and procedures in respect to air and sea traffic were abrogated or modified because of military necessity; others were disregarded through license, ignorance, or exigency. There was a greater potential health risk in international travel during the war and greater possibility of the spread of the quarantinable communicable diseases. The record was astonishingly good. It will now be considered having been purposely avoided in the presentation of problems of individual theaters.

The six internationally quarantinable diseases are cholera, plague, yellow fever, louseborne typhus fever, louseborne relapsing fever, and smallpox. In World War II, 3 of the 6 (cholera, louseborne typhus fever, and smallpox) definitely appeared in various major commands of the Army. In addition, it is possible that some of the relapsing fever cases were louseborne. No case of yellow fever or plague was reported; in fact no plague-infected rat was found in an American ship. The data for the Army as a whole and according to theaters of operations are given in table 13. The four diseases, cholera, louseborne typhus, relapsing fever, and smallpox, accounted for 402 cases and 32 deaths among troops of the Army as a whole. All theaters and areas had some experience with at least 1 of the 4 diseases. The China-Burma-India theater had experience with all four.

Relapsing fever was of commonest occurrence, for it appeared in home troops and in all theaters with the single exception of the European theater. There were no deaths. The Mediterranean theater accounted for 49 cases, an admirable record in view of the extensive epidemic that occurred in the

TABLE 13.—*Internationally quarantinable communicable diseases, U. S. Army, by theater or area, 1942–45*

[Preliminary data based on tabulations of individual medical records]

[Rate expressed as number of cases per annum per 1,000 average strength]

Theater or area	Cholera		Relapsing fever		Smallpox		Typhus fever (louseborne)	
	Cases (number)	Rate	Cases (number)	Rate	Cases (number)	Rate	Cases (number)	Rate
Continental United States	0	0.00	28	0.00	6	0.00	0	0.00
Overseas:								
Europe	0	0	0	0	0	0	10	.00
Mediterranean [1]	0	0	49	.03	12	.01	16	.01
Middle East	0	0	13	.09	15	.10	14	.10
China-Burma-India	10	.02	70	.16	33	.08	63	.14
Southwest Pacific	0	0	1	.00	20	.01	0	0
Central and South Pacific	0	0	1	.00	25	.02	1	.00
North America [2]	0	0	2	.00	0	0	0	0
Latin America	0	0	7	.02	5	.01	0	0
Total overseas	10	.00	143	.01	111	.01	104	.01
Total Army	10	.00	171	.01	117	.00	0	.00

[1] Includes North Africa.
[2] Includes Alaska and Iceland.

NOTE: Absolute zero is indicated by zero in the units column; 0.0 indicates a rate of more than zero but less than 0.05 and 0.00 a rate of more than zero but less than 0.005.

civilian population of the area. The greatest number, 70, was in the China-Burma-India theater; aside from 28 cases in the United States and 13 cases in the Middle East, the remainder was scattered sporadic infection.

Smallpox was next in frequency, with 117 cases and 30 deaths. The number of deaths indicates that the disease in most instances was variola vera and not the alastrim variety which characterizes smallpox in the United States and of which there were 6 cases in this military experience. All others were in theaters of operations. China-Burma-India had 33 cases and the 2 Pacific areas almost equal numbers. Only the European and North American areas had no smallpox.

The limitation of louseborne or classical typhus fever to 104 cases and no deaths was one of the remarkable achievements of preventive medicine in World War II. The infection was introduced into 5 of the 8 theaters. The potentiality for a major epidemic was great, for the existing circumstances were not dissimilar from those of other wars whose outcome typhus fever so frequently has decided. In World War I, 1914–18, and the immediate period thereafter, more than 5,000,000 persons had typhus fever in Russia alone, and deaths have been estimated at 2,000,000; in Serbia, essentially one-fifth of the

population was involved in a typhus epidemic, with 150,000 deaths in a 6-month period in 1915. In the European theater, in this war, American troops entered a region in Germany that had at the time about 17,000 cases among the civil population and displaced persons. The United States Army escaped with 10 cases and no deaths.

Cholera under ordinary circumstances is endemic only in the China-Burma-India theater among the eight principal divisions of territory in which the United States Army operated overseas. The disease has great potentiality for spreading; it has repeatedly ranged far afield from this endemic focus and on several occasions has invaded continental United States in epidemic proportions. Ten cases of cholera occurred among American troops of the China-Burma-India theater with two deaths, a usual fatality for cholera. There was much cholera in both India and China during the war years and in the particular places where troops were stationed. The disease did not spread within the command nor to neighboring theaters.

The great quarantinable diseases have settled many wars. All but two of them invaded one or more of the great command areas; no theater escaped visitation by at least one of them. None gained a foothold. The risk was real. Here then is further justification for the emphasis on communicable diseases in military preventive medicine.

INFECTIOUS AND PARASITIC DISEASES OF ESSENTIAL MILITARY SIGNIFICANCE

The close relationship of communicable disease and military operations has been so long recognized, so decisive in military history, and so stressed in military planning as to be accepted doctrine. The seriousness of a given disease in respect to death and disability may be the issue. A relatively mild condition may attain importance because of the noneffectiveness it engenders. Much depends on the nature of existing military operations. In this war, an outbreak of mumps had disorganized a training program. Epidemics of acute upper respiratory infection complicated movements of troops on long journeys. An undue prevalence of so benign a disease as scabies interfered with preparations for marshaling an invasion. Dysentery disrupted a campaign, and the German general staff could speak feelingly of epidemic hepatitis in Africa. Typhus fever threatened again to settle the outcome of a war to become, as Hans Zinsser has remarked, a more potent influence than generals.

The communicable diseases transmissible by way of the respiratory tract fall into two main groups, those involving the respiratory tract itself and those leading to general infection with various localization. The first group always has importance.

Fortune favored this war in respect to influenza. The disease was present in excessive amount in 1942 and 1943. Influenza A was pandemic in proportions but mild in form. This was a major consideration in the uniformly favorable rates for bacterial pneumonia, with the odd result, however, that

deaths from this cause were more frequent among the military populations stationed in warm climates, such as China-Burma-India with 3.19 per 100,000 and the Middle East with 2.05, than in the temperate zones of continental United States and the European theater, which reported rates of 1.23 per annum per 100,000.

Tuberculosis failed to maintain its established place as a cause of death and disability in military populations because of the effective screening of recruits at the time of induction.

The diseases of the respiratory group having localization other than in the respiratory tract itself failed to reach the statistical expectancy warranted by experience of previous wars. Mumps, the usual leader in numbers of cases, was outdistanced by rubella. The incidence of mumps was considerably lower than it had been in World War I, while the rubella rate was slightly higher. The incidence of meningococcal meningitis, justifiably recognized as a military disease, was only 0.51 per annum per 1,000 for the total Army and 0.67 for continental United States.

The record for intestinal infections was remarkably good, considering the long history of these diseases as the primary concern of camp and field. During the 4 years of war, the European theater had only two deaths from bacillary and unclassified dysentery combined, and operations were on a battleground where thousands have died of this cause in other wars.

Typhoid fever illustrates the effect of environment in the face of similar methods of prevention and control; 91 cases occurred in continental United States and 414 overseas, with rates of 0.01 and 0.04 per 1,000 average strength. The death rate for typhoid fever was relatively much greater in foreign operations, for 3 deaths occurred among home troops and 33 overseas.

Amebiasis was more costly in this war because of the many tropical areas in which troops operated; the same is true for infections by intestinal protozoa other than *Entamoeba histolytica*. The disability engendered through food poisoning cannot be well determined from the existing data, for food poisoning was not reported as such until 1944, and even then acceptance of the new direction for reporting was not immediate; previously, cases were usually included among the diarrheas and dysenteries. Indication is that this condition continued as a common cause of disability, despite the improvements in camp sanitation and nutritional practices.

The data cited in table 12 and the accounts of individual theaters show that the venereal diseases were still a main feature of medicomilitary practice; the records were better than in some other wars, but much remains to be done before control can be considered adequate or satisfactory.

Fungus infections took on new importance with operations in tropical countries, where mycosis of the feet interfered with military effectiveness and specific fungus infections were relatively frequent. Coccidioidomycosis was a special problem of troops in the United States.

Schistosomiasis and filariasis were helminth infections new to most military physicians of this war and were responsibile for much disability before control

was effected. Scabies, that common accompaniment of troops, had an interesting history in that admission rates progressively increased as the war continued; 1942 had a rate for overseas troops of 2.03 per 1,000 which increased in successive years to 2.84, 3.08, and 5.48. At home, rates were similar to those overseas, although the spread between first and final years was greater for domestic troops, the rates being 2.38 in 1942 and 7.60 in 1945. The experience of the United States Army in World War I was repeated in World War II, and the problem of prevention of scabies still remains unsolved. Progress has been made, however, in the control of infestation by lice. Admission rates were 0.32 per annum per 1,000 in continental United States and 0.18 overseas. The degree of louse infestation in the American Expeditionary Forces in 1918 was estimated to be 1 to 2 percent, but the significant feature is that in both regions rates declined in the same progressive fashion as they rose for scabies. The new insecticides give improved methods for control.

The arthropodborne diseases introduced a fresh element into historical United States military practice. Malaria was the chief problem. Troops had of course operated before in malarious regions and had developed effective control, for instance, in Panama. A new situation was encountered, however, when field operations had to be carried on both in unfamiliar environments and in many places where malaria was hyperendemic. The initial costs of this disease were heavy, but the final results were good. Filariasis, scrub typhus, and sandfly fever also posed relatively new problems. Dengue had almost been forgotten, especially the fact that it could reach such epidemic proportions as developed in the Pacific theaters.

Infectious hepatitis has had a part in war before but never to the same extent as in World War II, in which was added the further complication of serum hepatitis.

The results attained in control of tetanus, a disease long recognized as a peculiar hazard of war, are so striking as to deserve special mention. In the European theater where battle casualties were more numerous than in any other theater, both the mortality and the morbidity rates were approximately the same as those for troops stationed in continental United States, thousands of miles from a battlefield. The almost unbelievably good results, a single case and a single death during the whole period of operations in Europe, are attributable to the remarkable effectiveness of active immunization brought about by tetanus toxoid. Eight cases in the Zone of Interior were related to tetanus infection among recruits, principally before immunization had been accomplished. Only four cases of tetanus occurred among all troops overseas.

ACHIEVEMENTS AND CHALLENGES

Many new problems come to light in the course of an experience as broad as that described in this chapter. The development of administrative measures and the search for new knowledge on which those measures depend account for much of the effort expended on scrub typhus, plague, schistosomiasis, infectious

hepatitis, and the arthropodborne encephalitides. The story of the achievements of the war years fills many pages in these volumes on infectious diseases. The stimulus continues. No one of these problems was wholly solved but what was learned serves as a useful guide to the research of the postwar years.

Fort Bragg fever and Bullis fever, diseases newly recognized in the course of the war, were of minor military significance but were biologically informative. For instance, Fort Bragg fever was eventually found to be a form of leptospirosis. Clinical variants and immunological differences among older established processes were better defined. Numerous problems suggested by this experience are as yet scarcely touched.

Infectious mononucleosis was a fairly frequent disease among troops, 13,571 admissions in continental United States and 4,961 abroad. A significant feature was that rates rose each year for both great groups of troops, in the United States from 0.29 in 1942 to 1.71 in 1945, and overseas from 0.23 to 0.58 per annum per 1,000 average strength.

Lymphocytic choriomeningitis was present among domestic troops to the extent of 333 admissions in the last 3 years of the war, data being unavailable for 1942. Overseas troops with this infection numbered 425 for the same 3 years, 260 having occurred in 1945, primarily in Europe and the Mediterranean areas, but with all theaters represented, even the exemplary North American area. The disease is worldwide in its distribution, and, with due allowance for unconfirmed diagnosis, seemingly more frequent than ordinarily recognized.

The behavior of herpes zoster and chickenpox in this group of young adults, both at home and abroad, warrants further analysis. Like other communicable diseases of childhood, chickenpox is a fairly frequent infection of troops, especially recruits, less so than mumps and measles but more than whooping cough. Among troops stationed in continental United States, 8,555 cases of chickenpox occurred, but the returns also give 5,384 admissions for herpes zoster. Compared to continental United States, chickenpox was one-third as frequent among overseas troops, but herpes zoster occurred more frequently. Overseas, the 2,109 cases of chickenpox were exceeded by the 4,735 admissions for herpes zoster.

World War II involved more men and extended over a wider geographical area than any other war in history. The successful result that accrued to American arms was influenced in forceful degree by favorable casualty rates for the communicable diseases, rates that have no precedent.

The actual experiences of this war justify the firm emphasis which military medical officers continue to place on the significance of the communicable diseases. These experiences included the repeated threat of the great pandemic diseases; the occurrence of epidemics even under the best of conditions; and the strange problems brought by modern warfare, characterized as it is by rapid movement and wide dispersal of resources. Well-known infectious diseases, typhoid fever for example, carefully evaluated and adequately controlled under conditions of the American environment take on new significance when encountered in other parts of the world. New diseases come to light, and old

ones present new facets. Perhaps the most important consideration of all is the steady erosion of manpower brought about by everyday infections, losses which become evident when communicable disease is measured in terms of noneffectiveness and of the permanent injury which leads to lasting disability. The deaths that these diseases cause are not the sole concern, nor do they always provide a reasonable basis for judgment, in both the military and civilian practice of medicine and public health.

Part II

DISEASES TRANSMITTED CHIEFLY THROUGH RESPIRATORY TRACT

Diseases Caused by Viruses

CHAPTER II

Chickenpox

Joseph Stokes, Jr., M. D.

HISTORICAL NOTE [1]

Chickenpox has not been a serious problem among the armed services in any recent war. This is not only the result of low incidence but also because there are usually no complications, so that relatively few days are lost from duty. In World War I, it resulted in 31,534 days lost from duty, a noneffective rate of 0.02 per 1,000 average strength. There were 1,757 primary admissions for the total Army. In contrast, measles in World War I caused 1,877,944 days lost from duty, a noneffective rate of 1.25 per 1,000 average strength per annum, or about 62 times that recorded for chickenpox. Again in contrast, mumps in World War I caused 3,884,147 days lost from duty, a noneffective rate of 2.58 per 1,000 average strength per annum, or about 129 times that recorded for chickenpox. In World War I, the high noneffective rates for measles and mumps resulted to a considerable extent from complications.

Chickenpox and the other communicable diseases of childhood were common among troops in the United States in World War I, with a particularly high incidence in induction centers, whereas they were far less common in Europe. Also, in World War I, Negro troops had higher rates for chickenpox than white troops—a ratio of almost 4:1. All of these communicable diseases reached a peak in about January 1918. Since induction began late in the spring of 1917 and summer intervened, it was not unexpected that such epidemics would occur in the inducted troops during their first winter.

CONTROL MEASURES

Chickenpox appears to be one of the most readily transmissible of all infectious diseases, and no method of control was available in World War I. Between World War I and World War II, evidence was obtained by Wells and his coworkers [2] that, in various aggregations, ultraviolet light was effective in preventing cross infection, particularly in chickenpox and measles. It is highly probably that in certain installations, particularly at induction centers,

[1] The Medical Department of the United States Army in the World War. Communicable and Other Diseases. Washington: U. S. Government Printing Office, 1928, vol. IX, pp. 387-390, 411, 451.

[2] Wells, W. F., Wells, M. W., and Wilder, T. S.: Environmental Control of Epidemic Contagion. I. An Epidemiologic Study of Radiant Disinfection of Air in Day Schools. Am. J. Hyg. 35: 97-121, January 1942.

proper ultraviolet irradiation would have materially reduced the spread of such infection. If thus controlled, it is also highly probable that many men would later have contracted these diseases under much less favorable circumstances—on foreign soil or under combat conditions. It was thus particularly important that such possible means for control of chickenpox should not be used, at least in the United States.

The question has been raised repeatedly as to whether convalescent serum or gamma globulin was effective when injected during the incubation period in preventing or in attenuating chickenpox. There has been no conclusive evidence since World War I, or during World War II, that such attempts at passive immunization have been successful.

INCIDENCE

Table 14 outlines the incidence of chickenpox during World War II.

It is of some interest that, while the annual rates of chickenpox in the Latin American area for 1942 and 1943 were only slightly higher (0.45 and 0.74) than those recorded for the same years in the continental United States (0.42 and 0.54), during the next 2 years they rose considerably above the rates in the United States. The annual rates per 1,000 average strength for 1944 and 1945 in the Latin American area are reported as 1.41 and 2.27, respectively, as compared with 0.71 and 0.61 for the same years in the United States. There was at least a strength of 100,000 in the area at this period. An adequate explanation of the difference in annual rates is not available.

No satisfactory method of control of chickenpox was available for World War II nor were any investigations conducted on this disease, because of its relative unimportance to the United States Army.

TABLE 14.—*Incidence of chickenpox in the U. S. Army, 1940–45*

[Rate expressed as number of cases per annum per 1,000 average strength]

Year	Total		United States		Overseas	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
1940	[1] 112	0.33	[2] 84	0.33	[3] 21	0.32
1941	[1] 562	.42	[2] 456	.40	[3] 72	.61
1942	1,202	.37	1,124	.42	[4] 78	.13
1943	3,108	.45	2,815	.54	[4] 293	.17
1944	3,644	.47	2,831	.71	[4] 813	.21
1945	2,710	.36	1,785	.61	[4] 925	.20

[1] Includes all admissions.
[2] Data include Alaska and cover enlisted men only.
[3] Data are for white enlisted men and native troops only.
[4] Data include admissions on transports.

CHAPTER III

Common Respiratory Diseases

Philip E. Sartwell, M. D.[1]

The most important military diseases in terms of total morbidity, now that the intestinal and arthropodborne diseases have been largely controlled, is the group of acute minor respiratory tract infections generically termed common respiratory diseases. This statement does not necessarily apply to operations in tropical areas, but it is demonstrably true of forces in the United States and other parts of the Temperate Zone under usual conditions. It can be made with even greater emphasis during periods of mobilization. While the average period of disability or hospitalization is relatively short (estimated at 6.7 days in 1945), nevertheless the loss in effective manpower is very large. With the inclusion of influenza, these diseases were responsible for about 4 million admissions to hospital or quarters during World War II or 22 percent of admissions for all causes.

In comparison with the interest taken in malaria, typhus, hepatitis, and influenza, the minor undifferentiated respiratory infections attracted little attention during the war, less, at any rate, than their prevalence would seem to justify. This is perhaps due to the ill-defined character of the group and to the probability that it includes several diseases of distinct but undetermined etiology. Furthermore, it is well known that intensive programs of field and laboratory research on the common cold in the past have yielded comparatively little information of value; this tended to discourage further investigation when there were other fields which offered much better hope of progress. Since the war, reports of the isolation and cultivation of a common-cold virus in the embryonated hen's egg have appeared and are certain to stimulate advances in the field.

Because of the limited amount of investigation undertaken during World War II and the present inadequacy of control methods, the major part of this chapter will be devoted to epidemiologic features of common respiratory disease as revealed by analysis of routinely collected statistics. A brief section will deal with the research on the etiology of these diseases which was done during the war. Control measures that have been studied or recommended will be summarized, and some of the unsolved problems on which further work might profitably be done will be mentioned.

[1] The author gratefully acknowledges the assistance of Col. Fratis L. Duff, MC, USAF, in securing data and reports—both statistical and narrative—for this study. In addition, Colonel Duff's investigation of respiratory disease morbidity of Army divisions from the time of their activation (p. 69) has been utilized with his permission.

Credit for much of the effort toward the study and control of respiratory diseases during World War II should go to Brig. Gen. James Stevens Simmons, Chief, Preventive Medicine Service, Office of the Surgeon General, and his staff. It was General Simmons' policy to maintain a close and continuing scrutiny of morbidity rates and to initiate a special epidemiologic inquiry at posts or commands from which unusually high rates were reported. The members of the Army Epidemiological Board and its Commission on Acute Respiratory Diseases under the direction of Dr. John H. Dingle were responsible for a large share of the research in this field.

REPORTING OF COMMON RESPIRATORY DISEASES

A definition of terms is important at the outset of this discussion. The diagnoses to be included in the expression "common respiratory diseases" are determined by Army reporting practices. Since the chief reliable source of information on incidence is the weekly statistical health report, it is necessary to accept the grouping used in that report, which is satisfactory in most respects. The health report provides for the inclusion of the acute phases of the following: Coryza, rhinitis, nasopharyngitis, tonsillitis, pharyngitis, laryngitis, and bronchitis. It is recognized that, while the etiology of none of these conditions is known, anatomical differentiations are probably of little importance in their etiology or epidemiology and that all acute inflammatory conditions of the upper respiratory tract, exclusive of sinusitis, are best grouped together for study. Respiratory diseases with a specified etiology (as, for instance, streptococcal or diphtheritic diseases) were not included if the etiology was known and stated; there are undoubtedly many which failed to be recognized.

One exception to the statement that diseases of known etiology were separately reported is influenza. Influenza was reported separately on the statistical health report but was tabulated together with common respiratory diseases. Several points which are pertinent to this inclusion may be mentioned. First, it has become evident in recent years that in many instances it is impossible to differentiate influenza from other respiratory infections on a clinical basis. Second, laboratory tests for the recognition of influenza require much time and can be performed on so few cases that little or nothing can be done to improve the accuracy of routine morbidity reporting. Third, probably in recognition of these two facts, influenza is not reported as such from most Army installations except when a substantial epidemic is known to be in progress.

It is thus impossible to obtain any reliable estimate of how large a share influenza plays in the respiratory disease problem. Intensive studies will be necessary to determine this point. A number of recent studies have suggested that sporadic or endemic cases of virus influenza are more common in military populations than was once supposed.

In this chapter, it is pointed out that influenza is known to have been

prevalent among troops in the United States shortly before and during World War II at the following times: December 1940 to February 1941, December 1943 to January 1944, and in December 1945. The third of these epidemics was much less extensive than the first two. Overseas, United States troops experienced epidemics at about the same times, although the impact of these epidemics varied in different areas and, especially in tropical areas, was generally lighter than in the United States.

Evidence suggesting that the popularity of influenza as a diagnosis may have been waning during the war period is found in data secured from analysis of individual medical records. In 1942, 6.8 percent of cases of common respiratory diseases (including influenza) among troops in the United States were diagnosed as influenza; in 1943, the percentage for this diagnosis fell to 5.9; in 1944, to 3.5; and in 1945, to 1.6.

This sharp downward trend is not consistent with what is known of the prevalence of influenza over the period, which would lead one to expect 1943 to be the high year and 1945 the next highest. It is more in keeping with a progressive decrease in willingness to make a diagnosis of influenza in the absence of specific evidence of the disease—evidence which can be obtained in only a very small proportion of cases.

Although estimates will vary widely for different times and places, it is probable that only a minority of the respiratory infections see in military populations are caused by a known agent (if one excludes the common-cold viruses recently described, the frequency of which is as yet wholly unknown). Furthermore, an unknown share of these illnesses are probably not infections at all but may be responses to irritant agents (as an example, hay fever) or local manifestations of systemic conditions, such as reactions to immunizing agents.

Certain diseases, as they have come to be better recognized, have been excluded from the common respiratory diseases group. Thus, in March 1942, a circular letter from The Surgeon General described the character of primary atypical pneumonia, which had recently been observed to be a rather important disease in military life, and indicated that it was to be reported separately.[2] The 18 October 1943 revision of the statistical health report included a space for this diagnosis. The effect of this change on reported incidence of common respiratory diseases cannot be evaluated, as such a change is usually gradual. It is likely that a considerable number of cases once listed under common respiratory diseases later came to be termed atypical pneumonia and hence disappeared from the category discussed in this chapter. Some deletions from the group in the latter years of the war may have resulted from the increased emphasis on separate reporting of streptococcal sore throat. Provision for separate reporting of streptococcal sore throat was included in the October 1943 revision of the statistical health report. In September 1944,

[2] Circular Letter No. 19, Office of the Surgeon General, U. S. Army, 2 Mar. 1942, subject: Primary Atypical Pneumonia, Etiology Unknown.

the statement was made that "this diagnosis includes cases of tonsillitis or pharyngitis known or suspected to be caused by the beta hemolytic streptococcus." [3] Such changes in reporting practices are in part, at least, responsible for a shift in the proportion of different diagnoses on the individual medical records.

There are two possible sources of information on the incidence of respiratory diseases during the war period. One is the statistical health report, a summary report which was forwarded weekly by each post and major unit to The Surgeon General. This was a working report, used by the Preventive Medicine Service, Office of the Surgeon General, and by other commands as the basis for epidemiologic investigations, institution of special control measures, and periodic appraisal of the Army's health. The statistical health report included the following data which are pertinent to the subject of this chapter: (1) The average military strength of the post or command, (2) the number of admissions for common respiratory diseases, and (3) the number of admissions for influenza. These reports were consolidated in the Office of the Surgeon General to obtain service command, overseas theater, total United States and overseas, and total Army rates. They were also consolidated temporally to obtain monthly and annual rates (a month being either a 4- or 5-week period, depending on the number of Fridays which fall within the calendar month). All rates, whether for a period of 1 week, 1 month, or 1 year, were converted to an annual basis; that is, they represent the number of admissions per 1,000 troops that would have occurred had the rate in the particular time period studied continued over a full year.

The other source of information is the individual medical record, which was completed for each soldier after discharge from hospital and forwarded to the Office of the Surgeon General. These records constitute the most reliable data on Army morbidity. However, because of the magnitude of the task of processing these records, the required detailed data from this source were not available at the time this chapter was prepared, and some of the details (for example, rates by week) are not obtainable from this source. Therefore, the statistical health report is utilized as the basis for routine analyses, employing tabulations of the individual medical record only in special studies.

An attempt was made to estimate the comparability of common respiratory disease frequencies in the statistical health report and the individual medical record. For this comparison, influenza admissions were added to the individual medical record totals. It was found that in the United States and some of the overseas areas the agreement was fairly good but that certain areas, notably the European theater and the Pacific, showed large disparities in some or all of the war years. Within the United States, rates shown in statistical health reports were uniformly a little higher, but in none of the war years did the difference exceed 10 percent, as seen in table 15. It should be noted that the statistical health report rates consisted of incidence (total cases), whereas

[3] War Department Technical Bulletin No. 92, 15 Sept. 1944.

the individual medical record rates are admissions. It is to be expected, therefore, that the former would be higher—the amount of the statistical health report excess depending upon the extent of secondary diagnoses.

TABLE 15.—*Morbidity rates for common respiratory diseases and influenza in the Army in the United States, by source of data and year, 1942–45*

[Rate expressed as number of cases per annum per 1,000 average strength]

Year	Individual medical record [1] (admissions)	Statistical health report (incidence)	Percent excess in statistical health report
1942	239	243	1. 7
1943	225	247	9. 8
1944	140	147	5. 0
1945	108	116	7. 4

[1] Based on a sample of records.

A comparison of monthly rates computed from the two sources for troops in the United States during 1942 and 1944 showed close parallelism, and the excess in statistical health report rates was chiefly evident in the first half of both years. It is concluded that rates based on statistical health report data are a satisfactory index to the incidence of common respiratory disease, at least for troops in the United States.

Only illnesses of a certain degree of severity are reported in military practice. If the soldier who attends sick call is not considered ill enough to be taken off duty and admitted to hospital or quarters, he goes unreported. Criteria for taking men off duty varied widely, depending on the policy of the post or command surgeon and the dispensary surgeon, as well as type of duty and facilities for care. Administrative policies with respect to placing men on quarters status also affect the admission figures.

Certain variations in the rates may be considered as "artifacts" in the sense that they do not reflect true differences in incidence. An example of these is the usual marked drop in admissions during weekend periods, at least in situations where weekend leaves are obtainable. Most of these artifacts do not greatly affect the data used in this study, but the tendency for low rates or a slowing up in the normal seasonal increase at the Christmas and New Year's holidays needs to be kept in mind.

These remarks indicate that the disease group under discussion is a very vague group, subject to large irregularities in recognition and reporting. While this is true, it must be emphasized that few sources of information concerning mass behavior of the group approach the accuracy possessed by military records. The reasons for this are inherent in the availability of medical care, routine collection of data, and continuous enforcement of relatively simple rules for uniform reporting which the armed services provide.

EPIDEMIOLOGY

Epidemiologic information about common respiratory disease must be characterized as descriptive epidemiology; that is, certain facts are known about the selection of the disease as to time, place, and person without there being any real comprehension of the reasons underlying that selection. The existing information is of interest because it affords clues which, in the future, may point the way toward a more basic understanding and because it makes possible the prediction of and suggests ways of avoidance of periods of high morbidity.

The time distribution of common respiratory diseases will first be considered. The period covered by this history was preceded by 2 years (1940–41) of rather high morbidity; 1942 and 1943 were years of moderately high rates, while in 1944 and 1945 the rates were quite low, as seen in table 16.

TABLE 16. *Admission rates for common respiratory diseases and influenza in U. S. Army, by area of admission and year, 1940–45*

[Rate expressed as number of admissions per annum per 1,000 average strength]

Year	Total Army	United States	Overseas
1940	310	342	176
1941	311	329	132
1942	230	243	163
1943	231	247	181
1944	140	147	132
1945	109	116	105

It will be noted that for troops in the United States there was a sharp downward trend in rates over this period, while for troops overseas no trend was evident, although the rates for 1945 were lower than rates for any previous year. The United States trend is at least partially explained by the recruit epidemic phenomenon which is discussed later. The proportion of recruits comprising the United States military population declined progressively during the war. As there were few or no recruits (in the sense of men with less than 6 months' training) overseas, this trend would not be expected to be manifest in the theaters of operations.

It is not thought that influenza had a very great effect on the annual rates except in 1941 and, to a lesser extent, in 1943. Its influence on overseas morbidity was probably much less than on United States rates.

It is well known that there are large seasonal fluctuations in common respiratory disease in the Temperate Zone, but seldom have careful statistical measurements of this phenomenon been made. The "average" seasonal cycle of rates based on monthly data for the period 1924–47 has been computed.[4] Through a curve-fitting process, it was possible to construct a synthetic annual

[4] Health of the Army, June and September 1947.

cycle which agreed closely with the average seasonal curve after smoothing and correcting for the influence of end-of-month paydays and principal holidays. This artificial cycle is the sum of three terms: The mean; a sine curve of a 1-year period with maximum in February; and a sine curve of much smaller amplitude, a one-half year period, with maxima in February and August. The highest of the weekly smoothed average rates, which occurred in mid-February, was 352; the lowest, in mid-July, was 81.5. Thus, on the average, over the United States the midwinter rate was somewhat over four times as high as the mid-summer rate.

Viewing the month-by-month experience of the nine service commands, one is impressed with the essential agreement in the chronologic pattern over the entire United States. While the magnitude of rates differed between service commands and the seasonal curve for each of the four war years differed from all the other years, the pattern of rates in a particular year tended to be alike all over the country, as though the "epidemiologic unit" for some of the chief components of this disease group were the Nation or perhaps even the continent, as it was for meningococcal meningitis. This may be explained in part by the influenza epidemics, which were clearly nationwide, and the proportion of new recruits in each service command rose and fell in the same fashion at the same times. There is no evidence that there were important regional influences.

Seasonal trends in overseas commands are a result of geographic location and will be discussed later (p. 65).

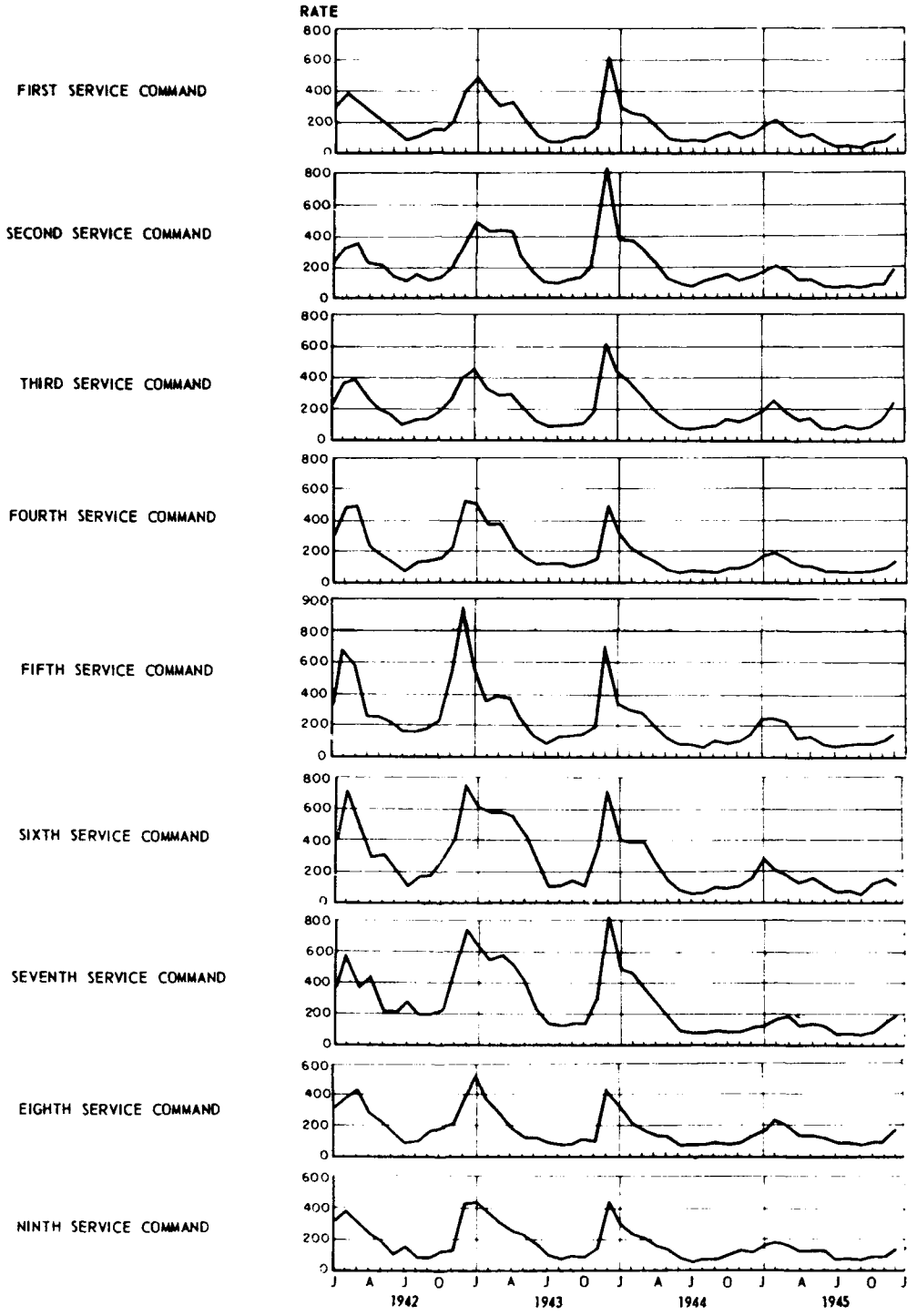
Chassan made a study of incidence by employing monthly respiratory disease admission rates for troops in the United States over a 40-year period—from 1906 to 1946.[5] He found a high correlation between rates for successive months, the coefficient of correlation averaging 0.84 and ranging from 0.71 for the December–January pair of months up to 0.89 for the August–September pair. This means, for example, that, if in a particular year the August rate is high, the September rate is likely to be proportionately high, and vice versa. The correlation is still evident when pairs of months separated by a considerable interval of time are taken; thus, the coefficient of correlation remains positive and larger than 0.5 when the interval between the months being compared is up to 9 months. Seasonal changes do not influence the coefficient of correlation.

As has been indicated, the most powerful factor determining common respiratory disease rates is the proportion of recruits in the command. With this in mind, geographic differences in rates may be described. Within the United States, the comparison among the nine service commands shows a general tendency toward higher rates in more northerly areas, as shown in table 17 and chart 7. The service commands, arranged in descending order of rates for the war period, are as follows: Sixth, Seventh, Fifth, Second, Third, First, Fourth, Ninth, and Eighth. This cannot be attributed to the recruit factor, since a somewhat higher proportionate recruit strength was present in the southern parts of the country owing to more favorable climatic conditions for

[5] Chassan, J. B.: The Autocorrelation Approach to the Analysis of the Incidence of Communicable Diseases. Human Biol. 20: 90-108, May 1948.

CHART 7. *Incidence rates for common respiratory diseases and influenza in the U. S. Army, 1942–45, by service commands and months*

[Rate expressed as number of cases per annum per 1,000 average strength]



training. Examination of the seasonal curves of incidence indicates that the magnitude of the seasonal rise and fall was greater in those service commands which had the highest rates.

TABLE 17 *Admission rates for common respiratory diseases and influenza, by service command and year, 1942–45*

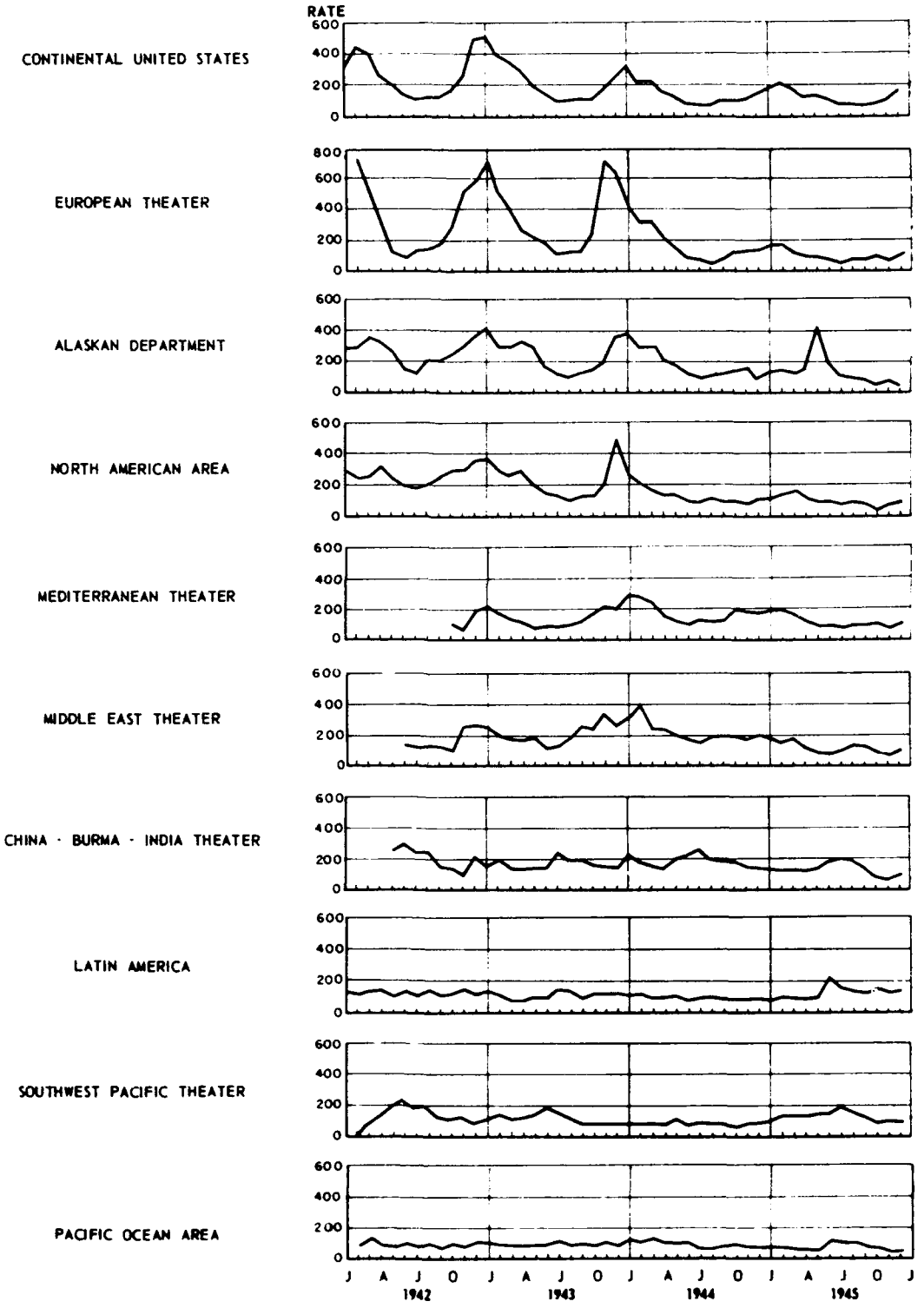
[Rate expressed as number of cases per annum per 1,000 average strength]

Service command	Admission rate				
	1942	1943	1944	1945	Average 1942–45
First	206	246	157	94	187
Second	200	310	191	111	211
Third	226	235	177	122	197
Fourth	242	230	125	102	182
Fifth	394	284	156	124	221
Sixth	348	366	194	116	265
Seventh	361	375	196	119	240
Eighth	216	202	123	130	167
Ninth	206	213	146	114	177

The over-all experience of the overseas areas will next be considered. The comparative rates should not be given great weight, since troop strengths varied widely in different theaters. The distribution of personnel within the areas was not constant. For example, no United States troops were in the Mediterranean theater until late in 1942. During most of 1943, all United States forces in this theater were in North Africa, while in the succeeding years they moved into Italy where environmental conditions were quite different. Thus, it is apparent that the theater designation is only an approximate and inconstant description of the geographic area of service. Certain generalizations can, however, be made. The average rate of respiratory diseases (203) was higher in the Alaskan Department than in any other major region. The North American area (with an average rate of 193) was next, succeeded by the Middle East (184), China-Burma-India (151), Mediterranean (including North Africa) (145), and European (137) theaters; and Southwest Pacific (111), Latin American (including Antilles Department) (103), and the Pacific Ocean (81) areas in that order. There are very marked differences in the extent of seasonal variations in these areas which do not entirely parallel the rates (chart 8). Seasonal variation was most extreme in the European theater, was moderate in the Alaskan, North American, Mediterranean, and Middle East regions; and was very small or negligible in the China-Burma-India theater, Latin America, Southwest Pacific, and Pacific Ocean areas. One may say, generally, that the more northerly regions had both the higher rates of admission and the more marked seasonal rises and falls. Rates were low and constant in parts of the world with tropical and equable climates. For example, in

CHART 8. *Incidence rates for common respiratory diseases and influenza in the U. S. Army, by area and months, 1942–45*

[Rate expressed as number of cases per annum per 1,000 average strength]



the Latin American area, the average rate was 103 per 1,000 per year over the 4-year period. Expressed as a proportion of total disease admissions, common respiratory diseases and influenza ranged downward from 37 percent in Alaska and the North American area to 13 percent in the Southwest Pacific Area.

It is commonplace to hear that the climate in a particular locality is conducive to minor respiratory diseases. A systematic analysis of rates at individual posts would have to take into consideration all the following factors: The length of service of the troops; rate of turnover; type of duty; age composition; and the prevalence of recognized epidemics of influenza, streptococcal disease, or other specific agents. Much of this information is not available, and such studies, therefore, will not be attempted here. It may be stated, however, that stations within the Sixth and Seventh Service Commands, particularly in the States of Colorado and Wyoming, consistently experienced comparatively high rates of admission for common respiratory diseases. There are several reasons which suggest that these high rates reflect in part an excessive prevalence of streptococcal disease at certain stations.

The influence of race, sex, and age cannot, unfortunately, be studied in detail owing to the dearth of information available at the present time. There have been three studies which have suggested that Negro troops experience a lower rate of admission than do white troops. In the first of these, Gordon found that rates in the European theater during World War II were consistently lower for Negro than for white troops.[6] The Commission on Acute Respiratory Diseases found a similar difference in its intensive studies at Fort Bragg, N. C., but could not exclude the possibility that it was due to differences in policy with respect to placing men under treatment. The author encountered substantial differences at all seasons between the admission rates of white and Negro recruits in his investigation at Fort Dix, N. J., in 1947–50.[7] Both groups showed large epidemics in units recruited and assembled during the winter months, but the magnitude of these rates was usually more than twice as high during the first few weeks of service for white troops as for Negro troops. No reasons based on administrative policy or factors of morale could be discovered to explain the difference.

With respect to sex, a limited number of observations on Women's Army Corps groups have indicated that admissions for common respiratory diseases, as for numerous other causes, are more frequent than among male troops. Thus, during the period from June 1944 to December 1945, inclusive, the admission rate for common respiratory diseases and influenza among Women's Army Corps personnel in the United States was some 60 percent higher than that for all Army personnel, though the pneumonia admission rate was slightly lower than that for all Army personnel.[8] This is consistent with experience reported by a number of industries and the findings of a majority of com-

[6] Gordon, J. E.: A History of Preventive Medicine in the European Theater of Operations, U. S. Army, 1941–45 [Official record.]

[7] Sartwell, P. E.: Common Respiratory Disease in Recruits. Am. J. Hyg. 53: 224–235, March 1951.

[8] Enlistment, Health, and Discharge of the WAC. Bull. U. S. Army M. Dept. 6: 276–287, September 1946.

munity surveys of illness morbidity. It may be doubted whether the incidence of respiratory infections is higher among females under military circumstances than among males. The difference may represent a greater tendency for Women's Army Corps personnel to report for medical treatment or a greater likelihood of their being admitted to hospital or quarters by the medical officer when they seek such care.

It has not been possible to secure any data with respect to admissions for common respiratory diseases by age. However, some studies have indicated that, over the span of ages represented by military organizations, the incidence of such illnesses does not vary much with age. As these illnesses do not appear to produce any prolonged immunity, this is the expected pattern.

INCIDENCE AMONG RECRUITS

The phenomenon of high incidence of respiratory infections among recruits has been universally noted and commented upon by students of military medicine. The most detailed observations made during World War II were those of the Commission on Acute Respiratory Diseases of the Army Epidemiological Board.[9] A somewhat similar study was conducted at Fort Dix by the author over the period 1947–50.[10] In both studies, morbidity rates specific for week of service were obtained by following units of company or battalion size from the time of their organization. The studies cited have, in general, shown that recruits arriving on the post in winter and thrown together with other recruits in training companies usually experience, almost at once, sharp epidemics of upper respiratory diseases. In the author's studies, these epidemics most commonly reached their peak in the third or fourth week of training, and incidence was down to a low level by the sixth to eighth week. Sometimes the rates go as high as 3,000 admissions per 1,000 troops per year for this brief period, returning to a baseline of less than 300 per 1,000 per year at the end of the epidemic. This process went on in the absence of any epidemic conditions among seasoned personnel on the post. Among the units observed, there were few which failed to undergo an epidemic, but the observations are too limited to say how general such experiences are in different years or in other parts of the country.

Troops inducted and beginning their training at other seasons of the year were also observed. Those inducted during the summer months generally had no epidemics, while those inducted in the spring and fall underwent a variety of experiences, seldom, however, having the explosive outbreaks that were characteristic of units brought in the service during the winter. The Fort Dix study showed that, of troops arriving in January or February of the winters of 1947–48 and 1948–49, 12 and 15 percent, respectively, were admitted

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1. Commission on Acute Respiratory Diseases. Acute Respiratory Disease Among New Recruits. Am. J. Pub. Health 36: 439–450, May 1946. 2. Commission on Acute Respiratory Diseases. Epidemiology of Atypical Pneumonia and Acute Respiratory Disease at Fort Bragg, North Carolina. Am. J. Pub. Health 34: 337–346, April 1944.

See footnote 7, p. 67.

to hospital during their first 8 weeks of military service and that during the summer of 1949 only about 1 percent were admitted.

The following discussion is based on a study conducted under the author's supervision by Col. Fratis L. Duff, MC. This study has provided new and confirmatory evidence regarding the effect of seasoning of troops as well as the climatic and geographic factors in common respiratory diseases.

During the period 1940–41, nearly all the Army divisions ordered to active duty were National Guard divisions. Officers and men of existing guard organizations were inducted as units, and the divisions were brought to strength by assignment of newly inducted men. Troops in these divisions consisted mainly of men who had lived in the same general area in which they received their training. In 1942, new Army of the United States and Reserve divisions were activated by assigning sufficient officers and noncommissioned officers to serve as a training cadre and then by rapidly filling the ranks with newly inducted troops. Troops in these new divisions came from all parts of the country. The period of training in the United States for both types of divisions was never less than 1 year. During the course of training, all divisions received some additional troops as replacements for men discharged from service or reassigned to other units. However, the number of changes in composition was usually quite small in proportion to the total divisional strength. Overseas, the personnel turnover rate was considerably larger owing to combat losses, reassignments of subordinate units, and later, the return of personnel to the Zone of Interior. After 1943, all recruits spent a period of at least 2 months in training at a basic training center before receiving their first operating assignment.

Quotas for most of the divisions formed in the early part of the war were filled within a period of a few weeks, so that early in their training the divisions consisted of a large majority of troops having approximately the same length of service, together with some seasoned men having diverse military backgrounds. This, as Colonel Duff recognized, provided opportunity for a study of common respiratory diseases in divisions, with the seasoning factor at work. Furthermore, since divisions began training at different calendar periods and in different parts of the country, there was hope of separating for study the epidemiologic factors of time, place, and military age. Colonel Duff was able to obtain the statistical health reports of 19 divisions and 1 artillery battalion covering periods of at least 1 full year from the time of organization. Some of these divisions could be followed for periods of 3 or 4 years, including long periods of overseas service and combat operations.

The information abstracted from each statistical health report included the weekly average strength and the number of admissions for common respiratory diseases, influenza, and other acute communicable diseases; however, only the common respiratory diseases and weekly average-strength figures were systematically utilized. From other sources, information was obtained as to periods of service of the division at each post or maneuver area in the United States, time of embarkation for foreign duty, and combat

periods while overseas. An arbitrary rule was made that the period of service of a division would be dated from the month in which the increase in strength from cadre to full or nearly full operational strength took place. In other words, for the purposes of this epidemiologic study, the time of mobilization of the division was taken as that month in which its recruit population, rather than its cadre, was assembled.

The experience of each unit during its first year of training was intensively studied. Admission rates for common respiratory disease were computed for the first 4-, 8-, and 12-week periods, for the first and second 6 months, and for the entire year (table 18). Monthly rates for the year were also computed; rates for organizations selected as typical are reproduced graphically in charts 9 and 10.

TABLE 18.—*Admission rates for common respiratory diseases in selected units*

[Rate expressed as number of cases per annum per 1,000 average strength]

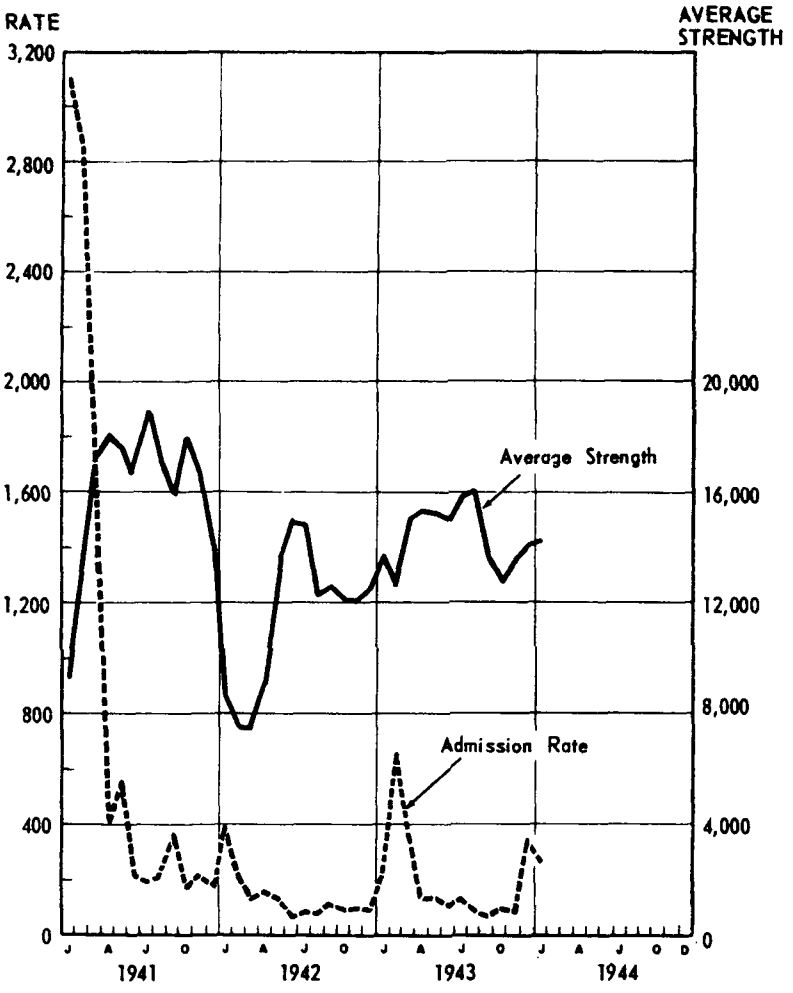
Unit	Month of mobilization	Training area	Admission rates			
			First 4 weeks	First 12 weeks	First 6 months	First year
30th Infantry Division	September	South Carolina	186	258	929	481
2d Armored Division	November	Georgia	76	366	323	195
35th Infantry Division	January	Arkansas	3, 100	2, 328	1, 229	694
26th Infantry Division	February	Massachusetts	3, 266	2, 462	1, 160	604
33d Infantry Division	March	Tennessee	3, 694	1, 687	973	749
77th Infantry Division	April	South Carolina	421	449	318	272
85th Infantry Division	May	Mississippi	169	86	123	430
81st Infantry Division	June	Alabama	118	82	92	132
89th Infantry Division	July	Colorado	384	252	660	710
95th Infantry Division	do	Texas	98	69	87	165
83d Infantry Division	October	Indiana	0	1, 187	631	367
91st Infantry Division	November	Oregon	702	726	432	272
11th Armored Division	do	Georgia	222	238	153	91
10th Armored Division	do	do	134	266	153	89
103d Artillery Battalion	December	Louisiana	558	606	352	214
99th Infantry Division	November	Mississippi	456	576	637	183
94th Infantry Division	December	Kansas	1, 322	1, 464	760	408
106th Infantry Division	March	South Carolina	638	440	228	236
63d Infantry Division	September	Mississippi	78	61	329	204
16th Armored Division	October	Arkansas	59	89	166	107

Divisions mobilized in the northern half of the United States had higher rates over the first year, as a rule, than those in the South. Of the 7 divisions trained in the States in the North, 4 had average rates of 600 or higher over the first year, while of the 12 divisions in the South, there were none with a rate above 600.

The incidence curves of these 19 divisions are quite varied, but there is an underlying common pattern which may be described in general terms. In

CHART 9.—*Incidence rates for common respiratory diseases and influenza and average strength of the 35th Infantry Division, U. S. Army, by years and months, 1941–44*

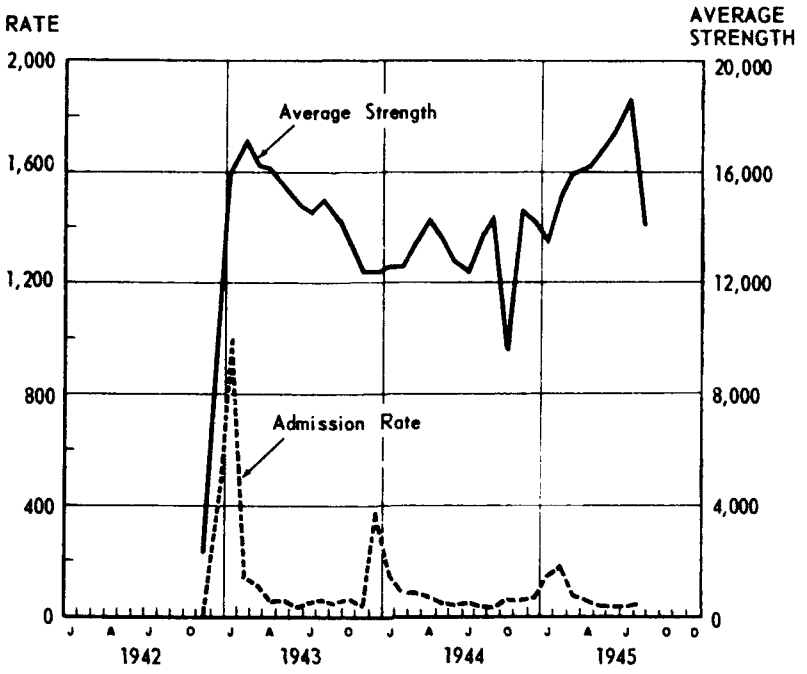
[Rate expressed as number of cases per annum per 1,000 average strength]



the divisions for which there were several years' records available, the rate for the first winter after formation of the division was always highest, the second and third winters having successively diminishing rates. Rates were regularly higher for divisions training in the North than for those in the South, as already stated. Divisions formed in the period from May to October experienced a sharp peak in either the succeeding December or January, but there was usually only 1 epidemic month. One exception to this statement was the 89th Infantry Division, which trained at Camp Carson, Colo., and experienced a peak in December 1942 but continued to have rather high rates until the following June, possibly representing the incidence of streptococcal disease. Units having the highest rates were those assembled during the winter months in the Northern States. There was no tendency for high

CHART 10. *Incidence rates for common respiratory diseases and influenza and average strength of the 99th Infantry Division, U. S. Army, by years and months, 1942–45*

[Rate expressed as number of cases per annum per 1,000 average strength]



rates to be associated either with periods of training in maneuver areas or with combat operations overseas.

Examples of the most extreme effect of the recruit epidemics on divisional rates are the 26th and 33d Infantry Divisions, both National Guard units, which were largely composed of men residing in the same section of the country where the divisions were assembled. The 26th was ordered into Federal service on 16 January 1941 and moved to Camp Edwards, Mass., in February 1941 (the month in which the strength rose above 8,000). In that month, the rate was 3,415, dropping precipitously in succeeding months to 120 in July. The next winter, while again stationed in Massachusetts, the division experienced its highest rate of 372 in February. The 33d Infantry Division was ordered into Federal service on 5 March 1941 at Chicago, Ill., and shortly afterward moved to Camp Forrest, Tenn. The strength first rose to 10,000 at the end of March 1941, and in the following month the admission rate was 3,697, thereafter dropping dramatically to 324 in August. Maximum rates of about 800 were experienced in the following two winters.

The effect of this high recruit susceptibility is, of course, high respiratory disease rates in the mobilization period of wars and in troops in the United States as contrasted with those in overseas commands. It has already been pointed out that the trend of rates for troops in the United States was downward from 342 in 1940 to 116 in 1945. No direct estimate of the proportion

of unseasoned troops in the United States at various times has been found, but figures for the number of inductions by months have been obtained from The Adjutant General, and so it is possible to compute for any period during the war the number of men inducted during the previous 3 months. Some of these men were discharged before completing 3 months' service, and a very few of them, perhaps, were assigned to overseas duties. The large majority, however, were still undergoing training in the United States 3 months after induction. Hence, this number (inductions during the past 3 months) was taken in relation to the total troop strength in the United States each month, as a rough estimate of the percentage of troops in this country with less than 3 months' service. By means of a series of scatter diagrams, the correlation between this figure and the corresponding United States common respiratory disease rate for each calendar month over a 5-year period (1941–45) was studied. In nearly all months, some correlation was evident; it was somewhat greater in the colder months. For the months of December 1943 and January 1944, incidence was higher than expected from the recruit percentage (influenza A being epidemic during this period). The correlation for the months of January and February is indicated in chart 11. This association does not of itself, of course, imply a direct relationship between respiratory disease rates and mobilization but is quite consistent with the abundant evidence from other sources.

RESPIRATORY INFECTIONS ABOARD TRANSPORTS

Considerable trouble was experienced in the European theater during 1942 with the high incidence of upper respiratory infections acquired by troops on the ocean voyage from the United States. Of three units surveyed, 51 to 75 percent had colds during a period of approximately 5 to 7 weeks including the preembarkation period, the voyage, and the postembarkation period despite the fact that the voyage was in late summer. The frequency of colds was low prior to embarkation, increased more than tenfold while in transit, and continued high, though subsiding, after arrival. Overcrowding on shipboard, which was unavoidable at the time, was considered responsible. On the other hand, a convoy of 24 transports left New York and Boston in December 1943, at the height of the influenza epidemic of that year and at a time when respiratory diseases were prevalent in the New York staging area. Although serious difficulty was feared, this convoy which carried 63,750 troops encountered no more respiratory diseases during the voyage than would be expected at the season, in the opinion of ships' surgeons and investigators in the theater.

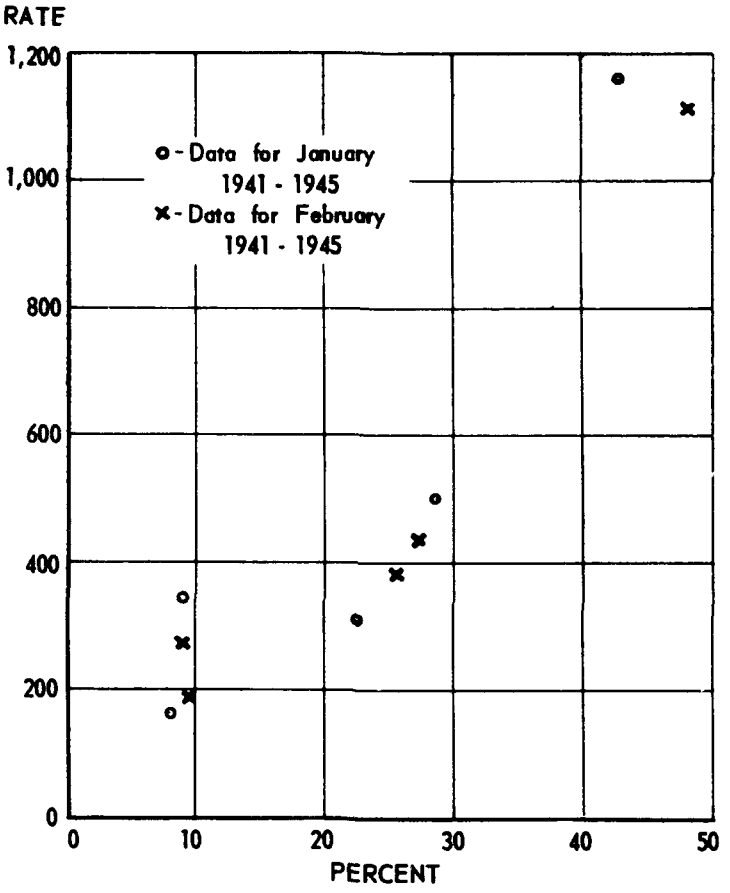
ASSOCIATION WITH OTHER DISEASES

The seasonal pattern of most of the diseases transmitted by respiratory secretions is much the same. Measles, meningococcal meningitis, mumps, and streptococcal infections are at their height during the late winter or spring; diphtheria (in the United States), in late autumn; pneumonia, usually in mid-

CHART 11.—*Incidence rates for common respiratory diseases in Army troops in the continental United States, January and February, 1941–45*

[Rate expressed as number of cases per annum per 1,000 average strength]

[Percent scale represents estimated percentage of troops with less than 3 months of service]



winter or late winter. Furthermore, most of these diseases show a relationship to length of service in general like that of common respiratory disease. These two factors are sufficient to account for the apparent association of common respiratory disease with other respiratory infections. It is possible that coughing, sneezing, and excessive respiratory secretions characteristic of colds facilitate the transmission of other diseases. In Army camps it was common, though by no means invariable, when common respiratory disease was epidemic, for primary atypical pneumonia and often measles, mumps, or streptococcal infection also to be prevalent. An association with meningococcal meningitis was observed in the winter of 1942–43 in the United States.[11] On the other hand, the sharp outbreaks of influenza in 1943 and 1945 occurred at a time when the common respiratory disease rate was rather low.

[11] Sartwell, P. E., and Smith, W. M.: Epidemiological Notes on Meningococcal Meningitis in the Army. Am. J. Pub. Health 34: 40–49, January 1944.

ETIOLOGIC STUDIES

Most of the investigations of possible etiologic agents of so-called undifferentiated respiratory diseases made during the war under Army auspices were carried out by the Commission on Acute Respiratory Diseases of the Army Epidemiological Board. This Commission, headed by Dr. Dingle, maintained its laboratories and made its field observations at Fort Bragg. The Commission's findings have been published in a comprehensive series of papers and no attempt will be made here to do more than restate their salient features. Studies were made of the bacterial flora of the upper respiratory tract which led to the conclusion that the flora of patients hospitalized for undifferentiated acute respiratory diseases did not differ from that of healthy recruits.[12] In another study, only 6 percent of the cases of mild respiratory infection was shown to be streptococcal infection by bacteriologic and immunologic studies.[13] There are indications that streptococcal disease was quite uncommon during the war in this region. In a group of patients with exudative pharyngitis and tonsillitis, a condition which was found to constitute some 10 percent of respiratory disease admissions, the Commission reported that 25 percent had beta hemolytic streptococci in their throats and exibited a rise in titer of streptococcal antibodies during convalescence, another 25 percent had streptococci but did not develop antibodies, and the remaining one-half had no streptococci.[14] The conclusion was drawn that in approximately one-fourth of this series of cases the disease was of streptococcal etiology. No means of accurate diagnosis other than cultural and serologic tests were found.

The Commission undertook a series of studies employing human volunteers in attempts to transmit, experimentally, the agents of minor respiratory illness in which no bacterial etiology could be found.[15] The investigators were successful in producing illnesses of two clinical types by the use of bacteria-free filtrates of pooled nasal and pharyngeal washings. Subsequently, homologous and heterologous immunity was tested on the same volunteers. Immunity to reinoculation was found in individuals receiving a filtrate which induced minor illness with an incubation period of about 5 to 6 days. Such immunity was not demonstrated in persons given the filtrate which induced a coryzalike illness with incubation period of 1 to 2 days. No cross-immunity was demonstrated.

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[12] Commission on Acute Respiratory Diseases: Bacteriological Findings in Undifferentiated and Other Acute Respiratory Diseases. Medicine 26: 465-484, December 1947.

[13] Commission on Acute Respiratory Diseases: The Role of Lancefield Groups of Beta-Hemolytic Streptococci in Respiratory Infections. New England J. Med. 236: 157-166, January 1947.

[14] Commission on Acute Respiratory Diseases: Endemic Exudative Pharyngitis and Tonsillitis. Etiology and Clinical Characteristics. J. A. M. A. 125: 1163-1169, 26 Aug. 1944.

[15] Commission on Acute Respiratory Diseases: Experimental Transmission of Minor Respiratory Illness to Human Volunteers by Filter-Passing Agents. I. Demonstration of Two Types of Illness Characterized by Long and Short Incubation Periods and Different Clinical Features. J. Clin. Investigation 26: 957-973, September 1947. II. Immunity on Reinoculation With Agents From the Two Types of Minor Respiratory Illness and From Primary Atypical Pneumonia. J. Clin. Investigation 26: 974-982, September 1947.

The question of etiology is obviously still far from solution. As stated earlier, the possibilities must include not only infectious agents but irritant or allergic incitants, though there is little doubt that the large proportion of the cases are infections. An interesting subject of speculation is the relationship between minor infections of the upper respiratory tract and primary atypical pneumonia, itself a vaguely defined disease group. Some parallelism between the two conditions has at times been noted, though this alone should not be taken as indicating any etiologic relationship.

The diagnosis of common respiratory disease remains a matter of exclusion of recognized disease entities, such as streptococcal tonsillitis and pharyngitis, influenza, pneumonia, and the like. Much information with a bearing on common respiratory disease will be found in publications relating to these other diseases, notably those of the commissions of the Army Epidemiological Board on influenza, hemolytic streptococcal infections, and pneumonia.

PREVENTION AND CONTROL

During the war, promising measures for the control of certain specific respiratory infections were introduced. Influenza vaccine received its first large-scale trials; chemoprophylaxis proved successful under certain conditions in the control of streptococcal and meningococcal infections; methods for air sanitation (glycol vapors, ultraviolet irradiation, and dust-suppressive measures) were tried. Both chemoprophylaxis and air sanitation were based on the demonstrated effectiveness of these agents as weapons against streptococci. The reason for mentioning them here is the possibility that they may also be effective against the agent or agents of common respiratory disease. Sulfadiazine chemoprophylaxis has not, so far as known, been subjected to any rigid test of this possibility. From the lack of evidence that it has clinical value, however, it may be presumed that (1) its prophylactic effectiveness is due to a reduction in streptococcal or other bacterial infections and (2) if such infections are not prevalent, it will not reduce respiratory morbidity. Air-sanitation measures have been demonstrated to reduce the bacterial count of the air, and whatever efficacy they may possess is presumably a result of this action. The Commission on Acute Respiratory Diseases was unable to show that oiling of floors and blankets, a dust-suppressive measure, reduced the incidence of respiratory diseases at Fort Bragg during a period when bacterial infections were infrequent.[5] In theory, at any rate, one of the factors determining the effectiveness of air sanitation in reduction of morbidity is the extent to which the disease in question is airborne. No precise knowledge is available on the relative importance of direct or indirect contact, inhalation at close range of droplets of respiratory secretions, and true airborne infection (inhalation of minute droplet nuclei or dust particles containing an infectious

[5] (1) Commission on Acute Respiratory Diseases and Commission on Airborne Infections. A Study of the Effect of Oiled Floors and Bedding on the Incidence of Respiratory Disease in New Recruits. Am. J. Hyg. 43: 130-144, March 1946. (2) Whayne, Tom F., Housing. *In* Medical Department, United States Army, Preventive Medicine in World War II. Volume II. Environmental Hygiene. Washington: U. S. Government Printing Office, 1955, pp. 27-74.

agent and suspended in the atmosphere of enclosed spaces. It may be that the relative importance of these various modes of transmission differs for each of the respiratory infections (fig. 1).

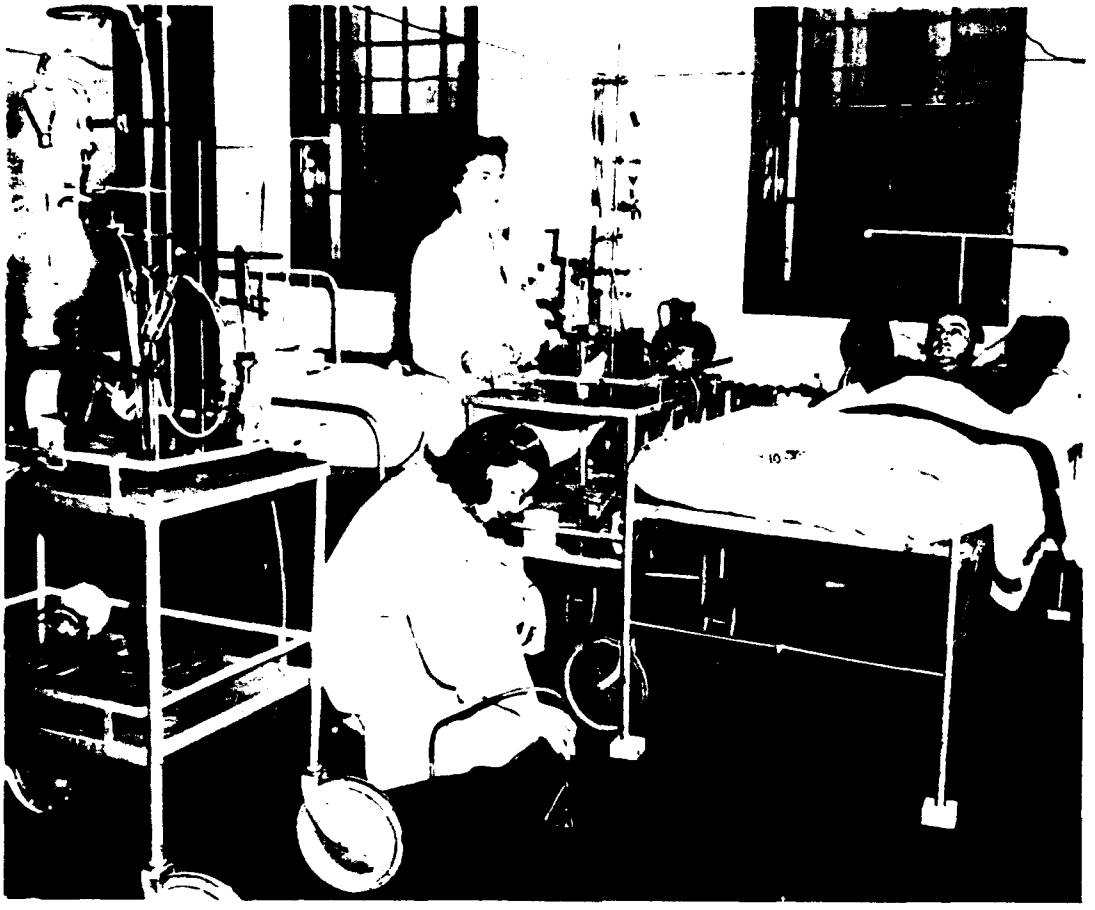


FIGURE 1. Culturing the environment of a ward. One bacteriological assistant cultures the air while the other cultures the floor dust.

Related to this problem is the question, to what extent is common respiratory disease transmitted in barracks, where men spend their sleeping hours, and to what extent is the disease transmitted in classrooms, messhalls, theaters, post exchanges, or other places where men congregate during the day. For the sake of completeness, still another possible source of infection needs to be mentioned; namely, foods and eating utensils contaminated by foodhandlers. There is little reason to regard the latter as important in this class of diseases, though it is true that occasional outbreaks of streptococcal pharyngitis have been traced to this source.

Observations of the Commission on Airborne Infections of the Army Epidemiological Board with respect to contamination of the environment with hemolytic streptococci are important to the understanding of atmospheric transfer of pathogens. Findings of this Commission demonstrated that in

wards and barracks the number of streptococci in bedding and floor dust is often large and that such activities as bedmaking and dry sweeping result in the resuspension in the air of these organisms. It was also observed that oiling of floors and blankets and introduction of triethylene glycol vapors into the air had a pronounced effect in reducing the content of viable streptococci in the atmosphere of ward or barracks. Unfortunately, no conclusive evidence that these measures will reduce the prevalence of common respiratory diseases has been produced.

It has long been assumed, largely on theoretical grounds, that reduction or avoidance of overcrowding is the most important measure within reach of the military surgeon for control of these diseases. Emphasis has been placed on the allotment of barracks space to provide every man with a certain minimum floor area or cubic footage of air volume and to maintain a certain minimum space between beds. Standards for minimum crowding have been established in War Department directives. The allowable minimum floor space per man, for example, was 60 square feet before World War II,[17] but this was temporarily reduced to 40 square feet during the war except for barracks housing recruits where space allotment was 50 square feet.[18] The change in standard is significant; it is probable that during any rapid mobilization a shortage of housing facilities for troops would develop, since it is possible to expand an Army faster than new housing can be built. This means that crowding is likely to be more severe at the same time that respiratory infections are at their peak owing to the large numbers of unseasoned recruits. To what extent crowding contributes to the difficulties is impossible to say. While the Medical Department has expressed its awareness of and opposition to the evils of crowding, it cannot be claimed that its views have been very effectively championed or upheld by command under the stress of military necessity.

Special epidemiologic investigations were made at various times during World War II when respiratory disease rates in particular posts rose to unusual levels. These investigations were conducted by post or organizational preventive medicine officers, by service command or theater headquarters, and in some instances by representatives of the Office of the Surgeon General. One investigation which will be described was carried out by representatives of the Army Epidemiological Board. Space allowance for troop housing was reduced in October 1942 from 60 to 40 square feet per man.[19] The Surgeon General subsequently requested that the Board survey conditions in Army camps resulting from the application of this order with particular reference to the current and expected incidence of acute respiratory diseases and meningococcal meningitis. Ten members of the Board and its commissions inspected 19 large posts in all parts of the country during December 1942.

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[17] Army Regulations No. 40-205, 15 Dec. 1924.

[18] (1) Army Regulations No. 40-205, 31 Dec. 1942. (2) Army Regulations No. 40-205, changes No. 2, 26 Mar. 1943.

[19] (1) Letter, The Adjutant General, to Commanding Generals, Commanding Officers, et cetera, 21 Oct. 1942, subject: Reduced Space Allowance at Posts, Camps, and/or Air Force Stations. (2) See footnote 18 (1).

Not only crowding but also heating, ventilation, messing facilities, and conditions in lavatories, post exchanges, and elsewhere were considered by this group. Crowding was found at some posts but not at others, and there was no particularly marked correlation between the extent of crowding and the seriousness of the respiratory disease problem. Most of the investigators felt that factors other than crowding in barracks were important, and several stated that they did not believe that crowding could be blamed for the respiratory disease rates. All, however, agreed as to the desirability of efforts to reduce crowding in barracks. At a special meeting of the Board held on 29 January 1943 and attended by representatives of the Office of the Surgeon General, Army Ground Forces, Army Air Forces, Services of Supply, and the Chief of Engineers, the findings of the investigators were summarized and discussed. It was emphasized that the order reducing space allowances to 40 square feet was a command decision made on the basis of military necessity. It was suggested that a return to 60 square feet per man in recruit reception and training centers would provide a partial solution and would automatically slow down recruitment. The Board finally adopted a resolution emphasizing the influence of crowding in barracks, messhalls, and recreation halls on the spread of meningitis, acute respiratory, and other epidemic diseases and endorsed the action of The Surgeon General in advocating a minimum of 60 square feet per man. It was pointed out that this would relieve crowding not only in barracks but in messhalls, washrooms, latrines, post exchanges, and elsewhere. Following this action, the regulation on space allowance was revised on 26 March 1943 and provided a minimum of 50 square feet per man in reception centers, except in emergencies, and 40 square feet at other stations.[20]

Other preventive measures which have long been taught in military medicine and are frequently employed are the provision of adequate ventilation arrangement of bunks to permit head-to-foot sleeping, and erection of shelter-halves as screens between beds. Like the spacing out of beds, these methods have received no rigid test. All are based on the assumption, which may be doubted, that transfer of respiratory infections occurs chiefly in barracks. It is most difficult to enforce corresponding measures in places of daytime congregation, particularly as the troops themselves are gregarious and tend to gather in social groups during off-duty hours. It is probable that many infections are spread on troop trains and in buses, since the frequency of travel is generally increased in military life.

One controlled study of the effect of barracks living arrangements was made by the Commission on Acute Respiratory Diseases.[21] This was a comparison of incidence rates for common respiratory diseases among recruits in barracks where double bunking was employed with those in barracks having standard single beds. Double bunking in this study meant the use of double-

[20] See footnote 18 (2), p. 78.

[21] Commission on Acute Respiratory Diseases: The Effect of Double-Bunking in Barracks on the Incidence of Respiratory Disease. Am. J. Hyg. 43: 65-81, January 1946.

decked beds so spaced that the total cubic footage of airspace per man was not reduced from that prevailing in single-bunked barracks. Under these conditions, no great difference in respiratory disease incidence was seen, but the occupants of double-bunked barracks fared somewhat better, in that during an epidemic of common respiratory disease their admission rates to hospital were lower.

Numerous other factors have been incriminated as predisposing to respiratory infections. Weather has, of course, long been suspected of influencing morbidity, but such careful studies as have been made have not supported this view. Avoidance of exposure to inclement conditions has, at any rate, been a common precept during epidemic conditions. In this connection, it is of interest that both in the author's studies and in the investigations of morbidity of divisions by Colonel Duff, the periods when troops were on maneuvers—sleeping in the open, often under severe weather conditions—were almost never marked by an increase in reported morbidity. The same is true of periods of combat during the winter season. It may, of course, be argued that opportunities for hospitalization of men with minor illnesses were interfered with, at such times, although this would not be true of the maneuver periods at Fort Dix. In summary, while temporary weather disturbances and exposure of inadequately clothed troops to vicissitudes of the weather are often blamed for respiratory epidemics, there seems to be little factual foundation for this beyond the well-established facts that morbidity is greater in colder climates and there is a well-marked seasonal cycle of morbidity which is roughly inversely proportional to temperature.

While nothing can be done about the weather beyond providing proper clothing and housing and, so far as possible, locating basic training establishments in the South, there are other respiratory disease control measures which may be advocated, again on largely theoretical grounds. By analogy with results of experimental epidemiology, it would appear that communicable diseases have a better chance of spreading among troops housed in large dormitories than when the unit of accommodation is small, even though space allowances per man are equally large. This is a factor which should be susceptible to study. The construction of barracks, particularly theater of operations type, was often such as to allow drafts. The temperature control provided by the heating systems in these barracks left much to be desired, and, although measures were taken to increase the humidity, it was a common experience in winter for the atmosphere of the barracks to be very dry. All of these environmental conditions are, of course, subject to control provided the expansion of the services is not too rapid for new construction to keep pace with it. There have been arguments as to the relative healthfulness of barracks and tents in prevention of respiratory diseases without, so far as is known, any sound evidence having been produced on either side.

Another preventive measure which might theoretically be expected to reduce morbidity is the prompt recognition, diagnosis, and admission to

hospital of infected individuals, thus removing sources of infection from the population. During epidemic periods, however, the hospitalization of mild cases would almost certainly overcrowd the hospitals. It may be doubted whether this measure would be helpful, since it is most unlikely that all infected individuals would be removed from duty even under favorable conditions. Moreover, if the behavior of this infection resembles measles or similar diseases, many would have had an opportunity to spread the infection before hospitalization. It must be acknowledged, in any case, that the hospitalization of all cases ill enough to go off duty is preferable to treating them in quarters, both because of the reduced risk of spreading infection and the relatively better care provided in hospital.

Finally, the discussion of control measures must include health education. In technical manuals, lectures on personal hygiene, posters, training films, and other training aids, emphasis was given to the mode of transmission of respiratory infections, the principles of avoidance of overcrowding, good ventilation, covering the mouth and nose when coughing or sneezing, proper clothing, and other steps which the soldier might himself take to modify his environment. It is impossible to form any estimate of the effectiveness of this teaching in influencing the behavior of soldiers. In general, however, such instruction is to be commended.

Another factor which has been blamed for the high recruit susceptibility is the fact that all recruits receive a course of immunizations, including vaccination against smallpox, three inoculations of typhoid-paratyphoid vaccine, and three injections of tetanus toxoid. The usual policy is to administer these as soon after entry into the service as possible. There are various reasons to discount this course of immunization as an element in recruit susceptibility. One is that immunizations are given at all seasons, while recruit epidemics are infrequent in summer and epidemics in units mobilized in the autumn and presumably immunized at that time have, on a number of occasions, occurred somewhat later in the winter. Furthermore, units alerted for overseas duty have commonly received intensive mass reimmunization and immunization with additional agents such as cholera, typhus, and yellow fever vaccines without experiencing such epidemics. It is true that some reactions to immunizing agents such as typhoid vaccine are probably misdiagnosed as acute febrile respiratory infections, but this would not be sufficiently common to affect the rates materially. Still another suggested explanation for the excess incidence among recruits is the physical depletion resulting from a rigorous training program. Most of the arguments advanced against immunizations and excessive exposure to the elements as factors are equally appropriate here.

Since none of these explanations of the recruit epidemic phenomenon seems adequate, a better explanation must be sought. Such an explanation can, it is believed, be found in the aggregation of large numbers of individuals coming from different environments. It seems reasonable that under ordinary civilian

circumstances there is relatively limited interchange of infectious agents between individuals who are in contact with one another. Living habits are such that individuals are in intimate association with a more or less constant group, although a less close association does occur with individuals encountered by chance in public vehicles, theaters, and the like. When a military organization is formed from men drawn from civilian life, it is almost certain that some of the men will be exposed for the first time in months or even years to pathogenic agents to which their immunity has waned and that they will, as a result, develop a clinical infection. Upon the termination of these clinical illnesses, it may be presumed that at least a transient immunity will remain, since some immunity mechanism is necessary for recovery from an infection. Thereafter, repeated exposures during the period when immunity is waning might suffice to reinforce that immunity without the individual suffering a second clinical attack. The fact that recruit epidemics occur only in the colder months is difficult to explain on this or any other hypothesis. It must be supposed that ecologic influences at other seasons simply do not support the development of an epidemic.

A phenomenon somewhat resembling the recruit epidemics on a smaller scale probably takes place at the beginning of a winter term in boarding schools and occasionally in other schools. Records adequate to study this are not available except in a few institutions, but those available tend to support this concept.

There is a real opportunity for research in military preventive medicine through further investigation of the various environmental factors discussed above in relation to acute respiratory diseases. The inability to attach a specific etiologic diagnosis to this group of diseases has probably been one of the chief deterrents to such studies, though it need not be if the recognized pathogens are reasonably well excluded. There are many features of Army life which would facilitate studies of this character. One possible avenue of approach to the control of respiratory diseases which has never been deliberately tried is the administrative rearrangement of programs for the training of newly inducted soldiers so as to avoid those conditions which have been shown to favor recruit epidemics, that is, the rapid aggregation of large numbers of recruits at a single post during winter months with a constant influx of new men. Admittedly this method of conducting basic training is the most efficient and economical. However, if some other method were to prevent the hospitalization of large numbers of troops, it might well prove less costly and more effective in the long run.

The argument is sometimes advanced that the seasoning process involving an attack of a respiratory disease is a necessary element of adaptation to military life. It is claimed, therefore, that attempts at prevention of recruit epidemics are unwise, since it is better for the recruit to have his seasoning behind him early in military training. As pointed out in the Fort Dix study,

this viewpoint is entirely defensible if one is considering diseases like measles and mumps in which the severity of attack is not thought to be influenced by dosage of the infectious agent or other factors and in which immunity is permanent. With respect to common undifferentiated respiratory diseases, there is little evidence that troops who have been through a recruit epidemic are any more adequately seasoned than those who have not. It may well be that consideration of the epidemiologic evidence now available in the planning of troop-training programs would have a definite effect in the lowering of over-all morbidity.

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CHAPTER IV

Influenza

Thomas Francis, Jr., M. D.

EPIDEMIOLOGY [1]

Influenza as a Military Problem

The long history of influenza does not indicate a close association between its epidemic occurrences and major military undertakings. Because of the frequency of both phenomena, however, it is to be expected that they may at times coincide. The appalling pandemic of 1918 in the last months of the exhausting conflict of World War I, with massive mobilization of armies and upheaval of civilian populations, has irrevocably linked those two catastrophes. It demonstrated that virulent influenza may be more devastating of human life than war itself. Jordan[2] has estimated that, in a few months, 20 million people perished; 548,000 in the United States alone; the number attacked was 50 times as great. Among the military personnel of the United States there was "an estimated total of 24,853 deaths from influenza as recorded, 469 from bronchitis, 10,341 from bronchopneumonia, and 11,329 from lobar pneumonia, a grand total of 46,992. This is nearly as large a total as that of the battle deaths, American Expeditionary Forces—50,385." These deaths occurred among approximately "797,993 cases of influenza, 272,735 of bronchitis, 37,334 of bronchopneumonia, and 51,115 of lobar pneumonia, a total of 1,159,177 cases of respiratory diseases." [3] About 1 out of every 5 men contracted influenza in the service.

Many students of the disease have sought to explain the factors responsible for the enormity of that devastation. The clinical, pathologic, epidemiologic, and bacteriologic data have been voluminously recorded.[4] It has in many quarters been accepted as the prototype of pandemic influenza, an unwarrantable conclusion since this most exceptional episode in the history of the disease

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[1] The author is greatly indebted to Col. John D. Morley, MC, USA (Ret.), Resident Lecturer, Department of Epidemiology, School of Public Health, University of Michigan, who gave extensive assistance in reviewing material and in the preparation of the graphs and tables dealing with incidence.

[2] Jordan, E. O.: Epidemic Influenza: A Survey. Chicago: American Medical Association, 1927.

[3] The Medical Department of the United States Army in the World War. Washington: U. S. Government Printing Office, 1928, vol. IX, pp. 67-68.

[4] (1) Thomson, D., and Thomson, R.: Influenza, With Special Reference to the Part Played by Pfeiffer's Bacillus, Streptococci, Pneumococci, etc., and the Virus Theory. Monograph 16, pt. I. Ann. Pickett-Thomson Research Lab. 9: 1-640, November 1933. (2) Thomson, D., and Thomson, R.: Influenza, With Special Reference to the Complications and Sequelae, Bacteriology of Influenzal Pneumonia, Pathology, Epidemiological Data, Prevention and Treatment. Monograph 16, pt. II. Ann. Pickett-Thomson Research Lab. 10: 641-1477, May 1934.

cannot be considered typical. Among other criteria suggested as being characteristic of pandemic influenza in contrast to that of interpandemic years are (1) a cycle of approximately 30 years; (2) the tendency for there to be three waves, the first a mild widespread disease, the second a severe autumnal epidemic with high fatality, the third of lower incidence but high fatality and less constant in occurrence; (3) a greater independence of season; (4) greater and more rapid dispersion; (5) a greater influence upon general mortality; and (6) a different age distribution so that the incidence in old people is less than that in the younger, especially middle age, groups. In many respects, these epidemiological generalizations have few supporting data other than those of 1918, amd most of the other criteria appear to measure severity of the disease. Confronted with this vast occurrence and the lack of adequate information as to the nature of the causative agent, interpretations of the conditions responsible for the 1918 pandemic have inevitably considered the dislocations and crowding of populations by wartime conditions to be essential elements in the development irrespective of the biologic characteristics of the microbial agent involved.

In the latter case, the various bacterial pathogens of the respiratory tract were extensively studied and *Hemophilus influenzae*, while considered an important contributor to severe disease, was not believed to meet the requirements of the specific inciting agent of the epidemic. Realization of the role of beta hemolytic *streptococci and* of staphylococci in severe pulmonary disease was heightened. But beyond these was a progressive development of the idea that a highly virulent virus was involved, and a number of studies were made in efforts to demonstrate its presence. No conclusive or consistent results were obtained, even from the intensive efforts of Rosenau and others,[5] to transmit the disease to human volunteers. It is interesting and surprising that apparently no material from patients was kept which could be subjected to later study for virus. Nevertheless, it seems probable that the autumnal epidemic of 1918 was initiated by a virus of high virulence in association with a high prevalence of potent bacterial pathogens. It is unlikely that this inciting agent was one with which the general population was completely inexperienced because all evidence emphasizes that the greatest proportion of the population did not take sick, indicating a resistance acquired earlier. Moreover, the fundamental characteristics of the disease clearly suggest that it was of similar behavior to influenza known before and since that time. As later data will make clear, the etiologic identity of the "waves" cannot be unreservedly assumed. Thomson and Thomson indicated that units affected in the spring prevalence were relatively resistant in the fall, although "seasoning" as such did not prevent infection since veterans and recruits were attacked in the autumn in organizations which had escaped the spring experience.

[5] Rosenau, M. J., Keegan, W. J., Goldberger, J., and Lake, G. C.: Experiments Upon Volunteers to Determine the Cause and Mode of Spread of Influenza, Boston, November and December 1918. U. S. Pub. Health Serv. Hyg. Lab. Bull. 123: 5-41, February 1921.

The relative helplessness with which prevention or treatment could be approached is apparent. Apart from the possible use of antipneumococcal serum in a limited fashion for treatment of specific cases, treatment was essentially palliative. A few studies were concerned with the use of convalescent serum. In the main, however, prompt bed rest, quiet, and simplified diet were relied upon. The handling of empyemas was revolutionized. In some instances, an intelligent effort was made to limit the opportunity for transmission of pathogens by segregating, for example, those with streptococcal infections from others. Along with these went efforts to prevent or reduce crowding by increasing space per man in barracks and in transportation and by avoiding congregations. The hygiene of housing and of person, together with isolation and quarantine, were the armamentarium of prevention.

Experience during World War I

During the period of World War I and the pandemic of influenza, one of the important agencies utilized in the campaign against respiratory diseases was the Pneumonia Board, appointed in 1918, at the request of The Surgeon General, by War Department Special Order No. 118, 20 May 1918. Composed of civilian and military experts in the field of respiratory and other infectious diseases, this board rendered advisory services and organized and directed investigations at posts, camps, and hospitals. The Pneumonia Board was a forerunner of the Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the Army which was established in 1941 through the initiative of the Preventive Medicine Service, Office of the Surgeon General.[6] The latter became known as the Army Epidemiological Board (p. 94).

Influenza is not especially a disease of wartime. However, conditions of mobilization, mass transportation, and crowding furnished a medium for the rapid spread and accentuation in severity of the disease. Because the microbial agents may be prevalent on a post or may become widely disseminated by transfer of personnel, influenza is a disease of great hazard to military effectiveness. Under these conditions, it appears that the military forces may suffer disproportionately in comparison with civilian populations.

With this perspective, the onset of another war inevitably recalled the specter of 1918 and the possibility that the development of similar or greater concentrations of population would again result in epidemiologic conditions which would heighten the severity of influenza to a catastrophic level. Influenza after 1918 had reverted to its normal behavior of recurrent epidemics at intervals of a few years, varying in distribution and severity but commonly mild. This increased the tendency to conclude that interpandemic influenza was a different disease and that influenza was in fact a clinical syndrome rather than a disease entity.

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[6] Bayne-Jones, S.: Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the Army. Army M. Bull. 64: 1-22, October 1942.

Experience in 1919–39

Between 1919 and 1939, 12 epidemics of influenza were recorded [7] by the United States Public Health Service on the basis of excessive mortality accompanying sharp epidemic waves of acute respiratory disease. The most prominent were those of 1919–20, 1922–23, 1925–26, 1928–29, 1932–33, and 1936–37, but others such as 1935–36 were widespread. That they were not inconsiderable is indicated by the fact that the estimated excess mortality from influenza and pneumonia in the 1919–20 epidemic was 100,000 and, in 1928–29, 50,000. Most of the remainder exhibited sharp peaks but were less widely distributed. A definite decline in pneumonia mortality began about 1929 and continued except for a rise in the epidemic period of 1935–37. Consequently, the scope of epidemics based upon excess mortality is less clearly demonstrated after 1930. A second interpretation for this alteration could be offered; namely, that the agent or agents of 1918 continued in prevalence through the 1928–29 epidemic and were replaced by milder strains thereafter. Nevertheless, the 1937 period had a sharp increase in mortality.

The experience of the Army during the interval between the two World Wars was not significantly different from that of the civilian population, and the mortality rates were extremely low. The small strength, furthermore, would give little meaning to an organizational rate except as it reflected general experience. In the annual reports of The Surgeon General, United States Army, for 1929 and 1930, it was pointed out that since 1920 there had been only the mild epidemic of influenza in 1926 until 1928 when "there occurred a more serious and generally distributed epidemic * * *. A comparatively large percentage of the strength of the Army was affected * * *." In December of 1928, a rate was reached of 523 per 1,000 white enlisted men in the United States; in January 1929, the rate was 336. The annual admission rates are less informative, since they bisect the epidemic, but that of 81.7 per 1,000 for 1928 is the highest of the decade 1920–29. The rates for pneumonia and common respiratory disease during these years were also among the highest for the decade. The death rate from pneumonia and influenza was, however, not proportionately elevated.

Special attention is called to a year such as 1936 when, in certain regions among the civilian population, influenza reached the level of sharp epidemics during the early months; then in December the peak of the 1936–37 pandemic spread was rapidly approached. The first was influenza B, the second influenza A.[8] The latter was worldwide and typically influenzal. Moreover, it was

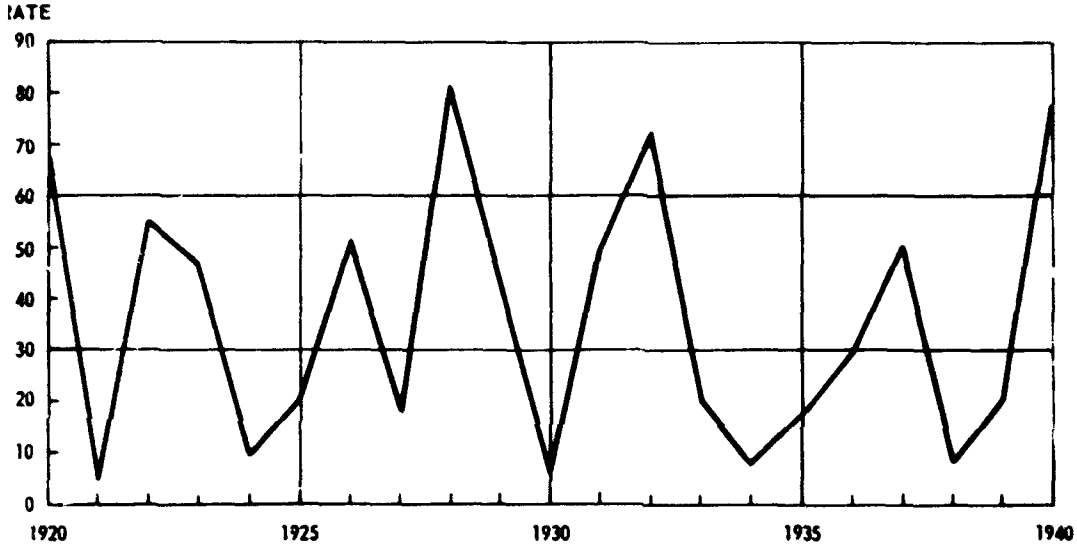
[7] (1) Collins, S. D.: Influenza-Pneumonia Mortality in a Group of About 95 Cities in the United States, 1920–29. Pub. Health Rep. 45: 361–406, 21 Feb. 1930. (2) Collins, S. D., and Gover, M.: Influenza and Pneumonia Mortality in a Group of About 95 Cities in the United States During Four Minor Epidemics, 1930–35, With a Summary for 1920–35. Pub. Health Rep. 50: 1668–1689, 29 Nov. 1935. (3) Gover, M.: Influenza and Pneumonia Mortality in a Group of 90 Cities in the United States, August 1935–March 1943, With a Summary for August 1920–March 1943. Pub. Health Rep. 58: 1033–1061, 9 July 1943. (4) Collins, S. D.: Age and Sex Incidence of Influenza in the Epidemic of 1943–44, With Comparative Data for Preceding Outbreaks. Pub. Health Rep. 59: 1483–1503, 17 Nov. 1944. (5) Collins, S. D.: Influenza and Pneumonia Excess Mortality at Specific Ages in the Epidemic 1943–44, With Comparative Data for Preceding Epidemics. Pub. Health Rep. 60: 821–835, 20 July; 853–863, 27 July 1945.

[8] Francis, T., Jr.: Epidemiological Studies in Influenza. Am. J. Pub. Health 27: 211–225, March 1937.

dentified etiologically in many parts of the world. Among white enlisted men n the Army in the United States, the admission rate for 1937 rose to 50.1 per ,000 average strength, but seasonal data are not available. There is evidence, nevertheless, that the 1936 37 epidemic was sharply expressed in the Army, lthough no significant increase in mortality occurred. In 1938 39, a mild prevalence of a spotty nature was present (charts 12 and 13).

'HART 12. *Incidence rates for influenza among white enlisted men serving in the Army in the United States, 1920 40*

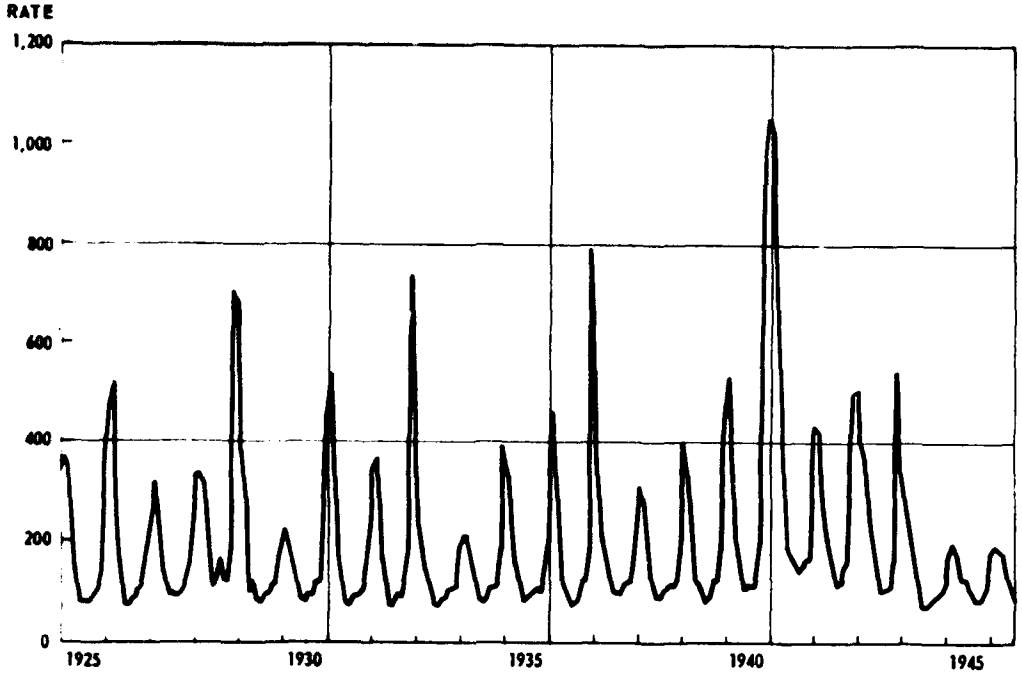
[Rate expressed as number of cases per annum per 1,000 average strength]



Influenza, then, in the 20 years since 1919 had returned to its former status of recurrent epidemics at short intervals, often so mild as to be unnoticed but with certain episodes of pandemic distribution and of sufficient severity to cause considerable alarm and disturbance in the general population. That it was a disease of potential severity was clearly apparent since even the mild prevalences tended to cause a heightened mortality from respiratory disease. The excess mortality remained a valuable index of the spread and severity of epidemics and even of their recognition. Reports based upon clinical diagnosis were, as always, unreliable since many factors, including publicity or special instructions, had a large influence in this respect. Army data show this effect clearly, as will be illustrated later, in the lack of reports of influenza from some areas when an epidemic was known to be occurring, or high reports of influenza in prevalences of acute respiratory disease known not to be identifiable as influenza. However, in most instances, the character of the epidemic curve and the distribution among troops of abrupt rapid epidemics of nonbacterial acute respiratory disease readily suggest its nature. The occurrence of the disease and the factors which govern it remained essentially unaffected at the onset of World War II.

CHART 13. *Incidence rates for common respiratory diseases and influenza in the Army in the United States, January 1925 to June 1946, inclusive*

[Rate expressed as number of cases per annum per 1,000 average strength]



Epidemic Influenza, 1940–45

Experience in 1940

The Army began its expansion program in 1940, and the draft was adopted. The increase in strength began slowly in June, then rapidly in October, November, and December. This and the industrial program brought together a large number of individuals from diversified areas under conditions furnishing ready opportunity for the rapid spread of respiratory infections. However, in general, the health of the Army and the United States during this period was excellent.

Early in 1940, a limited epidemic of mild influenza occurred, first in the Southeastern States and shortly afterward in New York. This was the outbreak from which influenza virus, type B, was first isolated.[9] In the civilian population, it was said to be limited to the eastern part of the United States, but serologic data demonstrated it to be more widely dispersed.[10] The admission rates for influenza in the Army show a moderate rise in the Third, Fourth, and Fifth Corps Areas during the months of January and February. In general, the United States Army within the continental limits had a low incidence of influenza for the first half of the year as compared with the previous

[9] Francis, T., Jr. A New Type of Virus From Epidemic Influenza. Science 92: 405–408, 1 Nov. 1940.
[10] See footnote 7-3, p. 98.

5-year average. The admission rate for common respiratory diseases for the same period, however, was in general higher than the 5-year average. Influenza B was recognized in southern England,[11] and, in the summer of 1940, it was prevalent in Cuba.[12] The recognition of this disease and means for identification were extremely important for proper understanding of the problem.

In June and July 1940, well-marked epidemics of influenza A occurred in Cuba and Puerto Rico. There was a sharp rise in the admission rate to 126.4 for influenza among the Puerto Rican troops in June followed by a moderate rise to 93.9 in July among the continental troops stationed on the island, but in August no cases were reported.

At this same time, an epidemic of mild influenza was reported from Argentina.[13] This outbreak reached its peak in the Argentine Navy in the week ending 21 July 1940, in the Army during the following week, and in the civilian population in the week ending 4 August.

In August, the admission rates for influenza showed a sudden rise in the Philippine Islands. The incidence during the month was twice as high among Filipino troops as that reported for United States troops stationed there. Unfortunately, the data for admissions in the overseas departments are not available for the remaining months of 1940 (September to December).

During 1940 in Australia, especially in military camps, extensive epidemics of pharyngitis and tracheitis occurred. Influenza was not isolated until September when more typical cases occurred in a military camp and in a small hospital outbreak.[14]

Influenza was noted in Hawaii in the middle of September 1940 and declared epidemic on 26 September. Reporting was then made mandatory until 31 December. Approximately 16,500 cases were reported. The majority of these were from the island of Oahu, including Honolulu. The epidemic peaked sharply during the middle of October. Doolittle [15] stated the impression that the disease came from the West and suggested that it was introduced by a Japanese training ship from the Japanese Mandated Islands where an epidemic was prevalent. The influenza incidence among white enlisted troops stationed in Hawaii during 1940 was 65.3 per 1,000 average strength per year. The highest incidence for white enlisted men for the year was reported in the United States (77.6) with Hawaii in second place.

In November 1940, the admission rates for influenza in the Ninth Corps Area showed a sharp rise which reached its peak in the 4-week period of

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[11] Andrewes, C. H., Glover, R. E., Lush, D., Hudson, N. P., and Stuart-Harris, C. H.: Influenza in England in 1940-41. Lancet 2: 387-389, 4 Oct. 1941.

[12] Lennette, E. H., Rickard, E. R., Hirst, G. K., and Horsfall, F. L.: The Diverse Etiology of Epidemic Influenza. Pub. Health Rep. 56: 1777-1788, 5 Sept. 1941.

[13] Sordelli, A., Taylor, R. M., and Parodi, A. S.: Estudio de los virus de la epidemia de influenzao currida en la Argeninga durante el año 1940. Rev. d. Inst. bact., Buenos Aires 10: 265-274, December 1941.

[14] (1) Rudd, G. V.: Influenza Epidemic at Puckapunyal. M. J. Australia 1: 7-9, 4 Jan. 1941. (2) Burnet, F. M., and Foley, M.: Two Methods for the Detection of Influenza Virus in Human Throat Washings Without the Use of Ferrets. M. J. Australia 1: 68-72, 18 Jan. 1941.

[15] Doolittle, S. E.: Clinical Observations During the 1940 Epidemic of Influenza in Honolulu. Proc. Staff Meet. Clin., Honolulu 7: 1-8, March 1941

December. During this same period, the Fifth Corps Area had a very high incidence (1,444 per 1,000 per annum based on the 4-week period). The Eighth Corps Area also recorded its highest rates during December. The other six corps areas reached their highest incidence during the 5-week period of January 1941. The First, Second, and Sixth Corps Areas had a relatively low number of cases of diagnosed influenza (below 200 cases per 1,000 per annum in the highest period), although definite epidemic peaks were attained. The rate of admissions in white enlisted men in the United States was 77.6 for 1940, which was approximately four times that for 1939, 20.3. Admissions for common respiratory disease in each corps area, except the Fifth Corps Area, showed a sharp rise coincident with the rise in influenza. The incidence of these diseases remained generally high until March in contrast with the much more rapid subsidence of influenza. The combined annual rate for common respiratory diseases and influenza was nearly 1,200 for the entire Army in the United States for 1940–41 (chart 14).

A widespread epidemic of influenza occurred in the civilian population at the same time.[16] The notable feature of this outbreak was the rapidity of spread from the Pacific Coast eastward throughout the United States and Canada. The peak of incidence in the Pacific Coast States was reached in mid-December. The West South Central and Mountain States reached their peak in late December or the first week in January, the East South Central and South Atlantic States the second week in January, and the East North Central and Middle Atlantic States during the third week of January. However, the New England States reached their peak during the second week in January. The spread of the disease as shown in the Metropolitan Life Insurance Company survey [17] seemed to follow three main paths: (1) The southernmost route across the Southern and Gulf States spread most rapidly and showed the most ordered progress; (2) the path across the middle of the country was not quite as rapid; (3) the northernmost route showed the slowest rate. The peak of the incidence in South Dakota was in early February and in Iowa and Wisconsin in mid-February. As the admission rates for the Army are given for 4- or 5-week periods, no such orderly progress of the disease was discernible.

Clinically the disease was a mild acute febrile illness with abrupt onset associated with mild myalgia, some lassitude, but little prostration. Complications were rare. Of 19,609 cases occurring among Army personnel in the United States, 57 developed pneumonia. Only 1 death from influenza was reported in the Army for 1940 and only 3 deaths from acute primary pneumonia.

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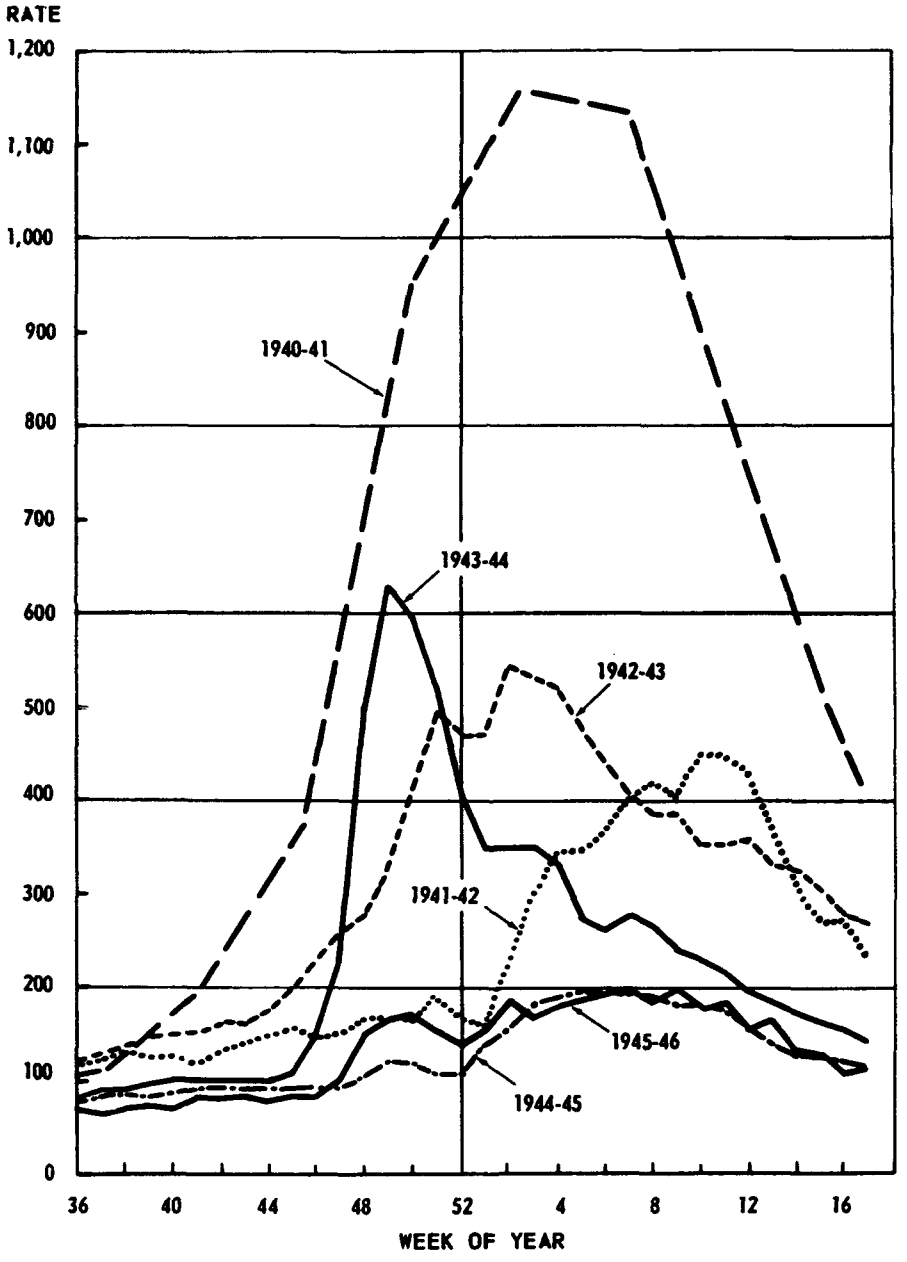
[16] (1) See footnote 7 (4) and (5), p. 88. (2) Brown, J. W., Eaton, M. D., Meiklejohn, G., Lagen, J. B., and Kerr, W. J.: An Epidemic of Influenza. Results of Prophylactic Inoculation of a Complex Influenza A-Distemper Vaccine. J. Clin. Investigation 20: 663-669, November 1941. (3) Sulkin, S. E., Bredeck, J. F., and Douglass, D. D.: Epidemic Influenza: Epidemiological, Clinical, and Laboratory Aspects of the 1940-41 Outbreak in St. Louis. Am. J. Pub. Health 32: 374-380, April 1942. (4) Pearson, H. E., Eppinger, E. C., Dingle, J. H., and Enders, J. F.: A Study of Influenza in Boston During the Winter of 1940-41. New England J. Med. 225: 763-770, 13 Nov. 1941.

[17] The Course of the Recent Influenza Epidemic. Statist. Bull. Metrop. Life Insur. Co. 22 (No. 4): 3-6, April 1941.

CHART 14.—*Variations in winter seasons for incidence rates for common respiratory diseases in the Army in the United States, September to April, 1940–46*

[Rate expressed as number of cases per annum per 1,000 average strength]

[Data for 1940–41 are based on rates per annum per 1,000 average strength by month. Data for the other years are based on rates per annum per 1,000 average strength by week]



The excess mortality recorded in the civilian population was slight, but, according to the recorded rates of the Army in the continental United States, this was the highest epidemic of the war period.

It was during this explosive outbreak of influenza that the planning and guidance exercised by the reorganized Preventive Medicine Service, Office of the Surgeon General, under the immediate direction of Lt. Col. (later Brig. Gen.) James S. Simmons, MC, resulted in the development of a new and powerful means for combating infectious diseases in the Army. Since the start of World War II in Europe in 1939, followed by the expansion of the Army, the Preventive Medicine Service had seen the necessity for preparing in advance for dealing with new and large problems. It was foreseen that there would be urgent need for the services of the best civilian specialists in the field of infectious diseases. The Pneumonia Board of 1918, previously mentioned, was recalled as an example of the kind of organization that would be needed.

Proceeding along these lines, Colonel Simmons drew up a plan for a greatly enlarged and strengthened civilian body of this type and, on 27 December 1940, recommended to The Surgeon General that the plan be forwarded to The Adjutant General for approval. This was done the same day by Maj. Gen. James C. Magee, The Surgeon General, and, on 11 January 1941 by order of the Secretary of War, the Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the Army was established. Within a a few months, a number of civilian commissions on various infectious diseases were formed, and the Commission on Influenza was one of the first to be established. The Board and its commissions were attached to, and administered by, the Preventive Medicine Service, Office of the Surgeon General.

Experience in 1941–42

During the remainder of the year 1941, the admissions for influenza and the common respiratory diseases followed the usual seasonal pattern and fell to the lowest level in July. However, the incidence of acute respiratory disease during the summer season remained higher than the average for the preceding decade.

With the onset of open hostilities in December 1941, the mobilization for war was tremendously increased. The very rapid induction of men when housing facilities were inadequate caused definite overcrowding. It was necessary to reduce the floor space per man in barracks from 50 to 40 square feet, and a considerable proportion of troops were housed in tents. Such housing conditions undoubtedly contributed to the moderately high admission rates for acute respiratory disease in the winter of 1941–42, but no epidemic of influenza was encountered.

Experience in 1942–43

In the summer of 1942, the military forces were still increasing rapidly. New groups of recruits were constantly entering camp, troop movements were

continuous, and the expansion frequently resulted in overcrowding on trains and in barracks, especially in induction and training centers. There was at the same time a migration of civilians into overcrowded industrial centers. In keeping with the recurrence of influenza A every other year since 1932, 1942–43 was scheduled for an epidemic with conditions in the population apparently favorable to the disease. Studies of vaccination were linked to this premise, and close observation was maintained. Influenza A was epidemic in Australia in May 1942,[18] but elsewhere the disease did not appear in significant amount, and in the United States it was not found.

On the other hand, noninfluenzal acute respiratory diseases and atypical pneumonia rose to epidemic heights and maintained a high level throughout the season when respiratory diseases were expected to occur. In the continental United States, a peak of 530 per 1,000 per annum for January 1943 slowly receded over several months. The disease was particularly prominent in recruits and in other epidemiologic characteristics differed from the usual epidemics of influenza.

Experience in 1943–44

Although influenza was inconspicuous during the preceding winter, each succeeding season carried the possibility of a severe epidemic which, with crucial operations mounting in many parts of the world, might be critical. Advance information was desirable. The virus laboratories of the Commission on Influenza of the Army Epidemiological Board were still alert in various areas, and the continued high incidence of respiratory disease held the attention of all medical agencies. Commission investigators identified influenza B widely but largely subclinically in two institutions in Michigan during March and April; two cases of type A were also found. Sporadic cases of type B were also detected at Fort Custer, Mich., and among students at the University of Michigan.[19] A small amount of influenza B was identified in Australia.[20]

In May, three sporadic cases of influenza A were identified at Fort Custer, and virus was isolated.[21] Serologic examination demonstrated that a limited outbreak in April among interns in a New York hospital was influenza A; and Eaton detected five cases of influenza A among students at the University of California in April and isolated a strain of virus. Here were striking evidences of a scattered low-grade circulation of influenza without epidemic conditions. Epidemic influenza in July was reported in Hawaii but not iden-

[18] Burnet, F. M., Beveridge, W.I.B., Bull, D. R., and Clark, E.; Investigations of an Influenza Epidemic in Military Camps in Victoria, May 1942. M. J. Australia 2: 371-376, 24 Oct. 1942.

[19] Salk, J. E., Pearson, H. E., Brown, P. N., Smyth, C. J., and Francis, T., Jr.: Immunization Against Influenza With Observations During an Epidemic of Influenza A One Year After Vaccination. Am. J. Hyg. 42: 307-322, November 1945.

[20] Beveridge, W. I. B., and Williams, S. E.: Sporadic Occurrence of Influenza in Victoria During 1943. M. J. Australia 2: 77-80, 22 July 1944.

[21] Francis, T., Jr : The Development of the 1943 Vaccination Study of the Commission on Influenza. Am. J. Hyg. 42. 1-11, July 1945.

tified by type. Scattered flurries in military camps were observed in Canada.[22] In the late spring and summer, limited scattered bursts of influenza A were identified in British civilians;[23] in August a localized unidentified epidemic occurred among United States troops in Southern Base Section. Much of the foreign information was only known through subsequent publication or through informal channels.

The possibility was recognized that these episodes might be the forerunner, or first wave, of a serious autumnal experience. With plans for evaluation of vaccine by the Commission on Influenza actively proceeding, the new strain of virus was incorporated in the vaccine; the continuous lookout for influenza was enlarged to include a greater number of listening posts maintained by Commission members throughout the nine service commands; and a formal arrangement for reporting was concluded. Samplings of throat washings and blood were obtained at intervals from patients with upper respiratory disease even though the diseases did not resemble influenza clinically. The results from each observation post were communicated to each of the others and to the Preventive Medicine Service, Office of the Surgeon General, at biweekly intervals.

On 17 and 18 November 1943, several patients presenting a picture which resembled influenza were observed in the ASTP (Army Specialized Training Program) unit at the University of Michigan. Throat washings given to ferrets elicited a typical reaction, and transfer of ferret material to eggs permitted identification of influenza virus, type A. On 22 November, Dr. E. R. Rickard in St. Louis, Mo., reported that between 11 and 18 November what appeared to be an epidemic of influenza had occurred in the ASTP unit at St. Louis University, involving 100 out of 550 men. He also reported that in one of the groups under observation at the University of Minnesota at Minneapolis, a sudden outburst of 20 cases had occurred on 21 November. Type A influenza virus was identified by direct inoculation of throat washings into the allantoic sac of eggs. All other investigating groups were notified that influenza A had been identified in both the Sixth and Seventh Service Commands.

Word was received from Lt. Col. F. B. Lusk, MC, Chief, Medical Service, Station Hospital, Fort Custer, on 15 November 1943, that a sharp increase of febrile respiratory disease had taken place. Material obtained from patients becoming ill there during the next week demonstrated that almost all of these patients had influenza A. After this time the spread of the disease was rapid, and subsequent reports from Commission members indicated that, within 7 to 10 days, cases had begun to appear over a great part of the United States.

From July 1943 to the week ending 6 November 1943, the weekly rates for influenza and common cold for the entire Army in the continental United

[22] Hare, R., Hamilton, J., and Feasby, W. R.: Influenza and Similar Respiratory Infections in a Military Camp Over a Period of Three Years. Canad. J. Pub. Health 34: 453–464, October 1943.

[23] Andrewes, C. H., and Glover, R. E.: The Influenza "A" Outbreak of October–December 1943. Lancet 2. 104–105, 22 July 1944.

States had remained at a level of approximately 100 per 1,000 per annum and up to 30 October in none of the service commands had rates reached 150.

In the week ending 6 November 1943, the first rises to levels greater than 150 were noted in the Sixth and Seventh Service Commands, to greater than 200 in the weeks ending 13 and 20 November, respectively; the two areas proceeded rapidly to reach their respective peaks of 1,279 and 1,050 the week ending 4 December (table 19). The first sharp increase recorded at posts in these commands was at Fort Custer where rates rose from 113 in the week ending 30 October to 441, 1,075, and 1,212 in the weeks of 6, 13, and 20 November, respectively. It was here, too, that influenza A was identified in May 1943. The First, Second, Third, and Fifth Service Commands reached heights of greater than 200 during the week ending 27 November and attained their peaks the week of 11 December. Rates in the Fourth, Eighth, and Ninth Service Commands, although giving evidence of increased prevalence of influenza, did not exceed 200 until the week of 4 December. The peaks in these three commands were considerably lower and later in their occurrence than elsewhere.

TABLE 19.—*Incidence rates for influenza and common colds in the Army in the United States, by service command, for a 14-week period, 1943–44*

[Rate expressed as number of cases per annum per 1,000 average strength]

Week ending—	First SvC	Second SvC	Third SvC	Fourth SvC	Fifth SvC	Sixth SvC	Seventh SvC	Eighth SvC	Ninth SvC	Total
1943										
30 Oct	111	141	93	99	132	112	124	81	100	102
6 Nov	113	142	101	85	118	155	157	76	105	103
13 Nov	111	132	133	93	127	240	177	80	107	113
20 Nov	145	182	181	118	188	354	237	97	146	150
27 Nov	247	322	315	180	346	619	473	119	161	231
4 Dec	607	691	691	489	895	1, 279	1, 050	214	239	501
11 Dec	847	1, 210	980	524	1, 021	844	1, 012	346	444	631
18 Dec	786	1, 048	713	426	687	558	898	479	576	593
25 Dec	513	594	428	529	525	349	538	581	481	521
31 Dec	336	438	356	408	415	342	423	507	328	408
1944										
7 Jan	295	416	371	353	338	355	428	361	295	351
14 Jan	354	417	402	328	347	411	487	341	297	353
21 Jan	280	374	451	332	338	454	504	311	297	350
28 Jan	239	361	484	317	352	399	514	271	265	332

It is of interest to note that on 24 November, at which time the recorded figures give little indication of influenza in the Eighth Service Command, patients with influenza A were identified in the station hospital at Fort Custer. These men had entrained 48 hours earlier in Texas (located in the Eighth

Service Command) and had remained in the troop train but became ill before arrival at Fort Custer.

The highest rates for the entire Army in the continental United States were 631 and 593 in the weeks of 11 and 18 December. By 7 January 1944, the epidemic period had practically ended in all areas, and the rate for the entire Army fell below that for the previous year but, due to the seasonal increase in upper respiratory infection of other etiology, in no command did the rate return to the level which had existed in early November. That the continued incidence was not due to influenza A is seen in the reports of the different investigators who, with the exception of those in California, considered the epidemic prevalence of cases to have ended before 1 January. Further support for this conclusion is found in the results of studies made in the respiratory wards at Fort Custer on 28 January 1944 when serologic tests in recent febrile respiratory admissions were all negative for influenza A although similar studies on 20 November and 27 December were almost uniformly positive. Precise information of the epidemic is largely derived from the Commission studies.[24]

In an analysis of the reported cases of influenza during the 1943–44 epidemic among the civilian population, Holland and Collins [25] found that the maximal incidence of the disease for the country as a whole occurred between 25 December 1943 and 4 January 1944, 2 to 3 weeks later than that noted in the Army. They conclude that the 1943–44 epidemic was larger than any since 1928–29 and that "from the standpoint of mortality from all causes the recent epidemic was comparable in severity to that of 1928–29, the major epidemic since the period 1918–20." For a group of 90 large cities, the excess mortality during the 11 weeks from 21 November 1943 to 5 February 1944 was 50 per 100,000 population in comparison with 65 per 100,000 for the epidemic of 1928–29 and 598 for the pandemic of 1918–19. Comparable data are not available for the 1936–37 epidemic, but a statement has been made that it was smaller than earlier outbreaks.

Collins [26] compared the results of house-to-house canvasses in Baltimore during the 1943–44 epidemic with those in other communities during 12 other epidemics since the beginning of 1918. He states, "considering actual rates, the recorded incidence for all ages in the 1943–44 outbreak was higher than in any other epidemic since that of 1918–19; the incidence among children under 10 years of age approximated that in 1918–19, and the incidence above 40

[24] (1) Rickard, E. R., Thigpen, M., and Crowley, J. H.: Vaccination Against Influenza at the University of Minnesota. Am. J. Hyg. 42: 12–20, July 1945. (2) Hale, W. M., and McKee, A. P.: The Value of Influenza Vaccination When Done at the Beginning of an Epidemic. Am. J. Hyg. 42: 21–27, July 1945. (3) Eaton, M. D., and Meiklejohn, G.: Vaccination Against Influenza: A Study in California During the Epidemic of 1943–44. Am. J. Hyg. 42: 28–44, July 1945. (4) Hirst, G. K., Plummer, N., and Friedewald, W. F.: Human Immunity Following Vaccination With Formalinized Influenza Virus. Am. J. Hyg. 42: 45–56, July 1945. (5) Salk, J. E., Menke, W. J., Jr., and Francis, T., Jr.: A Clinical Epidemiological and Immunological Evaluation of Vaccination Against Epidemic Influenza. Am. J. Hyg. 42: 57–93, July 1945. (6) Magill, T. P., Plummer, N., Smillie, W. G., and Sugg, J. Y.: An Evaluation of Vaccination Against Influenza. Am. J. Hyg. 42: 94–105, July 1945.

[25] Holland, D. F., and Collins, S. D.: The Influenza Epidemic of the Winter of 1943–44 in the United States: A Preliminary Summary. Pub. Health Rep. 59: 1131–1141, 1 Sept. 1944.

[26] See footnote 7 (4), p. 88.

years was greater than in 1918–19. The percentage of the total cases that were complicated by pneumonia in the 1943–44 epidemic was far below the figure for any other epidemic for which data are available." Nevertheless, the pneumonia rates among persons over 25 years of age correspond closely to those recorded in this age group during the epidemic of 1928–29.

The data from all sources indicate, then, an epidemic of high incidence taking its earliest prominent appearance in the North Central States in the first half of November 1943, spreading rapidly to a peak in December, and then abruptly declining to the seasonal level of respiratory disease, thereby occupying a total of approximately 6 weeks for the evident epidemic period. It is of interest that, in the Gulf, Southwestern and Pacific States, the onset was slower and the peak lower and later than in other parts of the country. The data indicated that in terms of incidence the outbreak was of major proportions but the complications and case fatality rates were low, although its effect on total mortality by virtue of the high incidence was greater than the case fatality rate would indicate.

In the entire Army, only 8 deaths occurred among influenza patients and 290 deaths among admissions for pneumonia other than primary atypical. Even a rough estimate of case fatality rates is unreliable since reported and summarized data are inconsistent. Of the total 89,764 cases recorded for the total Army in 1943, 69,840 were in the United States with 5 deaths and a case fatality rate of 0.01 percent. In the European theater, 4,717 cases are recorded with 1 death (table 20).

European Theater of Operations.—American troops in the European theater experienced an epidemic of influenza A in the fall of 1943, which reached its peak about 2 weeks earlier than in the United States.[27] After a suggestive rise in reported cases of acute respiratory disease in the last week of October and the first week of November, there was an extremely rapid increase to maximum annual admission rates of 1,079 per 1,000 strength for the week 26 November 1943. The decline of the epidemic was as sharply marked as its rise. By the end of December, the rate was 387. Virus A was demonstrated to be present in each of the principal base sections by serologic studies. Influenza B was not found. The disease was described as mild and uncomplicated with an acute onset and lasting only a few days. Constitutional rather than local symptoms predominated. The outbreak was almost completely unassociated with an increased prevalence of primary and secondary pneumonia. There were no deaths.

According to Gordon, admission rates for common respiratory diseases, including influenza, among Negro troops were much less than for white troops in the theater. The maximum rate for the two groups occurred during the week of 26 November 1943. The rate for white troops was 1,129 and for Negro troops, 477.

[27] Gordon, John E.: A History of Preventive Medicine in the European Theater of Operations, U. S. Army, 1941–45, vol. I. [Official record.]

TABLE 20. *Incidence of influenza in the U. S. Army, by area and year, 1942-45*

[Preliminary data based on sample tabulations of individual medical records of primary and secondary diagnoses]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942-45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	137,609	9.33	43,030	16.20	69,840	13.48	19,644	4.95	5,095	1.74
Overseas:										
Europe	14,737	3.35	1,685	20.30	4,717	17.68	4,290	2.56	4,045	1.71
Mediterranean [1]	15,796	10.65	910	39.69	6,797	14.88	7,039	10.83	1,050	2.97
Middle East	1,057	7.23	131	21.67	781	14.72	135	2.92	10	.24
China-India-Burma	1,576	3.59	74	8.46	183	4.62	819	4.85	500	2.26
Southwest Pacific	3,485	1.90	975	13.69	714	3.76	771	1.43	1,025	.99
Central and South Pacific	4,955	3.94	968	6.41	1,874	6.43	1,218	2.78	895	2.38
North America [2]	6,165	12.52	1,251	12.43	2,886	14.84	1,648	12.75	380	5.58
Latin America	2,078	5.45	614	6.02	1,103	9.13	116	1.35	245	3.36
Total overseas [3]	51,852	4.83	7,141	12.19	19,924	11.80	16,417	4.30	8,370	1.80
Total Army	189,461	7.44	50,171	15.47	89,764	13.06	36,061	4.63	13,465	1.78

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

The civilian population of the United Kingdom had a similar epidemic at approximately the same time.[28] It differed from that of the United States Army principally in respect to mortality. The maximum number of 1,148 deaths from influenza was reported during the week of 11 December 1943. This was 2 weeks later than the peak for the military cases. The deaths were largely among the older age group.

Other overseas theaters.— Alaska reported an explosive epidemic suggestive of influenza among military and civilian populations in the first 2 weeks of April 1943, and, again beginning on 21 December 1943, there was a sudden increase in the incidence of acute respiratory disease at Fort Greely, Alaska, which quickly reached a peak and subsided within 1 month. Small outposts were not involved. A total of 535 cases were admitted to hospital and 1,043 to quarters. Average duration of hospital and quarters stay was 5 and 4 days, respectively. The clinical description was "typical" of influenza. There was no recurrence and no deaths.

Dr. J. H. Dingle, a member of the Commission on Acute Respiratory Diseases, investigated a mild epidemic of influenza that occurred in Puerto

[28] See footnote 23, p. 96.

Rico in July 1943. It was estimated that 500 cases occurred among the civilian population of San Juan. There was no pulmonary involvement. Troops in Saint Thomas had a small outbreak of approximately 50 cases.

During July and August, an epidemic of acute respiratory disease, probably influenza, occurred in the 65th Infantry Division (Puerto Rican Regiment) with 441 admissions. There were 452 cases among employees of the Panama Engineer Division. Cases were also reported in Camps Sabanita and Coiner, Canal Zone.

Hawaii reported an epidemic of mild influenza in late June and July.

In the Southwest Pacific and China-Burma-India theater no influenza outbreak was reported.

In the North African theater, especially in Italy, no evidence of influenza in significant amount was discerned.

Variations in behavior.—The Commission on Influenza reported that differences in the behavior of the epidemic in adjacent units were clearly observed. For example, at the University of Minnesota, the incidence among the total study population was 5.9 percent, while in another group it was 38 percent. At the University of Michigan, a wide variation in involvement of different companies of the ASTP unit was observed, two companies having but a single hospitalized case each, while from another company not in the study 20 percent were hospitalized. The Commission on Acute Respiratory Diseases had recorded a number of significant features. At Camp Mackall, N. C., the troops were all seasoned men with 6 months or more of training. The epidemic appeared in all parts of the camp in an explosive fashion with 64 percent of the admissions occurring in a period of 5 days. Approximately half the admissions in the 11th Airborne Division occurred among the artillerymen with a rate of 8.8 percent, which comprised less than one-fifth the total strength. Very low admission rates, approximately 1.0 percent, were observed among 2 of the 3 infantry regiments. It was thought that "the explosiveness and short duration of the epidemic at Camp Mackall appear to be related to the environment and activities of the troops. There was marked crowding in the barracks; the inhabited area of the post was small; and all troops shared common transportation facilities." At this camp, 29 cases of pneumococcal pneumonia, most of which were type I, occurred during the epidemic period with a rather prompt disappearance thereafter.

At Pope Field, Fort Bragg, N. C., a detachment of glider mechanics had sick men with them on arrival at the post. They were put into quarantine under crowded conditions and in 7 days had a total admission rate of 35 percent. The remainder of the units in adjacent barracks had no contact with the affected unit. Cases occurred over a month's time with a total admission rate of 5 percent. A contrasting episode was that of the 326th Glider Infantry Regiment which arrived by train from the Midwest; a large number of admissions occurred in the first 2 days, but then the incidence dropped and remained low as they were moved into uncrowded barracks and field activities. No explosive outbreaks were seen at Fort Bragg in troops engaged in continuous

field training. These reports emphasized, too, that at Fort Bragg the virus was repeatedly introduced by men who had acquired their infection in widely separated areas of the country. This was also noted at Fort Custer where troops arriving promptly from Texas were sick although the disease had not been recognized there.

Gordon refers to the unified experience of the 358th and 360th Engineer (GS) Regiments, old units which constituted a single military population. The epidemic began 8 November and reached the extremely high level of 6,111 per 1,000 per annum during the week of 26 November. The disease declined rapidly without fatality. This represents again the abrupt rapid involvement of seasoned troops with a mild disease. The apparent spread from unit to unit and the irregularity in incidence of adjacent units is illustrated in the following example from the European theater.

General Depot G–18 of the Western Base Section had a strength of 5,737 men divided into eight camps. The first five were situated near Sudbury, England, and Nos. 6, 7, and 8 were at Eggerton, several miles away. On 3 November, the first probable patient with influenza from C Company, 131st Quartermaster Regiment (TRK), Camp No. 2, reported to the dispensary with headache, backache, prostration, and an elevated temperature. Within 12 hours, the medical officer developed the same symptoms. The following day 21 men of C Company were ill. On 5 November, cases began to develop in the 445th Engineer Base Company, on 7 November in 608th Engineer Light Equipment Company and on 8 November in the 887th Ordnance Ammunition Company. Altogether, Camp No. 2 with 837 men had 121 patients in hospital with influenza, practically all of whom developed the infection between the 4th and 11th of November and belonged to one or other of the four companies noted.

Camp No. 1 was located in the same vicinity as Camp No. 2, and shared the same dispensary. The outbreak in this camp extended over the same period. Of 849 men, 72 were admitted to hospital with influenza.

Camp No. 3, housing the 534th Quartermaster Service Battalion, a colored labor unit, had no known association with any other camp except for an exchange of labor with the 2d platoon of D Company located at Camp No. 8. The outbreak of influenza began at Camp No. 3 on 14 November with the hospitalization of six men. The following day so many new cases appeared that two empty barracks were converted into a camp infirmary. During the period 14 to 24 November, this unit had 111 cases of respiratory disease. The infection spread to 2d platoon, D Company at Camp No. 8 and 26 cases were reported between the 22d and 25th of November. With the exception of this platoon there were few cases of respiratory infection at Camp No. 8.

Camp 4, with 689 men, sent 15 to the hospital between 17 November and 25 November, and the daily sick call averaged about 15 which was only slightly higher than had held in October.

Camp 5, an Air Corps Depot having little association with other camps, had no cases of influenza.

Camps 6 and 7, located several miles from the other camps, likewise had very few cases of respiratory disease.

It is clearly demonstrated that respiratory disease spread rapidly from one group to another within an incubation period of about 24 to 48 hours. Association of groups was a much more important factor than kind of work or type of quarters.

It was recognized from the start that the epidemic disease affected recruits and seasoned men alike. In fact, at many posts this feature was

emphasized to differentiate between influenza and the acute respiratory disease which was preponderant in recruits. At Fort Bragg it was precisely observed that the incidence of influenza A was not different among the two classes of men living under comparable conditions. This may well be indicative of the fact that infection with influenza is not a year-in, year-out process but one which takes place primarily in epidemic periods; alternatively it may also be interpreted to demonstrate that immunity is not durable or that strain variation is involved.

As previously mentioned, in the European theater the peak incidence of influenza in Negro troops was less than half that in white troops. No other data of this nature are available.

In reviewing the variations that were encountered, it becomes increasingly convincing that, apart from the need for exposure of susceptibles to disease, the most important factor in determining the behavior of epidemic influenza within limited units is that of crowding, particularly when a high rate of change in the population exists.

A few instances are recorded of the epidemic moving into units in which beta hemolytic streptococcal infection was prevalent.[29] The incidence of respiratory diseases was highest throughout the epidemic in the Seventh Service Command, where streptococcal disease was highly prevalent. There was in these areas, however, no evidence that the bacterial invasion was accentuated by influenza as was clearly the case in 1918. Coburn[30] has indicated that influenza had a definite influence upon the behavior of streptococcal infection in naval units, and, at the United States Naval Training Center at Farragut, Idaho, the occurrence of influenza was clearly related to an exaggeration in spread and severity of streptococcal disease. There are also numerous studies indicating that pneumococcal pneumonia was more prevalent during the epidemic, but in other instances as at Sioux Falls, S. Dak., where pneumonia was epidemic, Hodges and MacLeod[31] conclude that influenza was not unduly prominent as a participating factor.

Special concern was attached to the transport of troops during the epidemic. Serious trouble was not encountered in overseas movements although a convoy of 63,750 troops had 7,529 (12 percent) sick calls for respiratory disease while en route to Great Britain in December; 962 men were hospitalized and 86 were evacuated to hospital on debarkation.[32] In another instance, 23 percent of a contingent were sick aboard ship. Official recommendation was made that at all ports of embarkation a minimum of 60 square feet housing space be re-

[29] See footnote 24 (2) and (3), p. 98.

[30] Coburn, A. F.: Mass Chemoprophylaxis. The U. S. Navy's Six Months' Program for the Control of Streptococcal Infections. *In* United States Navy Department, Bureau of Medicine and Surgery: The Prevention of Respiratory Tract Bacterial Infections by Sulfadiazine Prophylaxis in the United States Navy. Washington: U. S. Government Printing Office, 1944, pp. 149–162.

[31] Hodges, R. G., and MacLeod, C. M.: Epidemic Pneumococcal Pneumonia, IV. The Relationship of Nonbacterial Respiratory Disease to Pneumococcal Pneumonia. Am. J. Hyg. 44: 231–236, September 1946.

[32] Informal memorandum, Lieutenant Cree for Colonel Gordon, 20 Dec. 1943, subject: Respiratory Disease on Transports or Shipment UT 5.

quired; this was an effort to avoid crowding and illness at the time of embarkation.[1]

Control measures. The general control measures were those designed to prevent or retard the spread of infection from person to person. Emphasis was placed on the avoidance of overcrowding by increasing the floor space per man in barracks and reducing the number of troops per car on trains. Troop movements were kept at a minimum during the outbreak. Personal hygiene and proper sterilization of messgear were stressed. During the epidemic, fatigue and exposure to cold and wetness were minimized. In high-priority units, medical inspection of troops and the hospitalization of patients with incipient disease were advocated.

The action taken by the Preventive Medicine Service of the Surgeon General's Office to maintain close touch with the epidemic situation and to gain the advantage of environmental control measures is summarized as follows:[2]

1. The first definite information that epidemic influenza was occurring came on 21 November 1943. The isolation of virus A from such an outbreak was first reported on 25 November.

2. On 3 December, the Medical Statistics Division was requested to furnish the number of cases of influenza separate from common respiratory diseases.

3. All service commands, the Air Surgeon, and the Chief of Transportation were requested on 6 December to notify all stations under their jurisdiction to report by telegram all influenza outbreaks then occurring, as well as future outbreaks, to The Surgeon General. This action was taken at the request of General Simmons, and the reports were rendered under previously granted Control Approval Symbol MCE 64.

4. On 16 December, all posts over 5,000 strength were asked to telegraph weekly reports of the number of cases of common respiratory disease, including influenza and pneumonia, to service command headquarters, which was in turn, to forward a consolidated report by wire to The Surgeon General. This action followed a staff conference at which it was indicated that The Surgeon General was expected to know the current situation with respect to influenza. The reporting system thus set up reduced by a week the delay in compilation of rates, although the rates computed were, of course, estimates based on a sample only. Experience showed the estimated rates to be fairly accurate in comparison with the final returns on the monthly summary reports from all posts.

5. A report based on the telegraphic rates was made by The Surgeon General to the Secretary of War, Chief of Staff, and Commanding General,

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[1] Coded message (routine), Chief of Transportation to all Ports of Embarkation, 19 Dec. 1943, subject: Instructions in Control Measures To Be Put Into Effect. (2) Memorandum, Chief of Staff for Commanding General, Army Service Forces, 30 Dec. 1943, subject: Considerations of Influenza Precautions With Respect to Overseas Troop Movements. (This was accompanied by a list of 15 stations designated to provide overflow capacity for regular port staging areas in an emergency.) (3) Memorandum, Commanding General, Army Service Forces, for Chief of Transportation, 6 Jan. 1944, subject: Measures to Prevent Epidemics During Troop Movements.

[2] Memorandum for file, 24 Feb. 1944, subject: Summary of Developments and Action Taken During 1943 Influenza Epidemic.

ASF (Army Service Forces), on 21 December, and similar reports were rendered at the end of the month and again on 7 January 1944.

6. On 28 December, a request was made to the Control Division, ASF, for approval of the weekly telegraphic report; it had previously been understood that such approval was unnecessary. This request was disapproved, and The Surgeon General was instructed to advise service commands immediately that the weekly telegraphic report was no longer required. The Preventive Medicine Service, Office of the Surgeon General, informed the Commanding General, ASF, that his instructions had been carried out but indicated that this office did not concur in the objections to the report.

7. All service commands, the Air Surgeon, and the Chief of Transportation were notified by wire on 7 January 1944 that the telegraphic reports of respiratory diseases were discontinued.

8. On 15 December 1943, the Deputy Chief of Staff sent a radiogram to all major commands in the United States calling attention to control measures and authorizing hospitalization of civilians in military hospitals when necessary. Reference was made to the maintenance of production schedules in industrial plants. Reports of influenza outbreaks in units soon to go overseas were to be telegraphed to The Surgeon General.

9. On 19 December, the Transportation Corps telegraphed all ports, giving instructions in control measures. These instructions were prepared in consultation with the Epidemiology Branch, Preventive Medicine Division, Office of the Surgeon General.

10. On 30 December 1943, the Chief of Staff sent the Commanding General, ASF, a memorandum, entitled "Consideration of Influenza Precautions With Respect to Overseas Troop Movements," accompanied by a list of 15 stations designated to provide overflow capacity for regular port staging areas in an emergency.

11. The Commanding General, ASF, in his memorandum for the Chief of Transportation, dated 6 January 1944, entitled "Measures to Prevent Epidemics During Troop Movements," forwarded a copy of the memorandum from the Chief of Staff, previously referred to.

12. The Commanding General, ASF, also sent The Surgeon General a memorandum, same subject, on 6 January, including a copy of the memorandum from the Chief of Staff and attached list of stations. The Surgeon General was directed to issue necessary technical instructions and report recommendations for the modification of present procedures. The Surgeon General's endorsement on 20 January stated that an article on influenza would be published shortly in a medical technical bulletin and recommended that directives be published requiring a minimum of 60 square feet of space per man in barracks.

13. The Chief, Preventive Medicine Service, Office of the Surgeon General, sent a draft of medical technical bulletin entitled "Influenza," to the Executive Officer on 28 January.

14. On 29 January, a directive entitled "Measures to Prevent Epidemics of Respiratory Diseases," was sent to all defense commands, service commands,

ports, and technical services. This was substantially the same as the letter recommended by The Surgeon General.

Cooperation with civilian groups. Because of the potentialities of a severe outbreak of influenza similar to the 1918–19 epidemic, plans were made by the Preventive Medicine Service, Office of the Surgeon General, for the Army to give assistance in the medical care of civilians in cases of emergency. Medical Department personnel, supplies, hospitalization, and transportation were to be made available to the maximum extent after the first and second echelon facilities consisting of the local and State physicians and facilities, American Red Cross, United States Public Health Service, and Office of Civilian Defense, were exhausted. Hospitalization of civilian personnel was authorized, and each service command and post surgeon made plans to be put into effect if indicated. As the epidemic remained generally mild, these procedures were not used.

Experience in 1944

With the abrupt subsidence of the epidemic of influenza A in the winter of 1943–44, the admission rates for respiratory diseases within the continental limits of the United States continued to decline rapidly and remained below the average level of Army experience for the decade 1930–39 (chart 13). After January, they also reached a level well below that of the three preceding winters. The Army at this time was composed largely of seasoned troops since the rate of induction was low as compared with the 3 preceding years.

Experience in 1945

A small seasonal rise in rates occurred during the winter months 1944–45 but that for troops in the United States remained under 200 admissions per 1,000 per annum. The overseas theaters had similar low rates for acute respiratory disease. No outbreaks of influenza occurred. However, the Commission on Influenza continued a more extended alert for detection by investigating unusual rises in admissions for acute respiratory disease and sampling cases in respiratory wards of various Army hospitals. Investigation of an outbreak of illness in the Antilles Department reported to be influenza revealed it to be infectious mononucleosis; nevertheless, an opportunity was provided for setting up a center for the identification of influenza in the area.[35]

Beginning in March 1945, small localized outbreaks of influenza B occurred in many parts of the United States and overseas theaters. Detailed investigations of a number of these outbreaks are described in special reports from members of the Commission on Influenza to The Surgeon General, United States Army. However, it is of interest and importance to indicate the time, location, and extreme variability of these widely scattered upsurges of the disease which were identified by virus isolation or by serologic evidence.

[35] Essential Technical Medical Data, Caribbean Defense Command, 20 Feb. 1945.

The earliest outbreak occurred in March at Sioux Falls, although the serologic evidence of influenza B was not obtained until after the virus had been identified by Dr. J. E. Salk in sharp oubreaks at Buckley and Lowry Fields, Colo., in May. Streptococcal infection was also high. Evidence pointed to the fact that influenza was present, too, in the neighboring civilian population of Colorado as a very mild illness, not recognized clinically as influenza. Serologic studies at this same time in a hospital ward for respiratory illnesses at Sheppard Field, Tex., demonstrated that influenza B was present although its nature had not been suspected clinically. In April, a flurry was identified serologically at Fort Lewis, Wash., by the Ninth Service Command Laboratory. A sharp civilian outbreak in the town of Kasson, Minn., was recognized only in the school children, 80 percent of whom were affected; the peak was in the middle of May. Tests with sera from patients convalescent from an outbreak in Alaska in May demonstrated influenza B.

In June and July, sharp outbreaks occurred in the prisoner-of-war camps at Camp Edwards, Mass., and Camp Atterbury, Ind. The Fifth Service Command Laboratory identified the Camp Atterbury outbreak. The outbreaks seemed to be limited entirely to the prisoner-of-war compounds with prevalence of 10 to 12 percent, but Dr. T. P. Magill demonstrated by serologic studies that infection had been widely distributed in United States military personnel at Camp Edwards. He suggested that the Americans were being largely immunized by subclinical infection and that the prisoner groups became more prominently affected if they were not closely associated with United States troops. The Antilles Department laboratory reported an epidemic in San Juan beginning 8 June and reaching a peak about 22 June. In Jamaica, an estimated 50 percent of the population of Kingston were attacked in an epidemic. The disease was also identified in United States Army troops in Panama and in civilian employees in the Canal Zone. It is well to point out that Army laboratories were at this time actively engaged in the detection of the disease both by virus isolation and serologic tests.

Influenza was widely disseminated over a great part of the Pacific area. In Honolulu, a sharp civilian epidemic of 7,000 to 8,000 cases was reported by the Board of Health from 1 June to 15 July. Army admissions at this same time increased sharply with a peak on 27 June. The author and Capt. G. K. Hirst, MC, undertook an investigation at this time in the Pacific Ocean Area. Sampling of cases showed virus B was widespread. In this instance, too, streptococcal infection was prevalent without serious complications. Vaccination of essential personnel was carried out. The disease was demonstrated in naval forces at the same time, and vaccination was extended to certain specific personnel. At Tarawa in June, 83 percent of the Gilbert-Ellice labor troops were affected in an epidemic that followed the arrival of two Army ships. Caucasians in the area had little illness. Judged by admissions, little respiratory infection occurred at Saipan and Guam, but serologic samplings showed influenza B to be prevalent. On Okinawa, serologic studies of a denguelike

disease that was prevalent among the troops showed some significant rises in titer for influenza B from 23 July to as late as 27 August.

In July, evidence accumulated of localized outbreaks in Australia [36] and British Guiana.[37] In California, influenza B was identified by Dr. M. D. Eaton in troops who became ill aboard transports from the Pacific, and, at the same time, a local outbreak was seen at Stockton Ordnance Depot.

From late August through October, influenza B was encountered at Fort Bragg, Fort Dix, Fort Lewis, and at Fort Benjamin Harrison, Ind. In October, Burnet reported both influenza A and B in Australia with a concentration of cases in young people or in country districts while troops and adult city dwellers escaped almost completely. He reported two patients with influenza from whom virus B was isolated; 10 to 12 days later, during a second attack, virus A was also isolated from those individuals.

The disease was mild during the summer of 1945 but varied from typical febrile illness of 3 to 4 days' duration, with an occasional instance of pulmonary involvement, to transient indisposition and subclinical infection. It is quite likely that in the civilian population much more of the disease was unrecognized and ignored. However, the accumulated information identified clearly a continued but shifting prevalence of influenza B during a period of 8 months. The alerted interest was thus very effective in demonstrating a peculiar, irregular blustering occurrence, rising here or there for many months. Some of the minor episodes were of considerable size but localized, yet they recurred in the same posts at intervals of months in a typical endemic-epidemic manner.

The extensive distribution of the disease was considered to indicate that a definite epidemic wave was likely in the latter part of the year. On this basis, the Commission on Influenza recommended to General Simmons, on 21 June 1945, that vaccination with influenza A and B be carried out in the entire United States Army during the month of October 1945. War Department Circular No. 267, dated 5 September 1945, instructed that the forces in all Army commands be vaccinated in October and November.

The anticipated epidemic of influenza B occurred in November and December 1945. A definite increase in respiratory admissions in the Army in the United States began in the week ending 23 November when the rate rose from 88 to 103. It rose to 148 the following week and continued to a peak of 170 in the week ending 14 December. This rise was strikingly similar in time to the onset of the 1943 epidemic of influenza A. However, the increase was not nearly so great or abrupt.

The civilian population of the United States experienced an epidemic at the same time. Reported cases rose abruptly beginning the last week in November and reached a sharp peak of 148,688 cases during the second week

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[36] Burnet, F. M., Stone, J. D., and Anderson, S. G.: An Epidemic of Influenza B in Australia. Lancet 1: 807-811, 1 June 1946.

[37] Letter, Maj. H. G. Grady, MC, Commanding Officer, Medical Laboratory, to Brig. Gen. S. Bayne Jones, 24 Sept. 1945, subject: Study of Influenza-Like Illness in British Guiana.

in December.[38] They fell rapidly in the next 3 weeks to less than 50,000 cases per week. The areas most severely affected were the South Atlantic, South Central, East North Central, and Mountain States. The Pacific and New England areas, which were not affected severely, reached peaks late in December or in the first 2 weeks of January. In comparison with the 1943–44 epidemic of influenza A, the 1945–46 epidemic of type B in the general population reached a higher level for the peak week, but in total cases it was less. For the period 18 November 1945 to 26 January 1946, a total of 454,833 cases were reported in the States where influenza is a reportable disease. For the corresponding period in 1943–44, there were 587,193 cases and in the nonepidemic year 1944–45 only 32,620 cases. In the peak week of December 1945, the State of Kentucky contributed 60 percent of the total cases while this same State contributed 18 percent in the 1943–44 peak. This report is apparently made on the basis of estimates rather than actual reported cases. If the estimates for this State were excluded for comparison purposes, the civilian incidence in 1945–46 was at least half of the 1943–44 epidemic.[39]

In the Army, the increase as measured by admission rates for respiratory diseases, after correction of strengths due to the number of troops on furlough during this period, remained less than 25 percent of the 1943–44 epidemic; the actual increase over the preepidemic level was so small as to give evidence of only a minor increase. The Navy admission rates were 55 percent of those in 1943–44. In attempting to evaluate the efficacy of the vaccine, a detailed study of the incidence in the Army and Navy was made. The evidence available indicated that vaccination played a considerable role in the reduction of influenza in the Army during the winter 1945–46. More specific studies in the comparison of ASTP units with naval and civilian students at universities are given under the vaccination studies.

In England and Western Europe, the possibility of a widespread influenza epidemic appeared as a serious threat in the fall of 1945. The shortage of fuel, the nutritional status of the population, and the continued shifting and crowding of displaced persons on the Continent appeared to set the stage for such an occurrence. In order to establish listening posts and to organize laboratories for the detection of influenza, a mission consisting of Dr. Salk, Maj. G. J. Dammin, MC, and Lt. V. Sprague, MC, was dispatched to the European theater in November 1945.[40] Countrywide epidemics of mild influenza B were reported in December in Belgium and Holland.[41] Sporadic cases of influenza A and B were identified in United States troops and civilians in Germany, but no general epidemic occurred. A continuous survey center for the area was established in the Fourth Medical General Laboratory. In

[38] Prevalence of Disease in the United States. Pub. Health Reps. 60 and 61, July–December 1945 and January–June 1946, respectively.

[39] Monthly Progress Report, ASF, War Department, 31 Dec. 1945 and 31 Mar. 1946, Section 7: Health.

[40] Influenza Mission in Europe. Bull. U. S. Army M. Dept. 5: 495–496, May 1946.

[41] (1) Letter, Dr. J. E. Salk and Lt. Col. G. J. Dammin, MC, to Surgeon General, 1 May 1946, subject: Report of Special Group Assigned to the Office of the Chief Surgeon, European Theater, to Assist in the Problem of Influenza During the Winter of 1945–46. (2) Dudgeon, J. A., Stuart-Harris, C. H., Andrewes, C. H., Glover, R. E., and Bradley, W. H.: Influenza B in 1945–46. Lancet 2: 627–631, 2 Nov. 1946.

England, there were few sharp outbreaks recognized, but influenza B was identified in most areas with the height of the prevalence in January 1946. As measured by an increase in deaths, the epidemic was considered moderately severe.

The Army had essentially completed its yearlong experience with influenza B by the end of 1945, although some extension into 1946 was noted in Europe. It is of interest, however, to note that troops, mostly from the 13th Replacement Depot in Hawaii, had a sharp increase of respiratory disease about 1 February 1946, which was identified serologically as influenza A.[42] In the remainder of the Army, respiratory disease reached a point comparable to the same period of 1945, the lowest recorded level for the season (chart 13) The Army was being demobilized, and the threat of influenza which had hung over the entire period of World War II was dispelled. The experience of 1918 was not renewed, and influenza again behaved as a disease not primarily related to military conditions.

The tremendous dislocations of populations, the destruction of housing, and the rapid intermingling of people from many areas under crowded conditions was a milieu in which epidemics of typhoid fever, diphtheria, and tuberculosis rapidly gathered momentum. Circumstances were such that influenza comparable in severity to that of 1918 would be insusceptible to control. No other conclusion seems possible but that the biology of the infectious agents was the decisive factor in avoiding such an event. The studies of the disease and its prevention contributed greatly to a better understanding of its epidemiology and were responsible in a significant fashion for heightened efforts to identify and control respiratory disease of all types. There was, as always, a somewhat fatalistic attitude toward prevention or control measures, but in many situations a true effort was made to gain what benefit could be had by early institution of measures to limit crowding, to control transport of infected men, and at times to use sulfonamides prophylactically in the hope of reducing complications.

Circumstances were favorable. The periods of greatest effort were largely free from influenza or the disease was of sufficiently mild character to avoid serious disturbance. The widespread epidemic of 1943 was not prevalent in the major combat area at that time, Italy. In 1945, the European theater was essentially free from influenza B. Reports from the Pacific theater contained no significant references to influenza until 1945, with the exception of Hawaii which appears to have become a major crossroads for the transfer of influenza. Other theaters in the Far East made no reference to the disease. There is little doubt that the mild character of influenza in busy areas attracted little attention, and many were concerned with a diagnosis of influenza only if it were rapidly fatal or overwhelming. In many instances, epidemics clearly shown to be influenza, with classical clinical and epidemiologic

[42] Essential Technical Medical Data, U. S. Army Forces, Pacific, February 1946.

characteristics, were called nasopharyngitis. This practice sometimes avoided additional requirements imposed by the diagnosis of influenza. The constant insistence of the Preventive Medicine Service, Office of the Surgeon General, and its expert consultants on prompt investigation of outbreaks had left little doubt that the early distribution of information was an important factor in maintaining the alertness and interest of all commands.

RESEARCH WITH INFLUENZA VIRUS

Development of Knowledge

The lack of decisive information regarding the etiology of the pandemic disease of 1918 led to numerous efforts to establish the nature of the inciting agent of influenza. Various bacterial agents were isolated, heralded, and dismissed. Shope, in 1931,[43] however, described and established the evidence that swine influenza is caused by a combined infection of virus and *Hemophilus influenzae suis*, with the former serving as the effective agency in dissemination and immunity. The parallelism between the characteristics of this disease in swine and influenza in men, together with an etiologic complex to mollify the bacterial and viral schools of thought, promptly opened the field to further work. At the same time, viral studies of the common cold and psittacosis were enhancing interest in respiratory diseases.

Type A virus.—In 1933, Smith, Andrewes, and Laidlaw [44] isolated a virus from human cases of influenza A, which produced in ferrets, inoculated in the nose with garglings of the patients, a simple febrile upper respiratory disease of 3 to 4 days' duration involving the turbinate tissues. After recovery they were resistant and developed in their blood antibodies which would neutralize the virus so as to prevent infection when a mixture of convalescent serum and virus was inoculated into normal ferrets. In 1934–35, the author [45] confirmed and extended those results with recovery of virus from influenza patients in many communities of the Western Hemisphere, and also in showing that, with repeated passages of these human strains in ferrets by intranasal route, the animals developed extensive, fatal viral pneumonia. Virus was established by intranasal inoculation in mice so as to produce fatal viral pneumonia. Through their use, neutralization tests for antibody were readily possible. Complement fixation tests were developed. Virus was cultivated in tissue culture and in chick embryos.[46] Subsequently, the observation by

[43] Shope, R. E.: Swine Influenza: III. Filtration Experiments and Etiology. J. Exper. Med. 54: 373–385, September 1931.

[44] Smith, W., Andrewes, C. H., and Laidlaw, P. P.: A Virus Obtained From Influenza Patients. Lancet 2: 66–68, 8 July 1933.

[45] Francis, T., Jr.: Transmission of Influenza by a Filterable Virus. Science 80: 457-459, 16 Nov. 1934.

[46] (1) Francis, T., Jr., Magill, T. P., Rickard, E. R., and Beck, M. D.: Etiological and Serological Studies in Epidemic Influenza. Am. J. Pub. Health 27: 1141-1160, November 1937. (2) Friedewald, W. F.: The Immunological Response to Influenza Virus Infection as Measured by the Complement Fixation Test. Relation of the Complement-Fixing Antigen to the Virus Particle. J. Exper. Med. 78: 347–366, November 1943.

Hirst, [47] and McClelland and Hare, [48] that avian erythrocytes were agglutinated by influenza virus in infected allantoic fluid, added another important technique for identification of virus and serologic diagnosis. The application of these procedures to the problem of epidemic influenza became progressively established in a few years.[49]

It was found that, after the first years of life, a large proportion of the human population had antibodies to influenza A virus. Hence, at the onset of illness, a patient might well possess demonstrable antibodies from earlier infection, but, with recovery, a sharp rise in the level took place. It was necessary, therefore, for specific diagnosis to compare the titers of antibody in the acute phase of illness with that reached in convalescence. The specificity of the reaction was also clearly established. The serologic test thus became a procedure applicable to clinical diagnosis and to broader epidemiologic investigation in conjunction with the isolation and identification of virus.[50]

Employing these procedures, it was shown that outbreaks associated with type A virus had recurred at intervals of 2 years between 1932 and 1940–41.[51] They varied widely in extent and severity; the 1936–37 epidemic was worldwide and that of 1938–39 extremely spotty and of low order, but they were both influenza A. That not all the strains of influenza A are identical was established in 1936;[52] while most strains from the same epidemic are closely similar, those from different epidemics may show distinct differences. That they are of the same type can be demonstrated by complement fixation or by hyperimmunization of animals which bring out the common type antigenicity. Under the latter circumstances, the swine strains were also seen to be related to type A strains from man. Nevertheless, another feature arose to be considered in recurrences of influenza.

Type B virus.- From the epidemic in the early months of 1940, another influenza virus, type B, (p. 90) was established, and it was then possible to demonstrate that the widespread epidemic of 1935–36 was also influenza B. This virus was shown to be immunologically distinct from type A, thereby introducing a second disease, clinically and epidemiologically influenza, to be considered in the analysis of recurrences and of immunity to influenza.

No regular bacterial accompaniment of the virus was found in the epidemic

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[47] Hirst, G. K.: The Agglutination of Red Cells by Allantoic Fluid of Chick Embryos Infected With Influenza Virus. Science 94: 22–23, 4 July 1941.

[48] McClelland, L., and Hare, R.: The Adsorption of Influenza Virus by Red Cells and a New In Vitro Method of Measuring Antibodies for Influenza Virus. Canad. J. Pub. Health 32: 530–538, October 1941.

[49] (1) Andrewes, C. H., Laidlaw, P. P., and Smith, W.: Influenza: Observations on the Recovery of Virus From Man and on the Antibody Content of Human Sera. Brit. J. Exper. Path. 16: 566–582, December 1935. (2) Francis, T., Jr., and Magill, T. P.: The Incidence of Neutralizing Antibodies for Human Influenza Virus in the Serum of Human Individuals of Different Ages. J. Exper. Med. 63: 655–668, May 1936.

[50] Salk, J. E.: Laboratory Methods Used for the Recognition of Influenza. Bull. U. S. Army M. Dept. 5: 32–37, January 1946.

[51] Francis, T., Jr.: Factors Conditioning Resistance to Epidemic Influenza. Harvey Lect. 37: 69–99, 1941–42.

[52] (1) Magill, T. P., and Francis, T., Jr.: Antigenic Differences in Strains of Epidemic Influenza Virus: I. Cross-Neutralization Tests in Mice. Brit. J. Exper. Path. 19: 273–284, October 1938. II. Cross-Immunization Tests in Mice. Ibid.: 284–293. (2) Smith, W., and Andrewes, C. H.: Serological Races of Influenza Virus. Brit. J. Exper. Path. 19: 293–314, October 1938.

disease of man.[53] *Hemophilus influenzae* was uncommon, but, in occasional, rapidly fatal cases with extensive destruction of the respiratory epithelium, hemolytic *Staphylococcus aureus* had been encountered. Thus, the evidence firmly established influenza virus as the essential infectious agent in characteristic epidemic influenza.

Antibody formation.—Influenza virus produces an infection essentially limited to the respiratory tract. It has a highly selective, destructive action upon the ciliated respiratory epithelium of the nasal mucosa and upon that of the trachea and bronchi. After large doses given intraperitoneally to mice, virus can be recovered from the lungs and, if well adapted to that species, may produce extensive pulmonary lesions. The WS strain of type A can be established in the central nervous system of mice by the intracerebral route. Generally, however, the influenza virus can be considered rather strictly pneumotropic since, under most circumstances, inoculation of various species with active virus by other than the respiratory route elicits no evidence of infection but circulating antibodies and resistance may ensue.

Ferrets and mice recovering from infection are commonly immune to reinoculation of the same strain of virus and also to others of the same type. This effect is not permanent for, as well demonstrated in the ferret, after a few months, even though antibodies are present in the blood, reinoculation may again cause febrile illness, with destruction of the respiratory epithelium although pulmonary lesions do not ordinarily develop. The general clinical and epidemiologic experience of man with influenza has resulted in the conclusion that immunity to the disease is of a transient nature; the evidence has always been clouded, however, by lack of knowledge of the agent involved. Nevertheless, the experimental data in man and lower animals are in accord. The fact that many of the human patients have antibodies to virus of the same type before, or at the time of, onset of influenza is clear evidence of previous experience with the agent. This knowledge, together with the realization that there are two or more distinct types of virus and many variations of strains within the types, presents a formidable array of problems.

On the other hand, mice can be readily immunized by intraperitoneal vaccination of active or inactive virus and less readily by subcutaneous inoculation; they become resistant even after infection with virus not sufficiently well adapted to cause severe disease. Ferrets, too, can be vaccinated but usually less effectively than mice. In 1935, Francis and Magill [54] demonstrated that virus cultivated in tissue culture can be given to man subcutaneously or

[53] (1) Shope, R. E.: The Influenzas of Swine and Man. Harvey Lect. 31: 183–213, 1935–36. (2) Shope, R. E.: Swine Influenza. *In* Rockefeller Institute for Medical Research. Virus Diseases. Ithaca: Cornell University Press, 1943, pp. 83–109. (3) Andrewes, C. H.: Immunity in Influenza. The Bearing of Recent Research Work. Proc. Roy. Soc. Med. 32: 145–152, 1939. (4) Stuart-Harris, C. H.: Influenza Epidemics and the Influenza Viruses. Brit. M. J. 1: 209–216, 251–257, February 1945. (5) Francis, T., Jr.: Influenza: Methods of Study and Control. Bull. New York Acad. Med. 21: 337–355, July 1945. (6) Burnet, F. M., and Clark, E.: Influenza. A Survey of the Last Fifty Years in the Light of Modern Work on the Virus of Epidemic Influenza. Monographs from the Walter and Eliza Hall Institute of Research in Pathology and Medicine, No. 4. Melbourne: Macmillan, 1942, pp. 1–118.

[54] Francis, T., Jr., and Magill, T. P.: Cultivation of Human Influenza Virus in Artificial Medium. Science 82: 353–354, 11 Oct. 1935.

intracutaneously without eliciting signs of infection but resulting in the development of antibodies which reach a peak in about 14 days and are maintained for months. The levels attained, the curve of development, and the persistence are quite parallel to those observed in subjects undergoing the actual disease; hence, if immunity in man is correlated with antibodies as it is in other animals, the results of vaccination strongly suggest that resistance in man could be similarly effected. In this background of information lay a basis for efforts toward prophylactic immunization. Before the advent of the Commission on Influenza, different investigators had undertaken further studies of vaccination with preparations of virus from tissue culture, mouse lung, and chick embryo.[55] In each instance, vaccination had resulted in antibody formation, but the protective value against the disease had not been clearly established; nevertheless, some of the results had suggested a beneficial effect.

Organization for Research in War Period

The onset of war in Europe met in the United States a firm commitment of neutrality. However, with the progressive victories of the German armies, it became apparent that American democracy must be prepared for its own defense. Preparation meant more than the induction of men and the manufacture of materials. It meant the creation of forces trained in advance and maintained in a state of effectiveness. No longer did events await the arrival and the preparation of volunteers in the numbers now required. Manpower was important.

It is commonly said that the medical knowledge of one war has usually been forgotten and bitterly relearned in the next. The microbial enemy has often been more destructive than lead and steel. Certainly it is true that personal courage and indifference to risk have frequently been the substitute for sanitary and other preventive measures. But preparedness is medical too. In this instance, the history of respiratory disease in World War I in a hastily mobilized army without adequate provision for mass phenomena of disease had left its memory. The improvised efforts to meet a terrible situation had been splendid, but they emphasized the need to view the possible problems in ad-

[55] (1) Stokes, J., Jr., McGuinness, A. C., Langner, P. H., Jr., and Shaw, D. R.: Vaccination Against Epidemic Influenza With Active Virus of Human Influenza. Am. J. M. Sc. 194: 757-768, December 1937. (2) Smorodintseff, A. A., Tushinsky, M. D., Drobyshevskaya, A. I., Korovin, A. A., and Osetroff, A. I.: Investigation on Volunteers Infected With the Influenza Virus. Am. J. M. Sc. 194: 159-170, August 1937. (3) Martin, W. P., and Eaton, M. D.: Experiments on Immunization of Human Beings Against Influenza A. Proc. Soc. Exper. Biol. & Med. 47: 405-409, May-June 1941. (4) Horsfall, F. L., Jr., Lennette, E. H., Rickard, E. R., and Hirst, G. K.: Studies on the Efficacy of a Complex Vaccine Against Influenza A. Pub. Health Rep. 56: 1863-1875, 19 Sept. 1941. (5) Dalldorf, G., Whitney, E., and Ruskin, A.: A Controlled Clinical Test of Influenza A Vaccine. J. A. M. A. 116: 2574-2577, 7 June 1941. (6) Brown, J. W., Eaton, M. D., Meiklejohn, G., Lagen, J. B., and Kerr, W. J.: An Epidemic of Influenza. Results of Prophylactic Inoculation of a Complex Influenza A-Distemper Vaccine. J. Clin. Investigation 20: 663-669, November 1941. (7) Stuart-Harris, C. H., Smith, W., and Andrewes, C. H.: The Influenza Epidemic of January-March, 1939. Lancet 1: 205-211, 3 Feb. 1940. (8) Taylor, R. M., and Dreguss, M.: An Experiment in Immunization Against Influenza With a Formaldehyde-Inactivated Virus. Am. J. Hyg. 31: 31-35 (sec. B), January 1940. (9) Siegel, M., Muckenfuss, R. S., Schaeffer, M., Wilcox, H. L., and Leider, A. G.: A Study in Active Immunization Against Epidemic Influenza and Pneumococcus Pneumonia at Letchworth Village. IV. Results in an Epidemic of Influenza A in 1940-41. Am. J. Hyg. 35: 186-230, March 1942.

vance, to watch for signs of their development, and to seek control methods for early and extended application. Moreover, much greater scientific knowledge of specific approaches toward the prevention or treatment of epidemic infection had developed. The interest of many capable investigators was already moving in these directions. In addition, many problems could, because of special circumstances, be adequately studied only under military conditions. The Surgeon General's Office was not only aware of these facts but acutely interested in applying the concepts to the profit of the actively growing military forces. Under these influences, the Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the Army was initiated by the Preventive Medicine Division (fig. 2), and the Commission on Influenza was organized under the Board. Much of the research and observations relating to influenza was conducted by the Commission, but its functioning was at each step dependent upon the financial aid from the Board and the strong and imaginative support furnished by General Simmons, Brig. Gen. Stanhope Bayne-Jones, and others of the staff, and the splendid cooperation of the personnel of many organizations in which work was undertaken.



FIGURE 2.—Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the Army. Front row, left to right: Col. (later Brig. Gen.) J. S. Simmons, Dr. O. H. Perry Pepper, Dr. A. J. Warren, Dr. E. W. Goodpasture, Dr. F. G. Blake, Dr. O. T. Avery, Dr. K. F. Maxcy, Dr. A. R. Dochez, and Lt. Col. (later Brig. Gen.) S. Bayne-Jones. Second row, left to right: Dr. J. R. Paul, Dr. P. H. Long, Dr. C. M. MacLeod, Dr. T. Francis, Jr., Dr. W. A. Sawyer, Dr. J. Stokes, Jr., Dr. O. H. Robertson, and Dr. M. H. Dawson.

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From the start, emphasis was placed on a broad program of study of epidemics and of control measures as shown by the initial outline presented by the Commission on Influenza to the Board on 27 and 28 February 1941:

I. *Study of Control Measures.*
  1. Hygienic and Environmental Controls.
    a. Influence of housing, size of cantonments, troop movements.
    b. Isolation of individual or post; the use of masks.
    c. Disinfection—sterilization of dishes, sterilization of air with aerosols or ultraviolet light.
  2. Specific Control.
    a. The efficacy of vaccination against influenza virus infection in man.
    b. Prophylactic use of immune serum.

II. *Study of Epidemics.*
  1. Clinical.
    a. Attempts to establish clinical criteria for differentiation of disease caused by different types of influenza virus or by other agents.
    b. Possible study of chemoprophylaxis of bacterial complications.
  2. Epidemiological.
    a. To ascertain incidence of immunes, subclinical and clinical cases in correlation with laboratory studies.
    b. The method of introduction, the factors influencing spread, and the pattern of epidemics.
  3. Bacteriological.
    a. To determine whether any particular bacterium constantly accompanies influenza virus infection.
    b. Concentrated study of *H. influenzae*.
    c. Significance of bacteria in the course of the disease.
  4. Virus.
    a. To identify virus in epidemics, especially in recurrent waves, and the relation of illness to the immunologic state.
    b. To evaluate importance of factors other than circulating antibody in resistance.
    c. To institute prophylactic measures.
    d. To ascertain complications caused by virus alone.
  5. Pathological.
    a. To correlate the picture in fatal cases with etiological studies.
    b. To search for diagnostic criteria when illness was not caused by known virus.
  6. Cooperative studies with other Commissions, especially in the field of complications and chemotherapeutics.

In order to meet these objectives, a more detailed pattern of organization and plan of operation was designed by which the United States was divided geographically into eastern, midwestern, and far western areas, and the members of the Commission were largely divided in the same manner. It was proposed that, in the event of a severe epidemic, an equipped, highly qualified team of investigators comprising a clinician, a pathologist, an epidemiologist, a bacteriologist, and a virologist would enter the field in each area and function as an investigative unit to gain a rounded picture of the epidemic disease and special knowledge of the different aspects of the problen.

Interim studies would deal largely with practical approaches to the improvement of control procedures. At the meeting of the Board, 19 to 21 June

1941, specific authorization was given for the following interim investigations: (1) Experimental trial of influenza vaccine should a promising vaccine be available and suitable opportunity arise; (2) studies of the efficacy of respiratory masks; and (3) laboratory studies of materials and samples collected in the field bearing on the etiology, epidemiology, and immunology of influenza and its complications. The problems of air sterilization were referred to a newly formed Commission on Cross-infections in Hospitals. An appropriation of $25,000 for interim and $30,600 for field studies was voted. The original estimate was sharply reduced by the offer of the Rockefeller Foundation to provide, without cost, the facilities and participation of their laboratories in New York and San Francisco. Mobile laboratories were considered unnecessary because of the availability of corps area laboratories.

Here was provided a coordinated alert in which all members of the Commission would report immediately any outbreaks of respiratory diseases so that investigations could be rapidly instituted and in which civilian personnel with special interests were prepared to study influenza from a variety of approaches in their own laboratories or, in the field, to assist the Army during epidemics as ordered by The Surgeon General. A statement summarizing the current status of influenza and its control was prepared and published in Circular Letter No. 124, Office of the Surgeon General, on 23 December 1941.

Studies of Vaccination

Although a great deal of the subsequent discussion is concerned with the problem of vaccination against influenza, many of the major problems presented in the original plan were integrated into this broad concept in which research in the problems of the disease and the virus constitute the basis for studies in vaccination.

Intranasal vaccination.—Two major concepts of vaccination were considered. The one was intranasal introduction of active virus so attenuated as to give mild or subclinical infection with the expectancy that satisfactory resistance would develop. This would simulate natural conditions and should contribute any advantage, including possible alterations in tissue susceptibility, which accompanied actual infection. Preliminary observations had indicated that virus which had been maintained for long periods in tissue culture could be used without eliciting symptoms but the serologic response was quite irregular. The pathogenic balance was, therefore, of importance. Moreover, the objection was raised that within military forces active virus might be enhanced by passage and give rise to outbreaks of the disease. Nevertheless, in an approach to the problem, 3 series of 10 human subjects each were sprayed with different doses of type B virus from allantoic fluid.[56] After incubation periods of 18 to 24 hours, 27 of 30 men developed clinical influenza and with recovery showed significant rises in circulating antibody. But when retested

[56] Francis, T., Jr., Pearson, H. E., Salk, J. E., and Brown, P. N.: Immunity in Human Subjects Artificially Infected With Influenza Virus, Type B. Am. J. Pub. Health 34: 317-334, April 1944.

4 months later with the same virus, one-third again developed acute illness comparable to the first, while others had milder illness. Inactivated virus caused no reaction. There was thus no uniform, firm immunity acquired under the condition of the study.

Subcutaneous vaccination. The second approach to which greatest attention was given was that of subcutaneous vaccination. The various studies previously carried out (p. 113) had shown that virus administered in this manner could stimulate good levels of antibody, and some indications of resistance had been observed in man under epidemic conditions. Although active virus could be given, the problems of stability of virus, bacterial sterility and possible viral contaminants, and storage and distribution were practical objections. Finally, attention was centered upon the preparation of vaccine containing type A and type B viruses from the allantoic fluid of infected chick embryos. High titers of virus were available, and the problem here became one of developing procedures and production. For the latter purpose, the interest of commercial biologic firms was enlisted. The development of the program is given in detail in the history of the Commission on Influenza, and this discussion will present only summaries of the actual trials. The principles at all times were to provide for a supply of practicable vaccine; the selection of a homogeneous, stable vaccine; uniform procedure; close observation by highly competent investigators; confirmation of diagnosis by etiologic and serologic methods; and adequate controls at all steps.

Studies in 1941–42

Authorization was given for a study, under the immediate supervision of Dr. Eaton, in troops in California if the opportunity for a satisfactorily controlled experiment arose. Two thousand doses of a vaccine prepared from chick embryos were made available by Drs. J. H. Bauer and G. K. Hirst of the laboratories of the International Health Division of the Rockefeller Foundation. This effort was abandoned in January 1942 because of change in conditions due to our entry into war.

Studies in 1942–43

In addition to evidence that inactivation of virus caused a reduction in antigenic potency, data of Hirst, Rickard, Whitman, and Horsfall [57] indicated that within limits higher antibody levels were reached in man with preparations of virus concentrated from allantoic fluid by centrifugation. Hirst, Rickard, and Whitman,[58] and Hare, McClelland, and Morgan [59] described the concentration of virus by collection on a precipitate which formed when previously frozen material was allowed to thaw.

[57] Hirst, G. K., Rickard, E. R., Whitman, L., and Horsfall, F. L., Jr.: Antibody Response of Human Beings Following Vaccination With Influenza Viruses. J. Exper. Med. 75: 495–511, May 1942.

[58] Hirst, G. K., Rickard, E. R., and Whitman, L.: A New Method For Concentrating Influenza Virus, From Allantoic Fluid. Proc. Soc. Exper. Biol. & Med. 50: 129–133, May–June 1942.

[59] Hare, R., McClelland, L., and Morgan, J.: A Method for the Concentration of Influenza Virus. Canad. J. Pub. Health 33: 325–331, July 1942.

The probabilities of an epidemic in this period were high since it would be in keeping with the 2-year cycle of identified influenza A since 1932–33. A field trial of vaccine prepared by this method was then planned for the winter of 1942–43.

Cornell study.—Drs. Magill, N. Plummer, and W. G. Smillie and their associates undertook a study in the student body and faculty at Cornell University, Ithaca, N. Y. Recruitment into the study was on a voluntary basis with administration of vaccine or control material of normal allantoic fluid subcutaneously. Of a total of 2,885 persons, 1,672 received vaccine and 1,213 control material. Great difficulty was encountered by the firm which undertook the preparation of precipitated vaccine, and Dr. Hirst aided by providing 1,000 doses from the Rockefeller Foundation. Another lot of unconcentrated type A vaccine was also used. Careful clinical, viral, and serologic observation was maintained on respiratory illness in the population. No virus was isolated. No influenza was detected although serologic evidence suggested that a few infections with influenza B occurred. No difference in incidence of any class of respiratory disease was noted among the vaccinated and control groups. Good antibody responses to vaccination were maintained during the 4 to 5 months' observation. Reactions with systemic symptoms of fever, aches, and other conditions were observed in approximately 10 percent of those receiving either concentrated or unconcentrated vaccine and in 2 percent of the controls.

Michigan study.—In two institutions, a study was arranged to test the cold-precipitated type of vaccine. Owing to the difficulty mentioned in manufacture, attention was turned to vaccine prepared by concentration of virus from allantoic fluid by adsorption to, and elution from, erythrocytes of the infected embryo.[60] In this manner, an eightfold to tenfold concentration of virus could be obtained in salt solution while leaving a large proportion of extraneous material behind. The virus was readily inactivated by 0.05 percent formalin, and sterility was relatively easy to control. Its potency was equal to that prepared by freezing and thawing. The greater ease of its production resulted in obtaining sufficiently large amounts of finished vaccine from the same commercial firm. Each 1.0 cc. contained the virus obtained from 5.0 cc. of type A fluid and from 5.0 cc. of type B fluid.

In late December and early January, 3,914 persons received 1.0 cc. vaccine and 3,909 alternately received control solution. The expected epidemic of influenza A did not occur, although in one institution influenza B at a subclinical level was detected (p. 95). Antibodies to both viruses rose after vaccination and diminished slowly over a year's time but still remained above prevaccination levels. Data on antibody levels of the same persons before, and at intervals after, vaccination with combined A and B vaccine are presented in table 21.

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[60] Francis, T., Jr., and Salk, J. E.: A Simplified Procedure for the Concentration and Purification of Influenza Virus Science 96: 499–500, 27 Nov. 1942.

TABLE 21.—*Average antibody titers at intervals after vaccination*

Interval after vaccination	Number of subjects	Average antibody titer	
		Type A (PR 8)	Type B (Lee)
Prevaccination	92	59	33
2 weeks	92	548	542
4 months	68	356	353
1 year	82	282	246

The results of the year's endeavors had given information that concentrated vaccine was producible by two different methods of preparation and, in addition, that after eluate vaccine a high level of antibody was maintained for an extended period—a year at least. This observation was amply confirmed in later studies.

To gain information of the protective effect of vaccine by direct means, a test of immunity to induced influenza A and influenza B was undertaken by spraying with active virus groups of the vaccinated and control populations.[61] The results demonstrated a high degree of protection against influenza B. There was an incidence of 10 percent in those vaccinated as long as 4 months earlier and in 40 percent of the controls, a greater resistance than had been obtained by actual experimental infection with the same virus. In the case of influenza A, vaccination 2 weeks before test reduced the incidence from 50 percent in the controls to 16 percent in the vaccinated, although vaccination 4 months earlier had a less definite effect. There was, therefore, definite information of immunizing influence of the vaccine produced by elution. Stokes and Henle [62] had also demonstrated in a similar fashion the protection of a group of children from experimentally induced influenza A after subcutaneous vaccination with vaccine prepared from allantoic fluid.

Other studies.—Dr. Hirst undertook a study involving 8,058 persons in institutions who received vaccine concentrated by freezing and thawing with a similar number of designated uninoculated controls. The absence of epidemic influenza thwarted an evaluation of effect.[63] Another study by Dr. Eaton with alum-precipitated vaccine met the same situation, but he showed that the antibody titers were comparable to those obtained with the other preparations.

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[61] (1) Francis, T., Jr., Salk, J. E., Pearson, H. E., and Brown, P. N.: Protective Effect of Vaccination Against Induced Influenza A. J. Clin. Investigation 24: 536–546, July 1945. (2) Salk, J. E., Pearson, H. E., Brown, P. N., and Francis, T., Jr.: Protective Effect of Vaccination Against Induced Influenza B. J. Clin. Investigation 24: 547–553, July 1945.

[62] Henle, W., Henle, G., and Stokes, J., Jr.: Demonstration of the Efficacy of Vaccination Against Influenza Type A by Experimental Infection of Human Beings. J. Immunol. 46: 163–175, March 1943.

[63] Hirst, G. K., Rickard, E. R., and Friedewald, W. F.: Studies in Human Immunization Against Influenza. Duration of Immunity Induced by Inactive Virus. J. Exper. Med. 80: 265–273, October 1944.

Studies in 1943–44

The desirability of field studies of vaccination in military units was increasingly evident and for this reason authorization was obtained to conduct investigations in Army Specialized Training Program units. They were stable populations and subject to constant, uniform observations. It was possible to obtain participation of entire units so that vaccinated persons and controls could be properly designated rather than depending upon the less desirable and unpredictable basis of volunteers. Different members of the Commission were responsible for the work with units in different parts of the country; however, the same vaccine, the same record system, the same plan of observation, including clinical criteria, and viral and serologic studies were agreed upon. Alternate men in each unit were to receive vaccine and control material subcutaneously. The vaccine prepared by the elution method contained 50 percent type B (Lee) virus and 50 percent type A virus. Of the latter, half was the PR8 strain and half the Weiss strain, which had been isolated from a sporadic case in May 1943, and was employed because of the possibility that it might represent a forerunner of a subsequent strain.[64]

Six studies in nine ASTP groups constituting approximately 12,500 men were established with vaccination scheduled for October or November. Again, emphasis was placed upon a continuous, intensive watch for the occurrence of influenza. A coordinated system was arranged for sampling of respiratory illness in the selected units and throughout the different service commands with regular reporting of results. The epidemic of influenza A was promptly detected in the Sixth and Seventh Service Commands between 15 and 20 November at which time vaccination of the study groups was, with one exception, either completed or in progress. It spread rapidly through the service commands with sharp peaks during the early weeks of December, but in the Fourth, Eighth, and Ninth Service Commands the epidemic was more prolonged and at lower levels than in the others. It was essentially over by 1 January.

The results of the study gave conclusive evidence, because of the carefully controlled procedures, that vaccination had exerted a sharply protective effect against epidemic influenza A (table 22).[65]

The final figures differ little from those of the preliminary summary. The total incidence based upon admissions was 7.3 percent among 6,198 controls and 2.2 percent among 6,253 vaccinated, a ratio of 3.3:1. No fatalities occurred in the study groups and there were few cases of pneumonia.

Other data of importance were also obtained. It was found that the epidemic was essentially a pure one in that 80 to 90 percent of cases were identifiable as influenza A. In addition to the incidence of 8 to 10 percent as measured by the admissions, it was shown that as high as 35 to 40 percent of the control population had undergone infection, much of it subclinical. This

[64] See footnote 21, p. 95.

[65] Commission on Influenza: A Clinical Evaluation of Vaccination Against Influenza. Preliminary Report. J. A. M. A. 124: 982-985, 1 Apr. 1944.

TABLE 22.—*Summary of clinical evaluation of vaccination against influenza, 1943 (combined totals of all results)*

Group	Service Command	Army Specialized Training Program unit at—	Dates of vaccination	Subjects			Cases of influenza		Incidence of influenza		Percentage of total cases	
				Total	Vaccinated	Control	Vaccinated	Control	Vaccinated	Control	Vaccinated	Control
			1943	*Number*	*Number*	*Number*	*Number*	*Number*	*Percent*	*Percent*		
	Second	Columbia University	9 Nov.	982	498	484	15	43	3.01	8.86	26	74
1	...do...	New York Medical Schools [1]	26 Oct.–4 Nov.	1,953	976	977	14	33	1.43	3.38	30	70
2	...do...	Princeton University	2 Nov.	1,150	590	560	17	46	2.88	8.20	27	73
	...do...	Rutgers University	1 Nov.	1,212	606	606	7	42	1.15	6.93	14	86
	...do...	College of the City of New York.	19 Nov.	2,105	1,050	1,055	14	75	1.33	7.10	16	84
3	Sixth	University of Michigan	26 Oct.–2 Nov.	1,776	888	888	20	74	2.25	8.35	21	79
4	Seventh	University of Minnesota	5 Nov.–13 Nov.	1,206	599	607	16	55	2.68	9.06	22	78
5	...do...	University of Iowa	2 Dec.–4 Dec.	1,198	599	599	11	40	1.83	6.67	21	79
6	Ninth	University of California	19 Oct.–27 Oct.	892	457	435	24	34	5.25	7.80	41	59
	Total			12,474	6,263	6,211	138	442	2.22	7.11	23.8	76.2

[1] Includes Columbia Medical and Dental College, Long Island Medical College, New York Medical College, and New York University College of Medicine and Dentistry.

is of importance in estimating the immunizing effect of either vaccine or of the natural infection. There was a definite trend indicating the tendency for the highest frequency of disease to occur in that portion of the vaccinated population with the lowest antibody titers; the same relation existed in the unvaccinated population. There were suggestions that this correlation was more distinct when antibodies were measured against strains from the epidemic rather than against the PR8 strain of the vaccine.

The vaccine induced localized erythema, edema, and heat in the majority of those inoculated; some systemic manifestations such as mild aching and chilliness occurred in as many as 25 to 30 percent, and 1 to 2 percent of some groups were sick enough to report to sick call. In the total vaccinated population, four instances of apparently allergic reactions were recorded. In two instances, the epidemic began while vaccination was being done; it was observed that the incidence curves began to separate in the vaccinated and controls 5 to 8 days after vaccination, indicating that after this interval the vaccine effect had begun. In two groups, streptococcal infection was prevalent, but no accentuation in severity of disease took place. The duration of protective effect was not demonstrable by these studies, but it was uniformly noted that over several months only a mild fall in the postvaccination antibody titers to type A or type B virus took place. Two other groups which had been vaccinated the previous year were observed during this epidemic with results indicating that vaccine had exerted a protective influence even after that

interval.[66] The suggestion was made that the California study had been less decisive than the others because of a longer interval between vaccination and the epidemic occurrence, but this does not appear to be valid.

The success of the study in evaluating the vaccine and in gaining a large amount of accessory information was the result of a carefully planned program by highly interested and qualified investigators who maintained a uniformity of procedure and observation while using adequate controls in each phase of the study. The speed with which the epidemic developed, spread, and declined strongly demonstrates the difficulty to be encountered if the study is not established in advance of the epidemic; otherwise, it is probable that the opportunity will have passed before the work can be done.

With the termination of the 1943 epidemic, influenza apparently disappeared in the United States and, although the continuous alert for detection was maintained and expanded, no field studies were planned for 1944. The question of providing suitable vaccine for use throughout the Army was being studied. The problem of reactions was investigated, with a clear demonstration that this typhoidlike effect was strictly related to the amount of virus in the vaccine and that the amount contained in the eluate vaccines used in 1943 was about the limit tolerable.[67] The increase in antibody titer was not directly proportionate to the amount of virus; 2.0 mg. of virus protein resulted only in a twofold increase in titer over that obtained with 0.2 mg. It was clear, nevertheless, that too small a dose did not elicit adequate response, and it was concluded that a range between 0.2 and 0.5 mg. of "estimated virus protein" was the most practicable from all considerations—antibody response, percentage of reactions, and cost of production. The previous vaccines appeared to fall in this range. Concentration of vaccine by centrifugation was studied, standards for its production were devised, and it was accepted as an alternative procedure for Army vaccine.

Studies in 1945

In view of the increasing prevalence of influenza B noted in 1945, the recommendation was made through the Epidemiological Board that vaccination be carried out in the Army in October unless events warranted its earlier use. Final approval of The Surgeon General and of the Secretary of War was given in August. In some areas of the Pacific, vaccination had already been instituted because of higher incidence of the disease, but the program was carried out almost uniformly in all Army personnel in all theaters of operations during October and November 1945 with vaccine prepared by the elution method.

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[66] See footnotes 19, p. 95, and 63, p. 120.

[67] (1) Salk, J. E.: Reactions to Concentrated Influenza Virus Vaccines. J. Immunol. 58: 369-395, April 1948. (2) McLean, I. W., Jr., Beard, D., Taylor, A. R., Sharp, D. G., and Beard, J. W.: The Relation of Antibody Response in Swine to Dose of the Swine Influenza Virus Inactivated With Formalin and With Ultraviolet Light. J. Immunol. 51: 65-99, August 1945.

Because of the uniform regulation concerning vaccination throughout the Army, no controlled study could be established within that service. However, in two locations circumstances were such that an opportunity for evaluation arose. Naval personnel were not vaccinated, otherwise V–12 units were closely comparable to ASTP units, and the university agency for supervision of their health was usually the same. At the University of Michigan and at Yale University, the naval units and the ASTP units were under observation at the time the sharp epidemic of influenza B developed in November and December 1945. The opportunity was thereby provided for a comparison of the occurrence of influenza in the two organizations.[68] The results based upon admissions to the infirmaries were striking. As clearly demonstrated in table 23, vaccination had been highly protective against epidemic influenza B. The effect was even sharper than the results with the same vaccine against influenza A in 1943. Etiologic studies demonstrated that the epidemic was limited to type B virus. The strains of type B virus showed serologic characteristics which readily separated them from the vaccine strain (Lee) although the latter gave adequate cross-immunity. Two further indications were obtained. The first is in accord with earlier data that type B is a better immunizing agent than type A strains. The second is that vaccination of a total group gives better protection to the group than when, as in 1943, only half the group is vaccinated. Comparison of a completely vaccinated group with a completely unvaccinated unit might also yield a sharper differential—a possibility also suggested in 1943 when the incidence in certain unvaccinated companies was found to be strikingly higher than in the unvaccinated half of the study groups. Thus, vaccination of half a closely associated unit would have a limiting effect upon the risk to the unvaccinated portion so as to

TABLE 23.—*Admissions for influenza in vaccinated Army units and unvaccinated Navy units at the University of Michigan and at Yale University over an 8-week period*

Unit	Strength	Incidence (percent)	Total admissions	Admissions during week ending—							
				Nov. 3	Nov. 10	Nov. 17	Nov. 24	Dec. 1	Dec. 8	Dec. 15	Dec. 22
University of Michigan:											
Army	600	1. 17	7	0	0	1	2	1	0	2	1
Navy	1, 100	9. 91	109	6	7	7	9	21	10	35	14
Yale University:											
Army	550	. 55	3	0	0	0	0	0	0	3	0
Navy	1, 050	12. 67	133	2	6	19	21	38	26	11	10

[68] (1) Francis, T., Jr., Salk, J. E., and Brace, W. M.: The Protective Effect of Vaccination Against Epidemic Influenza B. J. A. M. A. 131: 275–278, 25 May 1946. (2) Hirst, G. K., Vilches, A., Rogers, O., and Robbins, C. L.: The Effect of Vaccination on the Incidence of Influenza B. Am. J. Hyg. 45: 96–101, January 1947.

reduce the incidence in the latter below that of a totally untreated population. There is no indication in the tabulated data that resistance of the vaccinated unit was any different in the late stages of the epidemic than earlier. Moreover, the close similarity of the results in the two institutions adds to their significance.

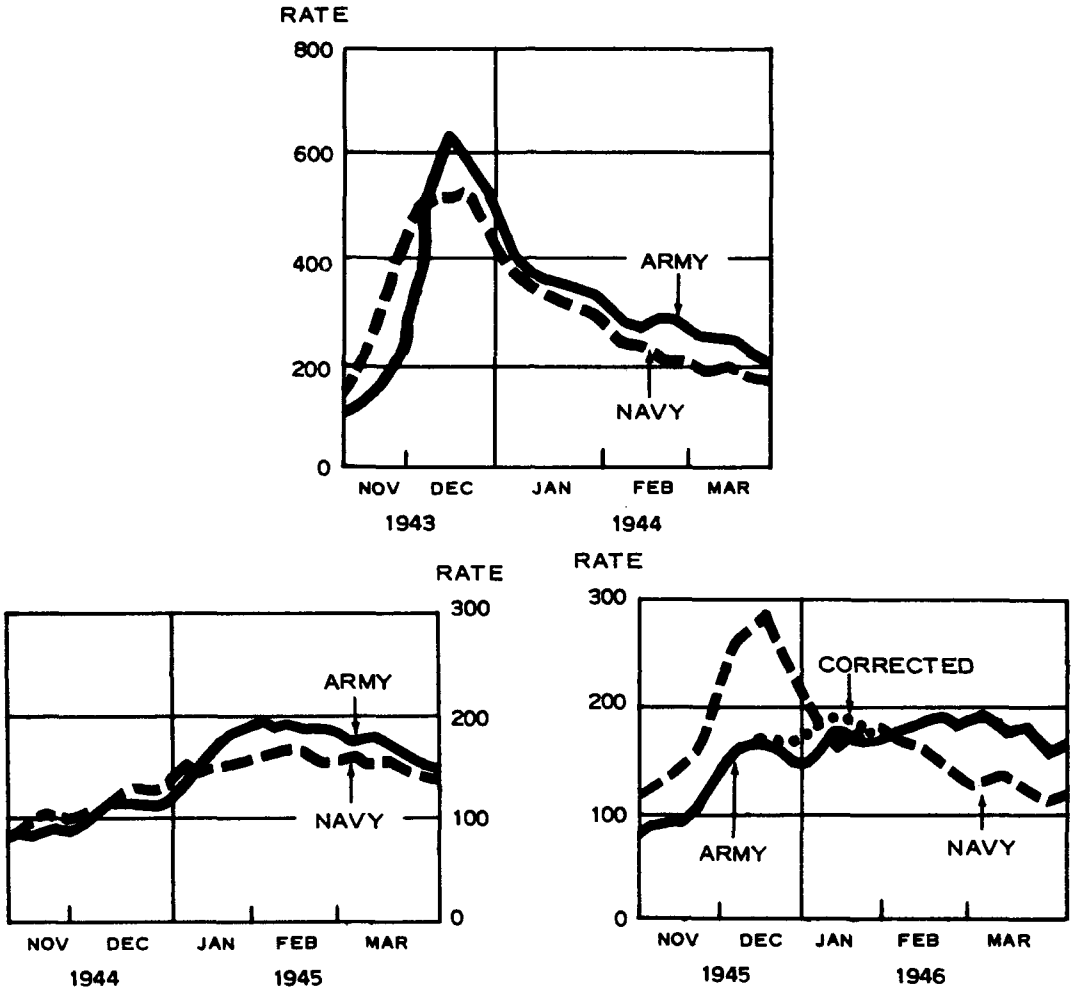
Further effort to gain information on the effect of vaccine upon the incidence of disease in the Army was presented in ASF monthly progress reports. Analysis was made of respiratory disease rates in Army and Navy personnel in the United States during the 1945 epidemic, when vaccine had been widely administered in the Army but the Navy remained unvaccinated, and compared them with the experience of the two services in 1943 when neither had been vaccinated. Whereas, in 1943, the incidence of admissions for common respiratory disease and influenza in the Army was 117 percent of that of the Navy at the peak of the epidemic, in 1945 the Navy rate at the peak was 173 percent of that of the Army, or, conversely, the Army rate was only 57 percent of the Navy's. While the Army rate in 1945 was only 27 percent of that observed in 1943, the Navy rate was still 55 percent (chart 15).

The excess rate over the preepidemic level for common respiratory diseases in the Army in 1945 was only about 16 percent of that in 1943, while in the Navy it was still about 46 percent of the 1943 excess. The comparison with previous experience between the two services points out: "The increase in Army morbidity should have been just twice what was actually reported for November and December. A large part of the 50 percent increase *not* prevented may have consisted of common colds rather than influenza." The latter suggestion is further supported by the fact that the increased incidence in the Army did not abruptly subside as it did in the Navy, indicating that it might be largely the seasonal rise of common respiratory disease. It was not markedly different from that of 1944 when little influenza was observed. Moreover, the contrast held when the incidence in naval districts and Army service commands was compared geographically. In the areas of the Fifth, Sixth, and Seventh Service Commands and the Ninth Naval District, where the ASTP study was convincing, the epidemic peak in the Navy was two and one-half times as high as in the Army (chart 16). The analysis indicates that vaccination was not complete in the Army and that most cases of confirmed influenza were in unvaccinated men. Further analysis emphasizes that the incidence curve in the Army did not differ strikingly from that of 1944, while the Navy experienced sharp epidemics. "In summary it may be said that all available evidence points to a considerable saving in morbidity. It seems entirely fair to judge the efficacy of the vaccination program on the basis of the period when the epidemic was in progress. One can only conclude that the epidemic of mild influenza B touches the Army only very lightly because of the protection afforded by the vaccine." [69]

[69] See footnote 39, p. 109.

CHART 15.—*Incidence of common respiratory diseases and influenza in the Army[1] and the Navy in the United States for the winters of 1943–44, 1944–45, and 1945–46*

[Rate expressed as number of admissions per annum per 1,000 average strength]

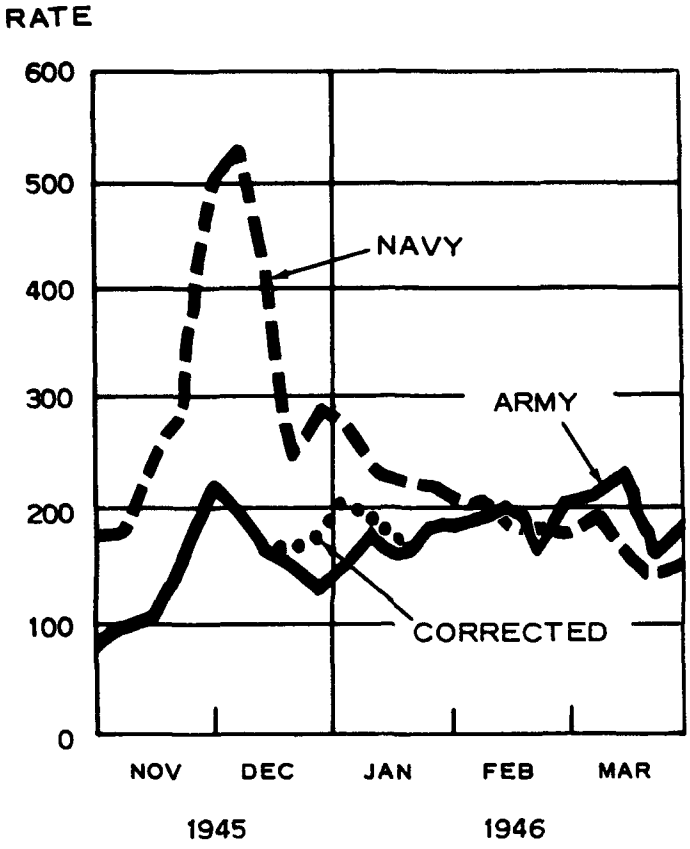


[1] Correction of the Army rate was necessary because the reporting system made no allowance for troops which were temporarily absent from posts for the Christmas holidays.

The field studies of vaccination against influenza led to an application of the procedure in the entire Army. Their development was guided by evidence as it could be acquired and upon the pattern of the scientific experiment with proper control. Even when an ideal study could not be planned in advance, active investigators, continuously on the alert, could seize the unplanned opportunity to gain evidence.

CHART 16.— *Incidence of common respiratory diseases and influenza in the United States in the Fifth, Sixth, and Seventh Service Commands of the Army* [1] *and the Ninth District of the Navy for the winter of 1945–46*

[Rate expressed as number of admissions per annum per 1,000 average strength]



[1] Correction of the Army rate was necessary because the reporting system made no allowance for troops which were temporarily absent from posts for the Christmas holidays.

Other Research

Other phases of research important to the problem of influenza were being carried on. A serious question was whether drugs effective in the treatment of bacterial pneumonia would be efficient when those pneumonias were associated with infection with influenza virus. Laboratory investigation had shown that the virus was not affected by sulfonamides or penicillin, but studies by Drs. Barry Wood and Carl Harford [70] demonstrated that sulfonamides would control pneumococcal infection in rats and mice in which pneumonia was produced by combined virus and bacteria. The clinical experience first

[70] Harford, C. G., Smith, M. R., and Wood, W. B., Jr.: Sulfonamide Chemotherapy of Combined Infection With Influenza Virus and Bacteria. J. Exper. Med. 83: 505–518, June 1946.

recorded by Eaton and Meiklejohn [71] during the 1943 epidemic developed also to establish the fact that the bacterial pneumonias encountered in influenza patients could be effectively treated with sulfonamides. The subsequent availability of penicillin enhanced the treatment, and a major threat was thus subject to control.

There still persisted a great possibility that *H. influenzae* might be seriously involved. For this reason, continuous studies were conducted on this organism, its prevalence, and its characteristics, by Dr. Hattie Alexander, and a diagnostic center was maintained.[72] No significant participation of that bacterium was encountered.

The idea that prophylaxis of influenza by use of intranasal spray with immune serum had developed. The concept was that antibody provided at the site of virus introduction might protect the susceptible epithelium of the respiratory tract. A series of studies in mice showed that it was possible; it was not effective in ferrets. Smorodintseff and his associates [73] had reported that influenza in mice could be prevented in this manner. In 1943, through the Commission on Influenza, a significant series of studies was conducted to determine whether human sera with high titers of antibody given by intranasal spray could prevent experimental infections with influenza virus. The studies were conclusive and indicated no prophylactic effect. The details are given in the history of the Commission on Influenza. The idea is, however, still of interest for further study.

The investigations of atypical pneumonia originally instituted through the Commission on Influenza gave rise to the Commission on Acute Respiratory Disease, and those on air sterilization were developed by the Commission on Air-Borne Infection. Details of other studies are also included in the histories of the respective Commissions.

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[71] See footnote 24 (3), p. 98.

[72] Alexander, H. E., Leidy, G., and MacPherson, C.: Production of Types A, B, C, D, E, and F *H. influenzae* Antibody for Diagnostic and Therapeutic Purposes. J. Immunol. 54: 207–211, October 1946.

[73] Smorodintseff, A. A., Gulmow, A. G., and Tschalkina, O. M.: Über die spezifische Propyhlaxe der epidemischen Grippe durch Inhalation antigrippösen Serums. Ztschr. f. klin. Med. 138: 756–765, 1940.

CHAPTER V

Measles

Joseph Stokes, Jr., M. D.

HISTORICAL NOTE [1]

In periods of mobilization, measles has always been a problem among the armed services. This was true of the Civil War, the Spanish-American War, the Philippine Insurrection, and World War I. Recruit depots were recognized as the posts where the incidence of measles rose rapidly and accounted for relatively high morbidity rates.

In World War I, total Army admission and death rates, respectively, were 23.79 and 0.57 per 1,000 average strength per year among the total of the average annual strengths of approximately 4 million men, with a daily average noneffective ratio of 1.25 per 1,000 average strength. More than eight-tenths of the primary admissions were among troops serving in the United States and in Alaska. Of an average annual mean strength of over 2 million enlisted men in the United States, the average annual admission rate was about 40 with a death rate of 0.94, and a daily average noneffective ratio of 2.07 per 1,000 average strength.

Among 93,629 primary admissions of enlisted men in the United States and Europe during World War I, there were 22,809 complications, the most common of which were pneumonia, otitis media, and mumps. Other important complications were suppurative pleurisy, mastoiditis, and scarlet fever. Extensive studies were made of bacterial flora in a number of severe epidemics in Army posts. The hemolytic streptococcus was found more frequently than any other bacterium as the apparent etiologic factor in these complications.

REDUCTION OF THE MILITARY PROBLEM IN WORLD WAR II

Measles as a military problem changed to a great degree between World Wars I and II, apparently as a result of two major factors: (1) Reduction in the total number of susceptibles of draft age as a result of sociological changes from 1919 to 1940 which helped to decrease the percentage of United States population that is both rural and isolated; and (2) lessening severity of the disease

[1] The Medical Department of the United States Army in the World War. Communicable and Other Diseases. Washington: U. S. Government Printing Office, 1928, vol. IX, pp. 411, 414, 431.

since 1936 as a result of sulfonamides and antibiotic agents, which have become increasingly useful in control of bacterial complications of measles.

Sociological changes.—The first factor is difficult to measure completely, although the decrease in percentage of rural population is obvious. It is well known that rural men of draft age in general have a higher percentage of susceptibles than urban draftees of the same age range, as indicated by experience in World War I. Such factors as the family automobile and the bus have greatly decreased isolation of the rural family, yet it is difficult to determine how much these changes have accomplished in lowering the age at which measles occurs.

Sulfonamides and antibiotics.—The second major factor, that of control of secondary bacterial complications, plays no part in decreasing actual incidence of measles but has a large role in reducing severity of measles and in thus diminishing its importance as a military problem. It has been realized for some time that such organisms as hemolytic streptococcus, pneumococcus, and staphylococcus had considerable effect on severity of the disease in World War I, and use of sulfonamides and antibiotics demonstrated more clearly the significance of those bacteria in relation to bronchopneumonia, otitis media, and other complications of measles.

Human-serum antibodies.—Also of increasing importance since World War I in control of sharp localized outbreaks of the disease, particularly as to its severity, was the availability of human-serum antibodies against measles in convenient and concentrated amounts which caused practically no discomfort and a rare local reaction in recipients. The first advance from the original use of convalescent serum by Nicolle and Conseil [2] in 1918 and of adult serum by Reitschel [3] in 1921, was the production of a concentrated placental gamma globulin by McKhann and Chu [4] in 1933. Later, serum produced from fractionation methods by Cohn and his associates [5] in 1944 proved to be even more satisfactory, causing fewer reactions than placental gamma globulin. Now gamma globulin from pools of plasma may be considered a standard preparation for use intramuscularly, but not intravenously, in measles prophylaxis. Certain studies have been conducted to determine its value in therapy conducted in the early prodromal phase, but additional research is required.

Immunization.—Studies on methods of immunization were made by several researchers following World War I. Plotz [6] in 1938 investigated the cultivation of measles virus on tissue culture, and Rake and Shaffer [7] in 1939 made a similar investigation of the virus in the chorio-allantois of embryonated

[2] Nicolle, C., and Conseil, E.: Pouvoir préventif du sérum d'un malade convalescent de rougeole. Bull. et mém. Soc. méd. d. hôp. de Paris 42: 336–338, 1918.

[3] Reitschel, P.: Zur Masernprophylaxe nach Degkewitz. Ztschr. f. Kinderh. 29: 127–132, June 1921.

[4] McKhann, C. F., and Chu, F. T.: Use of Placental Extract in Prevention and Modification of Measles. Am. J. Dis. Child. 45: 475–479, March 1933.

[5] Cohn, E. J., Oncley, J. L., Strong, L. E., Hughes, W. L., Jr., and Armstrong, S. H., Jr.: Chemical, Clinical, and Immunological Studies on the Products of Human Plasma Fractionation. I. The Characterization of the Protein Fractions of Human Plasma. J. Clin. Investigation 23: 417–432, July 1944.

[6] Plotz, H.: Culture "in vitro" du Virus de la Rougeole. Bull. Acad. de méd., Paris 119: 598–601, May 1938.

[7] Rake, G., and Shaffer, M. F.: Propagation of the Agent of Measles in the Fertile Hen's Egg. Nature, London 144: 672–673, October 1939.

hen's egg. It was shown by Shaffer and his associates [8] in 1941 that virus grown on the chick chorio-allantois sac, following several passages, will produce mild measles in *Macaca mulatta* monkeys and also in susceptible human beings. Monkeys and human beings thus infected demonstrated resistance to measles virus; the former apparently demonstrated a greater resistance than the latter. Later chorio-allantoic passages of virus produced fewer symptoms in man, but resistance appeared to be less than with earlier passage material. Difficulty in obtaining continued growth with fresh infective material on the chorio-allantois and in developing adequate serologic tests for susceptibility and resistance to measles has hampered further studies on possible production of active immunity in man by use of such passage material. Siim, in Copenhagen, has been able to repeat cultivation of the measles virus on the chorio-allantois of embryonated hen's egg and has also been able to produce measles of a similar mild nature with Koplik spots in monkeys and human beings. With continued passage on the chorio-allantois, the Siim virus also decreased in virulence and activity.

Difficulties inherent in such studies were chiefly responsible for emphasis in World War II upon developing means of passive immunization for any sharp outbreaks in the armed services, in case an emergency occurred.

Gamma globulin.—Storage of convalescent measles plasma in a dried state was not considered practical for control of acute outbreaks of measles. Gamma globulin was available as a byproduct of fractionation of pooled Red Cross plasma for obtaining concentrated albumin for treatment of shock. Such gamma globulin was first tested by Enders [9] for antibodies against many viral and bacterial diseases, such as influenza A and B antibodies and diphtheria antitoxin. It was found to contain these antibodies in a stable form which was concentrated to approximately 25 times the amount present in original pools of Red Cross plasma.

Following the original suggestion by Col. Elliott S. A. Robinson, MC, Chief, Laboratories Division, Preventive Medicine Service, Office of the Surgeon General, that gamma globulin be tested for its effect against measles, studies on prophylaxis were started. One study was conducted in Philadelphia by members of the Commission on Measles and Mumps, Army Epidemiological Board.[10] Another study was conducted in Boston by Dr. Charles Janeway and his coworkers.[11] These studies were made to determine the efficacy of gamma globulin in prevention, attenuation, and treatment of

[8] Shaffer, M. F., Rake, G., Stokes, J., Jr., and O'Neil, G. C.: Studies on Measles. II. Experimental Disease in Man and Monkey. J. Immunol. 41: 241-257, June 1941.

[9] Enders, J. F.: Chemical, Clinical, and Immunological Studies on the Products of Human Plasma Fractionation. X. The Concentrations of Certain Antibodies in Globulin Fractions Derived from Human Plasma. J. Clin. Investigation 23: 510-530, July 1944.

[10] Stokes, J., Jr., Maris, E. P., and Gellis, S. S.: Chemical, Clinical, and Immunological Studies on the Products of Human Plasma Fractionation. XI. The Use of Concentrated Normal Human Serum Gamma Globulin (Human Immune Serum Globulin) in the Prophylaxis and Treatment of Measles. J. Clin. Investigation 23: 531-540, July 1944.

[11] Ordman, C. W., Jennings, C. G., Jr., and Janeway, C. A.: Chemical, Clinical, and Immunological Studies on the Products of Human Plasma Fractionation. XII. The Use of Concentrated Normal Human Serum Gamma Globulin (Human Immune Serum Globulin) in the Prevention and Attenuation of Measles. J. Clin. Investigation 23: 541-549, July 1944.

measles in suitable groups of children in homes and hospitals. Conclusions fully established the value of gamma globulin in prevention and attenuation of measles. Some suggestion was also obtained in the Philadelphia studies that treatment of measles with large doses during the prodromal stages might result in modification of the disease, but findings were not sufficiently extensive to be conclusive. The very large dosage used for treatment, since it must be injected intramuscularly, caused such discomfort that it was impractical for routine use in either children or adults, unless the condition of exposed susceptibles warranted strenuous efforts for attenuation of measles.

Studies centering in Boston and Philadelphia also established that, while pools of Red Cross plasma from which the gamma globulin originated often produced serum hepatitis in a considerable percentage of the recipients, gamma globulin itself did not cause such hepatitis. In some cases, this may have been because of the infrequent presence of antibodies in the serum hepatitis virus. It appears more probable, however, from further studies conducted in Boston, that the virus may have an affinity for Fraction I of fractionated plasma rather than for Fraction II, since Fraction I is known to harbor the virus when it is present in plasma pools from which the fractions are obtained. It is also possible that the more rigorous handling of the gamma globulin fraction, as compared with other fractions, may inactivate the virus.

However, in view of safety, ready availability, small dosage resulting from concentration of antibodies, stability of antibodies, and lack of local or general reaction of gamma globulin, it appeared to be excellent material for storage in areas such as ports of embarkation in case emergency protection against measles was required. A number of emergencies which were not recorded are known to have existed during World War II. Two instances were reported, however. One occurred at an Army camp. Gamma globulin proved to be completely protective when injected in a dose of 10 ml., into each of 610 enlisted men who were exposed to measles.[12] In a similar situation, men in an Air Force unit, following exposure to measles, were injected just before embarkation and were completely protected by 5 ml. of gamma globulin.[13]

During the war, several thousand injections of gamma globulin in exposed susceptible children were administered by members of the Commission on Measles and Mumps, Army Epidemiological Board, for the purpose of determining proper dosage for attenuation and protection of personnel in the armed services. From a larger original dose of about .025 to .03 ml. per pound of

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[12] Letter, Col. T. G. Torsey, MC, Army Service Forces, New York Port of Embarkation, Camp Kilmer, N. J., to The Surgeon General, 10 June 1944, subject: Immune Serum Globulin for Prevention of Measles.

[13] Army Air Forces, Headquarters, I Troop Carrier Command, Stout Field, Ind., Preventive Medicine Bulletin No. 7, 20 Apr. 1945.

body weight for attenuation of measles, the dose has been reduced to about .02 ml. per pound of body weight. Approximately four times this amount would be fully protective in most instances, although there is considerable variation in susceptible individuals. The use of ultraviolet light to prevent cross-infection in measles was a method not fully explored during World War II.

INCIDENCE OF MEASLES IN WORLD WAR II

In World War II, remarkable reduction in the military problem posed by measles is well illustrated by available statistical data. As was expected when induction started, morbidity from measles among Army personnel in the United States rose considerably, apparently as a result of an influx of susceptible men from states with large rural populations. Also, as was expected, when a relatively small number of men were inducted in 1944, the incidence rate dropped considerably below that of the preceding 4 years. Incidence rates in the 4 years 1941–45 were 9.8 (enlisted men only and including Alaska), 4.5, 5.7, 2.7, and 0.9, respectively, as compared with an average during 1930–39 of 3.3 per 1,000. The morbidity rate for 1941 of 9.8 should be compared with the morbidity rate of 85.2 in 1917 inasmuch as both rates are for enlisted men only. The highest incidence rates for measles during the 3-year period, 1942–44, were reported by the Seventh Service Command. During this period, rates were lowest in the First and Second Service Commands.

The case fatality rate for measles in World War II was approximately 40 times less than the rate for World War I, which was 2.4 per 100 admissions. Remarkable change in the severity of measles apparently resulted from three causes: (1) Better control of secondary bacterial invaders in measles itself, (2) better control of bacterial components in patients with respiratory infection who were exposed to measles, and (3) reduction by chemotherapy of the number of respiratory infectious cases who would otherwise have been in hospital or barracks adding their bacterial population to already overpopulated areas.

Decrease in case fatality rates can best be explained by the control of pathogenic bacteria, although it is also probable that absence of pandemic influenza in World War II played some part in this reduction.

Table 24 shows annual incidence rates per 1,000 average strength for morbidity from measles, mumps, and scarlet fever from 1930 to 1945, inclusive, for the Army in the continental United States.

Measles must still be regarded as a difficult problem for induction centers but probably need no longer be considered as a major military problem in the field of infectious diseases.

TABLE 24.—*Incidence rates for measles, mumps, and scarlet fever in the Army in the continental United States, 1930–45*

[Rate expressed as number of cases per annum per 1,000 average strength]

Year [1]	Measles	Mumps	Scarlet fever
1930	4.7	2.8	0.4
1931	2.3	2.4	.3
1932	.7	2.3	.3
1933	2.8	2.0	.4
1934	7.0	2.6	.5
1935	3.3	3.8	.8
1936	4.5	16.5	2.3
1937	2.1	11.1	1.2
1938	4.5	2.3	.4
1939	1.4	2.4	.3
1940	3.7	3.9	.4
1941	9.8	7.3	2.0
1942	4.5	5.7	1.1
1943	5.7	6.9	2.4
1944	2.7	4.7	1.9
1945	.9	4.4	1.0

[1] Data for 1930–41 are for enlisted men only.

CHAPTER VI

Mumps

Joseph Stokes, Jr., M. D.

HISTORICAL NOTE [1]

Mumps remains a disease of the greatest importance in military medicine. In all wars, including World War II, periods of mobilization have been marked by sharp increases in incidence rates of this disease.

In World War I, the total number of cases admitted to hospital and quarters for mumps was 230,356, an annual rate of 55.80 per 1,000 average strength. Except for influenza and gonococcal infection, the incidence was higher for mumps than for any other communicable disease. The rates were considerably higher among Negro than among white enlisted men, being 134.75 (total admissions 38,619) and 49.99 (total admissions 179,948), respectively. The higher rates for Negroes can probably be accounted for by the rural origin of many of the Negro enlisted men in that war.

Although a few enlisted men were discharged from the service on account of disability, the reasons for such discharges presumably were for conditions other than mumps itself (for example, sequelae).

During World War I, the Army lost 3,884,147 man-days from duty, a figure which gives a noneffective rate of 2.58 per 1,000 strength—a noneffectiveness which was third on the list caused by important diseases. In this respect, the figures were also considerably higher (about three times) for Negro than for white enlisted men, the rates being 6.28 and 2.30 per 1,000 average strength, respectively. The disease was widespread throughout all Army posts in the United States, with particularly severe outbreaks in Camp Beauregard, La., Camp Wheeler, Ga., and Camps Bowie and Travis, Tex., where the admission rates were all over 205 per annum per 1,000 average strength. In 1918 and 1919, there was a marked seasonal incidence, with peaks in January and February of both years. Overseas, in the American Expeditionary Forces, the admission rate for Negroes continued in a ratio of about 3:1, and the total rate for all enlisted men in Europe was 50.93 as compared with a rate of 81.40 for enlisted men in specified camps in the United States. Undoubtedly, the greater number of unseasoned recruits in the Army in the continental United States was responsible for this higher admission rate.

[1] (1) The Medical Department of the United States Army in the World War. Statistics. Washington: Government Printing Office, 1925, vol. XV, pp. 86, 110. (2) The Medical Department of the United States Army in the World War. Communicable and Other Diseases. Washington: U. S. Government Printing Office, 1928, vol. IX, pp. 451–462.

MILITARY PROBLEM OF MUMPS

In all wars, including World War II, the primary problem presented by mumps has been the length of time lost from duty following the complication of orchitis, which occurs in about 30 percent of all cases. Moreover, the temporary disability caused by orchitis is frequently increased by psychological distress. In addition, the fact that mumps has a relatively long incubation period and a slower mode of transmission than measles and chickenpox prolongs the outbreaks, causes uncertainty and inconvenience, and, therefore, seriously interferes with active-duty performance.

RESEARCH DEVELOPMENTS

The concept of mumps held during World War I has been considerably revised as the result of experimental studies starting with the transmission of mumps by filtered (Berkefeld V and N filters) salivary secretions to the *Macacus rhesus* monkey by Johnson and Goodpasture [2] in 1934 and of mumps virus to the chick amnoitic sac by Habel [3] and by Levens and Enders [4] in 1945. The work in the monkey and chick embryo—particularly in the latter—led quickly to the following important advances which occurred just before and during World War II:

1. The development of a skin test for susceptibility both from the monkey's infected parotid gland and from the infected amniotic fluid of the chick embryo.[5]
2. The development of complement fixation tests.[6]
3. The development of hemagglutination studies.[7]
4. The demonstration of the frequent presence of mumps meningoencephalitis, and at times orchitis, without parotitis or other noticeable glandular reaction.[8]
5. The production of apparently solid immunity under field conditions as the result of reaction to mumps virus irrespective of severity of symptoms, size of glands, or whether single or multiple glands are involved.
6. The demonstration that about 40 percent of cases of mumps are

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[2] Johnson, C. D., and Goodpasture, E. W.: An Investigation of the Etiology of Mumps. J. Exper. Med. 59: 1-19, January 1934.

[3] Habel, K.: Cultivation of Mumps Virus in the Developing Chick Embryo and Its Application to Studies of Immunity to Mumps in Man. Pub. Health Rep. 60: 201-212, 23 Feb. 1945.

[4] Levens, J. H., and Enders, J. F.: The Hemoagglutinative Properties of Amniotic Fluid From Embryonated Eggs Infected With Mumps Virus. Science 102: 117-120, 3 Aug. 1945.

[5] (1) Enders, J. F.: Observations on Immunity in Mumps. Ann. Int. Med. 18: 1015-1019, June 1943. (2) Enders, J. F., Kane, L. W., Maris, E. P., and Stokes, J., Jr.: Immunity in Mumps. V. The Correlation of the Presence of Dermal Hypersensitivity and Resistance to Mumps. J. Exper. Med. 84: 341-364, October 1946.

[6] (1) Enders, J. F., and Cooney, S.: Detection of Antibody by Complement Fixation in Sera of Man and Monkey Convalescent From Mumps. Proc. Soc. Exper. Biol. & Med. 50: 180-184, May 1942. (2) Henle, G., Henle, W., and Harris, S.: The Serological Differentiation of Mumps Complement-Fixation Antigens. Proc. Soc. Exper. Biol. & Med. 64: 290-295, March 1947.

[7] See footnote 4.

[8] Kane, L. W., and Enders, J. F.: Immunity in Mumps. III. The Complement Fixation Test as an Aid in the Diagnosis of Mumps Meningoencephalitis. J. Exper. Med. 81: 137-150, January 1945.

clinically inapparent but, nevertheless, both transmit the virus to susceptible contacts and become solidly immune just as readily as the apparent cases.[9]

7. The demonstration of the period of infectivity of human saliva from about 48 hours before the onset of the symptoms to about 6 or 8 days after their onset.[10] These advances have permitted a fairly clear understanding of the etiology and epidemiology of the disease.

In addition, methods of active immunization [11] of increasing effectiveness have been developing and, while not as yet suitable for wide-scale use in the armed services, may soon become so if the early results are widely confirmed.

EXPERIENCE DURING WORLD WAR II

In World War II, as in World War I, large epidemics in young adult males differed considerably from epidemics in children before puberty. In epidemics in children, approximately 75 percent of all cases of clinical mumps (omitting inapparent mumps) belong to the full-blown type without complications. In contrast, epidemics in the United States Army may be divided roughly into three groups of approximately equal size with the following distinguishable characteristics: (1) A short course of the disease with signs and symptoms which are insignificant, (2) full-blown disease with marked swelling of the salivary glands but no complications, and (3) severe disease with the complications of epididymo-orchitis or meningoencephalitis, or both.[12] Table 25 shows incidence of mumps in the United States Army during World War II.

TABLE 25.—*Incidence of mumps in the U. S. Army, 1942-45*

[Rate expressed as number of cases per annum per 1,000 average strength]

Year	Total Army		United States		Overseas	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
1942	17, 983	5. 55	15, 264	5. 74	[1] 2, 719	4. 64
1943	40, 091	5. 84	35, 975	6. 94	[1] 4, 116	2. 43
1944	26, 396	3. 39	18, 456	4. 65	[1] 7, 940	2. 08
1945	18, 585	2. 45	12, 750	4. 35	[1] 5, 835	1. 26

[1] Data include admissions on transports.

[9] (1) Maris, E. P., Enders, J. F., Stokes, J., Jr., and Kane, L. W.: Immunity in Mumps. IV. The Correlation of the Presence of Complement-Fixing Antibody and Resistance to Mumps in Human Beings. J. Exper. Med. 84: 323-339, October 1946. (2) See footnote 5 (2), p. 136.

[10] Henle, G., Henle, W., Wendell, K. K., and Rosenberg, P.: Isolation of Mumps Virus From Human Beings With Induced Apparent or Inapparent Infections. J. Exper. Med. 88: 223-232, August 1948.

[11] (1) Stokes, J., Jr., Enders, J. F., Maris, E. P., and Kane, L. W.: Immunity in Mumps. VI. Experiments on the Vaccination of Human Beings With Formolized Mumps Virus. J. Exper. Med. 84: 407-428, November 1946. (2) Enders, J. F., Levens, J. H., Stokes, J., Jr., Maris, E. P., and Berenberg, W.: Attenuation of Virulence With Retention of Antigenicity of Mumps Virus After Passage in the Embryonated Egg. J. Immunol. 54: 283-291, November 1946.

[12] McGuinness, A. C., and Gall, E. A.: Mumps at Army Camps in 1943. War Med. 5: 95-104, February 1944.

As in World War I, the spread of mumps through Army posts was always comparatively slow in contrast with that of such diseases as epidemic influenza. This may be illustrated by a study conducted in 1944 under the auspices of the Commission of Measles and Mumps at Camp McCoy, Wis., where the peak of the epidemic (1,378 cases in all) was not reached until its 17th week, while the admission rate did not drop below 30 per week until the 26th week of the outbreak. The spread of mumps over this epidemic period is shown in chart 17. Chart 18 shows the spread of mumps in cases admitted from a single company over a 17-week period—a characteristically slow spread even among men who are closely and continuously in contact to each other. It has been uncommon for more than about 10 percent of the men in one company to develop mumps in a single respiratory season.

The Camp McCoy study, which included additional figures from several Army posts also shows the relationship between the day of onset of orchitis

CHART 17.—*Spread of mumps in 1,378 cases admitted to the station hospital at Camp McCoy, Wis., over a 31-week period*

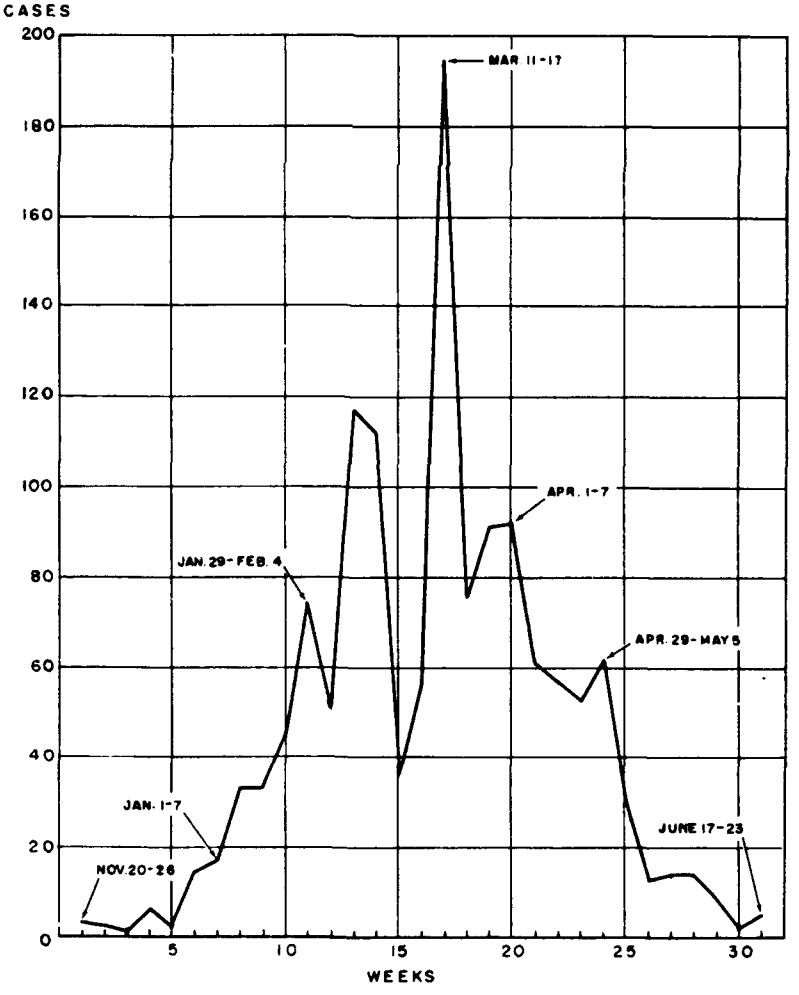
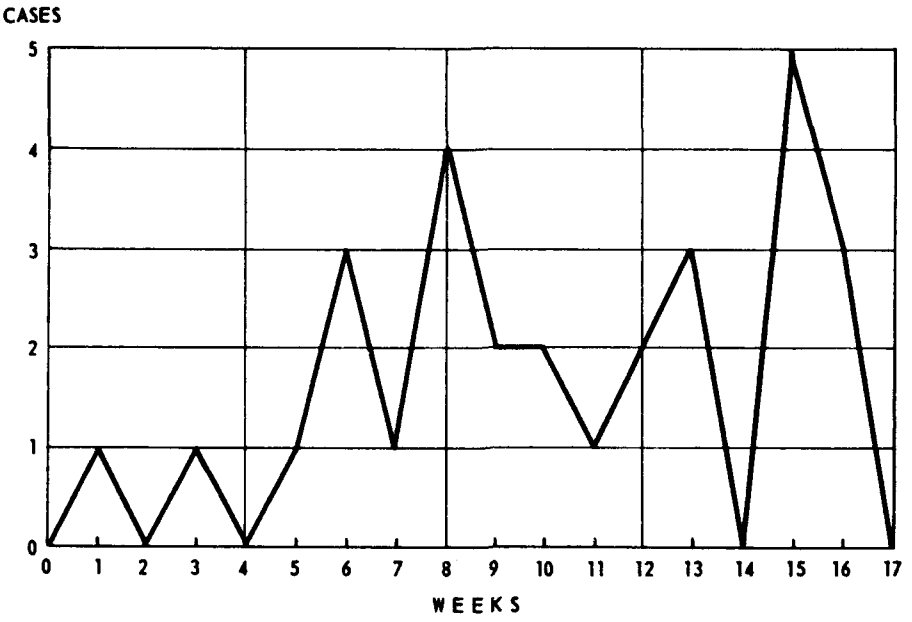


CHART 18.—*Spread of mumps in 29 cases from one company admitted to the station hospital at Camp McCoy, Wis., over a 17-week period*



and the day of onset of the preceding parotitis as well as the day of onset of meningoencephalitis following parotitis.

In severe cases of epididymo-orchitis, the operative procedure of orchidotomy, as first suggested by Smith [13] and later fully confirmed in its value by Wesselhoeft and Vose,[14] was successfully performed at Camp McCoy. The incision of the firm encapsulating tunica albuginea may well have prevented sterilization of one or both testicles when this complication was particularly severe and may be now considered as a well-recognized surgical procedure in this disease. The incidence of bilateral epididymo-orchitis in those who had this complication was 10.6 percent so that orchidotomy was useful generally for relief of pain rather than to prevent complete sterilization.

Because of the high incidence (approximating 30 percent) of this complication in widespread outbreaks on Army posts, it seemed appropriate for the Commission on Measles and Mumps to determine the possible protective value of ordinary immune serum globulin (gamma globulin) as prepared by the method of Cohn, Oncley, and others [15] against epididymo-orchitis after parotitis had started. This study was carried out at Fort Benning, Ga., by

[13] Smith, G. G.: Two Cases of Orchitis Due to Mumps Treated by Operation. Boston M. & S. J. 167: 323-325, September 1912.

[14] Wesselhoeft, C., and Vose, S. N.: Surgical Treatment of Severe Orchitis in Mumps. New England J. Med. 227: 277-280, 20 Aug. 1942.

[15] Cohn, E. J., Oncley, J. L., Strong, L. E., Hughes, W. L., Jr., and Armstrong, S. H., Jr.: Chemical, Clinical, and Immunological Studies on the Products of Human Plasma Fractionation. I. The Characterization of the Protein Fractions of Human Plasma. J. Clin. Investigation 23: 417-432, July 1944.

Gellis, McGuinness, and Peters.[16] Within the first 24 hours of onset, 50 ml. of the globulin were injected intramuscularly in alternate admissions for mumps—67 men being injected, while 67 remained as controls. The incidence of orchitis in those injected was 20.9 percent, while in the controls it was 26.8 percent. This could not be considered a significant difference.

Gamma globulin was then obtained from the fractionation of convalescent mumps serum and injected in doses of 20 ml. into alternate cases as previously indicated—51 being injected within the first 24 hours after onset, while 51 remained as controls. The incidence of orchitis in the injected group was significently lowered, apparently by the convalescent globulin, being 7.8 percent as compared with an incidence of 27.4 percent in the controls. In view of the fact that a figure of 7.8 percent was an exceptionally low figure for orchitis in mumps in any extensive Army outbreak, and inasmuch as the incidence in the control group corresponded well with the usual figure found in such outbreaks—approximately one-third of all cases of mumps in young adult males—the study proved to be highly suggestive. Thus, the gamma globulin from convalescent serum may well be worthy of further trial when mumps is active in recruitment centers or Army posts.

Annual rates for mumps in the continental United States were strikingly higher in World War I than in World War II. In 1918, the rate reached a high point of 75.5; the high point for the World War II period, on the other hand, was only 6.9, reached in 1943. The difference in rate has been regarded with apparent justification as evidence of the greater opportunities of rural youth for urban contact and of the actual decrease in the rural population during the intervening period between the two wars. The case fatality ratio for mumps was negligible—rarely was a death attributable to meningoencephalitis.

The Alaskan Department had the highest rate for mumps during the war period; this could be accounted for by the unusual amount of contact in inclosed places because of prolonged cold weather. On the other hand, in the Mediterranean theater, where the rates for most communicable diseases were high, the rate for mumps was inexplicably low. Thus, mumps in World War II continued to be a disease which was not completely predictable.

There continued to be certain Army posts in which commanding officers imposed working quarantine in attempts to control the spread of mumps. Aside from interfering with training activities and with the morale of the men, it had little effect.

[16] Gellis, S. S., McGuinness, A. C., and Peters, M.: A Study on the Prevention of Mumps Orchitis by Gamma Globulin. Am. J. M. Sc. 210: 661-664, November 1945.

CHAPTER VII

Psittacosis

Joseph E. Smadel, M. D.

A single case of psittacosis was recorded among United States Army personnel during World War II. The history of this case is of sufficient interest to warrant a summary.

Case report.—A 51-year-old white officer was admitted to the Station Hospital, Port of Embarkation, San Francisco, Calif., on 3 March 1944. His complaints on admission were vomiting and diarrhea followed by dizziness and headache of about 3 days' duration, also a temperature of 104° F.

On physical examination, the patient had tenderness deep in the right upper quadrant of the abdomen and an unsteady gait associated with a horizontal nystagmus. Lumbar puncture and stool cultures were negative; the urine showed moderate albuminuria with granular casts and a few white cells in the sediment. The white cell count was 5,450. Agglutinations for typhoid were positive at dilutions of 1:640 and at 1:20 for paratyphoid. A chest roentgenogram revealed an increase in confluent linear density extending from the right hilum to the right lower lung field, interpreted as due to an inflammatory process such as an atypical pneumonia.

On 7 March, the patient was transferred to Letterman General Hospital, San Francisco. Physical examination disclosed him to be acutely ill with moderate dyspnea and slight cyanosis. There was marked abdominal distention and dehydration. Auscultation of the lungs showed subcrepitant and crepitant rales involving the entire right lower lobe and, to a lesser extent, the left lower lobe. The liver extended 4 cm. below the right costal margin.

Laboratory studies were as follows: Red blood count, 4.3 million; hemoglobin, 83 percent; white blood count, 6,700 with 57 percent neutrophils, 30 percent lymphocytes, 1 percent monocytes, 1 percent eosinophils, and 1 percent basophils. Urine showed 3 plus albuminuria, negative sugar, few fine and coarse granular casts, 5 to 7 white blood cells, and 1 to 3 red blood cells over high-power fields. Agglutinations for typhoid and paratyphoid were nondiagnostic; for tularemia and brucellosis, negative. Stool examination and culture were not significant. Repeat leukocyte counts showed a persistently normal white blood count ranging from 5,450 to 12,100, the latter obtained on the day prior to death. Urinalyses remained essentially the same. Blood urea nitrogen and chlorides were normal. Total proteins were 4.35 gm. per 100 cc., with 2.35 gm. albumin and 1.30 gm. globulin per 100 cc. Cold agglutinins were negative. A roentgenogram of the chest showed an increase in the confluent density in the right lower lobe, and flat plate of the abdomen revealed a considerable amount of gaseous distention of the large and small intestines.

Despite intensive therapy with blood transfusion, parenteral fluids, and supportive measures to combat the distention and dyspnea, the patient's course continued febrile and he died on 14 March.

At autopsy, a definite patchy pneumonitis was found involving the left upper and lower lobes, and the right middle and lower lobes. On microscopic

section, there was a diffuse but patchy pneumonic involvement characterized mainly by large mononuclear leukocytes filling the distended alveoli with active phagocytosis of nuclear particles. Different stages of pneumonia were seen with some areas showing early organization. Lung smears were examined for elementary bodies, but no definitely diagnostic elementary bodies could be seen, though a few macrophages contained small granular objects which stained red. The gastrointestinal tract showed a severe acute gastroenteritis involving the stomach, duodenum, jejunum, ileum, and colon.

Lung tissue obtained at autopsy was examined by Dr. Gordon Meiklejohn at the University of California, Berkeley, with the following report:

> A 20 percent suspension of lung tissue in broth was inoculated intranasally in 0.45 cc. amounts into 2 adult western cotton rats under ether anesthesia. On the second day, they began to appear sick and dyspneic and by the fourth day were moribund and were sacrificed. Both showed extensive pulmonary consolidation, greyish in color. Impression smears of the lungs stained by the method of Macchiavello showed very large numbers of intracellular elementary bodies, frequently in unusually large clusters. The control cotton rats from the same stock inoculated at the same time with 0.45 cc. of broth showed no evidence of disease.
>
> Six white mice were inoculated intracerebrally with 0.03 cc. of the same suspension. One died on the second day. The remaining 5 died between the fourth and sixth day and smears showed rather small numbers of elementary bodies.
>
> Serum taken on the day of death was tested against lymphogranuloma venereum antigen for complement fixation with the following results: 1 to 6, 4 plus; 1 to 12, 3 plus; 1 to 24, 1 plus; 1 to 48, 0; New York strain, 0; serum control, 0. The same serum had a cold agglutination titer of less than 10.

Dr. K. F. Meyer also recovered a psittacosis-type virus from another portion of the lung tissue obtained at autopsy. These results suggested that the patient's pneumonia was due to a virus in the psittacosis-like group.[1]

Complement fixation tests were done at the Letterman General Hospital on blood obtained shortly before death. The results were:

> Complement fixation with lygranum antigen: 1 to 5, 4 plus; 1 to 10, 5 plus; 1 to 20, 4 plus; 1 to 40, 3 plus; 1 to 80, 1 plus; 1 to 160, 0; complement fixation with psittacosis antigen (Plotz antigen, Army Medical School No. 6, dated 4 May 1943): 1 to 5, 4 plus; 1 to 10, 4 plus; 1 to 20, 4 plus; 1 to 40, 4 plus; 1 to 80, 0; 1 to 160, 0.

Epidemiologic studies revealed that this officer was a transport commander on the *John Lykes* which had left New York on 20 December 1943, and had arrived at San Francisco on 15 February 1944, after a 6-day stopover from 20 to 26 January at Noumea, New Caledonia. As far as was known, he had had no contact with birds, although it was quite common for members of the crew to bring canaries onto the ship to be carried back to the United States for pets. The deceased officer had lived aboard ship until the time of his hospital admission and was not known to have visited any place in San Francisco harboring parrots or other birds. The question was raised as to whether the ship had ever been used for transportation of birds with a view to determining whether

[1] Letter, Dr. K. F. Meyer to Editorial Office, History of Preventive Medicine, U. S. Army, in World War II, 6 Aug. 1951.

possible dust from bird droppings might have been a source of the infection, but no information could be obtained. As far as is known, no other crew members of the *John Lykes* had become ill with a similar illness.

EPIZOOTICS

Epizootics caused by members of the psittacosis group of viruses occurred in carrier pigeons used by the Signal Corps [2] and were of some importance in the war effort. During the period of military mobilization, when primary atypical pneumonia was a matter of considerable concern to the Medical Corps, the possibility that this disease might be caused by one of the psittacosis agents was carefully investigated.[3] No evidence of such an etiologic relationship was established then or in subsequent studies on this disease which continued to plague the troops in the United States and overseas, especially in the European Theater of Operations.

Observations on primary atypical pneumonia among civilians in the New York metropolitan area during 1941 had shown that an appreciable proportion of these patients actually had psittacosis and had apparently contracted their disease as a result of association with flocks of pigeons in which epizootic psittacosis was present.[4] Since enzootic psittacosis exists in practically all flocks of pigeons examined to date [5] and since the disease flares to epizootic stages from time to time, it appeared desirable to study the relation of the avian to the human disease among birds and pigeoneers of the Signal Corps.

The 2d Platoon, 280 Signal Pigeon Company (mission to train and supply carrier pigeons) stationed at Tidworth, England, was under almost constant observation by the Virus Division of the 1st Medical General Laboratory from January 1943 to March 1945. During this period, there were a series of sharp epizootics of psittacosis among the flocks of the company,[6] but no typical or atypical cases of psittacosis occurred among the personnel of the company, despite the fact that many members of the organization were in intimate contact with the sick birds.

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[2] Smadel, J. E., Jackson, E. B., and Harman, J. W.: A New Virus Disease of Pigeons: Recovery of the Virus. J. Exper. Med. 81: 385-398, April 1945.

[3] Dingle, J. H., Abernethy, T. J., Badger, G. F., Buddingh, G. J., Feller, A. E., Langmuir, A. D., Ruegsegger, J. M., and Wood, W. B.: Primary Atypical Pneumonia, Etiology Unknown. War Med. 3: 223-248, March 1943.

[4] Smadel, J. E.: Atypical Pneumonia and Psittacosis. J. Clin. Investigation 22: 57-65, January 1943.

[5] (1) Coles, J. D. W. A.: Psittacosis in Domestic Pigeons. Onderstepoort J. Vet. Science & Animal Industry 15 (1 and 2): 141-148, July and October 1940. (2) Eddie, B., and Francis, T., Jr.: Occurrence of Psittacosis-like Infection in Domestic and Game Birds of Michigan. Proc. Soc. Exper. Biol. & Med. 50: 291-295, June 1942. (3) Meyer, K. F., Eddie, B., and Yanamura, H. Y.: Ornithosis (Psittacosis) in Pigeons and Its Relation to Human Pneumonitis. Proc. Soc. Exper. Biol. & Med. 49: 609-615, April 1942. (4) Blount, W. P.: Psittacosis in Pigeons; A Disease Communicable to Man: An Outbreak in Army Pigeons. J. Roy. Army Vet. Corps 15: 81-83, August 1944; and Canad. J. Comp. Med. 8: 260-264, September 1944. (5) Zichis, J., Shaughnessy, H. J., and Lemke, C.: Isolation of Psittacosis-like Viruses From Chicago Pigeons. J. Bact. 51: 616-617, May 1946. (6) Davis, D. J., and Ewing, C. L.: Recovery of Ornithosis Virus From Pigeons in Baltimore, Maryland. Pub. Health Rep. 62: 1484-1488, 10 Oct. 1947. (7) Labzoffsky, N. A.: Ornithosis Among "Wild" Pigeons in Ontario. Canad. J. Pub. Health 38: 187-192, April 1947. (8) Winsser, J.: Isolation of Ornithosis From Pigeons in The Netherlands. Antonie van Leeuwenhoek 15 (2): 86-90, 1949.

[6] (1) Annual Reports, 1st Medical General Laboratory, 1944, 1945. (2) See footnote 2.

Two observations contributed to an explanation of the absence of clinical disease among the pigeoneers. In the first place, most of the technical personnel responsible for the care and training of the birds had been pigeon fanciers in civilian life, hence they had had ample opportunity for prior exposure to the pigeon strain of psittacosis virus. In the second place, evidence that subclinical infection had taken place in the personnel of this company was forthcoming when it was shown that the sera of approximately one-third of the men possessed complement-fixing antibodies against the psittacosis-lymphogranuloma venereum group of viruses. In interpreting these serologic data, several points should be borne in mind: (1) The test fails to differentiate between antibodies elicited by psittacosis virus and the closely related virus of lymphogranuloma venereum; (2) antibodies against the psittacosis-lymphogranuloma venereum group persist in low titer for long periods of time; (3) the prevalence of a stationary low titer of these antibodies in an unselected small group of Negro troops with atypical pneumonia at Camp Claiborne, La., in 1941 was 46 percent, whereas the prevalence in white troops with the same disease at the same post was 4 percent;[7] and (4) personnel of the 280th Signal Pigeon Company were all white. With these points in mind, it seemed reasonable to assume that the majority of the pigeoneers with complement-fixing antibodies against the psittacosis-lymphogranuloma venereum group of viruses had had previous exposure to psittacosis virus and had suffered a subclinical infection or unrecognized disease. On the other hand, it was assumed that the high prevalence of psittacosis-lymphogranuloma venereum antibodies in Negro troops at Camp Claiborne was the result of exposure to lymphogranuloma venereum.

Civilian outbreaks of psittacosis have been characterized by a high degree of communicability between infected birds and man and, in some instances, by a moderately high degree of communicability from man to man.[8] In contrast to this general experience, there is evidence that one laboratory officer, who carried psittacosis virus in his sputum, did not, so far as is known, transmit the agent to other military personnel. This officer contracted psittacosis in 1938 as a result of work in a laboratory where the virus was being studied. He entered the Army early in the war and was released from active duty in April 1946. He was hospitalized in Letterman General Hospital in February 1946 because of a chronic cough and, at that time and on subsequent occasions, psittacosis virus was recovered from his sputum.[9] The officer was not aware of exposure to psittacosis after joining the military service, and the most probable explanation for the presence of virus in his respiratory tract is that he was a

[7] See footnote 3, p. 143.

[8] (1) See footnote 4, p. 143. (2) Meyer, K. F.: Pigeons and Barn Yard Fowls as Possible Sources of Human Psittacosis or Ornithosis. Schweiz. med. Wchnschr. 71: 1377–1379, 1 Nov. 1941. (3) Meyer, K. F.: The Ecology of Psittacosis and Ornithosis (De Lamar lecture). Medicine 21: 175–206, May 1942.

[9] Surgical Record No. S-26265, Tissues of Animals Inoculated With Patient's Sputum. (Filed at the Armed Forces Institute of Pathology under Accession No. 168625.)

chronic carrier of the agent.[10] Although such a chronic carrier state is frequently observed in birds or laboratory animals,[11] its occurrence in man had not been observed previously.

Another observation in the Army was also a little surprising in view of previous experience. Large numbers of parrots and related birds were acquired as pets by personnel in the Pacific and other theaters. These birds lived in close contact with their masters, and numerous attempts were made to smuggle them into the United States by returning troops. Occasional infections with psittacosis might have been expected under these conditions, yet not one was reported. Whether the single psittacosis death among military personnel (the transport officer previously mentioned) was a result of infection from such an exposure is unknown.

[10] Meyer, K. F., and Eddie, B.: Human Carrier of the Psittacosis Virus. J. Infect. Dis. 88: 109–125, March–April 1951.

[11] Quan, S. F., Meyer, K. F., and Eddie, B.: Attempts to Cure Parakeet Psittacosis Carriers With Aureomycin and Penecillin. J. Infect. Dis. 86: 132–135, March–April 1950.

CHAPTER VIII

Rubella

Joseph Stokes, Jr., M. D.

HISTORICAL NOTE[1]

Rubella (German measles) has been of considerably greater significance than chickenpox in the armed services but has not approached measles or mumps in importance. Whereas in World War I, the noneffective rate for chickenpox was 0.02 per 1,000; the same rate for rubella was 0.14 per 1,000; for measles, 1.25; and for mumps, 2.58. The total number of primary admissions for rubella was 17,378, or a rate of 4.21 per 1,000 per annum, while the similar figures for chickenpox were 1,757 and 0.43 respectively.

CONTROL MEASURES

During World War I, no method of control had been developed nor has any conclusive evidence been obtained during or after World War II that the disease can be prevented. An exception to this is the suggestive recent evidence that gamma globulin in certain batches prepared in the United States from the American Red Cross plasma pools and in Australia from convalescent plasma may modify the disease when injected during the incubation period. However, the evidence on passive immunization is still scant and inconclusive. No method of active immunization has been developed. There is no conclusive evidence that the virus has been transmitted to the embryonated hen's egg, although Habel[2] has reported the infection of the monkey (*Macaca mulatta*) with the virus. Many further studies should be conducted both among the armed services and civilian groups for the purpose of determining the possible value of both types of gamma globulin mentioned. Such passive immunization may be urgent for aggregations in strategic places.

INCIDENCE

The hospital and quarters admission rates in the United States per 1,000 per annum for the 5 years 1941–45 are 21.7 (enlisted men only and including Alaska), 5.5, 17.1, 3.1, and 3.4 respectively. During the winter and early

(1) The Medical Department of the United States in the World War. Statistics. Washington: Government Printing Office, 1925, vol. XV, pt. 2, p. 238. (2) The Medical Department of the United States in the World War. Communicable and Other Diseases. Washington: U. S. Government Printing Office, 1928, vol. IX, pp. 387, 463.

[2] Habel, K. Transmission of Rubella to Macacus Mulatta Monkeys. Pub. Health Rep. 57: 1126–1139, 31 July 1942.

spring, incidence rates were highest. On occasion in certain of the service commands, incidence rates reached from 100 to 132 per 1,000 strength, during January, February, March, and April of these 5 years.

When one separates the incidence of the disease in accordance with the various areas for the years 1942 through 1945 (table 26), it is obvious that this childhood disease follows the usual history of such diseases in wars; namely, that the incidence is relatively high during and shortly after the period when the rate of induction is high and relatively low during the period when the rate of induction is low.

Table 26 indicates such a separation. The difference between the years 1942 and 1945 would have been considerably greater had not the incidence in the Latin American area remained elevated. This apparently resulted from the high rate of induction of Puerto Ricans, since most of the cases were among Puerto Rican soldiers. The recruitments from Puerto Rico were high during the last half of 1941 and the first 6 months of 1942. Thereafter, they declined and remained low until 1944 when they began to rise and continued to accelerate until well into 1945. Such active recruitment and the absence of an influx of

TABLE 26.—*Incidence of rubella (German measles) in the U. S. Army, by area and year, 1942-45*

[Preliminary data based on sample tabulations of individual medical records of primary and secondary diagnoses]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942-45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	125,530	8.51	14,696	5.53	88,775	17.13	12,194	3.07	9,865	3.37
Overseas:										
Europe	3,809	.87	128	1.54	216	.81	1,595	.95	1,870	.79
Mediterranean [1]	719	.48	0		393	.86	171	.26	155	.44
Middle East	98	.67	2	.33	43	.81	28	.61	25	.61
China-Burma-India	195	.44	38	4.34	54	1.36	48	.28	55	.25
Southwest Pacific	1,002	.55	319	4.48	352	1.85	201	.37	130	.13
Central and South Pacific	726	.58	169	1.12	183	.63	114	.26	260	.69
North America [2]	1,084	2.20	220	2.19	804	4.13	40	.31	20	.29
Latin America	1,962	5.14	441	4.33	231	1.91	575	6.70	715	9.82
Total overseas [3]	10,300	.96	1,501	2.56	2,517	1.49	2,892	.76	3,390	.73
Total Army	135,830	5.33	16,197	4.99	91,292	13.29	15,086	1.94	13,255	1.75

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes nearly a thousand cases on transports.

continental units after 1942 would account for the fact that most of the cases were among Puerto Rican soldiers and that the rates were relatively high.

It was also of interest that a comparatively high incidence of rubella occurred aboard transports. This could well be caused by the movement and crowding of troops into the staging area, the port of embarkation, and the transport itself. Close contact and possibly the change of environment may have been causative factors.

CHAPTER IX

Smallpox

Col. Laurence A. Potter, MC, USA

In the past, war and pestilence went hand in hand, and smallpox changed the direction and outcome of many wars. For more than 150 years, man has had the tool with which smallpox could be virtually stamped out, but he has used the tool only with varying efficiency to control or limit the disease in some areas. The principal nations of the world have used vaccination to protect their troops so that smallpox did not affect the decisions, direction, or outcome of any of the various phases of the global war. Nevertheless, a slight, but avoidable, morbidity and mortality from smallpox did occur among United States Army troops. Furthermore, the possibility of an outbreak of smallpox among civilian populations in a global war must be a matter of concern and planning by those responsible for military operations. A study of the history of smallpox during World War II gives the basis for certain predictions.

EPIDEMIOLOGY [1]

Smallpox is one of the great epidemic diseases, worldwide in distribution, and capable of great explosive outbreaks. It occurs in two forms, the classical variola major of high virulence and the mild alastrim or variola minor. Vaccinia is rarely transmitted from man to man as a communicable disease.

Variola is transferred mainly by droplet infection but is also transmissible in fomites. In the classical form, the incubation period is almost invariably 12 days. Transmission of the disease is facilitated by crowding; hence the cases increase during the colder winter months, reaching a peak in the spring and falling off rapidly as summer approaches.

Complete immunity or protection for a limited period of years can be accomplished by introduction of the virus of cowpox into a small area of the skin of a susceptible person. An attack of smallpox, either the classical or mild form, usually gives lifelong immunity to both varieties.

Classical smallpox is endemic in certain areas, primarily Iraq, India, China, Mongolia, Manchuria, Mexico, and Portugal. Alastrim, the mild variety, is endemic in Africa, Europe, and the United States. Where alastrim

[1] (1) Russell, F. F.: Smallpox and Vaccination. *In* Oxford Medicine. New York: Oxford University Press, 1942, vol. 5, pp. 553-592. (2) Gill, Clifford A.: The Genesis of Epidemics and the Natural History of Disease. Baltimore: Wood, 1928. (3) Top, Franklin H.: Communicable Diseases. St. Louis: C. V. Mosby Co., 1947.

becomes endemic, the classical disease tends to die out and tends to recur only in explosive outbreaks traceable to new importations of the disease.

It is interesting to speculate as to the remote origin of smallpox. Arguments have been made for its probable origin in the paleo-Arctic province of Central Asia (Mongolia),[2] but it may have originated in Africa. Alastrim, a better adapted form, was first recognized in Africa.[3] Smallpox was probably derived from another animal host; it is poorly adapted in its classical form to humans, since it kills too many hosts.

INFLUENCE ON PAST WARS

In the past, the effect of war has been to cause flareups of this dreaded disease resulting from movements of armies, dislocations of peoples, and, since Jenner, disruption of programs of control. The extent of these outbreaks has been relative to the magnitude of the dislocations and the extent of programs of vaccination or the presence of the immunizing effect of alastrim in the population.

Smallpox broke the siege of Mecca A. D. 570, and the returning army carried the disease into North Africa where it became endemic. The Moslem invasions carried it from North Africa into Spain [4] where it remained endemic for centuries and only recently was supplanted by alastrim.

The subjugation of the American Indian resulted as much from the inroads of smallpox as from the fighting ability and superior weapons of the invaders. The weak settlements of New England could not have survived their early years had not the Indian tribes been riddled by smallpox contracted from the explorers and traders who preceded the settlers.[5]

The Franco-Prussian War was followed by the most furious epidemic of the 19th century. It has been estimated that 200,000 died of smallpox in France in 1869. The carrying of the disease into Germany by French prisoners eventually resulted in the deaths of 140,000 people in Prussia, 170,000 in Austria, 143,000 in England, and lesser numbers in other parts of the world. The further the disease progressed from the seat of war, the easier it was to bring under control.[6]

After World War I, Germany passed through two more severe epidemics as a result of spread of smallpox from Russia.[7]

The armies of the United States have experienced smallpox in all their wars but in ever-decreasing amounts as care in the use of vaccine and the organi-

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[2] Stallybrass, Clare O.: The Principles of Epidemiology and the Process of Infection. London: George Routledge and Son, 1931.

[3] See footnote 1 (1), p. 151.

[4] See footnote 2.

[5] Stearn, Esther Wagner, and Stearn, Allen E.: The Effect of Smallpox on the Destiny of the Amerindian. Boston: Bruce Humphries Inc., 1945.

[6] See footnote 2.

[7] Fabre, J.: Smallpox Prevalence Throughout the World During and After the Second World War. Epidemiol. & Vital Statist. Rep. 1: 268–289, June 1948.

zation of programs of immunization have improved and as alastrim has supplanted the virulent classical smallpox as an endemic, or at least recurring, disease in the United States.

The Revolutionary War was fought before Jenner's great discovery of the immunizing effect of cowpox in 1796. Nevertheless, among the many diseases and infections which took so great a toll of American troops, smallpox was the one infectious disease which the medical men of that period understood and could combat with a measure of success. Their weapon was inoculation – a heroic and, to present-day thinking, wasteful method nevertheless effective in the prevention of disruption of an army in the field by smallpox. Protection by inoculation was not carried out completely in all areas, or at all times, with the result that smallpox played a major part in the defeat of the units attempting to take Quebec and hold Canada.[8]

There are no available accounts as to the incidence of smallpox during the 26-month Mexican War, nor are there statistics for the Army for the years 1847 and 1848. A very high incidence of smallpox among troops occurred in 1849 [9] despite the fact that vaccine virus, which had been imported by Dr. Waterhouse of Boston in 1800, was being used routinely by the Army.

The next high peak of smallpox among Army troops occurred in 1864. The incidence was high throughout the whole period of the Civil War. There was a total of approximately 19,000 cases resulting in nearly 7,000 deaths during the war period.[10]

Smallpox took a much smaller toll during the Spanish-American War and the Philippine Insurrection. There had been no smallpox in the Army in 1896 and only one case in 1897. Smallpox among Negro laborers at Forts Barrancas and Pickins, Fla., and its prevalence in Columbia, S. C., in March, April, and May 1898 alerted the Army to danger and led to careful vaccination of volunteer troops at the time of their muster into the service of the United States. In spite of those precautions, the majority of cases and deaths occurred among the volunteer troops, with only a small number occurring among Regular Army troops.[11] There were 825 admissions resulting in 258 deaths (1898–1901); 674 of the admissions and 249 deaths occurred in the Philippines.[12]

Before World War I, the Army had experienced a sharp increase in smallpox admission and death rates during war periods. During World War I, the admission rate was but little higher than the low rates experienced in the years immediately preceding the war. There were 853 admissions with 14 deaths.[13] Of these, 789 admissions, resulting in only 1 death, occurred in the United States. The negligible mortality indicates the disease was variola

[8] Duncan, L. C.: Medical Men in the American Revolution, 1775–1783. Army M. Bull. No. 25, 1931.

[9] (1) Statistical Report on Sickness and Mortality in the Army of the United States. Washington: A. O. P. Nicholson, 1856. (2) The Medical Department of the United States Army in the World War. Communicable and Other Diseases. Washington: U. S. Government Printing Office, 1928, vol. IX, pp. 357–386.

[10] See footnote 9 (2).

[11] Annual Reports of the Surgeon General of the Army, 1898 and 1899. Washington: Government Printing Office.

[12] See footnote 9 (2).

[13] See footnote 9 (2).

minor (alastrim), which had by this time, perhaps aided by varying programs of vaccination among the civilian population, supplanted variola major in the United States.[11]

MILITARY EXPERIENCE

The principal advances made during the period 1919 to 1941 which had or could have had an effect in lowering toward zero the number of cases of smallpox in the Army arose not out of a better understanding of the disease or in improvement of the vaccine, but in technologic progress in refrigeration (fig. 3) and transport of the vaccine.

During World War II, refrigerators operated by electricity or a kerosene flame were present in numbers in forward areas as a part of the regular equipment of supply and hospital units. Regularly scheduled air transports operated from the United States to all theaters of war and were capable of rapid transport of vaccine adequately refrigerated by packing in dry ice (solid CO_2), which was easily available at the point of origin of air shipments of vaccine, when required.



FIGURE 3.—Use of matting and leaves to shield vaccine refrigerator from the sun, 25th Medical Depot Company, India, 1944.

[11] See footnote 2, p. 152.

Lack of understanding of the rapid deterioration of smallpox vaccine stored at temperatures above 0° C. often negated the advantage of adequate refrigeration and rapid, protected transport. Cases of smallpox among troops following their entry into a zone where smallpox was present in the civilian population sometimes resulted. In each such instance, revaccination with potent vaccine soon effectively prevented further cases. The major portion of the cases occurred after V–J Day and the end of combat action.

United States Army policy with respect to vaccination for smallpox during World War II was stated in Army Regulations No. 40–210, 15 September 1942; in Circular Letter No. 162, Office of the Surgeon General, 28 November 1942; and in War Department Technical Bulletin 114, 9 November 1944 (revised 28 February 1947). These regulations required vaccination against smallpox as soon as possible on entry into the military service, with revaccination at intervals of 3 years thereafter. In addition, revaccination was to be accomplished before departure for overseas duty unless the individual had been vaccinated within the 12-month period prior to departure. Vaccination was required, also, on exposure to smallpox or in the presence of an outbreak, regardless of the date or the result of the last vaccination.[15]

In World War I, combat and postwar occupation took place in one of the best protected areas of the world—continental Europe. Most of the cases of smallpox occurred in the United States and were variola minor resulting directly from a considerable incidence of this disease in the civilian population of the Midwest.[16] In the 20 years before World War II, improved local and State programs of immunization and the apparent low communicability of alastrim had resulted in a decline to a negligible incidence. However, during and immediately after the combat period of World War II, American troops entered most of the areas of the world wherein classical smallpox is endemic or recurs in epidemic form. The disturbance of war increased the epidemic potential and thus led to increased incidence in various areas of the world with consequent exposure of troops present in those areas. The low incidence (a total of 215 cases during the period from 1939 to 1947) among troops was remarkable. A single case overseas in 1942 was from the Middle East theater. There were 4 deaths among 15 cases overseas in 1943. No cases of smallpox occurred among Army troops in the India-Burma theater in 1943, but in 1944 there were 23 cases with 6 deaths from that area. The peak month, March, accounted for 14 cases and 3 deaths. There was a high incidence of smallpox among the civilian population of that area during the period.[17]

In 1944, an outbreak of smallpox occurred which could have had serious repercussions over a widespread area. Early in the year, seven cases of smallpox of a mild avirulent form occurred among civilian employees at the Army

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[15] *Long, Arthur P.:* The Army Immunization Program. *In* Medical Department, United States Army, Preventive Medicine in World War II. Personal Health Measures and Immunization. Washington: U. S. Government Printing Office, 1955, vol. III, p. 283.

[16] See footnote 9 (2), p. 153.

[17] Blumgart, H. L., and Pike, G. M.: History of Internal Medicine in India-Burma Theater. [Official record.]

airbase at Fortaleza, Brazil. Several of these employees were in daily close contact with transient military personnel passing through this installation en route between the United States and North Africa, the Middle East, and India. At the time of the incident, no known cases occurred among uniformed personnel either based at Fortaleza or transient through the base to points spread over half the globe. However, the potential implications of this occurrence are demonstrated by the following data on 155 Army personnel revaccinated as a direct result of the occurrence of the first case: 95 had a reaction described as vaccinia, 9 as vaccinoid, 39 as immune, and 12 had no noted reaction.[18]

From 1 January 1945 until the end of combat in August 1945, only 1 case of smallpox occurred in the Persian Gulf Command, 1 case occurred in the Mediterranean, and 1 case occurred in Europe. During this period, there were 13 cases with 4 deaths in the India-Burma theater. Two cases and one death occurred in February; 8 cases and 1 death occurred in March; and 3 cases with 2 deaths occurred in April.

All of the smallpox during these years occurred as sporadic cases among an otherwise completely protected uniformed population and were directly related to high incidences of the disease among the civilian populations of areas indicated. In these areas, the military population was very small in relation to the civilian population, and the soldiers were, as individuals either on or off duty, thrown into close contact with civilians. During the first winter of occupation, 1945–46, the same conditions existed in certain areas; a similar experience during this winter will be considered in detail, since it has a direct relationship with and is, in fact, part of the war, since it is illustrative of any grouping of cases in the previous years, and since more exact detail is available. The most important outbreak occurred among occupation forces in Korea during the winter of 1945–46.[19]

Prior to the movement of occupation forces into Korea from Okinawa and the Philippines, all immunization records were checked and personnel who had not been immunized against smallpox within 1 year were revaccinated.

The first troops landed in Korea early in September 1945, and additional units continued to arrive for several months. The first personnel eligible to return to the United States for discharge left early in October, and this process of returning individuals was a constant procedure throughout the winter. No substantial number of replacements for these individuals arrived in Korea until the first of the year 1946, but thereafter replacements arrived in a more or less continuous flow throughout the remainder of the winter. The Army population was a continually changing one; furthermore, the total number of troops in Korea fluctuated greatly from week to week, and the age distribution among the Army personnel shifted steadily toward the lower age group (18–20), the older men being returned to the United States by virtue of points based on length of

[18] Letter, Capt. B. M. Kagan, MC, Epidemiologist, Headquarters, United States Army Forces, South Atlantic, Office of the Chief Surgeon, Recife, Pernambuco, Brazil, to Commanding General, United States Army Forces, South Atlantic, 4 Sept. 1944, subject: Report on Smallpox at United States Army Air Base, Fortaleza, Brazil.

[19] (1) Boeck, V. H. F.: Smallpox Among U. S. Soldiers in Korea. Bull. U. S. Army M. Dept. 6: 45–58, July 1946. (2) Boeck, V. H. F.: Smallpox Among U. S. Army Forces in Korea. J. Mil. Med. Pacific 2: 2–6, April 1946.

service, service overseas, campaigns, decorations, and number of children. Personnel who remained were younger men who came as replacements late in the war when the age for compulsory service was lowered. The replacements received in Korea in 1946 were nearly all in the 18–20 age group. Because of these considerations, the estimation of rates based on total population, especially age specific rates, is not feasible. The over-all morbidity rate for smallpox was well under 1 per 1,000 or those exposed during the 4-month period 1 November 1945 to 1 March 1946, inclusive. (The annual rate per 1,000 average strength was 1.8.)

As nearly as could be determined, the program of vaccination of the civilian population [20] in Korea had been practically inoperative for at least 5 years; the vaccine manufactured in Korea presumably had been needed by the Japanese Army.

Korea was not a combat area during World War II, even in that early phase of it between the Japanese and Chinese prior to 1939. The country and its Japanese and Korean populations were relatively stable; at least, there were no mass movements into the area or within the area. Japanese troops moving through the country were transported directly on an excellent railroad system and were immunized against smallpox. When the war ended, this was changed. The Japanese capitulated a month before the entry of American occupational troops into Korea. On the date of capitulation, all industry stopped in Korea. Controls over the population ceased, and a great deal of movement within the country occurred. Furthermore, many Koreans had been employed by various Japanese agencies in north China and Manchuria, and many Japanese civilians were in these areas and in North Korea. These peoples began to move, in whatever manner they could, toward South Korea, Their movement was given impetus by the advance of Russian forces across Manchuria into Korea, north of the 38th parallel. This movement of refugees continued throughout the winter under conditions of indescribable hardship, since most of the movement was, of necessity, on foot. Even during freezing weather, refugees were entering at the rate of several thousand each day into the American Occupation Zone from across the northern border. Other Korean refugees were being repatriated by ship from Japan, China, and other areas of the Pacific. This latter group of refugees, however, was not a great threat because they were processed through United States Army or United States Navy agencies before embarkation.

The stage was set for smallpox to occur in epidemic proportions among the civilian population, since there was a large pool of susceptibles into which would be introduced large numbers of refugees bringing highly virulent smallpox from the outer reaches of Manchuria and north China.

The first cases among civilians which came to the attention of the occupying forces began to occur late in October. During the 4-month period November 1945 to March 1946, inclusive, 84 outbreaks numbering 200 to 500 cases

[20] Simmons, James S., Whayne, Tom F., Anderson, Gaylord W., and Horack, Harold M.: Global Epidemiology. Philadelphia: J. B. Lippincott Co., 1944, p. 151.

each were reported to Army authorities, and steps were taken to control them by intensive vaccination in the communities or localities where they occurred.[21] Any estimate of the total number of civilian cases would be only a guess, but they occurred in all areas of Korea south of the 38th parallel, and were well distributed among the 19 million people. The incidence was unquestionably higher in the northern sections and along the main routes of travel of the refugees as they spread out over Korea returning to their home areas.

The first case among United States Army troops was diagnosed on 3 November 1945, but the actual date of onset of symptoms was 27 October. During November, 5 more cases occurred, with 2 deaths toward the end of the month. In December, there were 21 more cases and 9 deaths; these were followed by 6 more cases and 2 deaths in January [22] and 4 more cases and 1 death in February. After 6 February 1946, there were no further cases.

Immediately upon recognition of the first case, the patient's record indicated a recent immune reaction, revaccination of all troops and American civilian personnel in Korea was ordered. This procedure took some time because of delay in obtaining sufficient vaccine. Hospitals were directed to examine all admissions carefully for recent vaccinations and to revaccinate if any doubt as to the reaction existed. If a case broke out in a hospital, mass revaccination of all hospital personnel and patients was to be done immediately.

All persons entering Korea were to be vaccinated on arrival. This order was later modified; vaccination was required aboard ship just prior to debarkation. The purpose was to prevent possible exposure at the reception center before vaccination could be accomplished and to make doubly sure that all persons were vaccinated. Previously, some personnel had not been vaccinated on arrival because they did not go through the center before assignment.

Because of the continued incidence of smallpox in December 1945 and doubt as to the potency of the vaccine available from depots in the Pacific, sufficient vaccine to revaccinate the entire command was ordered shipped by air, packed in dry ice, directly from the United States. This shipment was received 21 January 1946. Since ambient temperatures in Korea at this time were below freezing, no difficulties were encountered in preserving the vaccine during its distribution throughout the command. A directive was sent out requiring that careful entry of the reaction be made on each individual immunization register and that, upon completion of the program, each major echelon commander would certify by letter to the commanding general of the occupying forces that each individual of his command had been revaccinated with the "Special Lot of U. S. Vaccine." No further cases of smallpox occurred among Army personnel following completion, during the first week in February, of this program with vaccine of known high potency. The outbreak of

[21] See footnote 19 (1), p. 156.

[22] In addition, an Australian merchant seaman, 30 years old, contracted smallpox in January. He had never been vaccinated. The case was diagnosed as variola, severe, and resulted in death.

smallpox in Japan was a direct extension of the Korean epidemic. Many Japanese were repatriated through Korea, and, while the majority were vaccinated before leaving Korea, many made the trip surreptitiously in small boats and thus evaded processing stations.

Within the first few weeks following V–J Day (September 1945), steps were taken to reassemble and place in operation the Korean vaccine laboratory and its equipment, which had been dismantled and moved a few weeks prior to American occupation. This laboratory immediately processed and packaged vaccine lymph found at the former site and then procured calves and began operations. By February 1946, more than a million doses per month were being produced. This vaccine, augmented by vaccine from Army sources as it could be procured (3 million doses—September 1945 to April 1946), was used initially to vaccinate all refugees being evacuated from, or entering, Korea. When civilian outbreaks of the disease began to occur, it was used as a focal attack to prevent further spread. All Koreans in the Government agencies and transportation systems were vaccinated. As vaccine became more plentiful, vaccination programs were carried out in the larger cities and other communities where considerable numbers of American troops were concentrated. By the end of 7 months, over 7 million of the 19 million Koreans in the American area had been vaccinated, and the program was continuing at an accelerated pace. The very cold winter weather compensated for the lack of refrigeration in preservation of the potency of the vaccine. It was contemplated that operation of the vaccine laboratory would be suspended during the summer months.

The small number of cases and the ever-varying size and composition of the command do not permit statistical evaluation designed to present statistically significant proof supporting basic conclusions or recommendations; however, the data and the general conclusions and impressions derived from these facts are pertinent to the discussion.

Distribution of patients by age ranged from 18 to 48. Of the 37 patients, 14 were 19 years of age. Of the 22 patients 21 years of age and under, 9 died, the case fatality being 40.90 percent. Of the remaining 14 patients, over 21 years, 4 or 28.57 percent died. The age of one patient who died was not available. Over-all case fatality was 37.84 percent.

Available home addresses indicated that 27 of the patients were from rural areas and that 9 were from urban areas, a ratio of 3 rural to 1 urban. Deaths were 12 rural and 2 urban, or a ratio of 6 to 1. This undoubtedly reflects variations in vaccination programs rather than previous experience with variola. One case occurred among Negro troops whose strength was approximately 4 percent of the command. The rural-urban composition of the Army population is not known, but it is hardly likely that personnel from rural areas exceeded in number those of urban origin.

The 36 patients on whom such data was available originated in 18 States. Forty-four percent of the patients were from 4 States: Ohio—6, Alabama—4,

Texas 3, California 3. None of these States required vaccination as a prerequisite to school attendance.[23]

The likely source of infection in 30 of the patients was the native population, for soldiers in Korea were thrown into close contact with the people of this heavily populated area. Four patients were exposed while in Army hospitals. One very mild abortive infection occurred in the medical officer who attended the first Army patient. Two patients gave no history of contacts with civilians or with known cases.

The cases were differentiated clinically as follows: 3 varioloid (abortive) cases with no deaths; 21 cases variola with 4 deaths, case fatality 19 percent; 5 cases variola hemorrhagica pustulosum, 3 deaths, case fatality 60 percent; and 8 cases of purpura variolosa with 7 deaths, case fatality 87.5 percent.

The most enlightening and instructive feature of the Korean experience is the matter of protective immunization in all its aspects. As was indicated earlier in the discussion, the incidence of smallpox among the troops was considerably less than 1 case per 1,000 men. These 37 cases, however, occurred among individuals presumably immune. Why were they not immune? According to their records, nearly all had been vaccinated within a year. Some had had several vaccinations, but only 5 of the 35 whose records were available had been successfully vaccinated prior to entry into the Army. Nine gave a history of attempted vaccination with no take. The other 21 had not been vaccinated as civilians. Of the 35, 30 had records, confirmed by direct questioning, of reactions to vaccine ranging in interpretation from vaccinia to immune type after induction into the Army. The remaining five had records indicating immune-type reactions but stated that there had been no reaction.

As a part of an investigation planned by the Preventive Medicine Service, Office of the Surgeon General, Dr. William G. Workman of the National Institutes of Health, United States Public Health Service, visited Korea at the request of the Commanding General, United States Army Forces in Korea. Dr. Workman arrived in January 1946. He believed that failure of previous vaccinations to protect troops was due to the use of vaccine which had been allowed to deteriorate by failure to keep it at temperatures below 0° C. in all phases of transport and storage and that United States vaccine was as effective as any special vaccine made on such animals as camels or caribou.

Craigie and Wishart[24] have produced the immune type of reaction both with heat-killed vaccine lymph and, more significantly, with carefully washed formalin-killed elementary bodies of vaccinia on persons who had at some time in the past been vaccinated. No reaction occurred when this killed virus material was applied to persons who had never been vaccinated.

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[23] Hampton, B. C.: Smallpox in Relation to State Vaccination Laws and Regulations. Pub. Health Rep. 58: 1771-1778, 3 Dec. 1943.

[24] Craigie, J., and Wishart, F. O.: Skin Sensitivity to Elementary Bodies of Vaccinia. Canad. Pub. Health J. 24: 72-78, February 1933.

Wolpe [25] in a South African study demonstrated the value of repeated attempts to vaccinate. In his study, he revaccinated all those who did not show a reaction progressing at least to vesiculation. That is to say, he revaccinated not only "no take" individuals but also those whose reaction would normally be called "immune." The initial vaccination of one group resulted in 67 percent positive reactions according to his criteria. They were examined after 4 days. The remainder were again vaccinated, and 41 percent (nearly 14 percent of the total) had takes progressing at least to vesiculation. Those still not reacting or producing less than vesiculation were vaccinated a third time, and 14 percent (2 percent of the total) developed reactions of vesiculation or better. Eighteen percent still showed no demonstrable positive (vesiculation) when read at 4 days. By revaccination, he had raised the percentage giving a reaction beyond the type usually designated as immune in from 67 percent to 82 percent.

This would indicate that in vaccination programs in such organizations as an armed force, initial vaccinations should be done, not at the time of induction, when the individual is in every sense a transient, but at the training camp at which the individual will first be under the close supervision of the same medical officer for at least 10 days. This is necessary to insure that the results of vaccination are properly observed and that an accurate descriptive entry is made in the individual's record of immunizations. Of greater importance, the individual must be revaccinated until a vesicular reaction or primary-type reaction takes place, or until three attempts with known potent vaccine and proper technique have been made.

Entries in immunization registers should use descriptive terms such as "papular," "vesicular," and "pustular," rather than terms such as "immune," and "vaccinoid." The length of time intervening between the application of the vaccine and the height of the reaction should be included in the descriptive record.[26]

CIVILIAN EXPERIENCE

Smallpox among civilian populations presents a threat to troops operating or residing in the same area, disrupts the civilian economy, and, in the case of United States Armed Forces, always presents the humanitarian responsibility of doing something for the population under military control. The experience of this great global upheaval needs analysis in order to predict the behavior of smallpox in another world conflict.

In 1947, the author gathered information on the incidence of and number of deaths from smallpox in various countries of the world during the war years in order to see if any pattern existed. Recently a more complete com-

[25] Wolpe, J.: Report of 3,052 Vaccinations With Special Reference to Value of Repeated Attempts. South African M. J. 19: 339-340, 22 Sept. 1945.

[26] (1) Stuart, G.: Note on the 'Immunity Reaction' Following Vaccination Against Smallpox. Bull. World Health Organ. 1: 30-31, 1947-48. (2) MacKenzie, M.: Significance of the 'Immunity Reaction' Following Vaccination Against Smallpox. Bull. World Health Organ. 1: 32, 1947-48. (3) McKinnon, N. E., and Defries, R. D.: Interpretation of Reactions Following Revaccination. Canad. Pub. Health J. 22: 33-39, January 1931.

pilation of smallpox statistics has been published in the *Chronicle of the World Health Organization*.[27] Certain patterns of behavior present themselves.

While no great pandemic arose among the civilian populations during or after World War II, as used to follow most other wars throughout history, World War II nevertheless brought about directly or indirectly a great recrudescence of smallpox in many areas of both hemispheres.

The various countries of the world fall rather easily into three groups: (1) Those which have efficient, rigorously prosecuted vaccination programs; (2) those in which classical smallpox is endemic, control measures being exercised only sporadically to halt or limit periodic outbreaks in various communities; and (3) those (the largest group) wherein classical smallpox is not endemic but in which vaccination programs are lax and are pursued on an inefficient level. In the last group, the vaccination programs may be marshaled in full force whenever imported classical smallpox causes an outbreak but, on the other hand, are only mildly stimulated by the regular presence of alastrim at a low morbidity and mortality level. The main protection of this third group is the immunizing presence of alastrim, a tendency for a rather high rate of vaccination of infants on a voluntary basis, and, in some, an ability to do a prodigious amount of vaccination when classical smallpox appears.

Sweden and Switzerland fall into the group characterized by complete, efficient control. As nearly as could be determined, no deaths occurred from 1936 to 1946, inclusive. Cases are recorded only occasionally, and these probably represent importations. While these two neutral countries were immediately adjacent to the turmoil of war, they are relatively remote from any endemic area of classical smallpox.

India and Mexico represent the endemic group wherein explosive outbreaks of disease may be expected at 5-year to 10-year intervals.[28] The mortality peaks demonstrated for India and Mexico during the war years are exceeded by peaks occurring in 1936 and could have been expected, war or no war.

The third and largest group contains all gradations between the extremes of complete control and endemicity with little control. Among this group showing a marked rise in smallpox during or immediately after the war years, which probably was the result of disturbance of controls and movement of peoples incident to the war, are Egypt, Turkey, Japan, Korea, and to a lesser extent the United States and Great Britain. The deaths in the United States arose largely from one outbreak of classical smallpox (1946), which occurred because of importation into Seattle, Wash., from the Orient. Although quickly suppressed by mass vaccination, it cost 20 lives.[29]

Italy was fortunate, for although an epidemic of smallpox arose through importations from North Africa, it was the mild alastrim and carried a negligible mortality.[30] No direct connection between the imported case and the first

[27] (1) World Prevalence of Smallpox During and After the Second World War. Chron. World Health Organ. 2 (12): 278–281, December 1948. (2) See footnote 7, p. 152.

[28] See footnote 2, p. 152.

[29] Palmquist, E. E.: The 1946 Smallpox Experience in Seattle. Canad. J. Pub. Health 38: 213–218, May 1947.

[30] Stowman, K.: Smallpox at Bay and at Large. Epidemiol. Inform. Bull. 1: 371–376, 15 June 1945

local case could be demonstrated, though both patients were in separate rooms on the same isolation ward for a short time. The local patient was vaccinated at this time, and a simultaneous incubation of variola and vaccinia which occurred led to an initial erroneous diagnosis of generalized vaccinia. The patient was not isolated, nor was a program of vaccination of contacts carried out for 3 days; thus the initial spread of the disease within the city was permitted.

Canada has controlled smallpox well and appears not to have suffered an importation of virulent smallpox during the period.

Uruguay, a neutral, remote from the combat areas, nonendemic, and with a fairly effective program of control, developed a definite increase in reported cases, and in 1947 a localized outbreak of smallpox carrying a considerable mortality occurred.

Two countries (Sweden and Switzerland) have controlled smallpox to an amazing degree of completeness. Endemic areas seem to have their periodic epidemics without reference to events in other parts of the world and are affected only slightly (in timing and intensity) by internal events. Countries whose programs and conditions fall between these two extremes are subject to great fluctuations directly related to catastrophes (such as war) which increase importations of virulent forms of the disease and, depending on the distance from the catastrophic area, disrupt the incomplete and often very inefficient programs of control.

It may then be expected that, as the result of any such great disturbance, global war, for example, any nonendemic country not having prosecuted a complete, thorough program of control for a considerable period of time may experience an unusual incidence of smallpox which may reach proportions of a major epidemic, whether that country be combatant or neutral, and whether it is located near to or remote from the centers of disturbance.

Diseases Caused by Bacteria

CHAPTER X

Diphtheria

Aims C. McGuinness, M. D.

Diphtheria in World War II could not, from a statistical point of view, have been said to have reached epidemic proportions in any theater of operations; approximately 5,700 cases were reported for the total Army between January 1942 and December 1945. Because of the serious disability associated with the disease, however, it presented a problem of considerable importance in the European and Mediterranean Theaters of Operations, in the Pacific areas, and in the India-Burma theater. There were 125 deaths, 67 of which occurred in the European theater in 1945. Cutaneous diphtheria attending desert sores and tropical ulcers was a form of the disease virtually unknown to most medical officers, even though the desert sore of diphtheria had been recognized by the British for many years, both in the Middle East [1] and in northern India.[2] The severe polyneuritides following diphtheria, which were encountered especially in the Mediterranean and European theaters, likewise were a new problem to the United States Army medical officers.[3] In the European theater, diphtheria was a particular hazard to the occupation forces.

The number of cases of diphtheria and rates per 1,000 strength per annum in the various theaters and areas from January 1942 through December 1945 are shown in table 27. Table 28 shows deaths and mortality rates from diphtheria during the same period.

EXPERIENCE IN THE CONTINENTAL UNITED STATES

The United States entered World War II at a time when the incidence of diphtheria in this country had reached extremely low levels. In 1920, there were approximately 150,000 cases of diphtheria, with nearly 14,000 deaths, reported from 41 States. In 1938, there were approximately 30,000 cases from the entire country, with 2,600 deaths, a reduction of about 80 percent in both numbers of cases and deaths. In only a few States—namely, West Virginia, Kentucky, Tennessee, and sections of Virginia, North Carolina, Missouri, Arkansas, Texas, and New Mexico—were relatively high mortality rates still

[1] Craig, C. McK.: A Study of the Aetiology of the "Desert," Septic, or Veldt Sore Amongst European Troops: And Its Association With Faucial Diphtheria. Lancet 2: 478–479, *September* 1919.

[2] Bensted, H. J.: A Limited Outbreak of Diphtheria Exhibiting Both Cutaneous and Faucial Lesions. J. Roy. Army M. Corps 67: 295–307, November 1936.

[3] (1) Johnson, J. W., Jr.: Infectious Polyneuritis in MTOUSA, pts. 1 and 2. [Official record.] (2) Sampson, J. J.: Late Neuronitis Following Proved and Suspected Cutaneous, Faucial, and Wound Diphtheria. Am. J. M. Sc. 212: 432–448, October 1946.

TABLE 27. *Incidence of diphtheria in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	619	0.04	67	0.03	205	0.04	152	0.04	195	0.07
Overseas:										
Europe	2,557	.58	27	.33	45	.17	245	.15	2,240	.94
Mediterranean [1]	1,087	.73	2	.09	197	.43	628	.97	260	.73
Middle East	45	.31	1	.17	23	.43	11	.24	10	.24
China-India-Burma	208	.47	8	.91	15	.38	155	.92	30	.14
Southwest Pacific	615	.33	3	.04	7	.04	100	.19	505	.49
Pacific Ocean	519	.41	4	.03	69	.24	266	.61	180	.48
North America [2]	19	.04	2	.02	3	.02	14	.11	0	
Latin America	23	.06	5	.05	2	.02	1	.01	15	.21
Total overseas [3]	5,105	.48	55	.09	364	.22	1,426	.37	3,260	.70
Total Army	5,724	.22	122	.04	569	.08	1,578	.20	3,455	.46

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

to be found.[4] For the week ending 28 December 1940, diphtheria in this country reached a record low; each section of the country reported the lowest incidence in recent years.[5] A leading textbook of pediatrics published in late 1940[6] stated that 80 percent of urban adults were immune to diphtheria. Only in certain large cities and in a few rural areas did physicians have an opportunity to see the clinical picture with any degree of frequency. Thus, as a whole, the physicians of the country were complacent as to the problem and ignorant as to the disease, and relatively few laboratory personnel were competent in the bacteriologic techniques required for isolation and identification of virulent *Corynebacterium diphtheriae*.

In 1940, a study by Stebbins, Ingraham, and Chant[7] lent support to the theory that, with decreasing incidence of clinical infection and the associated decrease in the prevalence of carriers of toxigenic *C. diphtheriae*, natural immunization was materially reduced.

[4] Dauer, C. C.: Geographical Distribution of Diphtheria Mortality in the United States. Pub. Health Rep. 55: 622–628, April 1940.

[5] Prevalence of Communicable Diseases in the United States. Pub. Health Rep. 56: 89–92, 17 Jan. 1941.

[6] Holt, L. Emmett, Jr., and McIntosh, Rustin: Holt's Diseases of Infancy and Childhood. 11th ed. New York: D. Appleton-Century, 1940, p. 1083.

[7] Stebbins, E. L., Ingraham, H. S., and Chant, H. L.: Changing Factors in Diphtheria Immunity: Its Production and Duration. New York State J. Med. 40: 658–665, April 1940.

TABLE 28.—*Deaths due to diphtheria in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on tabulations of individual medical records]

[Rate expressed as number of deaths per annum per 100,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number of deaths	Rate	Number of deaths	Rate	Number of deaths	Rate	Number of deaths	Rate	Number of deaths	Rate
Continental United States	10	0. 07		0. 00	2	0. 04	2	0. 05	6	0. 20
Overseas:										
Europe	72	1. 64		0		0	5	. 30	67	2. 82
North Africa	11	. 74		0	4	. 88	3	. 46	4	1. 13
Middle East	3	2. 05		0	2	3. 77	1	2. 16		0
China-Burma-India	3	. 68	1	11. 43		0	2	1. 19		0
Pacific	21	. 68		0	2	. 42	8	. 82	11	. 78
North America	2	. 41		0		0	1	. 77	1	1. 47
Latin America		0		0		0		0		0
Total overseas [1]	115	1. 07	1	. 17	8	. 47	20	. 52	86	1. 85
Total Army	125	. 49	1	. 03	10	. 15	22	. 28	92	1. 21

[1] Includes 3 deaths on transports in 1945.

On 10 November 1941, Lt. Comdr. (later Capt.) LeRoy D. Fothergill, MC-V(S)USNR, made a very significant report to the medical officer in command, United States Naval Medical School, Washington, D. C., on a Schick-test survey at the Norfolk, Va., Newport, R. I., and Great Lakes, Ill., naval training stations.[8] This study, which was reported subsequently by Cheever,[9] and Worcester and Cheever,[10] was in part prompted by the epidemic of diphtheria in Halifax, Nova Scotia, which had reached serious levels the preceding winter; certain significant findings in that epidemic disclosed that over 50 percent of the cases were persons more than 15 years of age and that 48 percent of the population 20 years of age and over (not previously immunized) were found to be Schick positive.

In the study among recruits at the Norfolk, Newport, and Great Lakes stations, there was found an over-all Schick-positive rate of 33.8 percent. Recruits from the New England States showed the highest rate (32 to 76 percent) of susceptibility, and rates for the East and West North Central States were almost equally as high. Much lower rates (7 to 10 percent) were found among recruits from the South Atlantic, East South Central, and West

[8] Memorandum, Lt. Comdr. L. D. Fothergill to Medical Officer in Command, U. S. Naval Medical School, 10 Nov. 1941, subject: Schick Test Survey at Norfolk, Newport, and Great Lakes Naval Training Stations.

[9] Cheever, F. S.: A Schick Survey of 18,000 Naval Recruits. Am. J. Pub. Health 38: 374–377, March 1948.

[10] Worcester, J., and Cheever, F. S.: The Shick Status of 18,000 Young Adult Males. New England J. Med. 240: 954–959, June 1949.

South Central States, where diphtheria still was moderately prevalent. This study emphasized the importance of latent or subclinical infection as a factor in the production and maintenance of active immunity. Fothergill's group found further that about one-quarter of the Schick-positive group had a positive Maloney reaction together with a positive Schick test and therefore could not be immunized with toxoid without danger of serious reaction.

Fothergill's report came as a strong alert to the Preventive Medicine Division, Office of the Surgeon General. During the next 2 years, however, diphtheria did not appear to present a serious problem to United States Army troops, either in the Zone of Interior or overseas. In late 1943, there were reports of a definite increase in diphtheria in the civilian populations of continental Europe, as well as reports of the presence of diphtheria to an unexpected degree among United States troops in the South Pacific Area. As a result of these reports, the Preventive Medicine Service conducted a study [11] at Camp Ellis, Ill., and at Camp Tyson, Tenn., during the months of February, March, and April, 1944, to determine (1) what proportion of United States troops were susceptible to diphtheria; (2) what degree and portion of reactions were to be expected following the administration of diphtheria toxoid to troops; and (3) what method might be utilized to screen out the majority of reactors. The study was under the over-all direction of Lt. Col. (later Col.) Arthur P. Long, MC, Preventive Medicine Service, and the investigations were carried out by a team composed of Capt. (later Maj.) Emanuel B. Schoenbach, MC, of the Army Epidemiological Board; Dr. Paul B. Beeson, a civilian consultant of Atlanta, Ga.; and a technical assistant, Miss Charlotte Root. The findings are summarized as follows:

Among 2,933 Schick-tested individuals, 44 percent were found to have positive reactions including 5.8 percent with combined reactions. In general, the Northeastern and North Central States tended to have high percentages of susceptibility and the Southeastern States low percentages. This paralleled Fothergill's findings among Navy recruits.

To determine the degree of reactions to diphtheria toxoid, studies were carried out on 2 groups of approximately 300 men each, none of whom previously was Schick tested. Each individual received an intracutaneous injection of 0.1 cc. fluid toxoid diluted to contain 0.1 Lf per cubic centimeter, and at the same time a subcutaneous injection of 0.1 cc. of undiluted fluid toxoid. Approximately 5 percent of the individuals receiving the toxoid reaction test dose were hospitalized, and 3.7 percent were confined to quarters; 15.6 percent, while demonstrating some untoward reactions, continued on duty. Approximately 75 percent were retained for the 0.5 cc. dose of toxoid. Of the 429 individuals who received 0.5 cc. of toxoid, 5.8 percent were hospitalized, and 325 or 75.8 percent were retained for further doses.

While the number of cases of diphtheria in the United States Army during the 2 years following Pearl Harbor was not significant, the complacency which

[11] Diphtheria Susceptibility and Immunization. Bull. U. S. Army M. Dept. No. 76, 104-108, May 1944.

had existed in regard to this disease during the pre-Pearl Harbor mobilization had yielded to a realization of these three important facts:

1. A substantial percentage of United States Army troops was susceptible to diphtheria.

2. There were indications that the disease might become a serious threat to overseas troops.

3. The indiscriminate administration of diphtheria toxoid in the regular dosage might be expected to result in a relatively high proportion of reactions in troops.

Diphtheria among troops stationed in the continental United States never reached serious proportions. In table 27, rates of 0.03, 0.04, and 0.04 per 1,000 average strength per annum were reported for the years 1942, 1943, and 1944, respectively. Cases were scattered and special control measures such as routine immunization never received serious consideration. In 1945, the rate increased to 0.07 per 1,000 average strength per annum, largely as a result of patients with diphtheria arriving on transports from overseas theaters, chiefly from the Pacific. A number of these patients, many of whom had cutaneous diphtheria, reached the Letterman General Hospital in San Francisco, Calif., and Moore General Hospital in Asheville, N. C.[12] This situation led to a number of secondary cases among other patients as well as among hospital personnel, a significant number of the cross-infections occurring on the dermatologic wards. Again, except for these special situations, diphtheria was not an important problem in the Zone of Interior.

EXPERIENCE IN EUROPEAN THEATER OF OPERATIONS

Great Britain. From the Ministry of Health in Great Britain,[13] it was learned that the diphtheria situation in the civilian population was of no particular import, that it compared favorably with conditions in the United States, and that the general trend of diphtheria since 1940 had been downward. The reported incidence of diphtheria among soldiers of the British Army stationed in England indicated that the rates were decidedly low.

Table 29 shows the diphtheria rates in England and Wales for the years 1939 to 1945, inclusive.

Western Europe. In 1941, diphtheria began to increase in France, Germany, The Netherlands, Belgium, Norway, and Denmark. The year 1942 showed a substantial increase over 1941, and the rates and reported cases for 1943, as shown in table 30 were truly alarming. This information, which was available early in 1944, served as a warning that diphtheria might be a disease

[12] Memorandum, Commission on Meningococcal Meningitis, Army Epidemiological Board, for The Surgeon General, 6 July 1945, subject: Diphtheria Infections at Letterman General Hospital, San Francisco, California, and at Moore General Hospital, Asheville, North Carolina.

[13] Gordon, J. E.: A History of Preventive Medicine in the European Theater of Operations, U. S. Army, 1941-45. [Official record.]

TABLE 29.—*Diphtheria in England and Wales, 1939–45*

[Rate expressed as number of cases per annum per 100,000 population]

Year	Number of cases	Rate
1939	47, 698	115. 0
1940	46, 281	110. 6
1941	50, 797	121. 7
1942	41, 404	98. 8
1943	36, 077	85. 6
1944	29, 446	69. 4
1945	25, 000	[1] 58. 9

[1] Based on 1944 population.

Source: Gordon, J. E.: A History of Preventive Medicine in the European Theater of Operations, U. S. Army, 1941–45. [Official record.]

of appreciable importance among infections to be encountered by troops invading France, the Low Countries, and Germany.

United States Army.—The rising tide of diphtheria in western Europe in the early 1940's, together with the knowledge that a relatively high degree of susceptibility existed among United States troops,[14] gave ample warning to the problems that might confront our forces with respect to diphtheria at such time as they might invade the occupied countries and Germany. On the other hand, the diphtheria situation in the civilian population of England was on a level with the low rates prevailing in the United States, so that no particular difficulty was anticipated among the large bodies of United States troops accumulating in England during 1942, 1943, and up to the Normandy invasion in June 1944.

As anticipated, except for a few scattered cases, diphtheria presented no problem in the European theater in 1942 and 1943. There were 27 cases reported for 1942 with a rate of 0.33 per 1,000 average strength per annum, and 45 cases with a lower rate of 0.17 for the year 1943. Even lower rates prevailed until November 1944 when our troops reached the Low Countries and the cold and wet of winter had set in. From that time on, there was a steady increase in cases and rates. During the first 6 months of 1945—the final campaign of the war against Germany—there were 1,037 cases reported with an over-all rate of 0.76 per 1,000 per annum and a rate of 1.05 for the month of April. Unquestionably, as was the case in other theaters, many uncomplicated cases passed by without recognition.

Germany surrendered on 7 May 1945, and during the months of June and July the number of cases of diphtheria in the Armed Forces dropped off sharply. As United States troops began to mingle with the civilian population of Germany, the rates again increased, and it was soon evident that this disease was to become one of the major hazards of infection in the army of

[14] See footnote 13, p. 171.

TABLE 30.- *Incidence of diphtheria in civilian populations of Germany and Axis-occupied countries in Europe, 1939–43*

[Rate expressed as number of cases per annum per 100,000 population]

Country	Estimated population	1939		1940		1941		1942		1943	
		Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Germany	79, 375, 281	143, 585	180. 9	138, 397	164. 3	173, 161	208. 1	236, 645	288. 1	282, 859	287. 2
Belgium	8, 386, 553	2, 419	28. 8	[1] 2, 265	. . .	4, 271	50. 9	5, 464	65. 1	16, 072	191. 6
France	38, 000, 000	14, 019	35. 9	13, 568	34. 7	20, 018	52. 6	31, 466	81. 8	40, 697	118. 7
Denmark	3, 805, 000	1, 106	29. 1	860	22. 6	917	23. 1	1, 370	42. 6	2, 527	66. 4
Netherlands	8, 728, 569	1, 273	14. 6	1, 730	19. 8	5, 501	63. 6	19, 537	222. 7	[2] 53, 469	638. 9
Norway	2, 937, 000	72	1. 8	138	4. 7	2, 609	88. 9	8, 349	284. 3	[3] 20, 268	752. 8

[1] Exclusive of data missing for 15 May to 31 August.
[2] Total for 51 weeks.
[3] Total for 11 months.

Source: (1) 1944 population figures, World Almanac. (2) Medical Intelligence Summary No. 8, Office of the Chief Surgeon, ETOUSA, 27 Apr. 1944.

occupation. Rates again rose to over 0.92 per 1,000 average strength per annum in September 1945 and from October through December were less than 3 percent. During 1945, a total of 2,240 cases were reported in United States Army personnel.

No deaths from diphtheria occurred in the European theater in 1942 and 1943, and none in 1944 until operations started on the Continent. During the latter half of 1944, there were five deaths with a case fatality rate of 2 percent. In 1945, there were 2,240 cases with 67 deaths, or a case fatality rate of 3 percent.

While the experience of the occupation forces after 31 December 1945 is beyond the scope of this report, it is important to note that during 1946 diphtheria accounted for 15.3 percent of all deaths from disease conditions occurring in the theater and for 45.3 percent of all deaths occurring from communicable diseases. It caused twice as many deaths during that year as did the primary pneumonias.[15]

At the request of the Chief Surgeon, Headquarters, European Theater of Operations, and The Surgeon General, the author accompanied by Dr. J. Howard Mueller, consultant to the Secretary of War and member of the Commission on Epidemiological Survey, Army Epidemiological Board, made an investigation of problems of diphtheria in the military and civilian populations of the European theater during the period of 19 June to 18 August 1945, inclusive.[16] This survey revealed many things which had considerable bearing on the problems which were to develop among United States troops the following fall and winter. Diphtheria was prevalent among civilians but not excessively in the southern part of Germany and in Austria, the highest incidence being found in bombed-out cities such as Munich. In the northern cities, particularly Berlin, Bremen, and Hamburg, the rates were high even during the summer months. It was significant in Bremen, as well as elsewhere in Germany, that diphtheria was now a disease of adults, only 25 to 30 percent of the cases reported were children. With these great reservoirs of virulent *C. diphtheriae* in the civilian population, it is obvious why, as the rules against fraternization were relaxed, American troops in increasing numbers contracted the disease from their civilian contacts. Furthermore, it was very difficult to control the disease in the urban civilian populations which lived under conditions of extreme crowding and poor hygiene amidst the rubble of their bombed-out homes.

In the summer of 1945, it was recognized that diphtheria could be acquired venereally. In an evacuation hospital in Darmstadt, Germany, the author saw four cases of diphtheria in men from different units who apparently had contracted the disease from the same prostitute. The results of a carrier study made in April and May 1946 by Dr. Martin Frobisher, Jr., and Dr. Franklin H. Top, is summarized in table 31. This report is of interest in that it points

[15] Schulze, H. A.: Diphtheria in the U. S. Army in Europe. ETO M. Bull. 2 (No. 6): 2-26, June 1947.

[16] Letter, Dr. J. H. Mueller and Lt. Col. A. C. McGuinness, MC, to The Surgeon General, 15 Aug. 1945, subject: Survey of Problems of Diphtheria in ETO. Summary Report.

up the high carrier rates among German adult females in the Frankfurt region. Drs. Frobisher and Top, in a study of the age and sex distribution of diphtheria in three German population centers in September, October, and November, 1946, showed that almost twice as many cases were occurring in females as in males.[17]

The problem of management of carriers and steps taken to control diphtheria in the European theater is discussed in subsequent sections.

TABLE 31.—*Diphtheria carriers in U. S. Army personnel and German civilians in the European theater, April and May 1946*

Group cultured	Number cultured	Number of cultures used	Results			
			Number positive	Percent	Number doubtful [1]	Percent
Military personnel in Frankfurt 6 months or more:						
Male	382	332	6	1.8	15	4.5
Female [2]	210	210	2	.9	7	3.3
Male military personnel aboard ships in Le Havre immediately after arrival from United States	608	608	5	.8	4	.7
German civilians in Frankfurt:						
Male employees, Office of Military Government	210	210	2	.9	2	.9
Female employees, Office of Military Government	210	210	10	4.8	8	3.8
Females held as venereal disease suspects	200	150	11	7.3	8	5.3
German refugees [3]	605	605	7	1.1	15	2.5

[1] Cultures not morphologically typical, some of which may have been true diphtheria bacilli.

[2] Members of Women's Army Corps.

[3] Chiefly adult males and females, from towns near Stuttgart, Darmstadt, and Munich.

Source: Preliminary Report, Diphtheria Commission, March-May 1946, by Dr. M. Frobisher, Jr., and Dr. F. H. Top.

EXPERIENCE IN NORTH AFRICA, THE MIDDLE EAST, AND ITALY

Diphtheria had for years been known to be present in the civilian populations of the North African countries, with a moderate amount of the disease in the British Army, as already mentioned.[18] Statistics available (table 32) indicate that the amount of the disease among the civilian populations remained at a relatively constant level during the years in which United States forces were engaged in the North African and Middle East campaigns, although these figures cannot be considered entirely reliable.

[17] (1) See footnote 15, p. 174. (2) Preliminary Report, Diphtheria Commission, March-May 1946, by Dr. M. Frobisher, Jr., and Dr. F. H. Top.

[18] See footnote 1, p. 167.

TABLE 32.—*Incidence of diphtheria in civilian populations in North African countries, 1942–45*

Country	Number of reported cases			
	1942	1943	1944	1945
Egypt	3,950	4,143	3,326	3,130
Tunisia	107	86	147	106
Algeria	538	431	525	433
French Morocco	359	215	257	203

Source: Data for Egypt, Tunisia, and French Morocco from World Health Organization: Annual Epidemiological and Vital Statistics Reports, 1939–46, Part II, pp. 208, 216. Geneva, 1951. (2) Data for Algeria, L'Algérie, Service de Statistique Générale: Annuaire Statistique de l'Algérie, 1939–47. New series, vol. I, Alger, 1949, p. 83.

The situation in Italy was not clear early in the war, but figures now available are significant in that the total number of reported cases of diphtheria reached an 11-year low of 21,161 in 1941 and rose to 30,099, an 11-year high, in 1942.[19]

United States Army.—The Mediterranean theater (including North Africa) reported the highest rate for diphtheria during 1942–45. The rate for the Middle East theater, however, was one of the lowest recorded. During this period, there were 1,087 cases in the Mediterranean theater with the annual rate per 1,000 average strength being 0.73. Comparable data for the Middle East theater were 45 cases and an annual rate of 0.31 per 1,000 average strength (table 27).

The Theater Surgeon, NATOUSA (North African Theater of Operations, United States Army), reported in 1943 that mumps, measles, diphtheria, Vincent's infections, German measles, rheumatic fever, scarlet fever, chickenpox, smallpox, and whooping cough accounted for 1,678 (2.62 percent) of the total respiratory diseases. The only feature of this small group of cases is that they occurred predominantly in the early months of the year.[20]

Actually, as stated previously, there were only 45 cases of diphtheria reported from Africa and the Middle East from 1942 to 1945 (table 27). With the invasion and occupation of Sicily and the Italian mainland in the summer and fall of 1943, there developed a sharply increased awareness of the diphtheria problem. The Essential Technical Medical Data from NATOUSA for November 1943 called attention to the fact that diphtheria was slowly but definitely increasing and pointed out that patients were slow to arrive in medical installations. The Essential Medical Technical Data from NATOUSA for October 1943 reported 20 cases of diphtheria among prisoners of war admitted to the 56th Station Hospital and the 16th Evacuation Hospital during

[19] (1) Istituto centrale di Statistica del Regno d'Italia. Annuairo Statistico Italiano. Anno 1936-XIV; Anno 1937-XV, Anno 1938-XVI. (2) Istituto Superiore di Sanita: Andamento nel 1941 Dille Malattie Infettive e Diffusive Sogette a Denunzia Obbligatoria, 1946, p. 58. (3) Ibid: nel 1942, 1947, p. 67.

[20] Annual Report, Medical Section, Headquarters, NATOUSA, 1943, p. 110.

the months of June, July, and August of that year. Brig. Gen. Frederick A. Blesse, Surgeon, NATOUSA, issued Circular Letter No. 37, 2 October 1943, extending a warning to medical officers to be on the alert for the disease and pointing out the possibility that wound and anal diphtheria might occur. It is obvious, in retrospect, that many cases of diphtheria without complications were never diagnosed.

The year 1944 showed a sharp increase in the number of diphtheria cases in the Mediterranean theater—628 cases and a rate of 0.97 per 1,000 per annum as compared with 197 cases and a rate of 0.43 the preceding year (table 27). The NATOUSA Essential Technical Medical Data for March 1944 cited diphtheria reported in British (excluding Dominion or colonial troops) and American troops during a 3-month period as follows:

Month	*British*	*American*
December 1943	558	64
January 1944	490	42
February 1944	392	33

A high percentage of various types of paralyses has been noted in the course of diphtheria in the British forces. The majority of these paralyses occurred in patients whose diphtheria was cutaneous rather than pharyngeal in location and varied from mild local instances of neuritis to rather extensive polyneuritic disturbances.

This was highly significant in that it clearly pointed up the problems of cutaneous diphtheria and diphtheritic polyneuritis. Statistics are not available on the incidence of cutaneous diphtheria in United States Army troops in the North African and Mediterranean theaters, although it is known that it did exist, though obviously not to any marked extent. Infectious polyneuritis, however, was a problem of considerable interest and importance, and on 16 October 1944 the Surgeon, NATOUSA, listed infectious polyneuritis as one of the major disease problems of the theater and directed a study of the situation.[21] Diphtheria continued to be somewhat of a problem among troops in Italy through the winter of 1945, although, with the reduction in strength of United States troops in that theater as the war came to an end, the actual number of reported cases was not large. It would appear that cases developed sporadically in all sections of the theater and did not reach epidemic proportions in any specific areas or units. Because of the continuing presence of and interest in polyneuritis, Major Schoenbach and Dr. George D. Gammon of the Commission on Neurotropic Virus Diseases were sent by The Surgeon General to Italy during the summer of 1945 to investigate the polyneuritis situation with particular respect to its possible association with diphtheria. It was found impossible to differentiate the various types of polyneuritis by laboratory means; however, the history of diphtheria preceding the onset of polyneuritis in many cases and the high carrier rates for virulent *C. diphtheriae* found in the areas studied gave presumptive evidence that diphtheria was the etiologic agent in a substantial number of cases.

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[21] See footnote 3, p. 167.

EXPERIENCE IN THE PACIFIC

Available information was scattered at the time of Pearl Harbor and thereafter concerning diphtheria in the various sections of the Pacific where United States forces were to be deployed. From figures that could be obtained, there was at the time little indication the disease would present a serious problem. The following information on diphtheria in the various islands in the Pacific was compiled by the Medical Intelligence Division, Office of the Surgeon General:

Location	*Remarks*
Hawaiian Islands	Diphtheria cases reported regularly. The death rate from diphtheria was 1.4 per 100,000 population in 1940. The vast majority of reported cases occurred on the island of Oahu. In fiscal year 1942, 73 cases of diphtheria were reported, 65 of which occurred in Oahu (43 in Honolulu and 22 in the rural area), 2 occurred in Hawaii, 3 occurred in Maui and Lanai, and 3 occurred in Kauai. That year, no deaths from diphtheria were reported. In preceding years, the reported number of diphtheria cases were slightly larger, with a small number (2 to 6) deaths.
Gilbert and Ellice Islands, Ocean Island, and Nauru.	Diphtheria occurred occasionally in the Gilbert Islands but was not reported from Ocean Island or Nauru. Available sources did not state whether or not it occurred in the Ellice Islands.
Marshall Islands	Occurrence of diphtheria mentioned here, but no definite reports of cases found.
New Guinea	Diphtheria occasionally was reported from Dutch New Guinea. In 1937, an epidemic occurred along the north coast between Hollandia and Demta; 153 cases with 32 deaths were observed between August and November. Only sporadic cases were reported from British New Guinea. There was no information with regard to prophylactic vaccination, and the danger of development of major epidemics was considered serious.
Bismarck Archipelago	Diphtheria had occurred sporadically among both the European and native populations. It was felt that the danger of development of major epidemics was ever present in this population which had little natural immunity and no information regarding prophylactic vaccination.
Borneo	Diphtheria was endemic and frequently epidemic in most parts of the island. Between 1938 and 1944, several severe epidemics occurred.
Caroline Islands	Diphtheria was rare, but a few cases had been reported. In 1930, 10 cases (2 fatal) were reported from the naval hospital at Guam.
Fiji	Diphtheria was endemic. There were large numbers of carriers.
Formosa	Diphtheria had been reported but appeared to have been mild in character.

Location	*Remarks*
Izu, Bonin, Kazan, and Marcus Islands.	Diphtheria was common. There were 33 cases with 7 deaths reported in 1936, of which 14 occurred in the Izu Islands (Oshima, 4; Niijima, 8 (2 deaths); Hachijojima, 2 (1 death); and 19 with 4 deaths in Ogasawara (Bonin Islands).
British Solomon Island Protectorate.	No diphtheria was reported from these islands in the 1930's and early forties.
Mariana Islands	Diphtheria cases had been reported from the islands, but the disease was considered rare.
Molukken Islands and Islands in the Eastern Part of the Banda Sea.	Diphtheria had been reported only from Ceram. There the infection was formerly said to be rare but in recent years had been recognized.
Pitcairn Island	Diphtheria had not been reported.
Palau Islands	Although the disease is rare, cases of diphtheria had been reported from the islands.
Philippine Islands	A number of sporadic cases of diphtheria had been reported each year from the Philippines, but there had been no recent large epidemics. In 1938, there were 468 recorded cases with 157 deaths throughout the archipelago. Since 1925, there had been a progressive increase in the number of cases listed each year, but the mortality had remained more or less stationary. It was believed that the increase in reported incidence might be due to better reporting and to the establishment of more diagnostic laboratories, rather than to an actual spread of the disease.
Ryukyu Islands	Diphtheria was said to be common here although only 38 deaths in Kagshima Prefecture and 17 deaths in Okinawa Prefecture were reported from this cause in 1938.
Japan (excluding Okinawa)	The incidence of diphtheria was higher in Japan than in the United States, especially in some of the northern prefectures where the rates varied between 68 and 84 cases per 100,000 population in 1938. The disease increased moderately for Japan as a whole between 1938 and 1940, there being 28,420 cases with 3,853 deaths reported in 1938 and 38,412 cases with 4,288 deaths reported in 1940. It was felt that, with a deterioration in health conditions, including excessive overcrowding, diphtheria might become a disease of potential military importance.
Samoa	Though it was believed that diphtheria occurred in a mild form in many of the islands of the South Pacific, no reliable evidence was available to show that this disease was present in the Samoan Islands.
Tonga Islands	No reference to diphtheria was found.
Lesser Sunda and Southwestern Islands.	Diphtheria was not rare; acute outbreaks occasionally were observed. In 1935, an epidemic raged in Koepang. All the children were immunized, and the epidemic ceased.

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Contrary to expectations based on existing knowledge as to the prevalence of diphtheria in the Pacific, the disease proved somewhat troublesome, and cutaneous diphtheria was an unanticipated complication. As seen in table 27, the over-all morbidity rates for the years 1942–45 were 0.33 per 1,000 strength per annum for the Southwest Pacific (615 cases reported), and 0.41 per 1,000 per annum for the Pacific Ocean Area (519 cases reported). Undoubtedly, these represent only a fraction of the total; in this theater, as well as in others, many cases were not diagnosed unless complications developed.

United States Marines invaded Guadalcanal in August 1942, and that island finally was evacuated by the Japanese in February 1943. Early in 1943, the presence of diphtheria, both cutaneous and pharyngeal, was recognized among Army troops who had served in the Solomon Islands.[22] On 20 October 1943, Col. (later Brig. Gen.) Earl Maxwell, Surgeon, United States Army Forces, South Pacific Area, issued Medical Circular Letter No. 5 calling attention to the following:

> Contrary to common belief infections with *C. diphtheriae* have not been rare in this theater. As a certain number of these infections have been overlooked, occasionally until the development of neuritis has provoked further investigation, it is thought wise to direct attention to them.

In this same letter attention was called to cutaneous diphtheria in the form of tropical ulcers.

On 16 March 1944, Col. Benjamin M. Baker, Jr., MC, theater consultant in medicine, sent a detailed report to General Maxwell [23] which gave an account of studies in Bougainville and Fiji on troops evacuated from the Solomon Islands campaign. Approximately 25 cases were from the 164th Infantry Regiment, and 100 cases from the 25th Infantry Division. A later report [24] stated that of 291 cases of diphtheria in 1944 among troops who had served in the Solomons, 155 (or approximately one-half) were of the cutaneous type. In Colonel Baker's letter of 16 March it was stated further that both bulbar and peripheral neuritis have followed the two types of disease encountered. Approximately 2,800 men of the 164th Infantry Regiment were Schick-tested and 38 percent had positive reactions. It was of interest that 22 of 54 cases of cutaneous diphtheria, from whom virulent *C. diphtheriae* had been isolated, were in individuals who had been Schick positive at the time of original examination. Many, but not all, of these subsequently became Schick negative. All those who showed Schick-positive reactions in the 164th Infantry Regiment and 25th Infantry Division received 0.5 cc. of alum precipitated toxoid followed by 1.0 cc. Approximately 50 percent of those injected developed local reactions of moderate severity, and 10 percent developed incapacitating febrile reactions necessitating hospitalization for several days. "The measure [immunization] coupled with ordinary isolation of cases controlled the spread

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[22] Stevens, F. W.: Medicine—South Pacific Area. [Official record.]

[23] Letter, Col. B. M. Baker, MC, to Brig. Gen. Earl Maxwell, Headquarters, U. S. Army Forces, South Pacific Area, 16 Mar. 1944.

[24] Annual Report, Headquarters, South Pacific Base Command, 1945, p. 8.

of diphtheria in the 164th Infantry Regiment miraculously." This, incidentally, is one of the few reports of large-scale immunization of combat troops in World War II.

The story of diphtheria in troops engaged in the Solomon Islands campaign was repeated in Saipan and to a lesser degree in Biak, Leyte, Hollandia, and other areas. An excellent, detailed study of the problem of cutaneous diphtheria was made by Lt. Col. Averill A. Liebow, MC, Maj. Paul D. MacLean, MC, Lt. Col. John H. Bumstead, MC, and Maj. Louis G. Welt, MC, all of the 39th General Hospital.[25]

A survey of native populations of the Solomon Islands, the New Hebrides, and the Marianas revealed widespread [illegible]ence of virulent *C. diphtheriae*, chiefly in skin lesions. Most of the p[illegible] these islands over 3 years of age were Schick negative; obviously th[illegible] become immune as a result of contact with the organisms through skin lesio[illegible] rather than through pharyngeal infections which appeared to be unusual.

EXPERIENCE IN INDIA-BURMA THEATER

No health statistics were available for India and Burma following 1939. However, the Medical Intelligence Division, Office of the Surgeon General, reported that, in 1939, Burmese hospitals had treated 646 persons for diphtheria of whom 22 had died, thus pointing at least to the existence of diphtheria at that time. Information from India was scattered, and there was evidence merely to indicate that the disease did occur among the native populations of most provinces. Bensted,[26] in 1936, reported an outbreak of cutaneous and faucial diphtheria among British troops in northwest India, and Hamburger[27] described cutaneous diphtheria in northeast India in 1939.

Col. Herrman L. Blumgart, MC, and Maj. George M. Pike, MC, recorded the high incidence of diarrheal diseases and malaria in India-Burma and the serious degree to which they contributed to the noneffective rate in that theater.[28] They added "scrub typhus and cutaneous diphtheria, though less important statistically, hampered military operations because of their occurrence in combat areas and the serious disability which they occasioned." As shown in table 27, there were 208 cases reported for the theater for the period January 1942 through December 1945, with an over-all rate of 0.47 per 1,000 strength per annum. Undoubtedly, this rate is below the actual occurrence of the disease.

On 8 October 1944, Maj. Clarence S. Livingood, MC, Chief, Section of Dermatology and Syphilology, 20th General Hospital in Assam, reported[29]

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[25] Liebow, A. A., MacLean, P. D., Bumstead, J. H., and Welt, L. G.: Tropical Ulcers and Cutaneous Diphtheria. Arch. Int. Med. 78: 255-295, September 1946.

[26] See footnote 2, p. 167.

[27] Hamburger, H. J.: Observations on the Pathology and Therapy of the So-Called Frontier Sore. Indian M. Gaz. 74: 151-155, March 1939.

[28] Blumgart, H. L., and Pike, G. M.: History of Internal Medicine in India-Burma Theater. [Official record.]

[29] Letter, Maj. C. S. Livingood, MC, to Commanding Officer, 20th General Hospital, India-Burma Theater, 9 Oct. 1944, subject: Cutaneous Diphtheria.

on the problem of cutaneous diphtheria which had begun at that hospital the preceding June. Approximately 83 such cases had come under observation. The great majority of these cases had acquired the disease in combat in the Myitkyina area under circumstances of poor hygiene, wet and soiled clothing, insect bites, abrasions, and the like. As of December 1944, a total of 140 cases had been treated at the 20th General Hospital, and a detailed report was issued by Major Livingood in January 1945 [30] in which the following 20th General Hospital officers collaborated: Lt. Col. (later Col.) James S. Forrester, MC, Chief, Laboratory Section; Maj. Herbert S. Gaskill, MC, Chief, Neuropsychiatry Section; and Maj. Calvin F. Kay, MC, Chief, Cardiovascular Section.

One of the problems associated with cutaneous diphtheria proved to be the question of bacteriologic diagnosis. Among a group of 119 clinically diagnosed cases at the 20th General Hospital, virulent *C. diphtheria* were isolated from 21 percent, diphtheroids from 46.8 percent, and other organisms from 32.2 percent. As the experience of the laboratory personnel increased the percentage of isolations improved, yet virulent organisms never were recovered from many cases, especially those which were first seen a number of weeks after the onset of the lesions.

Postdiphtheritic polyneuritis occurred in about 34 percent of the cases of cutaneous diphtheria, and myocarditis in about 3 percent.

While faucial diphtheria unquestionably was present in the India-Burma theater, it appeared to have offered no particular problem.

Because of the importance of cutaneous diphtheria as reported from both India-Burma and the Pacific, War Department Technical Bulletin 143 was published and circularized by The Surgeon General in February 1945.

EXPERIENCE IN OTHER AREAS

From January 1942 to December 1945, inclusive, a total of 19 cases of diphtheria were reported from the North American area (including Alaska and Iceland), and 23 from Latin America, with morbidity rates of 0.04 and 0.06 per 1,000 per annum, respectively (table 27). Cases were scattered and the disease caused no particular problem. Similarly, in the Persian Gulf Command only a few scattered cases of diphtheria occurred and these also caused no particular difficulty.[31]

INCIDENCE AMONG PRISONERS OF WAR

During the summer of 1943, German prisoners captured in the North African campaign began to be brought to the United States for internment. Many of these prisoners apparently were carriers of virulent *C. diphtheriae*;

[30] Letter, Maj. C. S. Livingood, MC, to Commanding Officer, 20th General Hospital, India-Burma Theater, 25 Jan. 1945, subject: Cutaneous Diphtheria.

[31] Annual Reports, Headquarters, Persian Gulf Command, 1943; 1945 (1, 2d, and 3d quarters).

several outbreaks of diphtheria occurred, although in general, the disease was well confined to the prisoner groups. One such outbreak among prisoners interned at Aliceville, Ala., has been described in detail by Capt. Stephen Fleck, MC, Capt. (later Maj.) John W. Kellam, MC, and Maj. (later Lt. Col.) Arthur J. Klippen, MC.[32] Fifty-one cases of diphtheria were diagnosed among a group of approximately 5,000 prisoners over a period of about 2 months.

Diphtheria was a serious problem among German prisoners confined in enclosures on continental Europe, particularly in southern France. Col. John E. Gordon, MC, described the situation in the European Theater of Operations as follows:[33]

> During the period of active operations more than twice as many cases of diphtheria occurred among German prisoners of war than among the much greater numbers of American troops. From September 1944 to June 1945, inclusive, diphtheria cases among United States troops numbered 1,202; and for prisoners of war the figure was 2,859. The rates were of course far greater, in the order of about ten times * * * .
>
> Only incomplete data are available for deaths. The Advance Section of Communications Zone cared for 695,400 prisoners during the six-week period from 1 May to 15 June. During that time, 1,080 cases of diphtheria occurred among the prisoners, of whom 40 died. The mortality rate per thousand per year was thus 0.499 and the case fatality 3.7 percent. Prisoners of war included numbers of relatively young persons, some aged no more than 14 to 16 years, and the greater case fatality was therefore not altogether unexpected.

The carrier rates in some of the prisoner-of-war enclosures were exceedingly high. In some groups sampled by Dr. Mueller and the author during July 1945, as many as 10 percent harbored virulent *C. diphtheriae*. These high carrier rates prevailed chiefly in the enclosures in southern France where prisoners had been confined since the winter and early spring of 1944–45. Among groups of prisoners taken in the final months of the invasion of Germany there was less diphtheria, and the carrier rates were substantially lower, in the range of 1 to 2 percent.

TYPES OF VIRULENT *C. DIPHTHERIAE* ENCOUNTERED

The British for some years have placed emphasis on the relative virulence of the recognized types (gravis, mitis, and intermedius) of *C. diphtheriae*. Cruickshank[34] wrote in 1943 as follows:

> A knowledge of the infecting type should be of some help to the clinician in his handling of a case of diphtheria. The severity of the infection and the incidence of complications varies with the type, the more severe toxaemic infections with a fairly high incidence of paralysis (10–15%) being due to *gravis* and *intermedius* types, while *mitis* is nearly always associated with a mild infection except when it produces laryngeal diphtheria.

Brigadier R. E. Tunbridge, medical consultant to the 21st Army Group, British Land Army at Bad Oeynhausen in Germany, stated that in a series of approximately 400 consecutive cases of diphtheria in which careful typing of

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[32] Fleck, S., Kellam, J. W., and Klippen, A. J.: Diphtheria Among German Prisoners of War. Bull. U. S. Army M. Dept. No. 74, 80–89, March 1944.

[33] See footnote 13, p. 171.

[34] Cruickshank, R.: Diphtheria; Laboratory Aspects. Pub. Health 57: 17–19, November 1943.

all strains was done, the complication rate following infection with the gravis type was about 25 percent whereas the complication rate following infection with the mitis type was about 12 to 14 percent.[35] At the time, Brigadier Tunbridge stated that the British had picked up a few cases of diphtheria due to the intermedius-type organism, but a sufficient number of these cases was not available to permit conclusions as to how the complication rate following intermedius infection compared with the rates following infection with the other two types.

The only area in which type studies were carried out to any degree in United States troops was the European theater, and then only after the cessation of hostilities. From surveys and other information secured by Dr. Mueller and the author during the summer of 1945, approximately 80 percent of the strains on continental Europe were of the mitis type and 20 percent of the gravis type. No intermedius-type strains were picked up that summer. It was impossible to obtain any information which would permit correlation with strain type and severity of infection or rate of complications. Most United States Army medical officers, as well as a number of German physicians, were of the opinion that there was no relationship between type of organism and severity of infection.

There is practically no information concerning the types of *C. diphtheriae* encountered in the Pacific, although in a report on cutaneous diphtheria in United States troops in the Pacific, by Lieutenant Colonel Liebow and associates,[36] the statement was made that all organisms found were of the mitis type and that no organisms of the gravis type had been encountered.

No information is available concerning types of virulent *C. diphtheriae* encountered among United States troops or prisoners of war in the Zone of Interior. In this connection, it should be pointed out that American authorities have not shared the British opinion as to the importance of strain type in respect to virulence and likelihood of complications, and the only major attempt to type organisms was made in the European theater after the cessation of hostilities in the summer of 1945.

CONTROL MEASURES

Army Regulations

AR 40–210, 15 September 1942, provided that patients known to be ill with diphtheria be hospitalized and isolated, that contacts be inspected daily for 5 days, that contacts be excluded from foodhandling until shown to be free from virulent *C. diphtheriae*, and that known carriers be isolated and given suitable treatment. Provision was made for immunization against diphtheria "when in the opinion of the surgeon this procedure is necessary for the prevention or control of diphtheria in the command." These provisions were included

[35] Tunbridge, R. E.: Personal communication to author at Bad Oeynhausen, Germany, 30 July 1945.

[36] See footnote 25, p. 181.

in essentially the same form in AR 40-210 as revised and published on 25 April 1945. Obviously, the implementation of these regulations was dependent upon recognition of the disease. Through lack of experience with diphtheria in civilian training and practice, the United States Army medical officer of World War II initially was weak in the recognition of this disease. In those theaters where diphtheria was encountered to any degree, Army physicians learned rapidly much about the disease, and through their experience medical officers in other theaters were alerted.

The Laboratory

Laboratory officers, like clinicians, for the most part had to gain their first major experience with *C. diphtheriae*. The position of the laboratory was an important one in the detection and management of carriers as well as in confirmation of the clinical disease. A number of instances are on record where laboratory errors resulted in misdiagnosis of pharyngitides, and in hospitalization and quarantine of carriers of nonvirulent diphtheroids. Again, as war progressed and laboratory officers had increased experience, correlation between clinician and bacteriologist reached a high standard of efficiency. It would seem appropriate to quote here a statement by Dr. Mueller:[37]

* * * It may not be out of place to refer briefly to the purpose of the laboratory examination for the diphtheria bacillus and to how much the clinician should expect from it. The laboratory cannot "diagnose" diphtheria—that is the function of the physician. The bacteriologist may be able to state, following a delay of 12 to 15 hours, that organisms which he believes are consistent in morphology with *C. diphtheriae* are present in his culture. If he has had long practical experience in the matter, he may be able to make a similarly tentative statement even sooner, by examination of a direct smear made from the throat swab, but such an opinion is best not ventured by the inexperienced. Moreover, the failure to observe the organism in early, or even later, culture by no means excludes diphtheria in the patient. An improperly taken throat swab may yield entirely negative results, although more careful subsequent culturing may show the organism to be present abundantly in certain areas.

Under optimal conditions, the laboratory can report after from 2 to 4 days that a virulent diphtheria bacillus has been obtained from the culture. This does not of itself establish a clinical diagnosis of diphtheria, for the condition may have occurred in the throat of an immune carrier and may have been entirely nondiphtherial in nature. The decision as to the initial diagnosis and treatment of the case is the direct and immediate responsibility of the physician.

It must be recognized that even in the best of hands, and under ideal circumstances, the complete laboratory diagnosis of diphtheria (including virulence testing) requires time, considerable glassware and media, and an animal colony. Under conditions of combat and rapid movement, it was impossible to provide all the refinements necessary for good laboratory control of diphtheria, and such facilities during a large part of the war were limited largely to the army laboratories, and to a few of the more or less fixed general

[37] Mueller, J. H., and Miller, P. A.: A New Tellurite Plating Medium and Some Comments on the Laboratory "Diagnosis" of Diphtheria. J. Bact. 51: 743-750, June 1946.

hospitals. It is obvious that a great need exists for relatively simple laboratory procedures for the identification and virulence testing of *C. diphtheriae.*

After the cessation of hostilities in Europe in May 1945, very active measures were taken to improve the management and control of diphtheria in the European theater. At the request of Maj. Gen. Paul R. Hawley, Chief Surgeon, European Theater of Operations, The Surgeon General sent Dr. Mueller and the author to the European theater on 16 June, and during the next 2 months these consultants conferred with medical officers at all levels of command in most of the major installations on the Continent. Particular emphasis was placed on the establishment of uniform bacteriologic techniques. Circular Letter No. 69, Headquarters, Theater Service Forces, European Theater of Operations, 28 September 1945, outlined in great detail the techniques of the bacteriologic diagnosis of diphtheria, and introduced the use of the new tellurite-plating medium which had been developed by Dr. Mueller. This plating medium offers a greatly improved method of screening large numbers of throat cultures as compared with the traditional examination for the diphtheria bacillus based on the microscopic appearance of a stained smear from a culture on Löffler's medium. Furthermore, it makes possible a gross differentiation of the several types (mitis, gravis, and intermedius) of *C. diphtheriae.*

Immunization

As stated before, AR 40–210, 15 September 1942 and 25 April 1945, provided for immunization against diphtheria "when in the opinion of the surgeon this procedure is necessary for the prevention or control of diphtheria within the command." Routine immunization against diphtheria was not recommended for several reasons. Although it was recognized that the age distribution of diphtheria had been shifting in recent years and that the disease was becoming more and more one of young adults, it was thought that a sufficiently high proportion of United States troops possessed actual or latent active immunity to diphtheria to prevent a significantly high incidence of the disease, except under unusual conditions. An important consideration in the decision not to immunize troops routinely was based on the knowledge that injections of diphtheria toxoid would be followed by moderate to severe reactions in an appreciable number of cases.

Instructions on the subject of active immunization against diphtheria in World War II were first issued in the Surgeon General's Circular Letter No. 162 in 1942. These instructions recommended plain or fluid toxoid in doses of 0.5, 1.0, and 1.0 cc., given subcutaneously at intervals of approximately 3 weeks. Reference also was made to the reactions to be expected, and it was recommended further that immunization be limited to Schick-positive individuals and then only in the presence of a definite hazard from the disease. Because of the unexpected occurrence of diphtheria in United States troops in the Pacific, the presence of the disease among troops in Africa and the threat of diphtheria on the continent of Europe, TB MED (War Department Technical

Bulletin (Medical)) 47, published on 28 May 1944, contained a detailed description of the disease and recommendations concerning immunization similar to those contained in Circular Letter No. 162 (1942) as described. On the basis of the studies previously mentioned which were conducted at Camp Ellis and Camp Tyson by Colonel Long and associates,[38] TB MED 114 was published on 9 November 1944 in which a number of changes were made in the immunization recommendations. These recommendations follow:

* * * When time and facilities permit, preliminary Schick testing may be done and only the positive reactors should be immunized. However, because of the time required, the meticulous care necessary to obtain reliable results, and other inherent difficulties, mass Schick testing will seldom be feasible and the entire group requiring immunization should be given toxoid [plain] in the measure described below.

* * * * * * *

Method of immunization. Reactions to diphtheria toxoid are more common in adults than in children and, therefore, it is desirable to begin with a dosage of 0.1 cc., subcutaneously, and to limit further immunization to those who do not react severely to this test dose. The occurrence, after any dose in the series, of local edema or induration more than 6 cm. in diameter, or a marked constitutional reaction with fever over 101° F., is a contraindication to further doses. The group given the test dose should be inspected after 48 hours. Those who have not experienced severe reactions may be given the first regular immunizing dose of 0.5 cc. at this time. From this point subsequent doses are given at 3-week intervals, the second and third immunizing doses being 1.0 cc. Even though the entire series cannot be completed for some individuals because of reactions, this procedure should raise the general level of immunity sufficiently to prevent an epidemic of diphtheria.

In general, these recommendations concerning the technique of immunization were employed for the remainder of the war.

In the fall of 1945, because of the sharp increase in diphtheria among the personnel of certain general hospitals in the United States, Army Service Forces Circular No. 415, dated 9 November, directed that all such persons coming into contact with patients be Schick tested and those showing positive reactions be immunized. This is the only instance during the war of the application in this country of a diphtheria-immunization program instituted by War Department directive. Up to the end of 1945, diphtheria immunization was carried out only in a few instances in overseas installations. Some immunizations were done in the North African-Mediterranean theater because of the high incidence of diphtheria among civilians. In 1944, Schick testing followed by immunization was carried out in the 64th Infantry Regiment of the 25th Infantry Division in Bougainville and the Fiji Islands. Although 10 percent of those injected (0.5 cc. followed by 1.0 cc.) developed incapacitating febrile reactions, the measure was considered to have been instrumental in controlling the spread of diphtheria in the regiment.[39] Small immunization programs also were conducted in the Persian Gulf Command,[40] and in the Alaskan Wing, Air

[38] See footnote 11, p. 170.

[39] See footnote 23, p. 180.

[40] Monthly Sanitary Report, Persian Gulf Command, 8 Mar. 1944.

Transport Command.[41] In all instances, attention was drawn to the relatively high frequency of febrile reactions.

The great increase in diphtheria among troops stationed in the European theater in the summer and early fall of 1945 raised strongly the question as to whether immunization should be made mandatory for hospital personnel, at least. Because of the problem of reactions, however, mandatory immunization was not adopted, and as a compromise Circular Letter No. 69, Headquarters, Theater Service Forces, European Theater of Operations, 28 September 1945, prescribed that "where practicable, only immune personnel will be utilized in caring for diphtheria patients."

Control of Carriers

In those theaters and regions where diphtheria was prevalent, control of carriers was a difficult problem throughout the war. Particular difficulty was encountered in the management of individuals with chronic cutaneous diphtheria. It was frequently very difficult to isolate organisms from these skin lesions, and yet experience indicated that patients with chronic skin lesions were at times serious sources of contagion.

The following statement was made in TB MED 47, 28 May 1944:

> *Carrier control.* Before releasing carriers from isolation, negative cultures should be required, as stated in paragraph 15 d (3), sec. IV, AR 40-210. If cultures have not become negative within 4 weeks, consideration should be given to the removal of tonsils and adenoids or [to] other appropriate treatment. Antitoxin is of no value in the treatment of carriers.

Although statistics are not available, the impression has been gained that many of the chronic carriers were individuals with hypertrophied tonsils and that final clearance of infection frequently was not accomplished until the tonsillar tissue had been removed.

Fleming, in 1929,[42] demonstrated that penicillin inhibited the growth in vitro of diphtheroid bacilli and *C. diphtheriae*. On the basis of this information, a study was conducted in the spring of 1945 among personnel and patients of the 3d General Hospital at Aix-en-Provence, France. The results of this study, which was carried out by Lt. Col. (later Col.) Samuel Karelitz, MC, Capt. Ralph E. Moloshok, MC, and Capt. (later Maj.) Louis R. Wassermann, MC,[43] indicated that large doses of parenterally administered penicillin might be of value in clearing up the chronic carrier state. These investigators also concluded that if penicillin was administered in large doses, in addition to antitoxin, early during the acute disease, fewer patients advanced to the chronic carrier state. They emphasized emphatically that penicillin was not a substitute for antitoxin in the treatment of the acute case. Colonel Karelitz

[41] Monthly Sanitary Report, Alaskan Wing, Air Transport Command, March 1944.

[42] Fleming, A.: On the Antibacterial Action of Cultures of a Penicillium, With Special Reference to Their Use in the Isolation of *B. Influenzæ*. Brit. J. Exper. Path. 10: 226–236, June 1929.

[43] Karelitz, S., Moloshok, R. E., and Wassermann, L. R.: Penicillin in the Treatment of Diphtheria and the Diphtheria Carrier State. ETO M. Bull. 32: 67–72, July–August 1945.

and his group found that in their experience local application of penicillin in the form of gargles was of no value in either the acute disease or the carrier state. Capt. Harold W. Muecke, MC, conducted studies of penicillin in the treatment of carriers at the 28th General Hospital near Sissonne, France.[44] His studies resulted in conclusions essentially similar to those drawn by Colonel Karelitz and his associates. Essentially, the same findings were obtained in a study conducted under the direction of Col. Ross Paull, MC, at the Letterman General Hospital, San Francisco, Calif., during the winter and spring of 1945.[45]

The situation with respect to carrier control as of the end of 1945 might be summarized as follows:

1. Accurate determination of the carrier state was dependent upon good techniques in taking nasopharyngeal cultures supported by good bacteriologic techniques in the laboratory, both prerequisites frequently were lacking.

2. Many chronic carriers were individuals with chronically hypertrophied tonsillar and adenoid tissue, the removal of which often was necessary to secure a cure.

3. Evidence was available which indicated that parenteral penicillin in large doses was effective in shortening the carrier state when administered as well as late in the course of the disease.

4. Antitoxin again was found to be of no value in the treatment of the carrier state.

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[44] Muecke, H. W.: Personal communication to author, 9 Aug. 1945.

[45] Letter, Col. Ross Paull, MC, to Lt. Col. A. P. Long, MC, 8 Sept. 1945.

CHAPTER XI

Meningococcal Meningitis

John J. Phair, M. D.

Relationships between civilian and military medicine are well illustrated by the history of meningococcal meningitis in this country as a whole and in the United States Army. The concern of both in the prevention and treatment of the disease has been deep and constant over the years. Progress made by one has been applied also by the other. Civilian and military experts, often the same individual under different dress, have served both the civilian populations and the Armed Forces. This was notably true of the vigorous and intelligent programs of the Preventive Medicine Service, Office of the Surgeon General of the Army, and of the Commission on Meningococcal Meningitis, Army Epidemiological Board, which was established and supported by The Surgeon General.

This chapter is divided into three main sections:

1. Meningitis as a military health problem up to and including World War I. This portion covers the years from about 1805 to 1919 when it was almost impossible to control the disease.

2. Meningitis between World War I and World War II, which covers the period from 1919 to 1939, inclusive. It was during these 20 years, in 1934, that the sulfonamides came into use and completely changed the therapy, prognosis, and possibilities of at least temporary prevention of meningococcal meningitis.

3. Meningitis in the Army during World War II (1941–45). There were many advances made by the study of the disease in military personnel, the dynamics of subclinical meningococcal infections were studied, and methods of chemoprophylaxis using sulfonamides (chiefly sulfadiazine) were developed. It was a period of intense activity in this field by the Preventive Medicine Service and the Commission on Meningococcal Meningitis.

PERIOD UP TO AND INCLUDING WORLD WAR I

Meningococcal meningitis has always been one of the most serious and important of the various communicable diseases of man, insofar as the United States Army has been concerned. Apparently impossible to control in any practical sense and difficult to treat, it has been a constantly present and vexing problem for everyone responsible for the health of any of the branches of the Armed Forces. The gravity of this infection arises not because of its incidence,

which has always been low when compared with other upper respiratory diseases, but because of the usually extraordinarily high case fatality rate. As a matter of fact, during World War I, while this disease ranked only 76th as a cause of admission to a hospital, of the cases reported approximately 40 percent were fatal, bringing it up to 6th as a cause of death among Army personnel.

The sudden onset, the striking clinical picture, and the high case fatality rate have always served to delineate cerebrospinal fever sharply for the laity as well as physicians. Other important factors which distinguished this infection were the lack of a successful therapeutic program, the usual failure to demonstrate any degree of association between the clinical cases, and the fact that quarantine or similar control measures generally have proved ineffective. Therefore, the appearance of a single case in a command or in a neighboring civilian community was, and is, followed by an unwarranted apprehension and alarm on the part of all individuals who had had any possible degree of contact with the patient. The orderly flow of training, reception and shipment of personnel, and housing was usually disrupted immediately and completely. As a consequence, few illnesses were given more attention by line and medical officers. Since the anxiety and fear of attack is always inversely related to the extent of knowledge possessed as to the modes of transmission and efficacy of preventive procedures, this interest and concern of the military was natural and excusable.

The historical record of cerebrospinal meningitis as a clinical and epidemiologic entity opens early in the 19th century with the description of an outbreak in Geneva, Switzerland, which began in March 1805, and another in Medford, Mass., during March 1806. Isolation of the causative organism by Weichselbaum [1] in 1887, confirmed by the extensive studies of von Lingelsheim [2] in 1905, permitted a firm etiologic diagnosis based upon the demonstration of the causative organism in the spinal fluid.

Hirsch,[3] in his classical work, has compiled descriptions of epidemics which were published in the medical literature from 1805 to 1882. Netter and Debré [4] supplemented those accounts and completed the record up to 1911. Heiman and Feldstein [5] have cataloged outbreaks in the United States. Similar monographs were published by Dopter,[6] Sophian,[7] and Worster-Drought and Kennedy.[8] From accounts gathered from these references, it is possible to piece together a partial mosaic of the frequency and extent of major epidemics

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[1] Weichselbaum, A.: Ueber die Aetiologie der akuten Meningitis cerebro-spinalis. Fortschr. d. Med., Berlin 5: 573-583, 1887.

[2] von Lingelsheim, W.: Die bakteriologischen Arbeiten der kgl. hygienischen Station zu Beuthen O.-Schles. während der Genickstarreepidemie in Oberschlesien im Winter 1904-5. Klin. Jahrgb. Jena 15: 373-488, 1906.

[3] Hirsch, August: Handbook of Geographical and Historical Pathology, vol. III, ch. 18. London: The New Sydenham Society, 1883-86.

[4] Netter, A., and Debré, R.: La Méningite Cérébro-spinale. Paris: Masson et Cie, 1911.

[5] Heiman, Henry, and Feldstein, Samuel: Meningococcus Meningitis. Philadelphia: J. B. Lippincott Co., 1913.

[6] Dopter, C.: L'Infection Méningococcique. Paris: J. B. Baillière & Fils, 1921.

[7] Sophian, Abraham: Epidemic Cerebrospinal Meningitis. St. Louis: C. V. Mosby Co., 1913.

[8] Worster-Drought, Cecil C., and Kennedy, Alexander M.: Cerebro-Spinal Fever: The Etiology, Symptomatology, Diagnosis and Treatment of Epidemic Cerebro-Spinal Meningitis. New York: The Macmillan Co., 1919.

which have occurred in Western civilizations during the 19th and beginning of the 20th century.

Granting the inadequate and chance nature of the picture which can be elicited from these historical descriptions, it is nevertheless apparent that there were periods when the disease was excessively prevalent over large geographic areas and others when it was relatively quiescent. There were at least five intervals when the disease was apparently epidemic and not limited by natural boundaries; namely, 1838–50, 1865–70, 1886–88, 1896–1905, and 1909–10.

Meningococcal meningitis became epidemic again shortly after the outbreak of World War I. In England, France, Germany, and probably in other western European countries, the crest of the epidemic wave was observed in 1915, but in Denmark it came a year later. These accounts have not been reviewed in detail since they are readily accessible in the references which have been cited.

The military, for many reasons, has always been selected as one of the principal occupational classes on which the disease falls most heavily. Without a doubt, it has been present among personnel of the United States Army during all wars and mobilization periods. Interesting clinical reports of outbreaks are recorded in the histories of the War of 1812, the Mexican War, and the Civil War. No reasonable comparison of the incidence rates is possible because of the considerable confusion in nomenclature and the inability to make a satisfactory differential diagnosis without the demonstration of the etiologic agent. In spite of the fact that the meningococcus had been recognized as the specific cause of cerebrospinal meningitis 10 years prior to the Spanish-American War, very few cases which occurred among Army personnel during that conflict were confirmed by laboratory methods. It is more than probable, therefore, that many of the cases were confused with typhus, typhoid, and other fevers. Since the turn of the century, however, with the increased use of laboratory facilities, diagnosis became more exact, and records have included usually only those cases in which the clinical impression has been confirmed by appropriate examinations.

It is noteworthy in studying these records that the admission rates for the Army have always been negligible except during the periods of rapid and extraordinary mobilization of unseasoned personnel. For example, in 1907 at the time of the Cuban occupations, the rate increased nearly tenfold. Again in 1913, during the military activity on the Mexican border, the admission rate more than doubled. However, in contrast, it is also important to remember that the subsequent concentration of solely Regular Army troops on the Mexican border in 1911 was not accompanied by a similar rise.

For several years before the onset of World War I, meningococcal meningitis, as indicated by the weekly morbidity and mortality reports of the United States Public Health Service, had been noticeably prevalent and widely distributed among the civilian population of the United States. In 1917, the rapid mobilization of over a million raw susceptible recruits from all sections of the country was followed by the outbreaks of the disease in every camp

and installation. The close intimate contact under unusually crowded barrack conditions provided ideal soil and opportunity for the growth and dissemination of meningococci. As a consequence, the annual admission rate among enlisted men in the United States increased rapidly to a peak of 4.6 per 1,000 in January 1918. From this crest, the number of cases fell gradually with the exception of a small rise in October 1918 to the low point of 0.07 during the months of October, November, and December, 1919. The incidence of this disease in many camps was well above 2 per 1,000 per annum; for instance, the high at Camp Beauregard, La., was recorded as 12.8, and at Camp Jackson, S. C., 25.7 per 1,000 per annum. These attack rates are far greater than those ordinarily found in adult civilian populations living under customary urban relationships.

The disease occurred sporadically in the Army Expeditionary Forces rather than in extensive epidemics and at a lower rate than that found in the continental United States. A large proportion of the European cases could be traced to contacts within receiving ports or on shipboard, and usually the morbidity rates were highest in organizations originating from training camps in the United States having an epidemic incidence. It was commonly noted that more than 50 percent of the cases in any week, during the period troops were arriving from the United States, were reported from ports, and, in most cases, it is easy to assume that the exposure occurred during the voyage to England or France.

Comparison between the monthly incidence rates of the American Expeditionary Forces and of the British and French almost always shows higher rates in the American troops for this period. The British Expeditionary Force in France was attacked with some severity, the French Army suffered less, and the German Army hardly at all.

Etiologic Agent and Mode of Transmission

Meningococcal meningitis is an infectious disease caused by an obligate parasite of man, the meningococcus, a member of the genus *Neisseria* (*Neisseria intracellularis*, *Diplococcus intracellularis meningitidis*, *Neisseria meningitidis*).

The diagnosis of the infection rests upon a combination of clinical and laboratory findings. In cases with frank meningitis, the causative organism is always present in the cerebrospinal fluid; in a considerable proportion of cases, it is also demonstrable in the nasopharynx. Early in the disease, blood cultures are, likewise, positive in 40 to 60 percent of the patients. The organisms can be seen in stained smears of spinal fluid and blood expressed by puncturing the petechiae. The demonstration of the etiologic agent requires, however, an adequate, meticulous technique and a thorough understanding of the pathogenesis of the disease so that specimens can be obtained from the proper sites during optimal periods.

In 1887, Weichselbaum [9] of Vienna published his classical paper describing the finding of this organism in six patients. His work was quickly confirmed by Goldschmidt,[10] but it was not until the extensive studies of von Lingelsheim [11] during an epidemic of 1904–5 that his observations were finally confirmed. Dopter [12] described parameningococci in 1909 since some confusion had arisen over the failure of monovalent serum to agglutinate all strains of meningococci. As soon as the so-called parameningococci were found in typical clinical cases, various systems for serologic classification of this organism were developed. These systems employed agglutination, agglutinin absorption, complement fixation, and chemical fractionation. Classification of strains into four types was generally accepted as a result of this work.

The host response originally recognized and described is similar to other types of purulent inflammation of the meninges and is characterized clinically by an intense headache, emesis, stiff neck, positive Brudzinski's signs, and in the more severe reactions by coma and convulsions.

In some of the pioneer pathologic studies, the presence of a purulent rhinitis was recorded and some workers suggested the nasopharynx as a possible portal of entry. Weichselbaum [13] in his original paper mentioned that one of his patients had a purulent sinusitis and expressed an opinion that the organisms might find their way to the meninges via the nose. Subsequently, many other investigators isolated the organism not only from the nasal passages of patients but from healty contacts and even individuals without a history of the slightest exposure to the disease. So it was generally accepted that the meningococcus could proliferate in the nasopharynx without giving rise to symptoms and that it could be transmitted from patients to healthy persons or between individuals who had no contact with the clinical disease.

While there was general agreement as to the nasopharynx as the portal of entry, there were two main views as to the possible routes traveled by the organisms in order to gain access to the meninges. The first, supported by Netter and Debré,[14] suggested the direct transmission of the organism from the nose to the meninges. The second, upheld by scientific workers in Germany, America, and England,[15] postulated that the organisms were carried to the meninges by the blood stream. Each group of the interested investigators marshaled evidence to support their views, but the general opinion of physicians

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[9] See footnote 1, p. 192.

[10] Goldschmidt, F.: Ein Beitrag zur Aetiologie der Meningitis cerebro-spinalis. Centralb. f. Bacteriol. u. Parasitenk., Jena 2: 649–654, 1887.

[11] See footnote 2, p. 192.

[12] Dopter, C.: Étude de quelques germes isolés du rhino-pharynx, voisins du méningocoque (paraméningocoques). Compt. rend. Soc. de biol. 67: 74–76, 1909.

[13] See *footnote* 1, p. 192.

[14] See footnote 4, p. 192.

[15] (1) Councilman, W. T., Mallory, F. B., and Wright, J. H.: Epidemic Cerebrospinal Meningitis and Its Relation to Other Forms of Meningitis. Report of the State Board of Health of Massachusetts, 1898. (2) Elser, W. J., and Huntoon, F. M.: Studies on Meningitis. J. Med. Research 20: 371–541, June 1909. (3) Flexner, S., and Barker, L. F.: A Contribution to Our Knowledge of Epidemic Cerebro-spinal Meningitis. Am. J. M. Sc. 107: 155–172, February 1894. (4) Albrecht, H., and Ghon, A.: Ueber die Aetiologie und pathologische Anatomie der Meningitis cerebrospinalis epidemica. Wien. klin. Wchnschr. 14: 984–996, 1901.

dealing with these patients, as summed up essentially by Topley and Wilson,[16] was that the most probable sequence had to be as follows: The organisms reached the nasopharynx as an airborne parasite; here they set up a rhinopharyngitis but usually gave no trouble. In some cases, however, the organisms gained access to the meninges; whether by direct invasion or via the blood stream was not known. Meningococcemias without meningitis also occurred frequently.

Much has been written about the various factors which may play a role in determining the occurrence of this disease. Early in the recorded history, keen observers noted that the incidence was affected by crowding, poor housing, or a combination of these two factors, although they could give no reasons for this association. A low temperature, a cold wind, and increased humidity also appeared to be predisposing agents, but, in retrospect, it seems probable that these act mainly by forcing undue crowding indoors. Fatigue was also thought to play an important role, for it was the recruits, unaccustomed to rigors of military life, who furnished the greatest number of cases.

During the war of 1914–18, many investigations were carried out in attempts to ascertain the importance of the symptomless infections of healthy individuals in the transmission of this disease. Glover[17] noted that when the carrier rate rose above 20 percent, isolated cases would appear and, as the epidemic gained a foothold, the rate might reach as high as 88 percent. He also observed a direct relationship between the proportion of soldiers harboring the organism and the degree and type of overcrowding. He called attention to the aggregation of infections in a given hut which pointed strongly toward direct transmission from one man to another. This observation brought about spacing of the men in the barracks during World War I as a control measure which seemed to give relatively good results.

Following these and many similar studies,[18] it was generally recognized that the carrier infections were clearly of great importance in the propagation of meningococci and that the infection was airborne. While it was possible that indirect infections such as contaminated dust particles may play a small part in the spread of this disease, in view of the extreme susceptibility of the organism to drying and to cold, it was regarded as negligible. Neither in barracks nor in civilian households can this method of infection be compared with direct transmittal by nasal mucus sprayed by the sneezing of infected individuals, even though meningococci remain viable after drying for 10 days if protected from direct sunlight.

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[16] Topley and Wilson's Principles of Bacteriology and Immunity. 3d ed. Baltimore: The Williams and Wilkins Co., 1946, vol. II.

[17] Glover, J. A.: Cerebrospinal Fever: Studies in the Bacteriology, Preventive Control and Specific Treatment of Cerebrospinal Fever Among the Military Forces, 1915–19. Section 20. Observations of the Meningococcus Carrier Rate and Their Application to the Prevention of Cerebrospinal Fever. Medical Research Council of the Privy Council. Special Report Series No. 50. London: His Majesty's Stationery Office, 1920.

[18] (1) Dudley, S. F., and Brennan, J. R.: High and Persistent Carrier Rates of *Neisseria meningitidis* Unaccompanied by Cases of Meningitis. J. Hyg. 34: 525–541, December 1934. (2) Maxey, K. F.: The Relationsip of Meningococcus Carriers to the Incidence of Cerebrospinal Fever. Am. J. M. Sc. 193: 438–445, March 1937.

Control

With usual conditions and standards of human intercourse, there were no effective methods that could be devised to prevent dissemination of the meningococcus and the consequent occurrence of cases. Immediate methods of control during the period prior to and including World War I generally included isolation of acute clinical cases until 14 days after onset of the disease and close observation of immediate contacts such as household associates, barracksmates and messmates. General procedures called for prevention of overcrowding in living quarters, working places, and conveyances, such as is common in institutional and military populations. Early in World War I, it was considered advisable to detect and isolate the symptomless infections (carriers). Later, however, it was realized that such an isolation policy, even if effective, would be impracticable if rigidly applied to either civilian or military groups. For such populations as were exposed to epidemic prevalence of the disease, the separation or spacing of individuals was increased, and the ventilation of living and sleeping quarters improved. Likewise, all possibilities of chilling, bodily fatigue, and physical strain were minimized.

Polyvalent serum which could protect mice was prepared as early as 1906 by Jochmann,[19] who injected horses first with dead then living cultures. Kraus and Doerr,[20] prepared antitoxic sera by injection of broth filtrates and endotoxin. Such sera were used for treatment either by intravenous or intraspinal injections. Employment of these materials with spinal lavage seemed to give satisfactory results, but this could not be proved because of the extreme variability of the case fatality rates in different areas and outbreaks.

Sophian and Black,[21] Greenwood,[22] and Gates [23] made early attempts to ascertain the value of prophylactic vaccination but could not obtain unequivocal results. There was and is, however, no acceptable method of determining the resistance of an individual to an infection with this parasite other than determining the incidence of disease in a vaccinated and control group. This situation prevented any adequate assay. Since the immunity conferred by a clinical attack was apparently of long duration, subclinical (carrier) infections were accepted as probably explaining the resistance of most adults. This was supported by the observation that contact (secondary) cases in families or military units were very rare.

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[19] Jochmann, G.: Versuche zur Serodiagnostik und Serotherapie der epidemischen Genickstarre. Deutsche med. Wchnschr. 32: 788–793, May 1906.

[20] Kraus, R., and Doerr, R.: Ueber Meningokokkengifte und Gegengifte. Wien. klin. Wchnschr. 21: 12–14, January 1908.

[21] Sophian, A., and Black, J.: Prophylactic Vaccination Against Epidemic Meningitis. J. A. M. A. 59: 527–532, August 1912.

[22] Greenwood, M.: The Outbreak of Cerebrospinal Fever at Salisbury in 1914–15. Proc. Roy. Soc. Med., London 10: Sec. Epidemiol. and State Med., 44–55, November 1916.

[23] Gates, F. L.: A Report of Antimeningitis Vaccination and Observations on Agglutinins in the Blood of Chronic Meningococcus Carriers. J. Exper. Med. 28: 449–474, October 1919.

PERIOD BETWEEN WORLD WAR I AND WORLD WAR II

In the years between 1919 and 1939, a more exact description of the occurrence of meningitis in various population groups became possible on the basis of mortality and morbidity reports. The published records of the Health Section of the League of Nations [24] began after World War I and the number of countries from which annual reports of cases of meningococcal meningitis were received increased steadily year by year. Hedrich [25] published a careful analysis of this material for the years between 1915 and 1930. Gover and Jackson [26] have expanded and extended these studies to take in the interval up to and including the year 1945, devoting most of the discussion to a description of the occurrence of this disease in the United States but including a brief report of the experience in other countries.

Germany experienced a sharp and pronounced outbreak in 1922, and a number of other Middle European countries showed similar waves that year or the next. Incidence was high in France and Denmark in 1925, in Sweden in 1926–27, and in Poland, Czechoslovakia, the Baltic states, Germany, Italy, and Greece in 1929. From 1928 to 1931, meningococcal meningitis was excessively prevalent in Canada and, from 1931 to 1934, in the British Isles. Another epidemic spread began in 1937–38 in Germany, Italy, Poland, Czechoslovakia, Greece, and Turkey when the recent world conflict was imminent. It is not at all improbable that this increase was related to or precipitated by the extensive mobilization activities which preceded the war.

A wave began in South Africa in 1928 and spread to Southern and Northern Rhodesia in 1929 and 1930. In North Africa, it began in Morocco and Algeria in 1929–30, extended to Egypt and the Anglo-Egyptian Sudan from 1930 to 1936, reaching Uganda and Kenya in 1934 to 1938. In the Far East, epidemics were recorded in Indo-China and Hong Kong between 1931 to 1934, and in Formosa and Japan in the period 1934 to 1936. These data, gleaned from the incomplete records of the League of Nations, insofar as they go, lend substantial support to the constantly recurring impression that meningococcal meningitis becomes epidemic over large geographic regions in such a manner as to suggest a wavelike spread through populations living in essentially contiguous countries. Furthermore, it is evident that between 1919 and the beginning of World War II, most of the reporting countries experienced one, two, or perhaps three possibly related or synchronous epidemic waves.

After World War I, really beginning in 1916, coincidental with the broadening of the United States registration area, recognition, reporting, and classification of deaths and cases of meningococcal meningitis became relatively satisfactory. The area and population was sufficiently large and representative

[24] League of Nations: Health Organization. Annual, monthly, and weekly epidemiological reports.

[25] Hedrich, A. W.: The Movements of Epidemic Meningitis, 1915–1930. Pub. Health Rep. 46: 2709–2726, November 1931.

[26] Gover, M., and Jackson, G.: Cerebrospinal Meningitis; Chronological Record of Reported Cases and Deaths Pub. Health Rep. 61: 433–450, March 1946.

to afford a comprehensive delineation of the occurrence and movement of this disease in the United States.

For the purpose of studying the chronology and geographic extent of epidemic periods, either regional morbidity or mortality rates may be employed as indices.[27] The wide variation in case fatality rates, as previously noted by many observers, is an ever-present source of error. Likewise, morbidity rates are equally unreliable because of failure to report or to make a proper diagnosis. However, after analyzing the available records from all points of view, it is apparent that the use of either the morbidity or mortality rate does not essentially change the picture that can be drawn regarding the occurrence of meningococcal meningitis.

Epidemic incidence was noted in many parts of the United States during 1917–18, but the outbreaks were so small and scattered in the Mountain and Pacific States, that they were scarcely perceptible in the regional picture. The next wave in 1928–29 apparently began in 1926 in the regions which had been spared in the earlier outbreak and gradually extended into the Mountain States. Under this impact, the area recorded the highest attack rate realized in all regions. Excessive rates were found throughout the country, but the epidemic wave was least noticeable in New England and the South Atlantic States.

After a relatively quiet period of 5 years, meningococcal meningitis became important again in 1935–36 with a noteworthy increase in rates which was reflected all over the United States but with the highest incidence in the South Atlantic and East South Central States. Following this swing, a period of low incidence followed, lasting until 1942.

It must be kept in mind that the excessive rates for any given region may be due to a number of small foci within the area; that is, the regional picture may be only the reflection of epidemic incidence of a few localities. The data are not sufficiently detailed, the distribution of cases and deaths are not uniform, and it is impossible to analyze the occurrence in small geographic units or to attempt to trace the movement of this disease from one area to another. It is possible and interesting, however, to note the time required for an epidemic to move through the United States. When the incidence curve swings upward, it is reflected across most of the continent within a year. This is an entirely different situation from that which was found in the classical Swedish experience [28] of 1856–61. The rapid spread noted today, as compared with the slow movement found in the middle of the 19th century, probably can be attributed directly to the tremendous improvement in methods of transportation: modern railroads, paved highways, the automobile, and the airplane have all played an important part in changing the effective contact rate not only for meningococcal meningitis but all parasitic diseases in Western civilizations.

It was noted before that the use of mortality and morbidity rates could not be relied upon entirely to describe the extent and severity of epidemics. How-

[27] United States Public Health Service: The Notifiable Diseases; Prevalence of Certain Important Communicable Diseases, by States: Pub. Health Rep. Supp. (issued annually).

[28] See footnote 3, p. 192.

ever, it was remarkable that the ratio of mortality to morbidity was relatively constant for all regions, the United States as a whole, and in epidemic or endemic periods prior to 1940. About that time a marked change occurred, and suddenly three or four times as many cases per death were reported than heretofore. This sudden drop in the case fatality rate was recorded throughout the United States registration area. Several explanations may be offered to explain this observation, such as a change in the strains of the meningococcus prevailing in the United States, improvement of reporting, and better diagnostic facilities.

However, it was and is generally accepted that the introduction of a much more effective therapeutic program prevented an extremely significant number of deaths. Sulfonamide drugs were made generally available to the physicians in the United States in 1934, and it was quickly demonstrated that they have an extraordinary specificity in the treatment of meningococcal infections.[29] Of course, while the improvement in the case fatality rate lagged 5 or 6 years behind, it is not unexpected that this apparently long period of time was required before these compounds were used in the treatment of a sufficient number of cases to produce the improvement in recorded mortality figures. The addition of these drugs to the armamentarium of the physician was the most significant advance in medical knowledge in the 20 years between the two world conflicts. It changed completely the therapy and prognosis of classical meningococcal meningitis.

Etiologic Agent and Mode of Transmission

The several studies attempting to classify the various strains of meningococci isolated during this interval between world conflicts served to show only that no sharp line of demarcation could be drawn between the several types. Group I was the organism commonly found in the United States during the so-called epidemic episodes, while group II was isolated more frequently during the interepidemic periods. Branham and others [30] and Rake and Scherp [31] reached the broad conclusion that, on the basis of extensive clinical, chemical, and serologic observations, it was impracticable to distinguish between type I and type III and they should be classified jointly as group I. Type II is not a homogenous group serologically and was, therefore, more appropriately desig-

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[29] Dingle, J. H., Thomas, L., and Morton, A. R.: Treatment of Meningococcic Meningitis and Meningococcemia With Sulfadiazine. J. A. M. A. 116: 2666-2668, June 1941.

[30] (1) Branham, S. E.: Significance of Serologic Types Among Meningococci. J. A. M. A. 108: 692-696, February 1937. (2) Branham, S. E.: Value of Typing Meningococci. Am. J. Pub. Health 35: 233-238, March 1945. (3) Branham, S. E., Mitchell, R. H., and Brainin, W.: Gonococcic meningitis. J. A. M. A. 110: 1804-1806, May 1938. (4) Branham, S. E., and Pittman, M.: A Recommended Procedure for the Mouse Protection Test in Evaluation of Antimeningococcus Serum. Pub. Health Rep. 55: 2340-2346, December 1940. (5) Branham, S. E., Pittman, M., Rake, G., and Scherp, H. W.: A Proposed Mouse Protection Unit for Antimeningococcus Serum. Proc. Soc. Exper. Biol. & Med. 39: 348-350, November 1938. (6) Branham, S. E., Taft, C. E., and Carlin, S. A.: Studies on Meningococci Isolated in the United States, 1928-1930: Serological Classification and Geographic Distribution. Pub. Health Rep. 46: 897-916, April 1931. (7) Branham, S. E., and Carlin, S. A.: Comments on a Newly Recognized Group of the Meningococcus. Proc. Soc. Exper. Biol. & Med. 49: 141-144, February 1942.

[31] Rake, G., and Scherp, H. W.: Studies on Meningococcus Infection. III. The Antigenic Complex of the Meningococcus -A Type-specific Substance. J. Exper. Med. 58: 341-374, September 1933.

nated as group II. A number of strains were isolated late in the thirties as distinctive members of group II and are now referred to as group IIa. Type IV has not been isolated from clinical cases in the United States since World War I, and, because it is so uncommon, it was generally accepted that maintenance of a separate classification seemed rather unwarranted.

Branham believed firmly that the meningococci classified in group I were responsible for the epidemics and those in group II for sporadic cases. She stated that "during recent years it has been possible to foresee periods of unusual incidence by the increase in the relative number of Group I strains sent in for typing, and to recognize that an outbreak was on the wane by an increased proportionate occurrence of Group II strains."

These and similar studies were directed primarily at the development of an adequate therapeutic serum. The occasional observation could be used in control procedures, but the great interest lay in the standardization of serum treatment. The introduction of the sulfonamides completely changed the picture. For some time it was undecided whether it would be necessary to employ both serum and these drugs, but it was not long before clinical reports demonstrated that the sulfonamide alone would suffice.

Control

During this interval, efforts to develop and assay various methods of prophylactic immunization continued apace in many countries. Zrůnek and Feierabend [32] endeavored to immunize the Czechoslovakian Army. Riding and Corkill [33] in 1932 carried out a large series of vaccinations among the natives in northern Sudan. No significant protection was obtained in either of these attempts although the studies were well planned with large samples and carefully carried out by the investigators.

With the concept that meningococci produce a soluble exotoxin, Ferry [34] in the early thirties recommended that therapeutic antitoxic sera be prepared by injecting horses with the type-specific toxins. Later he developed toxoids which were employed by Kuhns [35] in studies utilizing various Civilian Conservation Corps installations. The results obtained were not sufficient to warrant serious consideration of this method of immunization. The principal obstacle encountered in all these investigations came about because there is no laboratory method by which human immunity can be gaged. Reliance always had to be

[32] Zrůnek, K., and Feierabend, B.: An Attempt of Active Immunisation Against Epidemic Cerebrospinal Meningitis. Trav. Inst. d'hyg. pub. de l'Etat Tchécoslovaque 2: 1-6, January 1931.

[33] Riding, D., and Corkill, N. L.: Prophylactic Vaccination in Epidemic Meningococcal Meningitis. J. Hyg. 32: 258-267, April 1932.

[34] (1) Ferry, N. S.: Meningococcus Antitoxin. I. Prophylactic and Therapeutic Tests on Guinea Pigs. J. Immunol. 23: 315-324, 1932. II. Therapeutic Tests on Monkeys. Ibid. 23: 325-347, October 1932. (2) Ferry, N. S., Norton, J. F., and Steele, A. H.: Studies of the Properties of Bouillon Filtrates of Meningococcus: Production of Soluble Toxin. J. Immunol. 21: 293-312, October 1931.

[35] Kuhns, D. M., Kisner, P., Williams, M. P., and Moorman, P. L.: The Control of Meningococcic Meningitis Epidemics by Active Immunization With Meningococcus Soluble Toxin: Further Studies. J. A. M. A. 110: 484-487, February 1938.

placed upon the comparison of incidence rates and this was not at all satisfactory when dealing with a disease in which such a large proportion of the infections are subclinical.

ARMY EXPERIENCE DURING WORLD WAR II

Although meningococcal meningitis was not yet a serious problem for the Army early in 1941 and sulfonamide therapy seemed extraordinarily promising, it was believed desirable by the Preventive Medicine Service of the Surgeon General's Office to establish and support a special group of civilian workers concerned primarily with this disease. The tempo of mobilization was increasing, and the unusual susceptibility of new recruits was well recognized. For the prevention and control of meningococcal meningitis in the Army, with its shifting population, unusual environmental factors, and tactical requirements, extension of knowledge of the biology of the causative organism and the epidemiology of the disease were deemed of primary importance in the war effort.

To carry out this proposal, the Commission on Meningococcal Meningitis was organized under the Board for the Investigation and Control of Influenza and Other Epidemic Diseases in the Army. Employing their recommendations, a tentative program for study and control of meningococcal meningitis was formulated, approved by the Board, and submitted to the Office of the Surgeon General on 26 April 1941.[36] This memorandum, in addition to specific immediate recommendations, defined the aims and basic procedures that would permit the coordination of the activities of all interested groups, civilian and military, and to contribute effectively in every way to the solution of this problem. Among the more important suggestions, it was recommended that a central laboratory be established at the Johns Hopkins School of Hygiene and Public Health, Baltimore, Md., to act as a center for interim laboratory studies, preparation and distribution of meningococcal typing sera, and analysis of case data for correlation with the characteristic strains isolated. An investigative team was planned to carry on the necessary field studies and to evaluate therapeutic and prophylactic measures. In addition, the importance of a specialized consultation service for the Office of the Surgeon General and commanding officers of Army posts and medical laboratories was recognized and provided.

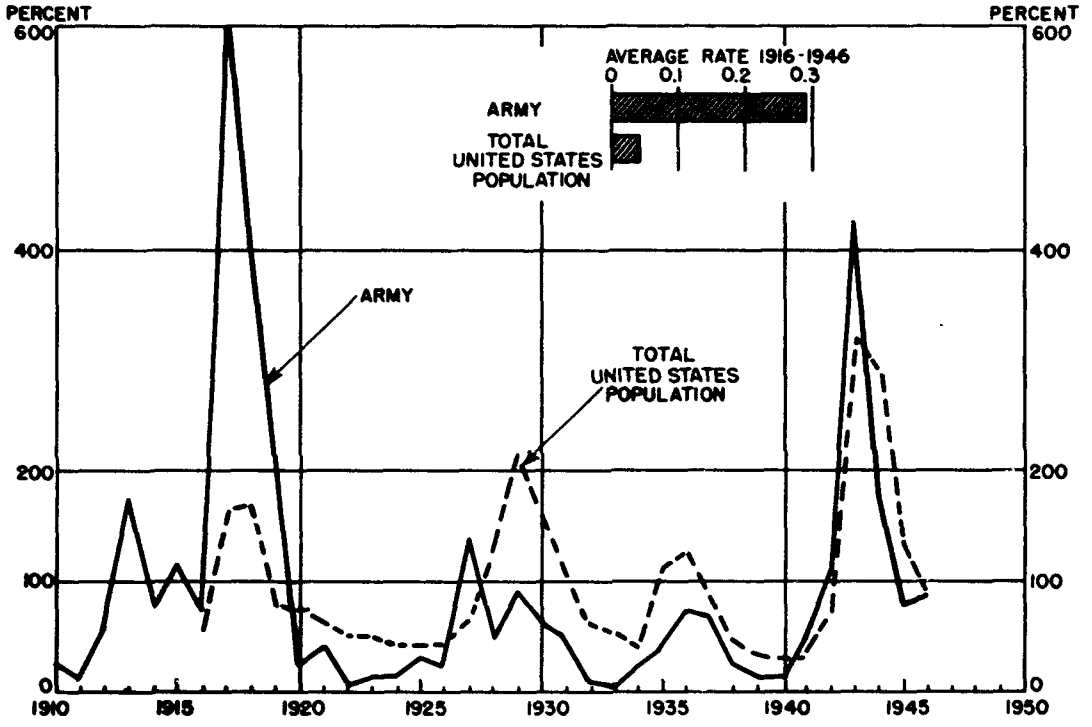
In 1941, as previously noted, the incidence of meningococcal meningitis in the United States was low and usually sporadic. The reports to the Health Section of the League of Nations at the actual beginning of World War II in 1939, however, showed a rise in incidence in practically every European country where records are available. The disease became increasingly prevalent in the United States at the end of 1942 shortly after this country entered the war. Beginning with the fall of 1942, the number of cases began to rise among both Army personnel and civilian populations. From 1942 through 1944, the

[36] Commission on Meningococcal Meningitis, United States Army, Preliminary Report, 1941.

United States experienced the most severe epidemic ever recorded by the United States Public Health Service [37] (chart 19). Illustrative of the force of this outbreak, the Commission from July 1941 through June 1943 received over 5,000 case records from the various military commands stationed in the continental United States.[38] Chile in 1942, likewise, had one of the largest and most severe epidemics recorded in modern times.[39]

CHART 19.—*Meningococcal meningitis in the Army in the United States, 1910–46*[1] *and in the total United States population,*[2] *1916–46*

[Annual rates expressed as percent of average rate for 1916–46; Average rate 1916–46=100 percent]



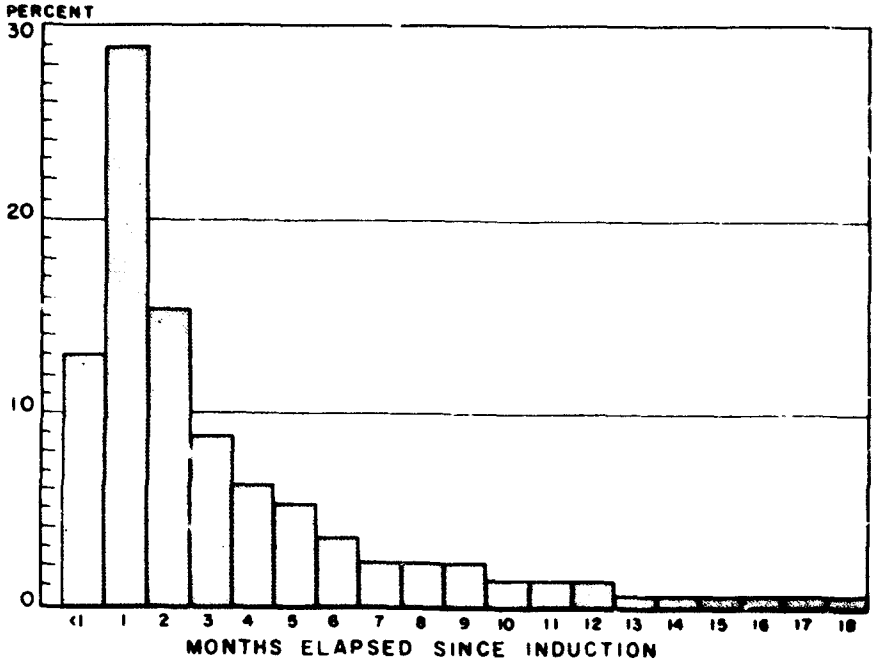
[1] The data for 1910–13, 1917, and 1920–21 are for enlisted men only. The data for 1918–41 include Alaska.
[2] The data for the total United States population include military personnel in the United States.

In the Army, the patients with this disease were concentrated among the new unseasoned recruits. Sixty-seven percent of the 5,000 cases reported to the Commission on Meningococcal Meningitis occurred among men who had been in service 3 months or less, 82 percent with 6 months or less, 89 percent with 9 months or less, and 93 percent with 12 months or less. Although this was a time of rapid expansion in the Army, the appearance of cases in the recruits during their first 3 months of service was consistent not only throughout mobilization but was noteworthy for the entire war period. Therefore, this

[37] Meningococcus Meningitis in the United States During 1943. Pub. Health Rep. 59: 469–471, April 1944.
[38] Records and Cultures of Cases Received By and On File With the Commission on Meningococcal Meningitis, Army Epidemiological Board.
[39] Pizzi, M.: A Severe Epidemic of Meningococcus Meningitis in Chile, 1941-1942. Am. J. Pub. Health 34: 231–238, March 1944.

difference apparently is a valid representation of the unusual hazard or risk associated with the younger group, insofar as Army experience is concerned. The susceptibility of the very young age group has been noted repeatedly in civilian populations, and it is interesting in this respect to call attention to the Army experience with environmental age as based on a study of 1,337 cases (chart 20).[40]

CHART 20.—*Meningococcal infections by duration of Army service, percent of 1,337 cases*



Mortality from meningococcal meningitis and meningococcemia was exceedingly low, when compared with usual experience, due to widespread and intelligent use of the sulfonamides. From records received by the Commission for the period July 1941 to July 1943, 4,843 cases were classified as proved or probable clinical meningococcal infection.[41] Of this group, the final disposition of 4,724 cases was recorded. The case fatality among this latter group was 3.7 percent. Disposition was usually not stated because the patient was still retained in the hospital at the time the record was forwarded to the central office. It is highly improbable that any deaths occurred among this group retained for further observation, and the case fatality among the entire group of 4,843 cases was also only 3.7 percent.

The experience with this disease in areas such as Europe, Australia, and the Mediterranean, mirrored that which was found in the continental United States. The incidence was high throughout 1942 and 1943. For instance, the disease among the first contingents to reach the British Isles reached an

[40] See footnote 37, p. 203.
[41] See footnote 38, p. 203.

annual incidence of 4.56 per 1,000 per annum. Also the improvement in the case fatality rate was duplicated with the general use of the sulfonamides. The case fatality rate of 4.6 percent for United States troops stationed in Europe during this conflict was extraordinary when compared with the rate of 43.4 percent recorded in World War I.

The epidemiologic pattern, however, was much the same as has been described in the previous war, in that the disease appeared sporadically. No extensive epidemics were reported from any theater and all groupings of cases were of a minor nature. That a goodly proportion of the infections were imported from the Zone of Interior became manifest by the frequency with which the disease was recognized on transports and the higher rates among newly arrived troops.

Etiologic Agent and Mode of Transmission

Approximately 2,488 strains from cases of meningococcemia and classical meningitis occurring in Army personnel were carefully studied in respect to colony morphology, biochemical activity, and antigenic patterns.[42] The results have been summarized in table 33.

It is important to call attention to the fact that the frequency distribution of meningococcal types as isolated from cases and illustrated in this table was not duplicated when the subclinical infections were carefully studied. Typing of the strains obtained from cases throughout the Army revealed that 91 percent were group I. In a study group, 36.1 percent of the meningococci isolated from the subclinical infections were group I, 23.2 type IIa, 39.4 group II, and 1.3 polyvalent IIa–II.

It was recognized, with the careful study of the detailed clinical records of these Army cases, that meningococcal infections should be considered as having three distinct stages. The first is a localized infection of the nasopharynx; the second is a septicemia (meningococcemia); and the third is a purulent inflammation of the meninges, particularly at the base of the brain. It must be noted, however, that the progression through these stages may be so rapid that the stages may not appear separate to the casual observer but to coexist.

The importance of the simple, essentially innocuous nasopharyngeal infections was difficult to ascertain because the term "infection," in its various gradations, cannot be precisely defined. Its scope includes a wide range of manifestations varying from a local reaction, so slight that only the associated antibody response of the host indicated the presence of the organism, to the gross changes which result in clinical signs and symptoms.

A study [43] was formulated, as part of the total effort of many workers, to describe the dynamics of subclinical meningococcal infections according to type

[42] See footnote 38, p. 203.

[43] Phair, J. J., Schoenbach, E. B., and Root, C. M.: Meningococcal Carrier Studies. Am. J. Pub. Health 34: 148–154, February 1944.

TABLE 33.—*Serologic classification of case strains isolated from Army personnel and received by the Commission on Meningococcal Meningitis* [1]

Period	*Meningococci*						*Gonococci*	Unclassifiable	Contaminated	Not viable	Total
	I	IIa	II	IV	II-IIa cross	Polyvalent					
1941											
September December	4							2			6
1942											
January–March	31	2	3			1		5		6	48
April–June	50	2	1					4			57
July–September	41	2	4			1		1			49
October–December	48	5			2	1		4		1	61
1943											
January–March	1,021	59	17		1		3	10	16	54	1,181
April–June	822	51	45		1	1		8	25	37	990
July–September	49	4				1		1		1	56
Total	2,066	125	70		4	5	3	[2] 35	41	99	2,448
Percent of total	84.4	5.1	2.9	0	0.2	0.2	0.1	[2] 1.4	1.7	4.0	100.0
Percent of total proved	91.0	5.5	3.1	0	.2	.2	0	0	0	0	100.0

[1] Approximately 500 additional strains were received from other sources and were part of permanent collection.
[2] Most of these were isolated from nasopharyngeal cultures.

in respect to distribution, prevalence, incidence, and duration. Among 99 men in an untreated group, the average composite prevalence rate was 40 percent, but 91.9 percent were infected at some time during the study period. Of the 91 men with positive cultures, 44 had infections classified as persistent under a reasonable definition. An approximately equal number had only transient infections. Classification of the plates according to the type and number of meningococcal colonies present did not differentiate between the persistent and transient infections.

No fixed pattern could be derived from the study of the individual records. Cultures from some men were negative throughout the study period. Spontaneous parasitic cures occurred in many. In others, infections with one type would be followed, interrupted, or accompanied by an infection with another type. No correlation could be demonstrated between the daily prevalence rates, climatic conditions, the occurrence of upper respiratory diseases, and the incidence of the common contagious diseases, including clinical meningococcal infections.

The high proportion (91.9 percent) of the 99 men found infected extended and confirmed the concept that the spread of the meningococcus is primarily at the subclinical level. Few escaped infection during the brief study period of 68 days, indicating the extent and extraordinary rapidity of dissemination. It was quite apparent that the composite or type-specific prevalence rate for any given day is a static representation of a biologic equilibrium and does not describe the rate of transmission of the meningococcus. A dynamic process exists and must be illustrated by a dynamic equation. The number of new infections in each succeeding time period, that is, the incidence rate, is a much better index than the level of prevalence attained. If the incidence is increasing, transmission is taking place; if it is decreasing, conditions are operating against the survival of the parasite. The same prevalence may be found in two population groups, yet in one there may be a declining incidence and in the other an increasing transmission of the meningococcus. If all other factors are equal, such as group susceptibility or degree of contact, the latter situation would cause more concern.

With this concept the hypothesis of a fixed epidemic level of subclinical meningococcal infections falls. Glover [44] had concluded that there was a correlation between prevalence of these infections and the incidence of clinical cases by stating: "A carrier rate of 20 percent (without awaiting the occurrence of cases) should be regarded as a signal for prompt and effective action * * *." However, in the development of this thesis, he was actually concerned with the changes in the prevalence rather than the static level. In his report, the following inferences were drawn: "A wave of high (non-contact) carrier rates *precedes* and accompanies an outbreak of cerebrospinal fever. In other words there is a carrier epidemic (for the most part entirely devoid of symptoms) *preceding* and accompanying the much smaller case epidemic." He did not stress sufficiently the importance of this latter observation and later workers in the field of preventive medicine neglected it entirely.

These results, combined with the many similar investigations of other workers dealing with Army personnel, indicated that clinical reactions or cases of the disease must then be considered only as uncertain and irregular indicators of the dissemination of the specific micro-organism in a population. The rise or decline of the incidence of disease over long periods of time may be due alone to a change in the prevalence of a parasite, to a variation in the ratio of clinical to subclinical infections, or a combination of these. However, it is evident that the number of cases would be determined largely by the total incidence of new infections, clinical and subclinical, rather than the level of the prevalence, in any given period.

Control

These studies as described offered a reasonable explanation of the failure of control measures which have been suggested in the past. The meningococcus

[44] See footnote 17, p. 196.

must be considered as an efficient parasite which maintains itself in any human population through infections at a subclinical level. Only a very small proportion of the total number of infections are followed by clinical manifestations. The latter group acquire the parasite not necessarily from contact with other cases but from apparently healthy individuals who have a subclinical infection. As there are no methods for the protection of susceptibles, a reduction in the number of clinical cases can be obtained only by decreasing the probability of exposure to the meningococcus.

The formulation and application of adequate practical preventive programs for the control of such upper respiratory infections, in civilian or military populations under these circumstances, is extremely difficult. The obstacles encountered stem essentially from the fact that the host-parasite relationship is well established and apparently of long standing. The extent of this adaptation in the equilibrium between host and parasite was evidenced by (1) the relatively small proportion of infections which present clinical manifestations; (2) a relatively prolonged infectious period; (3) a vulnerable portal of entry and an open portal of exit; (4) the antitoxic, rather than antiparasitic, immunity which usually follows an infection; and (5) the relatively transient immune period.

Attempts at nasal disinfection had been made in World War I without success. The administration of subtherapeutic doses of the sulfonamides had been advocated for the chemoprophylaxis of certain selected, particularly upper respiratory, bacterial infections by numerous workers before World War II. Schwentker and coworkers [45] employed sulfadiazine in an attempt to influence the course of a scarlet fever epidemic among the personnel of a naval training station. Coburn,[46] Holbrook,[47] Hodges,[48] Van Ravenswaay,[49] among others, have reported on the apparent reduction in the attack rate of streptococcal respiratory infections at Army and Navy installations following the institution of sulfadiazine prophylaxis. The experience of several investigative teams organized by the Navy at various bases and conducted through the winter of 1943–44 has been summarized in a bulletin issued by the Bureau of Medicine and Surgery of the Navy Department. Siegel [50] reported extensively on studies for the control of acute infections of the respiratory tract with small daily doses of sulfadiazine administered to an institutional group. The effect of this regime on the nasopharyngeal flora was followed by carefully repeated nasal and throat cultures at periodic intervals.

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[45] Schwentker, F. F., Hodes, H. L., Kingsland, L. C., Jr., Chenoweth, B. M., and Peck, J. L.: Streptococcal Infections in a Naval Training Station. Am. J. Pub. Health 33: 1455–1460, December 1943.

[46] Coburn, A. F.: The Carrier Problem in the Dissemination of Hemolytic Streptococcus. U. S. Nav. M. Bull. 42: 325–335, February 1944.

[47] Holbrook, W. P.: The Army Air Forces Rheumatic Fever Control program. J. A. M. A. 126: 84–87, September 1944.

[48] Hodges, R. G.: The Use of Sulfadiazine as a Prophylactic against Respiratory Disease. New England J. Med. 231: 817–820, December 1944.

[49] Van Ravenswaay, A. C.: Use of Sulfonamides in the Army. J. Missouri M. A. 41: 29–30, February 1944.

[50] Siegel, M.: Studies on the Control of Acute Infections of the Respiratory Tract. III. Results of Sulfadiazine Therapy From May 3, 1942 to May 2, 1943. Am. J. Dis. Child. 67: 365–370, May 1944.

Meehan and Merrillees,[51] Fairbrother,[52] Mueller,[53] Cheever and coworkers,[54] Kuhns and coworkers,[55] and the Commission on Meningococcal Meningitis,[56] among others,[57] reported the prevention of meningococcal infections with sulfonamide prophylaxis. This has been the disease in which the greatest success has attended its use because of the unusual sensitivity of the meningococcus to these compounds. It was readily demonstrated by carefully controlled observations that even a single 2-gm. dose of sulfadiazine was followed by a rapid and complete disappearance of meningococci from repeated subsequent nasopharyngeal cultures of the entire group.

Prophylaxis by chemotherapy, however, cannot control the incidence of reinfections indefinitely. It must be recognized that only parasitic cure or suppression was obtained. There may be a brief refractory period following the administration of the drug, due possibly to a change in the nasopharyngeal bacterial flora. However, there are no grounds, either theoretical or experimental, for the assumption that the sulfonamides, except during the relatively brief period of their activity, confer freedom from, or enhance resistance to, subsequent infection over any long period of time. The rapidity with which the treated groups may again attain the prevalence level of the general community will depend upon their degree of reexposure. Such exposure is the resultant of the effective contact rate between the treated and untreated groups and the incidence of meningococcal infections as contrasted to the prevalence among the latter.

The use of these chemotherapeutic drugs, however, offered a feasible and effective method of controlling the dissemination of meningococci in the vast variety of situations found in the Army, such as troop trains, troopships, training installations, and prison camps. Armed with this knowledge and fortified also with a rapid and adequate therapeutic program, the Preventive Medicine Service arranged prophylactic programs which met the urgent needs of the Army during the war period. It is impossible to determine the contribution of mass sulfadiazine prophylaxis to the decline of meningococcal meningitis in the Army as a whole, but there is no doubt as to its effectiveness in the control of specific outbreaks. However, with intelligent application of these procedures,[58] fear was allayed, and the widespread shifting and deployment of troops and other personnel was not hampered or curtailed because of this disease during the remainder of World War II.

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[51] Meehan, J. F., and Merrillees, C. R.: An Outbreak of Cerebro-spinal Meningitis in a Foundling Hospital. The Treatment of Carriers with "M & B 693." M. J. Australia 2: 84–90, July 1940.

[52] Fairbrother, R. W.: Cerebrospinal Meningitis. The Use of Sulfonamide Derivatives in Prophylaxis. Brit. M. J. 2: 859–862, December 1940.

[53] Mueller, J. H.: The Relation of the Carrier to Epidemic Meningitis. Ann. Int. Med. 18: 974–977, June 1943.

[54] Cheever, F. S., Breese, B. B., and Upham, H. C.: The Treatment of Meningococcus Carriers With Sulfadiazine. Ann. Int. Med. 19: 602–608, October 1943.

[55] Kuhns, D. M., Nelson, C. T., Feldman, H. A., and Kuhn, L. R.: The Prophylactic Value of Sulfadiazine in the Control of Meningococcic Meningitis. J. A. M. A. 123: 335–339, October 1943.

[56] See footnote 36, p. 202.

[57] Aycock, W. L., and Mueller, J. H.: Meningococcus Carrier Rates and Meningitis Incidence. Bact. Rev. 14: 115–160, June 1950.

[58] Circular Letter No. 170, Office of the Surgeon General, U. S. Army, 30 Sept. 1943, subject: Prophylaxis of Meningococcal Meningitis by Use of Sulfadiazine.

CHAPTER XII

The Pneumonias

Section I. Primary Atypical Pneumonia

Norman L. Cressy, M. D.

The occurrence of respiratory illness among troops during periods of mobilization has always been a matter of great importance, and pneumonia as either a primary or secondary disease has usually been a major cause of death. During the winter of 1812 and 1813, there was a high incidence of acute respiratory disease among troops stationed on the northern frontier.[1] Measles complicated by pneumonia was epidemic from September to December 1812. During the following winter, although the morbidity and mortality were lower for the Army as a whole, new troops joining the service were as severely affected as the men who were mobilized during the preceding year. In the War Between the States, acute respiratory disease was again an important cause of morbidity and mortality.[2] During the winter of 1862, there was excessive seasonal variation of respiratory disease rates with a curve for catarrh which was similar to the influenza epidemic of 1918, but there was no epidemic such as the one which occurred in the fall of 1918. In the Mexican-border mobilization of 1916, there was an epidemic of pneumonia with about 400 cases occurring among 40,000 troops and a 20-percent case mortality.[3] During World War I, the great pandemic of influenza swept through troops and civilian populations alike with large numbers of deaths caused in the main by secondary pneumonia. At the same time, measles was widespread, and this disease was also complicated by secondary pneumonia.[4]

Knowledge regarding the recognition, epidemiology, and treatment of pneumonia was more definite at the outbreak of World War II than it had been at the time of any previous mobilization. Primary atypical pneumonia had been recognized for several years. It was first recorded by the Army in a separate diagnostic category on the individual medical records during 1941 and on the weekly statistical health report in March 1942. Whether this disease or group of diseases was present before or during World War I will

[1] Mann, James: Medical Sketches of the Campaigns of 1812, 13, 14. Dedham: H. Mann & Co., 1816, p. 306.

[2] The Medical and Surgical History of the War of the Rebellion. Medical History. Washington: Government Printing Office, 1888, vol. 1, pt. III, p. 719.

[3] Nichols, H. J.: The Lobar Pneumonia Problem in the Army From the Viewpoint of the Recent Differentiation of Types of Pneumococci. Mil. Surgeon 41: 149–161, August 1917.

[4] The Medical Department of the United States Army in the World War. Pathology of the Acute Respiratory Diseases, and of Gas Gangrene Following War Wounds. Washington: U. S. Government Printing Office, 1929, vol. XII, p. 7.

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probably never be known, but there is evidence to suggest that it was not a new disease. The following quotation is taken from the history of the Medical Department of the United States Army in World War I: [5]

* * * the usual type of pneumonia occurring among young male adults in civil life is of course primary lobar pneumonia * * *. That such cases occurred among the troops is beyond question * * *. However, it was early recognized clinically that in the larger number of cases observed in the camps the pneumonia was of an atypical nature. The onset tended to be slower than that of the lobar pneumonia of civil life; the course more prolonged. Crisis was relatively rare; physical signs were slow of development and of patchy distribution and scattered in several lobes. These facts led careful observers to consider a large proportion of the cases as bronchopneumonia rather than as the usual lobar type. The results of post-mortem study of fatal cases lent confirmation to this distinction: The typical croupous consolidation of lobar pneumonia was relatively rare, patchy consolidation of a suppurative character more frequent. Even when the consolidation involved nearly or quite an entire lobe, careful study often showed evidence of the formation of such lobar consolidation by the confluence of smaller areas, lobular in origin.

The similarity of this description of pneumonia to the picture seen in primary atypical pneumonia during World War II will be obvious to all those familiar with the disease. It would seem quite likely that atypical pneumonia was indeed present in World War I and was classified largely as bronchopneumonia.

The first of the really efficient chemotherapeutic agents, the sulfonamides, had been in use for several years prior to World War II mobilization. These drugs, *and later penicillin*, proved to be so efficient in the treatment of pneumococcus infections among service personnel that in May 1944 it was recommended that antipneumococcus serum be dropped from the Medical Department supply table. This situation was in sharp contrast to that which existed in World War I when the only specific pneumonia therapy was an antipneumococcus type I serum. The total effect of the use of the sulfonamides and penicillin will probably remain immeasurable. One might expect that, in addition to lowering the mortality case rate of primary bacterial pneumonia and other bacterial infections, it must also have lowered the number of cases occurring as a complication of other diseases.

RECOGNITION OF THE DISEASE

It was during the late 1930's that primary atypical pneumonia was first recognized as a disease distinct from the bacterial pneumonias. In 1938, Reimann [6] published an account of a respiratory illness which he called atypical pneumonia and suggested its probable viral nature. Others had previously described a similar disease which in retrospect might well have been the same thing. Bowen [7] published a radiologic description of an epidemic among

[5] The Medical Department of the United States Army in the World War. Communicable and Other Diseases. Washington: U. S. Government Printing Office, 1928, vol. IX, p. 61.

[6] Reimann, H. A.: An Acute Infection of the Respiratory Tract With Atypical Pneumonia. A Disease Entity Probably Caused by a Filtrable Virus. J. A. M. A. 111: 2377-2384, December 1938.

[7] Bowen, A.: Acute Influenza Pneumonitis. Am. J. Roentgenol. 34: 168-174, August 1935.

troops in Hawaii which he called acute influenza pneumonitis. Gallagher [8] described bronchial pneumonia and acute pneumonitis in adolescents. In 1916, Clough and Richter [9] published an account of a patient with a respiratory illness in whom autohemagglutinins were demonstrated. The failure of some cases of pneumonia to respond promptly to the sulfonamides undoubtedly influenced the thinking of investigators and helped to bring about the concept of this disease as a separate entity of virus etiology.

Since many of the early reports dealt with epidemics in young adults in camps and boarding schools, it came as no great surprise when cases began to be recognized in the Armed Forces.

EPIDEMIOLOGIC ASPECTS

Statistics for pneumonia in the Armed Forces in World War II are neither accurate nor complete. Reasons for this vary all the way from the intrinsic difficulties of gathering statistics in wartime to the ability of the medical personnel to make accurate differential diagnoses. The disease became officially reportable on the weekly statistical health report as Primary Atypical Pneumonia, Etiology Unknown, by direction of Circular Letter No. 19, Office of the Surgeon General, United States Army, 2 March 1942. It was probably some months following this before all medical personnel became sufficiently familiar with this new classification to make the differential diagnosis regularly. Officers concerned almost exclusively with the respiratory disease problem often had trouble in making accurate distinction between the various types of pneumonia even while working under the best conditions. It would be reasonable to expect that officers responsible for all the medical problems of entire units working under less favorable and often hazardous conditions would have even greater difficulty. The fact that there was some difficulty in making accurate differential diagnoses was emphasized in a report from ETOUSA (European Theater of Operations, United States Army) for April 1944 by Maj. Charles D. May, MC.[10] He stated:

> It was possible to substantiate the diagnosis of atypical pneumonia in 72% of the patients so diagnosed. But in only 35% of the patients diagnosed by the hospital as primary [i. e., bacterial or lobar] pneumonia was the evidence considered adequate to justify the diagnosis.
>
> One hypothesis to account for the apparent increase in the incidence of both primary and atypical pneumonia without a corresponding rise in the incidence of common respiratory disease is that there was an actual increase in atypical pneumonia with a confusion in diagnosis leading to many of the cases being reported as primary pneumonia.

Available data show that primary atypical pneumonia was present in all theaters and that its clinical characteristics with but very few exceptions were similar wherever it was reported.

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[8] Gallagher, J. R.: Bronchopneumonia in Adolescence. Yale J. Biol. & Med. 7: 23-40, October 1934.

[9] Clough, M. C., and Richter, I. M.: A Study of an Autoagglutinin Occurring in a Human Serum. Bull. Johns Hopkins Hosp. 29: 86-93, April 1918.

[10] Memorandum, Maj. C. D. May, MC, for Lt. Col. J. E. Gordon, MC, 2 May 1944, subject: Pneumonia Study, ETOUSA, 1944—Considerations for Further Study.

During the summer of 1941, an unexpectedly high incidence of pneumonia was observed in the soldiers at Camp Claiborne, La. The unusual character of this disease was noted by the commanding officer and by the chief of the medical service at the station hospital who recognized its similarity to the clinical syndrome of atypical pneumonia which had recently been described. The outbreak was reported to The Surgeon General and to the Surgeon, Fourth Corps Area, which resulted in the institution of preliminary surveys at Camp Claiborne by Drs. A. R. Dochez, Yale Kneeland, Colin M. MacLeod, and Kenneth Goodner. These workers felt that further investigation was warranted, and, accordingly in December 1941, Drs. John H. Dingle and W. Barry Wood took up residence at Camp Claiborne and remained until 1 May 1942 to direct a group study of the problem. The results of the work led to the establishment of a laboratory for the Commission on Acute Respiratory Diseases at Fort Bragg, N. C., on 19 October 1942.

It is probable that the figures of morbidity and mortality obtained at Camp Claiborne during 1941 and 1942 and at Fort Bragg during the remainder of the war are fairly accurate because of the special studies which were conducted at these two stations. The early work at Camp Claiborne showed that the disease represented a real problem to the Army with an average attack rate of 28 per 100,000 per week and a recorded peak incidence of 88 per 100,000 per week during an epidemic. It was shown further that the average hospitalization period for patients with atypical pneumonia was 32 days.[11] This clearly presented a threat to the well-being of troops in training and potentially to those in combat.

Available figures suggest that, excluding the common respiratory diseases, atypical pneumonia represented the major respiratory disease problem for the Army as a whole. In the 4-year period from 1942–45, total Army admissions for atypical pneumonia were 160,940 with an annual admission rate of 6.32 per 1,000 (table 34). Comparable figures for all other pneumonias were 109,882 and 4.31 (table 35). This general relationship was true both in the United States and in overseas areas taken as a whole. It is of interest that in 1942, the year in which atypical pneumonia was first accepted as an official diagnosis for the statistical health reports, the incidence of reported atypical pneumonia in all areas except the Central and South Pacific was less than that for all other pneumonias. In 1943, the relationship of the two groups, for the total Army and for the United States, was reversed, in that atypical pneumonia admissions exceeded admissions for other pneumonias; for total overseas admissions, however, other pneumonia still exceeded atypical pneumonia, although not as markedly as in 1942. In 1944 and 1945, so far as the figures are available, the diagnosis of atypical pneumonia far exceeded the total for all other pneumonias. This was true in all areas except in 1944 in China-Burma-India, the Middle East, and North America, exclusive of the United States. Whether this represented in part a growing awareness among medical personnel of the

[11] Dingle, J. H., Abernethy, T. J., Badger, G. F., Buddingh, G. J., Feller, A. E., Langmuir, A. D., Ruegsegger, J. M., and Wood, W. B.: Primary Atypical Pneumonia, Etiology Unknown. War Med. 3: 223–248, March 1943.

TABLE 34. *Admissions for primary atypical pneumonia in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records]

[Rate expressed as number of admissions per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	110,133	7.47	17,902	6.74	46,375	8.95	25,056	6.31	20,800	7.09
Overseas:										
Europe	23,206	5.28	766	9.23	1,695	6.35	8,055	4.80	12,690	5.35
Mediterranean [1]	12,908	8.70	153	6.67	1,219	2.67	6,536	10.06	5,000	14.13
Middle East	552	3.78	37	6.12	124	2.34	216	4.67	175	4.28
China-Burma-India	1,453	3.31	21	2.40	73	1.84	379	2.25	980	4.43
Southwest Pacific	5,117	2.79	83	1.17	189	1.00	915	1.70	3,930	3.79
Central and South Pacific	5,217	4.15	601	3.98	893	3.06	1,383	3.15	2,340	6.23
North America [2]	584	1.19	73	.73	231	1.19	105	.81	175	2.57
Latin America	1,321	3.46	235	2.31	311	2.57	285	3.32	490	6.73
Total overseas [3]	50,807	4.73	1,989	3.40	4,802	2.84	17,966	4.70	26,050	5.61
Total Army	160,940	6.32	19,891	6.13	51,177	7.45	43,022	5.52	46,850	6.18

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

presence of atypical pneumonia or whether it represented a true increase in the incidence of this disease cannot be determined.

The peak incidence of atypical pneumonia for the entire Army was reached in 1943, when the rate per 1,000 per year reached 7.45 (table 34). This was largely a reflection of the rate for troops in the United States where the rate reached 8.95 in that year. The peak incidence in Europe, however, occurred in 1942, when it reached 9.23 per 1,000 per annum. Thereafter it declined to a low of 4.80 in 1944 but rose slightly to 5.35 in 1945. The Middle East had its greatest incidence in 1942. All other areas had a peak rate in 1945, when the incidence in the Mediterranean (North African) theater reached 14.13 per 1,000 per annum, the highest rate recorded anywhere for an entire area.

Generally, there seems to have been a somewhat higher incidence of atypical pneumonia during colder months when all respiratory disease was more prevalent, but it is obvious that sharp outbreaks did occur during the warmer months as well. From the date on which the disease first became reportable, it was present in all areas almost constantly. Comparative figures for all other pneumonias indicate that atypical pneumonia was the major respiratory illness.

TABLE 35. *Admissions for pneumonia, other than primary atypical type, in the U. S. Army, by area and year, 1942-45*

[Preliminary data based on sample tabulations of individual medical records]

[Rate expressed as number of admissions per annum per 1,000 average strength]

Area	1942-45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	81, 962	5. 56	24, 267	9. 13	35, 735	6. 90	14, 470	3. 64	7, 490	2. 55
Overseas:										
Europe	10, 699	2. 43	788	9. 49	1, 196	4. 48	4, 060	2. 42	4, 655	1. 96
Mediterranean [1]	4, 807	3. 24	233	10. 16	1, 508	3. 30	2, 026	3. 12	1, 040	2. 94
Middle East	678	4. 64	60	9. 92	281	5. 30	287	6. 21	50	1. 22
China-Burma-India	1, 804	4. 11	39	4. 46	262	6. 61	798	4. 73	705	3. 18
Southwest Pacific	3, 732	2. 03	370	5. 19	458	2. 41	669	1. 24	2, 235	2. 16
Central and South Pacific	2, 304	1. 83	494	3. 27	514	1. 76	521	1. 19	775	2. 06
North America [2]	1, 424	2. 89	496	4. 93	573	2. 95	215	1. 66	140	2. 06
Latin America	1, 548	4. 06	708	6. 95	388	3. 21	192	2. 24	260	3. 57
Total overseas [3]	27, 920	2. 60	3, 316	5. 66	5, 426	3. 21	9, 003	2. 36	10, 175	2. 19
Total Army	109, 882	4. 31	27, 583	8. 51	41, 161	5. 99	23, 473	3. 01	17, 665	2. 33

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

In spite of the widespread morbidity due to atypical pneumonia, the mortality was fortunately low in all areas. There were 170 deaths attributed to atypical pneumonia in the entire Army from 1942 through 1945. This is in contrast to 1,041 deaths caused by other pneumonias during the same period. It is of interest that the case fatality rates were higher overseas than in the United States for all types of the disease; however, the death rate (number of deaths due to pneumonia per 100,000 average strength per year) was higher for both atypical and other pneumonias among those troops stationed in the United States than it was for those stationed overseas. The over-all case fatality rate (number of deaths per 100 admissions for pneumonia) for atypical pneumonia was 0.12 per 100 admissions for the 4-year period 1942 through 1945 as compared with a rate of 0.88 per 100 admissions for all other pneumonias.

What conclusions can be drawn from the foregoing discussion? As stated previously, the figures are incomplete and are based on sample tabulations. It can be said, nevertheless, that, apart from the common respiratory diseases, atypical pneumonia was the major respiratory disease problem during World War II. Atypical pneumonia was present in all areas in significant

numbers and accounted for a large share of the total morbidity from respiratory disease.

Since atypical pneumonia was not recognized as an entity in 1918, no direct comparison of figures for the two World Wars for this disease can be made. Study of the figures for all pneumonia for the two periods is very interesting. The admission rate for all pneumonia per 1,000 average strength per year for the total Army from April 1917 to December 1919 was 18.98, and the approximate case fatality rate per 100 admissions was 24.46. The admission rate for all pneumonia in the entire Army from 1942 to 1945 was 10.63 per 1,000 average strength per year, and the average case mortality rate (deaths per 100 admissions) for the same period was approximately 0.42. This great reduction of morbidity and mortality figures could have been caused by many factors, ranging from the type of warfare carried on to the many aspects of personal hygiene. Undoubtedly, the sulfonamides and penicillin were a great influence and may well have been the most important. The absence of pandemic influenza played an undetermined but probably important role.

SPECIAL STUDIES

The results of the early investigations at Camp Claiborne, in 1941 and 1942, showed the need for continued study of the problem of atypical pneumonia. To this end, the Commission on Acute Respiratory Diseases was founded, and a laboratory was later equipped at Fort Bragg. This Commission functioned as an active investigating unit from 1 August 1942 throughout the duration of the war. The scope of its activities included not only primary atypical pneumonia but also influenza and other respiratory diseases. The complete work is fully reported in numerous articles under the authorship of the Commission on Acute Respiratory Diseases which were published from 1943 through 1946. Only the most important aspects of the work will be set forth here. The most significant work of the Commission concerned the study of atypical pneumonia in human volunteer subjects who were drawn from the ranks of conscientious objectors.[12] These studies were carried out over a period of 3 years. The disease was successfully transmitted to humans by inoculation with bacteria-free filtrates of respiratory secretions which had been collected from patients with atypical pneumonia. This accomplishment alone lends strong support to the widely held theory that atypical pneumonia is a virus disease. These carefully controlled cases furnished a unique opportunity to study the clinical, roentgenographic, and laboratory characteristics of atypical pneumonia.

Clinical Aspects

Onset.—The Commission's studies of these cases confirmed and extended previous descriptions of the clinical picture of atypical pneumonia. The exact

[12] Commission on Acute Respiratory Diseases: The Transmission of Primary Atypical Pneumonia to Human Volunteers. Bull. Johns Hopkins Hosp. 79: 97-167, August 1946.

time of onset proved to be almost as difficult to determine in these cases as in the naturally occurring disease. Often the earliest symptoms were mild and inconstant with no objective evidence of illness. This was quite in keeping with the previous observations of the disease. The incubation period varied from 7 to 14 days and was in general shorter for those who received untreated inoculation and longer for those who received filtered material. The reason for this variation is not clear but could represent a difference in the amount of infectious material present since presumably some of the agent was adsorbed by the filter in processing.

The character of onset varied considerably among the 16 patients studied. In five, the simultaneous occurrence of fever and constitutional and local symptoms marked a rather sudden onset of illness. In the remaining 11, the onset was gradual and marked by varying local and constitutional symptoms. The latter type of onset was in accord with that described by numerous authors in the naturally occurring disease. Early symptoms included dry or sore throat, nasal stuffiness, and headache. Feverishness and headache developed early, and chilliness was common and most prevalent on the second day following the onset. Malaise occurred in more than half the patients and was also an early symptom. Anorexia was present at some time in all patients. Local symptoms included nasal stuffiness, mild sore throat, and hoarseness. Cough was a conspicuous feature and was usually dry at first but later became paroxysmal and productive. All patients developed coughs between the first and fourth day. Sputum was mucoid at first, later becoming purulent. No patient developed grossly bloody or rusty sputum, but two of the most severely ill produced a slight blood streaking. Fourteen of the sixteen patients with pneumonia experienced chest discomfort, usually described as a sense of pressure or substernal soreness. Only one developed sharp pleuritic pain.

Physical findings.—Fine and coarse rales were present in 15 of the 16 patients. Only one had no rales at any time. Rales first appeared from the first to the ninth day after onset. They developed on the third and fourth day in the greatest number of patients. Slight dullness to percussion was present in 10 patients, changes in tactile and vocal fremitus in 5, and alterations of the breath sounds in 7. Only one patient developed pleural fluid. Two patients showed signs of central nervous system disturbance. One developed partial loss of bladder and rectal function with motor impairment of the lower limbs. In the other, there was transient areflexia of the lower limbs. Both eventually recovered completely.

Roentgenographic findings.—Roentgenographic findings conformed generally to those seen in the naturally occurring cases. The earliest findings were peribronchial infiltration, most commonly seen at the lung bases. The appearance was usually that of soft patchy densities of irregular size and shape. In some cases, the densities were small, discrete, and nodular, tending to become larger and confluent as the disease process continued. There was much variation in the development of these lesions. In four patients, it was limited to one or both lower lobes. In six patients, it spread toward the hilar region.

The distribution involved the lower lobes solely in 75 percent of the patients. In the others, the lower lobes were involved with concomitant lesions in other lobes. Average duration of roentgen-ray lesions was about 10 days with a few being present for only 1 to 3 days. It was not uncommon to find roentgenographic evidence of pneumonia before physical signs developed.

Fever.—All of the patients had fever. It began as early as the first day of illness in some and reached its peak incidence of onset between the second and fifth days. The maximum temperature observed was 104.8° F., and the average maximum was 102.8° F. By the ninth day following onset, most temperatures had returned to normal. The pulse and respiratory rates were not strikingly elevated except in a few patients who were severely ill with extreme pneumonic infiltration. The cases varied considerably in severity from very mild with minimal infiltration and fever for only 3 days to rather severe with extensive involvement of all lobes and fever for 15 days.

Complications.—Complications were observed in only four patients. One had maxillary sinusitis during the recovery period; one had a toxic psychosis associated with fever. Pleural effusion and encephalomyelitis each occurred in one patient.

Minor respiratory illness.—It is of interest that many of the subjects who were inoculated but did not develop atypical pneumonia did, however, develop evidence of a minor respiratory illness. Whether these represented mild infections with atypical pneumonia or infection with other agents present in the inoculum could not be determined.

Laboratory studies.—Total and differential leukocyte counts were in general within normal limits. Slight elevations were observed in a few of the more severely ill patients. Sedimentation rates showed no constant variation and, although they rose above normal in some individuals, the average for the group showed no striking increase.

Bacterial studies indicated that none of the common organisms found in the respiratory tract seemed to play any role in the infection. There were no concentrations of any one organism that suggested bacterial influence. Special efforts were made to recover the streptococcus MG described by Mirick and others.[13] The Commission's studies failed to relate these organisms causally to atypical pneumonia. Cold autohemagglutinins were found in 13 of the 16 cases of pneumonia in significant titers. The significance of this test in its relationship to the etiology of atypical pneumonia is not known. It is apparently of some diagnostic value in those cases in which it is present.

Etiologic Studies

From the beginning of the first work of the Commission group at Camp Claiborne until the end of the war, research was in progress to uncover the

[13] Mirick, G. S., Thomas, L., Curnen, E. C., and Horsfall, F. L., Jr.: Studies on a Non-Hemolytic Streptococcus Isolated From the Respiratory Tract of Human Beings. J. Exper. Med. 80: 391-440, November 1944.

agent of atypical pneumonia. Commission studies involved the use of chick embryos, mice, rats, cotton rats, hamsters, guinea pigs, cats, and monkeys. A few members of the Commission were sent to Puerto Rico to work with mongooses. In no instance was it possible to reproduce the disease until human volunteers were used. The electronic microscope and ultracentrifuge at Duke University, Durham, N. C., were used in an attempt to find virus particles but without success. Acute and convalescent sera from patients with atypical pneumonia failed to show antibodies against any of the known viruses or rickettsiae. Much of this work was done in the laboratory of Dr. Thomas Francis, Jr., at the University of Michigan at Ann Arbor. Extensive bacterial studies over a period of 5 years failed to reveal any bacterial agent responsible for the disease.

Epidemiologic Studies

The early work of the investigators at Camp Claiborne showed that a moderately severe epidemic of atypical pneumonia occurred during the summer of 1941. The peak epidemic rate was about three times as high as the average endemic rate prevalent at that camp. Studies failed to reveal any possibility of contamination of water, milk, or food supplies as a transmitting agent. At the same time, the disease was too widely spread to be easily charged to direct person-to-person contact of overt cases except for a small number which occurred among medical personnel. It was noted that many cases were mild and indistinguishable from common respiratory disease infections except by roentgenogram. It was concluded that these cases probably formed an inapparent reservoir which spread the disease from person to person. This was substantiated by subsequent work. It will be recalled that many of the inoculated human volunteers described earlier developed minor respiratory illness without evidence of pneumonia. In addition, previously unknown cases of atypical pneumonia, without symptoms, were found during roentgenographic surveys of entire units.

Later studies at Fort Bragg showed that new recruits experienced high rates of respiratory illness during the first 4 weeks after their arrival at camp.[14] The peak incidence of atypical pneumonia was likewise greatest during this period. The attack rate at Fort Bragg for respiratory diseases, in general, and for atypical pneumonia, in particular, followed a more or less constant ratio of 10:1. This led to some speculation as to the possibility of a common etiology. However, this possibility was not supported by results from subsequent studies in human volunteers who failed to develop pneumonia following inoculation with material recovered from patients with common respiratory disease. Reports from England specifically note the increased incidence of atypical pneumonia in the presence of normal figures for common respiratory

[14] Commission on Acute Respiratory Diseases: Acute Respiratory Disease Among New Recruits. Am. J. Pub. Health 36: 439–450, May 1946.

diseases.[15] A report from India takes note of the sharp increase in all respiratory diseases, including atypical pneumonia, during the hottest months of the year.[16] A similar experience was reported from Camp Claiborne during the summer of 1941, at which time the incidence of common respiratory diseases was at its usual seasonal level.[17]

The evidence indicates that atypical pneumonia is an infectious disease of virus etiology. It is generally, but not invariably, more prevalent at the time of greatest incidence of other respiratory diseases. It can be spread from person to person by infected respiratory secretions, and this is probably its natural mode of spread. The frequent finding of inapparent cases suggests the probability that such cases form a reservoir of infection from which clinical cases may arise.

SUMMARY

Atypical pneumonia, which first became recognized as a clinical entity in the late 1930's, made its appearance in the Army soon after large-scale mobilization began. It became clear after preliminary studies at Camp Claiborne that the disease was of major importance to the Army, and it eventually became an outstanding respiratory problem. It appeared in significant numbers in all theaters.

Special research in this field was carried on by the Army throughout the war. These studies showed that atypical pneumonia is an infectious disease which can be transmitted to human volunteers by the inhalation of infected bacteria-free filtrates. The specific etiologic agent was not determined, and extensive serologic studies failed to suggest a relationship to any known virus or rickettsia.

Morbidity and mortality rates for all pneumonia taken as a group were much lower in World War II than in World War I. There are probably many reasons for these differences, but unquestionably the use of chemotherapeutic agents and the absence of pandemic influenza were important factors.

Extensive clinical, laboratory, and epidemiologic investigations were pursued which confirmed and extended the findings of previous workers.

Available evidence suggests that the natural mode of spread is by person-to-person contact. Inapparent cases are known to exist which probably furnish a reservoir of infection. In general, atypical pneumonia was most prevalent during the colder months when there was an increase in all respiratory diseases. There were, however, some notable exceptions to this rule, and several epidemics were reported during the warm months.

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[15] Gordon, J. E.: A History of Preventive Medicine in the European Theater of Operations, United States Army, 1941-1945. Pt. III, Epidemiology, sec. 3, Acute Respiratory Infections, No. 3, The Pneumonias, pp. 1-14. [Official record.]

[16] Blumgart, H. L., and Pike, G. M.: History of Internal Medicine in India-Burma Theater. Pt. II, Internal Medicine in the India-Burma Theater. Respiratory Diseases, pp. 116-123. [In preparation.]

[17] See footnote 11, p. 214.

Section II. Bacterial Pneumonia

Richard G. Hodges, M. D.

In great contrast to World War I and probably to all previous wartime mobilizations, bacterial pneumonia during World War II did not present a major problem. The reasons for this were several. Although influenza, both A and B, involved the military population, nothing resembling the pandemic of 1918–19 occurred with its wake of pneumococcal, streptococcal, and influenzal pneumonia. Furthermore, there were no epidemics of measles to introduce pneumonia as a complication. This may be due to the automobile and the motion picture, both of which brought about an earlier and more general exposure of the rural population to measles. Finally, the widespread use of the sulfonamide drugs in the early treatment of febrile respiratory infections probably resulted in the prevention or abortion of many cases and certainly reduced the mortality to an extremely low level.

No meaningful figures can be given as to the incidence of bacterial pneumonia during the war years. The general decline of accurate bacteriologic diagnosis made it impossible to distinguish between bacterial and primary atypical pneumonia. During the early years of the war, it is probable that many cases of nonbacterial pneumonia were diagnosed as bacterial; when the diagnosis of primary atypical pneumonia had become popularized, it is probable that the error was in the opposite direction. The best available data on the comparative incidence of the two conditions is given in the preceding section dealing with primary atypical pneumonia.

Historically, the most important aspect of bacterial pneumonia was the information gathered about the spread and particularly the prevention of pneumococcal pneumonia.[18] The studies were carried out at a single installation, the Army Air Force Technical School at Sioux Falls, S. Dak. This was the only large military establishment that suffered severely from pneumococcal pneumonia. The investigation represented a joint project of the Commission on Pneumonia, Army Epidemiological Board, Office of the Surgeon General, and the Army Air Force Rheumatic Fever Control Program, Office of the Air Surgeon.

EPIDEMIOLOGY

Studies on the epidemiology of pneumococcal pneumonia covered a 3-year period at the Army Air Force Technical School at Sioux Falls, S. Dak. During this period of observation, more than 1,600 cases of pneumonia occurred. On several occasions, the attack rate exceeded 150 cases per annum per 1,000 average strength. The experience of the first year was studied in retrospect from hospital records; the data for the second year were gathered directly by an epidemiologist; during the third year, extensive bacteriologic and statistical

[18] Hodges, R. G., and MacLeod, C. M.: Epidemic Pneumococcal Pneumonia, pts. I–V. Am. J. Hyg. 44: 183–243, September 1946.

facilities were available. From this large experience, certain factors which contributed to the high pneumonia rates could be detected.

Influence of Population Characteristics and Environment

The two important population characteristics which influenced the pneumonia attack rates appeared to be length of service and duration of stay on the post. During the first months after the establishment of the post, it was impossible to distinguish between these two factors since men were brought to the school direct from basic training. Later, when the population was a mixture of new recruits and of seasoned troops, it was possible so to distinguish. There was a small but definite excess incidence in men newly introduced into military service. Regardless of length of service, the majority of pneumonia cases occurred in men spending their first 8 weeks at the post. Thus, it appeared that the troops became twice seasoned, first to military life in general and secondly to the particular environment of this particular technical school. Of these factors, the latter was by far the more important.

No way was devised of subjecting the environmental factors to controlled study. However, there was much to indicate that environment was important. In many ways, the operations of the technical school were admirably devised to promote the spread of respiratory disease. The barracks were of the theater of operations type, ill-suited to the climate of South Dakota. No efforts were made to promote ventilation or to control dust. The school buildings were no better than the barracks in these respects. The exact role of dust could not be determined, but pathogenic pneumococci were cultured from 29 percent of 147 dust samples. Moreover, there was a thorough mixing of the school population. Each class was composed of men from several different squadrons and from many different barracks. These arrangements facilitated cross-infection. It was shown that common respiratory diseases, pneumonia, streptococcal sore throat, epidemic influenza, and even specific serologic types of pneumococci spread rapidly and evenly throughout the school population.

No evidence was obtained that implicated previous geographic environment, age, chilling, or fatigue as being important factors in the production of pneumococcal pneumonia in the population as a whole.

Influence of Pneumococcal Carrier State

During the third year of the study, extensive carrier surveys were made in an attempt to relate the behavior of the causative organism to the incidence of pneumococcal pneumonia. Serologic typing was carried out with great care. Three methods of survey were used. A single squadron was sampled three times a week throughout the year. Cultures were made from all men admitted to hospital for respiratory diseases. Cultures were also taken from the occupants of a single barracks three times a week for 9 consecutive weeks. The carrier rates for the single squadron and for the hospital admissions, which

came from all squadrons, were identical and were combined to represent the population as a whole.

The total carrier rate was affected by season. Starting with a rate of approximately 40 percent in September, there was a sharp rise to 60 percent in November, and this high level was maintained throughout the winter season. Within the total carrier rate, the individual serologic types behaved rather independently, each attaining its own peak of incidence. Surprisingly, neither the total nor the specific carrier rates appeared to be affected by the incidence of common respiratory diseases.

Men newly arrived at the post were relatively free of pneumococci and were almost entirely free of the types which were known to produce pneumonia frequently. However, the new arrivals rapidly acquired pneumococci. After 4 weeks in the environment, their carrier rate was equal to that of the total population. Moreover, the new men became rapidly infected with the pathogenic types. This was demonstrated in the single barracks study. Only 7 percent of new men carried either type IV or XII, the types that were currently the leading cause of pneumonia. After 7 weeks in the barracks, 40 percent of the new men had become carriers of one or both of these types.

The single barracks study also demonstrated that the carrier state was dynamic in character. Men rapidly acquired and lost several different serologic types of pneumococci during the period of observation.

The incidence of pneumonia did not correspond to the total carrier rate nor did it correlate closely with the carrier rates for the highly infective types of pneumococci. At the times when the pneumonia rate was high, it was usual for the carrier rate to be high also, but there were several periods when the carrier rate for infective types was high and the pneumonia rate low. This indicated that some other factor besides the presence of the infective agent was necessary to produce high pneumonia rates.

Influence of Nonbacterial Respiratory Disease

During the entire 3 years of observation, there was a close relationship between the incidence of nonbacterial respiratory disease and that of pneumococcal pneumonia. On the average, 1 case of pneumonia was admitted to the hospital for every 10 patients admitted with nonbacterial respiratory disease. The seasonal occurrences of the two conditions paralleled each other closely, there being only two periods when the 1:10 ratio was not closely approximated. The first of these was in the 4 months after the post was opened. At that time, the incidence of nonbacterial respiratory disease was high but that for pneumococcal pneumonia low. Presumably, this was before the population had become thoroughly seeded with pneumococci. The other period was during the second winter of the study when, for a time, the ratio was more nearly 1:5 than 1:10. This corresponded to a period when the incidence of type II pneumonia was very high, approximately 60 percent of the cases of pneumonia. Later, it was possible to show that there were considerable differ-

ences in the "infectivity" of the various types of pneumococci. Infectivity was expressed as the number of men admitted with pneumonia due to type X divided by the number of respiratory admissions carrying type X. The value for type II was 0.52; whereas for type IV, it was 0.17; for type XII, 0.26; for type III, 0.10; and for type VIII, 0.09. Thus, when type II was prevalent, more cases of pneumonia per case of nonbacterial respiratory disease would be expected to occur.

Two epidemics, one of influenza A and one of influenza B, were identified. Both resulted in a sharp rise in the pneumococcal pneumonia rates, but during both the usual 1:10 ratio held good.

It was concluded that the incidence of pneumonia was governed by the prevalence of pneumococci, by the infectivity of the serologic types which were present, and by the incidence of nonbacterial respiratory disease.

PREVENTION OF THE DISEASE

The circumstances in the Army Air Force Technical School, Sioux Falls, S. Dak., appeared to be ideal to test the efficacy of immunization against pneumococcal pneumonia. Of the recorded attempts to conduct such immunization,[19] each had been handicapped by one or more of the following difficulties: (1) Differences in the composition of the immunized and control groups, (2) uncertainty as to whether the specific pneumococcal types included in the immunizing preparation were the same as those currently causing pneumonia, (3) failure to determine whether the observed decline in cases in the immunized group was due to a decrease in cases caused by the pneumococcal types in the vaccine, and (4) inadequate control of the antigenicity of the preparation used.

In the investigation conducted at Sioux Falls, the situation was such that each of these obstacles could be eliminated.[20] In the first place in the 2 preceding years, the population had been subjected to a thorough epidemiologic study of respiratory disease. It was known that the population was very uniform in respect to such epidemiologic characteristics as age, length of service, and duration of stay on the post, and that the environment of the troops was admirably devised to facilitate a rapid and uniform dissemination of respiratory disease throughout the entire population. This had been proven for streptococcal sore throat, pneumonia, influenza A, and the common respiratory diseases. Second, for the 2 preceding years, the pneumococcal pneumonia rates had been extremely high, and, equally important, the distribution of pneumococcus types causing pneumonia appeared to be uniform. For each of the 2 preceding years, the approximate distribution was: Type II, 34 percent; types I, V, and VII, 9 percent each; types XII and IV, 7 and 5 percent, respectively. Third, statistical machinery was established whereby the

[19] Heffron, R.: Pneumonia With Special Reference to Pneumococcus Lobar Pneumonia. New York: The Commonwealth Fund, 1939, pp. 446–483.

[20] MacLeod, C. M., Hodges, R. G., Heidelberger, M., and Bernhard, W. G.: Prevention of Pneumococcal Pneumonia by Immunization With Specific Capsular Polysaccharides. J. Exper. Med. 82: 445–465, December 1945.

population could be carefully followed, and a laboratory capable of doing extensive pneumococcal typing was organized. Finally, through the efforts of Dr. Michael Heidelberger, highly purified capsular polysaccharides of known antigenicity were available.

Accordingly, in September 1944, the entire personnel of the technical school was subjected to this test. A barracks was roped off longitudinally, and as the men were marched through they were given a random choice of which side of the rope they preferred. Those passing down one side received 1.0 ml. of saline containing 0.03 to 0.06 mg. of types I, II, V, and VII type-specific polysaccharide; those choosing the opposite side were injected with 1.0 ml. of saline containing 0.5 percent phenol. Subsequently, when a new troop shipment arrived at the post, alternate men received the polysaccharide solution or the saline, respectively. In all, 8,586 men were injected with the polysaccharide solution and 8,449 with saline. In terms of man-days exposure, the experience was 745,997 days for the immunized and 772,898 days for the nonimmunized. Many samplings of the population were taken to test for random distribution, and in each instance immunized and nonimmunized men were found to be present in equal numbers.

The effect of the immunization on the development of clinical pneumonia is shown in table 36. Pneumonia due to the types against which immunization was not practiced was equally divided between the treated and control groups. Pneumonia due to types I, II, V, and VII occurred 4 times in the treated group and 26 times in the control group. Moreover, each of the four cases in the immunized group occurred within 2 weeks after the individual was injected, whereas the cases in the control group were distributed at random over the period of observation (table 37). The number of type II cases was large enough to afford sound evidence of the protective value of the type-specific polysaccharide. There was no reason to believe that the specific protection against the other types was not equally good.

TABLE 36.—*Incidence of pneumonia in immunized and nonimmunized groups*

Type of pneumonia	Immunized group [1]	Nonimmunized group [2]
I	2	2
II	1	14
V	1	4
VII	0	6
Total	4	26
IV	8	6
XII	21	25
Other types	27	28

[1] 745,997 man-days exposure.
[2] 772,898 man-days exposure.

TABLE 37.—*Interval between injection and the development of the several types of pneumonia in immunized and nonimmunized subjects*

Interval (weeks)	Types I, II, V, VII		All other types	
	Immunized subjects	Nonimmunized subjects	Immunized subjects	Nonimmunized subjects
	Number	*Number*	*Number*	*Number*
1	2	0	1	1
2	2	3	5	3
3	0	3	7	5
4	0	2	8	12
6	0	2	6	7
8	0	2	3	4
10	0	1	4	4
12	0	0	2	4
14	0	2	2	1
16	0	3	2	4
16+	0	8	16	14
Total	4	26	56	59

The incidence for pneumonia due to types IV and XII, against which immunization was not practiced, was approximately that expected from the experience of the preceding 2 years. For types I, II, V, and VII, even in the nonimmunized group, the incidence of pneumonia was far below that expected. This strongly suggested that immunization of one-half of the population conferred a real protection on the nonimmunized subjects. There were plausible explanations for this. Reduction of the number of cases would comparably reduce the number of case contacts and might thus inhibit the spread of the organism. A more potent reason stemmed from the results of the carrier study which was being carried out simultaneously (table 38). The carrier rates for the types against which immunization was not practiced were almost equal in the immunized and nonimmunized groups. However, the rates for types I, II, V, and VII were significantly lower in the immunized group as compared with the control. Thus, the immunization of a given individual appeared to render him relatively resistant to becoming a carrier of the specific types contained in the vaccine. Consequently, in a population consisting of intimately mixed immunes and nonimmunes, every second transfer of a pneumococcus would result in the organisms falling on relatively infertile ground. The consistent behavior of types IV and XII in each of the 3 years of observation provided a means of calculating the amount of reduction in the incidence of type I, II, V, and VII pneumonia among the nonimmunes which was achieved by immunizing one-half the population. Only 17.6 percent of the expected cases were observed.

TABLE 38.—*Distribution of individual types of pneumococci between immunized and nonimmunized groups (excluding cases of pneumococcal pneumonia)*

Pneumococcal type	Immunized men [1]		Nonimmunized men [2]	
	Number	Percent	Number	Percent
I	6	0. 3	12	0. 7
II	14	. 8	30	1. 7
V	1	. 1	5	. 3
VII	11	. 6	12	. 7
Total	32	1. 79	59	3. 26
XII	127	7. 1	139	7. 6
All other types	1, 053	59. 0	1, 097	60. 6
Grand total	1, 212	67. 9	1, 297	71. 6

[1] Total cultures taken, 1,785.
[2] Total cultures taken, 1,810.

CONCLUSIONS

Bacterial pneumonia was not a major problem in World War II. However, studies conducted under the auspices of the Armed Forces did demonstrate the steps that should be taken to control pneumococcal pneumonia during future periods of mobilization. A high carrier rate for pneumococci and a high incidence of nonbacterial respiratory disease were shown to stimulate the occurrence of pneumonia. Although neither of these factors could be attacked directly, the implication was clear that more care in choosing the geographic location of military schools and more attention to the sanitation of installations in unfavorable climates would serve to lessen the spread of both bacterial and of nonbacterial respiratory disease.

Finally, it was possible to prove that a high degree of protection against pneumococcal pneumonia can be given by immunization with type-specific capsular polysaccharides.

CHAPTER XIII

Hemolytic Streptococcal Infections

Lowell A. Rantz, M. D.

THE MILITARY PROBLEM

Period before World War II.—Infection by hemolytic streptococci has been a major problem to the United States Army in all of its wars for which informative historical data are available. However, for a number of reasons, disease caused by these organisms was not recognized as an important military problem before World War II. The nature of infection by the hemolytic streptococcus, the essentials of its epidemiology, and particularly the intimacy of the relationship between it and the development of rheumatic fever have become well established only in the last 20 years. Furthermore, in all wars prior to World Wars I and II, the enormous incidence of enteric infection and malaria so overshadowed that of other infectious diseases that these attained relatively little prominence in the minds of medical officers responsible for the health of the Army.

Inspection of the recorded experience of the United States Army in the Civil War [1] demonstrates that hemolytic streptococcal disease occurred very frequently, and the magnitude of the problem was such that, had it been present in the Army during World War II, it would have been regarded as of the very greatest importance. During the Civil War, scarlet fever was an uncommon disease, only 696 cases having been reported. Approximately 25,000 cases of erysipelas, another form of streptococcal infection, were reported with a note that it was known that this was only a part of the total problem. One of every two hundred and twenty-five wounded developed the disease, which occurred essentially as primary facial erysipelas in epidemic form and also as a complication of battle injuries. It had a very definite geographic distribution in that the infection was much more common in troops stationed in the western areas, particularly in the States of Michigan, Ohio, Indiana, Illinois, Wisconsin, Iowa, Minnesota, Nebraska, and the Dakotas.

In addition to the widespread occurrence of erysipelas, there was also on enormous incidence of acute rheumatism in the Civil War.[2] In 5.2 years, 145,000 cases occurred among white troops at a rate of 65 per 1,000 per annum.

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[1] (1) Medical and Surgical History of the War of the Rebellion. Medical History. Washington: Government Printing Office, 1888, pt. III, vol. I, pp. 624, 662-675. (2) Medical and Surgical History of the War of the Rebellion. Surgical History. Washington: Government Printing Office, 1883, pt. III, vol. II, p. 851.

[2] Medical and Surgical History of the War of the Rebellion. Medical History. Washington: Government Printing Office, 1870, pt. I, vol. I, pp. 637-639.

Not all of these may be regarded as examples of rheumatic fever, but examination of the case records which have been preserved in the Medical and Surgical History of the War of the Rebellion indicates that a substantial number of them were certainly this disease. This impression is substantiated by 642 deaths reported to have been caused by rheumatism, endocarditis, and pericarditis. In retrospect, it is impossible to define in absolute terms the magnitude of the problem of rheumatic fever in the Civil War, but it is obvious that it was great. An attempt to determine the geographic distribution of acute arthritis on the basis of available data was unsatisfactory.

The hemolytic streptococcus was a common cause of bacterial pneumonia complicating influenza during the pandemic of 1918. Primary infection by these organisms was not recognized as an important problem. There were 11,675 admissions for scarlet fever, but the mortality was extremely low, and the disease caused little concern.[3] Similarly, little interest was aroused by 2,598 admissions for erysipelas. The significance of 24,770 admissions for acute articular rheumatism was entirely overlooked. This disorder was not considered to be probable rheumatic fever, and no attempt was made to determine the frequency of occurrence of chronic valvular heart disease as a complication.

Rheumatic fever is not mentioned in the official history of the Medical Department of the United States Army in the World War. Acute articular rheumatism appears only in the statistical reports, and this disease was not deemed worthy of special comment elsewhere although the number of cases was very great. The available data do not permit a definition of the geographic incidence, but inspection of the statistical information reveals that disease in this category was very common in the same areas in which outbreaks of rheumatic fever were observed during World War II. On the basis of these data covering only 2 years of active mobilization, streptococcal respiratory infection and the frequently associated rheumatic fever must have occurred in epidemics comparable to those observed during World War II.

Advances in understanding and accumulation of information about hemolytic streptococcal infection had been continuous and of far-reaching importance in the decade before the onset of World War II. In spite of this fact, the Medical Department of the United States Army found itself poorly prepared to cope with the problem of infection by these organisms. Several well-defined factors were responsible for this lack of preparation. Of prime importance was the failure of the Medical Department and of the civilian physicians associated with it to recognize the extreme importance of hemolytic streptococcal respiratory infection occurring without a skin rash. Emphasis was largely on the control of scarlet fever. Of equal significance was the lack of appreciation of the intimate relationship between hemolytic streptococcal disease and rheumatic fever. Also, medical officers were not trained to distinguish clinically between hemolytic streptococcal and nonbacterial respiratory infections, and diagnostic bacteriologic

[3] The Medical Department of the United States Army in the World War. Statistics. Washington: Government Printing Office, 1925, vol. XV, pt. 2, p. 86.

methods were not readily available to them. Lastly, the Army and its consultants had not familiarized themselves with available civilian and military data on the basis of which it would have been possible to predict with considerable accuracy those geographic areas in which outbreaks of hemolytic streptococcal disease and rheumatic fever might be expected to occur.

Little information is available as to the situation during the prewar expansion of the Armed Forces. During 1941, outbreaks of scarlet fever followed by rheumatic fever occurred at Chanute Field, Ill., Scott Field, Ill., and Fort Knox, Ky. The incidence of these diseases elsewhere in the Army during this year was low. Drs. James D. Trask, Francis F. Schwentker, and M. Henry Dawson, of the Commission on Hemolytic Streptococcal Infections, visited each of these camps in November 1941. They noted that medical officers did not recognize the association of scarlet fever and streptococcal disease occurring without a rash nor did they connect either of them with rheumatic fever.[4]

World War II period.—Plans were made to investigate outbreaks of hemolytic streptococcal infections during the coming year. Between December 1941 and April 1942, Dr. Schwentker made bacteriologic studies at the camps mentioned and at Fort Francis E. Warren, Wyo., where many cases of scarlet fever were occurring.[5]

Available records do not reveal that streptococcal disease was viewed with alarm during 1941 and 1942, although the frequency of scarlet fever in the total Army during these years was comparable to that in 1943 and 1944 when interest in infection by streptococcal organisms was very great. The absence of concern in the earlier period was the direct result of the lack of accurate reports [6] on the occurrence of rheumatic fever prior to 1943 and to the mildness of acute streptococcal disease.

The first detailed information in regard to the problem of rheumatic fever was obtained when a survey early in February 1943 of the continuing scarlet fever epidemic at Fort Warren revealed that more than 100 cases of streptococcal disease had been hospitalized but neither correctly diagnosed nor reported to The Surgeon General.[7] Subsequently, the rapid increase in size of installations in areas of high incidence of this disease in Colorado and Utah was associated with epidemics of streptococcal infection and rheumatic fever, and additional surveys were made which again delineated the deficiencies in background and information on the part of medical officers in regard to streptococcal respiratory disease and its complications, but no control measures

[4] Report, M. Henry Dawson, M. D., Director, Commission on Hemolytic Streptococcal Infections, Army Epidemiological Board, 24 Nov. 1941, subject: Report on Reconnaissance Trip to the Fifth and Sixth Corps Areas by Dr. James D. Trask, Dr. Francis F. Schwentker, and Dr. M. Henry Dawson, Members of the Commission on Hemolytic Streptococcal Infections, November 11-19, 1941.

[5] Schwentker, F. F.: Survey of Hemolytic Streptococci in Certain Army Camps. Army M. Bull. No. 65: 94-104, January 1943.

[6] Rheumatic fever was first included in the weekly statistical summary in February 1942.

[7] Report, Lowell A. Rantz, M. D., Member, Commission on Hemolytic Streptococcal Infections, Army Epidemiological Board, to Col. S. Bayne-Jones, Office of the Surgeon General, 23 Feb. 1943, subject: Report of Epidemic of Scarlet Fever and Septic Sore Throat, at Fort Francis E. Warren.

were recommended.[5] Principal interest of this investigation centered in the large number of cases of rheumatic fever and the varying clinical patterns of streptococcal infection. Scarlet fever was common in certain stations; in others, tonsillitis without rash was the rule. In one post, suppurative complications were exceedingly common.

During 1943, 6,710 admissions for rheumatic fever were reported from the Army in the United States. According to summaries of the statistical health reports, about 74 percent of these were in the Seventh and Ninth Service Commands. About 43 percent of all cases occurred in the States of Colorado, Utah, Idaho, Montana, and Wyoming. The 10 stations having more than 100 cases of rheumatic fever and an annual rate of greater than 5 per 1,000 in 1943 are presented in table 39. Their geographic proximity is apparent. There is the presumption that the incidence of rheumatic fever has been underreported on the statistical health report.

TABLE 39.—*Incidence of rheumatic fever in the U. S. Army, selected installations, 1943*

[Preliminary data based on summaries of the statistical health report]

[Rate expressed as number of cases per annum per 1,000 average strength]

Station	Number of cases	Rate
Fort Francis E. Warren, Wyo	806	49. 9
Lowry Field, Colo	620	30. 0
Camp Crowder, Mo	420	12. 2
Buckley Field, Colo	343	26. 4
Kearns Field, Utah	250	14. 4
Camp Carson, Colo	257	9. 3
Sioux Falls Army Air Field, S. Dak	203	10. 6
Lincoln Army Air Field, Mo	192	12. 4
Scott Field, Ill	161	8. 1
Chanute Field, Ill	108	5. 4

This large number of cases of a serious disease requiring prolonged hospital care and resulting in many separations from service attracted very considerable interest in the Offices of the Surgeon General of the Army and the Air Surgeon. Three programs aimed at the acquisition of new knowledge about hemolytic streptococcal disease with special reference to its relationship to rheumatic fever and to control methods were instituted toward the end of this year. One of these programs was under the auspices of the Commission on Hemolytic Streptococcal Infections of the Army Epidemiological Board. Its purpose was the intimate investigation of a large number of cases of hemolytic streptococcal respiratory infection for the purpose of defining the natural history,

[5] Report, Chester S. Keefer, M. D., Director, and Lowell A. Rantz, M. D., Member, Commission on Hemolytic Streptococcal Infections, Army Epidemiological Board, April 1943, subject: Report of Investigation of Rheumatic Fever at Fort Francis E. Warren, Cheyenne, Wyo., Lowry Field, Denver, Colo., Buckley Field, Denver, Colo., and Camp Carson, Colorado Springs, Colo.

bacteriology, and immunology of these disorders. Associated with this group was the Commission on Air-Borne Infections whose main interest lay in the investigation of methods for control of airborne infection with particular reference to the hemolytic streptococcus. The third was the Army Air Forces Rheumatic Fever Control Program, which will be the subject of a later section of this chapter.

These three programs were in active operation throughout 1944. Much new knowledge was acquired, but effective methods for the control of streptococcal disease were not forthcoming. Dust control and air sterilization with glycol vapors were proved not to be of great value and were not applied in other than experimental field studies.

The Air Forces investigated intensively the role of sulfonamide prophylaxis during the early months of 1944. Impressive results were obtained in the reduction of streptococcal infection and rheumatic fever, and the use of this technique in certain defined situations, primarily for the prevention of these diseases, became established Army policy on 1 November 1944 with the publication of TB MED 112. Unfortunately, by this time highly sulfonamide-resistant strains of streptococci had emerged and were causing disease among naval personnel where chemoprophylaxis had been widely used on a mass basis since December 1943.

Streptococcal infection continued to be epidemic throughout 1944, and 4,877 cases of rheumatic fever were reported in the United States. The highest incidence was in the Sixth and Seventh Service Commands where 37 percent of the cases occurred. Twenty-four percent occurred in the States of Colorado, Utah, Wyoming, and Nevada, where relatively few troops were stationed. The widespread movement of troops throughout the country may have been responsible for larger numbers of cases in other areas than during the previous year. By this time, highly communicable strains of hemolytic streptococcus were doubtless seeded throughout the Army and its numerous establishments.

In 1944, streptococcal infection and rheumatic fever became an important problem among troops overseas for the first time, 4,639 cases being reported. This represented a rate of only 1.21 per 1,000 per annum or approximately one-third of that among troops in the United States.

As the winter of 1945 began, it became apparent that sulfonamide prophylaxis, the only tool of value for the prevention of streptococcal infection and rheumatic fever, had become ineffective. Disease caused by resistant streptococci was epidemic in the United States Navy, and an outbreak of infection caused by similar strains had occurred in an Army Air Forces station. The problem was considered at a National Research Council conference on 28 February 1945. The failure of sulfonamide prophylaxis was discussed in detail, and the hazards involved in its continued use were described. As a result of these experiences in the Navy, this technique was applied only selectively in the Army Air Forces and practically not at all in the Army Ground Forces. The possible value of penicillin prophylaxis was explored at another National

Research Council conference on 20 March 1945, and certain studies for its evaluation under field conditions by the Army Air Forces were outlined but were not undertaken.

Although the incidence of scarlet fever and rheumatic fever declined sharply in the Zone of Interior during 1945, in spite of the absence of effective control measures, the incidence of streptococcal sore throat rose from 0.82 per 1,000 in 1944 to 3.64 in 1945 (table 40). The combined effect was an increase in incidence from 3.98 in 1944 to 5.21 in 1945. Only 1,675 cases of rheumatic fever were reported. Two thousand and fifty additional cases occurred in the Army overseas.

An important National Research Council conference on streptococcal disease was held on 7 July 1945, and recommendations were made which will be the subject of later comment. The war ended in the fall of that year, and information on the occurrence of these diseases during demobilization is not available.

STATISTICAL INFORMATION

The previous section has indicated that hemolytic streptococcal respiratory infection and its complications were an important problem during World War II.

TABLE 40.—*Incidence rates of scarlet fever, streptococcal sore throat, and rheumatic fever in the U. S. Army, continental United States, by service command and year, 1944–45*

[Preliminary data based on individual medical record sample tabulations of primary and secondary diagnoses]
[Rate expressed as number of cases per annum per 1,000 average strength]

Service command	Scarlet fever			Streptococcal sore throat			Rheumatic fever [1]		
	1944–45	1944	1945	1944–45	1944	1945	1944–45	1944	1945
First	1.1	1.2	0.9	1.3	0.8	1.9	0.6	0.7	0.4
Second	1.2	1.4	.8	1.7	.8	2.7	.6	.6	.5
Third [2]	1.8	2.7	.7	2.8	.6	5.6	.6	.7	.5
Fourth	.5	.5	.6	1.1	.4	2.3	.5	.5	.4
Fifth	1.6	2.5	.5	1.5	.4	2.8	.8	.8	.8
Sixth	3.5	4.5	2.2	6.3	5.7	7.1	1.7	2.5	.7
Seventh	2.7	3.7	1.4	2.9	.8	5.7	2.4	3.7	.7
Eighth	1.4	1.6	1.2	1.9	.6	3.8	.8	.8	.7
Ninth	2.1	2.8	1.2	1.9	.9	3.2	1.3	1.7	.6
Total	1.5	1.9	1.0	2.0	.8	3.6	.9	1.2	.6

[1] Includes Sydenham's chorea.
[2] Includes Military District of Washington.

During 1942 through 1945, 26,063 cases of scarlet fever were reported in the Army in the United States and 3,449 additional cases from the Army overseas (table 41). During the 2 years, 1944 and 1945, when streptococcal sore throat was coded separately, 20,471 cases were diagnosed (table 42). This figure was entirely too low as streptococcal respiratory infection occurring without a rash was recognized in only its most typical clinical form.

The author believes that not less than 5 cases of streptococcal sore throat were hospitalized for every case of scarlet fever. If this approximation be accepted, at least 150,000 men suffered infection by hemolytic streptococci of this degree of severity during 1942–45, inclusive. A minimum of 5 days of hospitalization was required by each, a loss of 750,000 man-days during the war. These statistics do not include a group at least equally large in whom infection occurred which was not sufficiently severe to require hospital care, but which reduced efficiency for several days.

The data pertaining to rheumatic fever are even more significant: there were 18,339 cases reported (table 43). Average hospitalization was not less than 3 months and usually more. At least 2 million man-days were lost. Many of these men were separated from service and may very well have later received service-connected disability compensation and care under the Veterans' Administration.

TABLE 41.—*Incidence of scarlet fever in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on individual medical record sample tabulations of primary and secondary diagnoses]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	26, 063	1. 77	2, 873	1. 08	12, 585	2. 43	7, 680	1. 93	2, 925	1. 00
Overseas:										
Europe	2, 516	. 57	34	. 41	177	. 44	1, 085	. 65	1, 280	. 54
Mediterranean [1]	308	. 21	3	. 13	81	. 18	74	. 11	150	. 42
Middle East	30	. 21	1	. 17	13	. 25	11	. 24	5	. 12
China-Burma-India	57	. 13	11	1. 26	16	. 40	10	. 06	20	. 09
Southwest Pacific	149	. 08	90	1. 26	33	. 17	16	. 03	10	. 01
Central and South Pacific	123	. 10	12	. 08	25	. 09	31	. 07	55	. 15
North America [2]	183	. 37	58	. 58	75	. 39	25	. 19	25	. 37
Latin America	6	. 02	1	. 01	1	. 01	4	. 05	0	
Total overseas [3]	3, 449	. 32	213	. 36	382	. 23	1, 274	. 33	1, 580	. 34
Total Army	29, 512	1. 16	3, 086	. 95	12, 967	1. 89	8, 954	1. 15	4, 505	. 59

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes incidence on transports.

The cost to the United States Army in terms of dollars and effectiveness as the result of hemolytic streptococcal infection and its complications cannot be assessed. The loss of about 900,000 man-days per year presented a problem of considerable magnitude.

TABLE 42.—*Incidence of streptococcal sore throat in the U. S. Army, by area and year, 1944–45*

[Preliminary data based on individual medical record sample tabulations of primary and secondary diagnoses]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1944–45		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	13, 926	20. 17	3, 266	0. 82	10, 660	3. 64
Overseas:						
Europe	2, 990	. 74	525	. 31	2, 465	1. 04
Mediterranean [1]	982	. 98	427	. 66	555	1. 57
Middle East	131	1. 50	31	. 67	100	2. 45
China-Burma-India	370	. 95	105	. 62	265	1. 20
Southwest Pacific	921	. 58	181	. 34	740	. 71
Central and South Pacific	916	1. 12	216	. 49	700	1. 86
North America [2]	67	. 34	32	. 25	35	. 51
Latin America	82	. 52	27	. 31	55	. 76
Total overseas [3]	6, 545	7. 73	1, 560	. 41	4, 985	1. 10
Total Army	20, 471	13. 32	4, 826	. 62	15, 645	2. 07

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes incidence on transports.

During 1942–45, the number of deaths due to the three streptococcal diseases was as follows:

Scarlet fever	61
Rheumatic fever	28
Streptococcal sore throat (1944–45)	12

There were 689 cases of streptococcal pneumonia in 1944–45, 524 cases in continental United States, and 165 cases overseas. There were 20 deaths (13 in the United States and 7 overseas) attributed to streptococcal pneumonia. The disease was notably uncommon even in areas in which streptococcal infection was epidemic. At Chanute Field, only 16 cases with 1 death were observed between 1 January 1942 and 21 April 1945.[9] Pulmonary involvement was a not infrequent complication of the type 17 sulfonamide-resistant outbreak of streptococcal disease that occurred at Keesler Field and Amarillo Army Air Field during late 1944 and early 1945. At the latter station, 312 cases of streptococcal respiratory infection were recognized, of which 19 were compli-

[9] Countryman, H. D.: The Treatment of Pneumonias and Their Complications. News Letters, Army Air Forces Rheumatic Fever Control Program, vol. 2, No. 9, September 1945, pp. 6–12.

cated by pneumonia. There was one death. Prompt treatment with penicillin was highly efficacious in controlling the illness.[10]

During 1942–45, 2,398 cases of erysipelas were reported, 1,627 in the continental United States and 771 overseas.

TABLE 43.—*Admissions for rheumatic fever in the U. S. Army, by area and year 1942–45* [1]

[Preliminary data based on sample tabulations of individual medical records]

[Rate expressed as number of admissions per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	14, 176	0. 96	1, 529	0. 58	6, 710	1. 29	4, 447	1. 12	1, 490	0. 51
Overseas:										
Europe	1, 929	. 44	27	. 33	82	. 31	725	. 43	1, 095	. 46
Mediterranean [2]	755	. 51	8	. 35	202	. 44	370	. 57	175	. 49
Middle East	60	. 41	4	. 66	21	. 40	25	. 54	10	. 24
China-Burma-India	110	. 25	2	. 23	19	. 48	44	. 26	45	. 20
Southwest Pacific	572	. 31	33	. 46	50	. 26	194	. 36	295	. 28
Central and South Pacific	388	. 31	27	. 18	66	. 23	130	. 30	165	. 44
North America [3]	215	. 44	52	. 52	84	. 43	54	. 42	25	. 37
Latin America	59	. 15	19	. 19	17	. 14	13	. 15	10	. 14
Total overseas [4]	4, 163	. 39	179	. 31	554	. 33	1, 575	. 41	1, 855	. 40
Total Army	18, 339	. 72	1, 708	. 53	7, 264	1. 06	6, 022	. 77	3, 345	. 44

[1] Includes Sydenham's chorea.
[2] Includes North Africa.
[3] Includes Alaska and Iceland.
[4] Includes admissions on transports.

EPIDEMIOLOGY

Geographic Incidence

The geographic distribution of streptococcal infection in the continental United States has been emphasized. Table 40 demonstrates that rates for disease caused by these organisms were high in the Sixth and Seventh and very low in the Fourth Service Commands. These facts are further presented in tables 44 and 45, which show the occurrence of scarlet fever and rheumatic fever during 1943 and 1944 when the most widespread epidemics were in progress.

Inspection of these figures, particularly the incidence by States, reveals that streptococcal infection was most common in belts lying to the east and

[10] Report, Maj. Norman B. Roberg, MC, subject: A Survey of an Epidemic Caused by a Virulent Sulfadiazine-Resistant Strain of the *Streptococcus Hemolyticus* (Group A, Type 17) at Amarillo Army Air Field.

west of the Rocky Mountains, in the area of the Great Plains, and around the Great Lakes. The experience was worst in Colorado, Utah, and Wyoming. The Navy also encountered very severe outbreaks of these diseases in their installations in these same general areas.

Civilian experience would have suggested that the Northeastern States should have experienced a high incidence of these disorders. That it did not is doubtless the result of the fact that few troops were trained there. It was used principally as a staging area for well-seasoned men.

TABLE 44.—*Incidence rates of scarlet fever and rheumatic fever in the continental United States, by service commands, 1943–44*

[Rate expressed as number of cases per annum per 1,000 average strength]

Service Command	Scarlet fever	Rheumatic fever
First	1. 81	0. 87
Second	2. 29	. 53
Third [1]	1. 78	. 56
Fourth	. 67	. 40
Fifth	1. 99	. 72
Sixth	5. 53	2. 49
Seventh	7. 83	6. 76
Eighth	1. 65	. 57
Ninth	2. 47	1. 22

[1] Includes the Military District of Washington.

Equally noteworthy was the infrequent occurrence of hemolytic streptococcal infection among troops stationed in the Southern United States. Epidemics did occur there, particularly during 1944 and 1945, but they were short lived and of little consequence.

Additional pertinent information was obtained by the Army Air Forces Rheumatic Fever Control Program in a study of group A hemolytic streptococcal carriers at several Army Air Forces installations. The data for the period 1 January to 21 April 1944 are summarized as follows: [11]

Location	*Percent*
Buckley Field, Colo	30. 3
Amarillo Field, Tex	13. 1
Lincoln Army Air Field, Nebr	9. 7
Kearns Field, Utah	9. 2
San Antonio Aviation Cadet Center, Tex	. 3
Drew Field, Fla	1. 4
Davis-Monthan Field, Ariz	. 7

The higher carrier rates among troops stationed in the Rocky Mountain area, the Middle West, and northern Texas are to be contrasted with the very

[11] Van Ravenswaay, A. C.: The Geographic Distribution of Hemolytic Streptococci. Relationship to the Incidence of Rheumatic Fever. J. A. M. A. 126: 486–490, 21 Oct. 1944.

TABLE 45.—*Incidence rates of scarlet fever and rheumatic fever in the continental United States, by State, 1944*

[Rate expressed as number of cases per annum per 1,000 average strength]

Location	Scarlet fever	Rheumatic fever	Location	Scarlet fever	Rheumatic fever
First Service Command:			Sixth Service Command:		
Connecticut	0	0.5	Illinois	3.8	2.6
Maine	1.0	1.0	Michigan	2.3	1.0
Massachusetts	1.3	.8	Wisconsin	8.3	3.7
New Hampshire	1.0	1.0	Seventh Service Command:		
Rhode Island	2.7	0	Colorado	5.5	10.8
Vermont	0	0	Iowa	9.6	4.3
Second Service Command:			Kansas	.9	.8
Delaware	.5	.5	Minnesota	5.2	.8
New Jersey	2.0	.7	Missouri	3.4	1.5
New York	1.2	.5	Nebraska	4.5	2.1
Third Service Command:			North Dakota	0	0
District of Columbia	.2	.1	South Dakota	5.8	4.8
Maryland	2.4	.4	Wyoming	2.5	2.7
Pennsylvania	3.5	1.1	Eighth Service Command:		
Virginia	3.5	1.0	Arkansas	.8	.5
Fourth Service Command:			Louisiana	.2	.7
Alabama	.5	.5	New Mexico	1.4	2.0
Florida	.2	.6	Oklahoma	1.7	.6
Georgia	.6	.5	Texas	2.1	.9
Mississippi	.5	.3	Ninth Service Command:		
North Carolina	.7	.9	Arizona	2.0	1.4
South Carolina	.4	.3	California	2.0	1.4
Tennessee	.6	.7	Idaho	4.3	.4
Fifth Service Command:			Montana	4.6	1.9
Indiana	1.9	.5	Nevada	8.9	6.1
Kentucky	2.0	.9	Oregon	5.9	3.4
[illegible]io	5.1	.7	Utah	9.4	5.0
[illegible]st Virginia	0	0	Washington	1.8	1.1

low rates in the South. This study doubtless reflects the previously emphasized geographic variations in the occurrence of streptococcal disease.

The results of the two studies by the Commission on Acute Respiratory Disease, Army Epidemiological Board, were equally striking. These studies were conducted at Fort Bragg, N. C., one from April to June 1943 and the other from March to May 1944.[12] In each year, approximately 900 successive admissions to the hospital because of respiratory disease were investigated. About 4 percent in both years were proved by appropriate bacteriologic and immunologic procedures to be of group A hemolytic streptococcal origin. The

[12] (1) Commission on Acute Respiratory Diseases: Endemic Exudative Pharyngitis and Tonsillitis. Etiology and Clinical Characteristics. J. A. M. A. 125: 1163–1169, 26 Aug. 1944. (2) Commission on Acute Respiratory Diseases: Role of β-Hemolytic Streptococci in Common Respiratory Disease. Am. J. Pub. Health 35: 675–682, July 1945.

troops involved were unseasoned during both periods and highly susceptible to undifferentiated respiratory infection.

Another study, from January to April 1944, was carried out at Camp Carson.[13] Fifteen hundred cases of hospitalized respiratory disease were examined and about 350, or 23 percent, were proved to be caused by hemolytic streptococci.

A contrasting situation within the author's experience at Fort Francis E. Warren is of interest. This post of about 20,000 men was experiencing a severe outbreak of streptococcal disease in February 1943. Forty cases of scarlet fever per week were entering the hospital. On 8 February 1943, a special barracks hospital was opened. Three days later, 100 new cases of acute streptococcal respiratory disease occurring without skin rash, sufficiently severe to deserve bed care, had been admitted to the hospital. All were personally examined and studied bacteriologically by the author. This represented a streptococcal disease rate at that time of around 650 per 1,000 per annum.

The causes of these striking geographic relationships in the frequency of occurrence of hemolytic streptococcal infection and its complications have been the subject of much speculation, but no satisfactory explanation has been offered. The implications in regard to the establishment of camps for future training of troops are obvious.

Approximately 12 percent of all scarlet fever admissions and 23 percent of all rheumatic fever admissions (tables 41 and 43) occurred in the Army overseas. The rates for the former disease were about one-fifth and for the latter about two-fifths of those in the continental United States. The worst experiences were encountered in the European, Mediterranean, and Middle East theaters, and the North American area. Streptococcal infection was a much less important problem in the Pacific Ocean, Southwest Pacific, and Latin American areas. This distribution is in accord with the available information regarding the occurrence of these disorders among civilian populations in the several areas.

There were 77 cases of scarlet fever and 75 admissions for rheumatic fever on transports, during the years 1942–45, according to individual medical record tabulations.

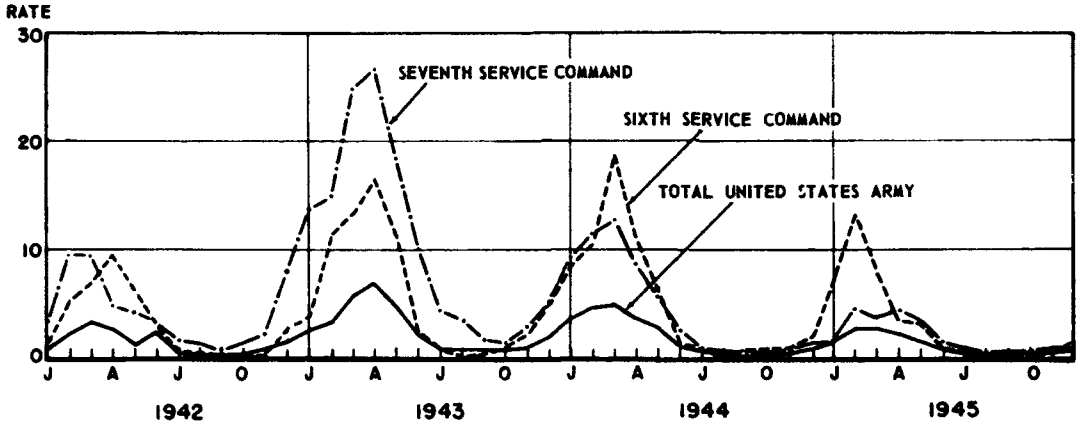
Seasonal incidence.—Hemolytic streptococcal infection has been regarded as a disease of the winter months. This was true, to a certain extent, in the Army in the continental United States. Inspection of chart 21 reveals, however, that whereas the incidence of scarlet fever began to increase in December and January 1942–43, 1943–44, and 1944–45, respectively, peak levels were not attained until March or April, and rates remained high until the end of May and then fell sharply through the summer. This was particularly true in the Sixth and Seventh Service Commands where epidemics were in progress.

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[13] Rantz, L. A., Rantz, H. H., Boisvert, P. J., and Spink, W. W.: Streptococcic and Nonstreptococcic Disease of the Respiratory Tract; Epidemiologic Observations. Arch. Int. Med. 77: 121–131, February 1946.

CHART 21.—*Seasonal incidence of scarlet fever in the Army in the continental United States, 1942–45*

[Rate expressed as number of cases per annum per 1,000 average strength]



The highest incidence of rheumatic fever always lags about 1 month behind the streptococcal respiratory infection. A comparison of charts 21 and 22 shows that this was the case in the total Army in 1943 and 1945, the highest level being attained each year in May. However, many cases appeared throughout the summer. In 1944, the peak occurred in April. Satisfactory explanations for these seasonal relationships have not been forthcoming.

Seasoning of Personnel

Epidemics of nonbacterial respiratory infection are very likely to occur when raw recruits are assembled in basic training centers. Throughout the last war, it was tacitly assumed that this was also the case in regard to hemolytic streptococcal disease. This opinion emerged since the most serious outbreaks in both the Army and the Navy occurred in stations in which men fresh from civilian life were undergoing basic training.

It is curious to learn that, in spite of the great importance of the problem, a critical study was never made by the Army or the Navy for the purpose of determining the relative susceptibility to streptococcal disease of personnel of various degrees of seasoning.

Experience in the Navy, as described by Coburn,[14] indicates that well-seasoned men were highly susceptible to streptococcal disease when brought together in centers in high-incidence areas. One study was conducted in an Army camp in such an area in which nearly all of the personnel had had from 7 to 24 months of service. Annual rates of hospitalization for streptococcal infection of 200 per 1,000 were attained.[15]

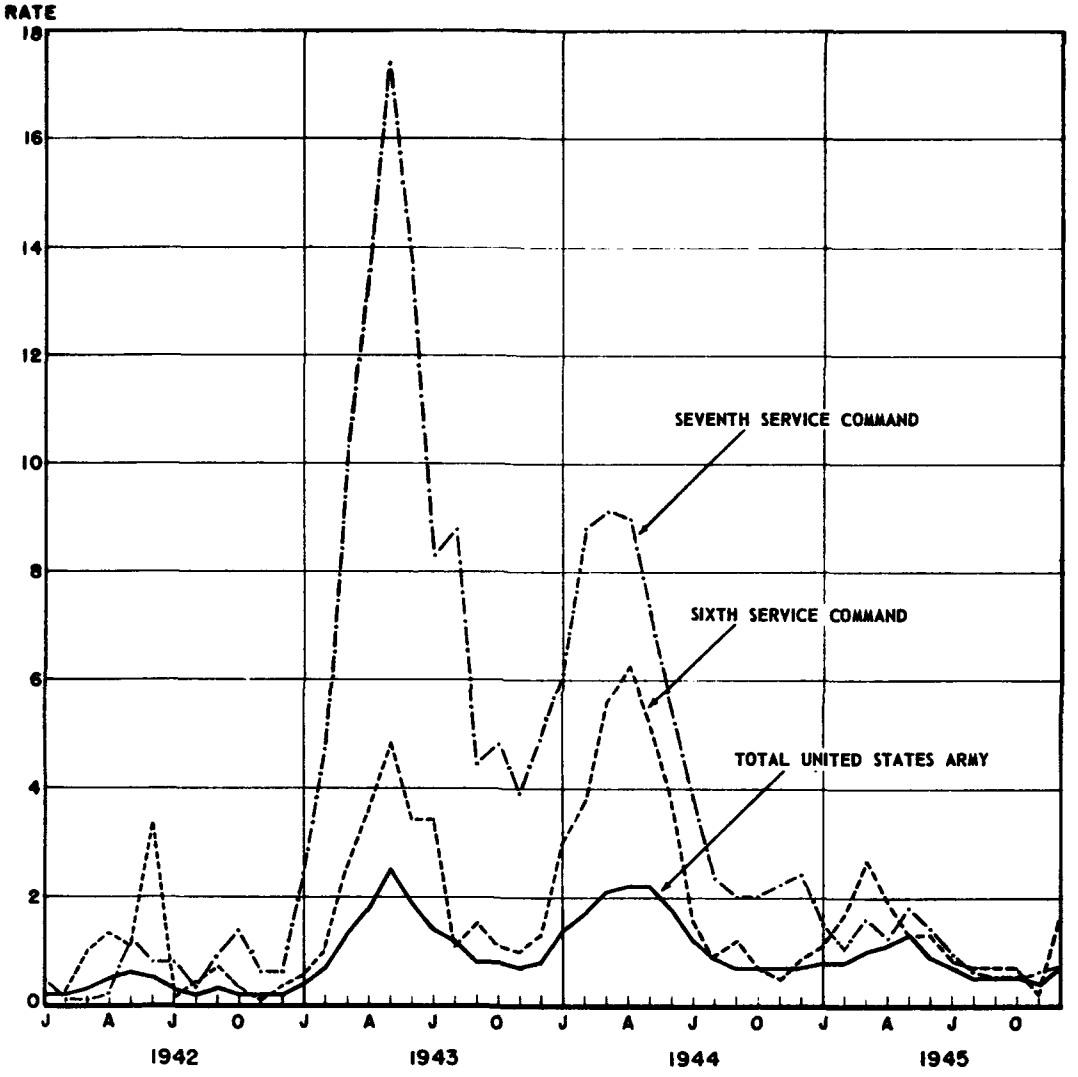
Another pertinent investigation suggested that the rates might have been much higher had a similar number of raw recruits been assembled.

[14] Coburn, Alvin F., and Young, Donald C.: The Epidemiology of Hemolytic Streptococcus During World War II in the United States Navy. Baltimore: Williams and Wilkins, 1949.

[15] See footnote 13, p. 240.

CHART 22.—*Seasonal incidence of rheumatic fever in the Army in the continental United States, 1942–45*

[Rate expressed as number of cases per annum per 1,000 average strength]



Approximately 300 cases of sulfonamide-resistant type 17 hemolytic streptococcal infection were hospitalized at Amarillo Army Air Field during February and March 1945. An annual rate of 105 per 1,000 was observed among 7,000 well-seasoned men. A basic training squadron of about 1,000 men experienced an annual rate of 864 per 1,000, 8 times that in the seasoned troops.[16]

In summary, the meager evidence indicates that all types of troops brought together in areas where streptococcal infection is common will experience outbreaks of disease caused by these organisms. One study suggests that the epidemic will be more intense among new recruits.

[16] See footnote 10, p. 237.

Movement of Troops

During one investigation, it was clearly shown that the transfer of troops from a low- to a high-incidence area was accompanied by an outbreak of streptococcal disease even among seasoned troops.[17] Inconclusive evidence was obtained which suggested that men who had been stationed for 6 months in the latter area were relatively immune to infection. Annual rates among such troops were about 50 per 1,000 as contrasted with previously mentioned rates of 200 per 1,000 among fresh troops, but seasoned men moved into the study post from a low-incidence area.

The severity and persistence of certain epidemics of streptococcal infection which occurred in basic training establishments in high-incidence areas were probably specifically related to the constant movement into such posts of men in small groups from other parts of the country. This constant influx of susceptibles doubtless served to maintain the epidemic situation and may have been of greater importance than the lack of seasoning. The practice of distributing these new arrivals throughout the post, rather than maintaining them in separate units, insured the exposure of all to the epidemic streptococcus.

Movement of troops from high- to low-incidence areas must have been associated with the establishment of outbreaks of varying degrees of severity in stations which had previously been relatively free of streptococcal illness. This definitely occurred on several occasions in the Navy.[18] The Amarillo Army Air Field epidemic in 1945 was initiated by troops transferred from Keesler Field.[19] No other documented episodes of this type in the Army are known to the author.

If such spread from high-incidence areas occurred frequently with the establishment of new and prolonged epidemics, there should have been a leveling off of the relative frequency of scarlet fever and rheumatic fever between various areas during 1942–44. Such a comparative study is possible at present only on a service command basis. Inspection of table 46 reveals that the rates for scarlet fever in each service command were quite stable throughout the critical period when men who had received their basic training in high-incidence areas were being distributed throughout the country.

The seven service commands (excluding the Sixth and Seventh) in which rheumatic fever rates had been low during 1942 and 1943 all showed very definite increases during 1944. This may well have reflected a spread of streptococcus into these areas by troops from the Sixth and Seventh Service Commands. Rates for rheumatic fever remained notably low throughout the war for the Second Service Command as well as the Eighth. Epidemics occurred in these areas, as has been previously noted, but were explosive and short lived.

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[17] See footnote 13, p. 240.
[18] See footnote 14, p. 241.
[19] See footnote 10, p. 237.

TABLE 46.—*Incidence rates of scarlet fever and rheumatic fever, by year and service command, 1942–45*

[Rate expressed as number of cases per annum per 1,000 average strength]

Service command	Scarlet fever				Rheumatic fever			
	1942	1943	1944	1945	1942	1943	1944	1945
First	0.5	2.0	1.6	0.6	0.2	0.7	1.2	0.9
Second	.7	2.7	1.8	1.1	.3	.4	.7	.6
Third	.4	1.6	2.3	1.9	.2	.4	1.0	.4
Fourth	.3	.7	.6	.6	.2	.3	.6	.5
Fifth	1.7	1.6	2.4	.7	.4	.3	1.2	.8
Sixth	2.5	5.6	5.4	2.3	.7	2.1	3.0	1.0
Seventh	3.8	10.5	4.6	1.7	.7	8.1	5.2	1.0
Eighth	1.3	1.7	1.5	1.2	.3	.4	.8	.9
Ninth	1.4	2.2	2.9	1.1	.3	.9	1.7	.9
Total	1.1	2.5	2.1	1.1	.3	1.2	1.4	.8

Housing

Overcrowding in barracks and classrooms was often considered to be a contributing factor to epidemics of streptococcal infection in the Army. Critical evaluation was impossible since virtually all Army establishments were overcrowded from 1942 to 1944 when disease caused by these organisms was most prevalent. Many exceedingly crowded posts in low-incidence areas escaped epidemic streptococcal disease entirely. The congestion in barracks doubtless aggravated the spread of hemolytic streptococci, but its elimination would not have been an effective prophylaxis if other conditions had been favorable to the organism.

Relationship to Virus Infection

The experience of World War I had alerted physicians in the Army to the potentialities of influenza as a precursor of hemolytic streptococcal pneumonia. It was also believed that infection by this and other respiratory viruses might predispose to upper respiratory infection by streptococci. Evidence supporting this possibility was not forthcoming. The increase in rates for scarlet fever during and after the pandemic of influenza A in November and December 1943 was no greater than the usual seasonal increment. One critical study in a post in an area of high incidence at this time failed to demonstrate any increase at all in the streptococcal disease rate.[20] It is of considerable interest that this epidemic was not associated with the appearance of hemolytic streptococcal pneumonia even in areas where streptococcal respiratory infection was common.

[20] See footnote 13, p. 240.

Information is not available to the author which would indicate that rubella, rubeola, or mumps significantly enhanced the susceptibility of troops to streptococcal infection. Doubtless, an occasional case of these disorders was complicated by streptococcal pneumonia.

Hemolytic Streptococcus Grouping and Typing

It was well known in 1940 that the hemolytic streptococci could be divided into groups and types by serologic techniques. Nearly all primary human infections of the respiratory tract had been shown to be caused by strains belonging to group A. The members of this group could be subdivided into types by agglutination or precipitin methods. It was the perfection of the latter technique which permitted the widespread application of streptococcal typing during World War II. The agglutinative method, although a valuable tool, cannot be standardized for use by laboratories without personnel with a great deal of special training.

The first application of serologic typing in the Army was by Schwentker, who studied a small number of cases of scarlet fever and tonsillitis at Chanute Field, Scott Field, Fort Knox, and Fort Warren, between 18 December 1941 and 25 March 1942. A large number of different types were recovered from scarlet fever contacts and from healthy carriers. Only 11 types were shown to be causing disease. At Fort Warren, type 19 was responsible for nearly all infections.[21]

Another study made between March and June 1942 at Chanute Field by the Commission on Air-Borne Infections revealed that 85.5 percent of all infections were due to types 18, 19, 1, 6, 17, and 26 in that order. Between November 1942 and August 1943, 86.7 percent were caused by types 19, 1, 3, 6, 17, 36, 18, and 5.[22] In February 1943, a survey showed that type 19 was still the only important type responsible for disease at Fort Warren. The frequency of various types was determined at Camp Carson between December 1943 and May 1944 by the Commission on Hemolytic Streptococcal Infection. A somewhat broader spectrum was encountered here but 13 were responsible for 94.5 percent and 7, types 36, 19, 3, 17, 30, 46, and 6, for 66.1 percent of all infections.[23]

After April 1944, complete data are available on type distributions in Army Air Forces installations from the Rheumatic Fever Control Program. Between April and August 1944, 85.2 percent of 2,021 presumed cases of group A streptococcal respiratory infection were caused by types 19, 30, 3, 1, 17, 14, 6, 36, 5, 26, and 12. The first 6 were responsible for 77 percent of all illness.[24]

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[21] See footnote 5, p. 231.

[22] Hamburger, M., Jr., Hilles, C. H., Hamburger, V. G., Johnson, M. A., and Wallin, J. G.: Ability of Different Types of Hemolytic Streptococci to Produce Scarlet Fever. J. A. M. A. 124: 564-566, 26 Feb. 1944.

[23] See footnote 13, p. 240.

[24] News Letter, Army Air Forces Rheumatic Fever Control Program, vol. 1, Nos. 1-5; vol. 2, Nos. 1-9, August 1944-September 1945.

After this date, the type incidence is expressed only on the basis of rates which makes epidemiologic calculation and comparison more difficult. An analysis has been made based on reports of 10 months of study in which each type has been scored on the basis of its having caused a significantly large number of cases (2.58 × standard error) in any month in any post or, if this value was not calculated, a rate of 10 per 1,000 per annum.

Only nine types caused significant amounts of disease: Type 17 in 11 months, type 19 in 7, type 30 in 2, and types 1, 3, 5, 6, 26, and 36 each in 1 month. The same nine types in 27 additional months were responsible for rates of 5 per 1,000 per annum. Only four others, 11, 10–12, 14, and 22 attained these levels and then only in single post months.

In summary, the results of streptococcal typing presented here demonstrate that the same small group of types caused the bulk of studied respiratory infection by these organisms between the spring of 1942 and the fall of 1945. During the period 1942 to the summer of 1944, types 1, 3, 6, 17, 19, and 36 were most frequently recovered. During late 1944 and 1945, types 3, 17, 19, and 30 were of the greatest importance.

It is apparent from these observations that the Army functioned for several years as a closed epidemiologic unit in which streptococci were serially transmitted. Strains of a certain small group of types retained their high degree of communicability over a long period of time. The dominance of types 3, 17, and 19 during the last year was very largely due to the fact that they had become sulfonamide resistant and were thus able to spread in populations protected by these drugs.

An additional feature of the typing program was the establishment of a relationship between type and the ability of the streptococcus to produce a skin rash. The observation was first made by Hamburger and his associates of the Commission on Air-Borne Infections. These workers noted that between November 1942 and August 1943, types 1, 3, 17, 19, and 29 were frequently associated with scarlet fever. Sufficient observations were available to indicate strongly that types 5, 6, 18, and 36 were not able to stimulate the development of a rash.

These observations were extended by the Commission on Hemolytic Streptococcal Infections at Camp Carson between December 1943 and May 1944. During this period only types 3, 17, 19, and 30 were rash formers. Nonscarlatinogenic types were 1, 5, 6, 24, 26, 36, 44, and 46. Critical study revealed that strains of the latter types were erythrogenetically ineffective even in Dick-positive persons.[25]

Considerable additional and related information was obtained by the Army Air Forces Rheumatic Fever Control Program. Between April and August 1944, 89.4 percent of 229 cases of scarlet fever were caused by types 1, 3, 17, 19, and 30. Strains of only types 6 and 18 of those previously shown by the commissions to be nonscarlatinogenic were recovered from 2 cases of

[25] Rantz, L. A., Boisvert, P. J., and Clark, W. H.: The Relationship of Serological Types of Group A Hemolytic Streptococci to Toxin Formation and Antibody Response. Stanford M. Bull. 6: 55–65, February 1948.

scarlet fever during this period although they were responsible for more than 70 cases of respiratory infection occurring without rash. It is possible that the streptococci from these two cases were incorrectly classified.

At any event, certain strains remained highly scarlatinogenic for 2 years and others were unable to stimulate rash production. After October 1944, all data are presented in the form of rates per 1,000 per annum, and no strain is recorded unless a frequency of 1 per 1,000 was attained in some month in a post under study. Over an 11-month period, only disease caused by types 3, 17, 19, and 30 attained these levels, although during 1 post-month types 6, 24, and 26 were recorded as etiologically responsible for scarlet fever.

The relationship of the various serologic types in the causation of rheumatic fever was of interest, but little information was obtained which can be reviewed profitably. *Streptococci* of type 1[26] and an unidentified type[27] were the cause of extensive foodborne outbreaks that were not followed by the development of rheumatic fever. Similarly, the type 17 *Streptococcus* responsible for the epidemic at Keesler Field and Amarillo Army Air Field was not capable of inciting the rheumatic state.[28] The impression is gained that certain strains were rheumato-genetically more potent than others and that this property was not related specifically to the numerical type.

An attempt was made to define the geographic distribution of the several types of group A hemolytic streptococcus by the typing laboratory at the Santa Ana Army Air Base.[29] This study was carried out between 25 April and 1 July 1944. Throat cultures were obtained from 5,828 men within 48 hours after their arrival at this post from the various college training detachments of the Army Air Forces Western Flying Training Command. The total group A carrier rate was circa 200 per 1,000, but this varied from 490 per 1,000 to 1 per 1,000 depending on the establishment from which the troops had come. The predominant types were similar to those noted, 1, 3, 6, 14, 17, 19, 30, and 36, and they were found to be widely distributed throughout the country. Space does not permit a further detailed analysis of this interesting study.

Foodborne Streptococcal Infection

Food contaminated by hemolytic streptococci is well able to transmit these organisms to susceptible persons. When this occurs, an explosive outbreak of streptococcal respiratory disease occurs which rea[illegible]ts peak in from 48 to 72 hours and then declines rapidly. The diseas[illegible]ess is not different from that seen in more usual circumstances, and rheumatic fever is a frequent complication.

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[26] Rantz, L. A., Spink, W. W., and Boisvert, P. J.: Hemolytic Streptococcus Sore Throat; Detailed Study of Simultaneous Infection of a Large Number of Men by a Single Type. Arch. Int. Med. 76: 278–283, November-December 1945.

[27] Bloomfield, A. L., and Rantz, L. A.: An Outbreak of Streptococcic Sore Throat in an Army Camp; Clinical and Epidemiologic Observations. J. A. M. A. 121: 315–319, 30 Jan. 1943.

[28] See footnote 10, p. 237.

[29] Mitchell, R. B.: Geographical Distribution of the Serological Groups and Types of Beta Hemolytic Streptococci. Report from Streptococcus Typing Laboratory, Army Air Forces Regional Hospital, Santa Ana Army Air Base, 21 Oct. 1944. [Official record.]

There were 19 recorded epidemics of streptococcal infection during 1942–45, involving 2,879 cases, and others doubtless occurred but were not recognized or reported. Three were studied by commissions of the Army Epidemiological Board, and the results were published in scientific journals.[30]

Of greatest interest to this history is the fact that careful bacteriologic investigation of foodhandlers was made in five of these outbreaks, and a hemolytic streptococcal carrier was discovered among the kitchen staff in four of them. The serologic type of streptococcus recovered from two of these individuals was the same as that isolated from infected cases. The other two strains were not typed. Messworkers with acute tonsillitis were discovered during the investigation of two other epidemics, but no bacteriologic studies were made.

Traditional epidemiologic study of such outbreaks includes elaborate survey of food and water supplies with particular reference to milk. The observations just recorded indicate that the usual source of the infectious agent is a hemolytic streptococcal carrier among the foodhandlers who contaminates certain articles of food after they are cooked. Simple bacteriologic methods, readily applicable by almost any military establishment, should permit the detection and control of such individuals and the elimination of foodborne streptococcal disease as a health hazard in the Armed Forces.

DIAGNOSIS

At the onset of World War II, medical officers were inadequately trained in the recognition of hemolytic streptococcal infection. The author, during 1943, repeatedly examined large numbers of patients in epidemic areas in whom the presumptive diagnosis of streptococcal respiratory infection could be made on inspection of the nasopharynx. The correct etiology of these cases had usually not been suspected by the officers in charge, even though concurrent epidemics of scarlet fever were in progress. This situation was partly the result of insufficient previous experience and partly because of the failure to appreciate the importance and significance of streptococcal infection occurring without a skin rash.

An additional factor was the inability of the usual station hospital laboratory to isolate and identify adequately the hemolytic streptococcus. Bacteriologic control of clinical diagnoses was essentially impossible in most installations during the early years of the war.

Similarly, rheumatic fever was not regularly recognized clinically, even in epidemic areas, until early in 1943 and was not reported to The Surgeon General. Facilities for the adequate study of this disease were usually available but were not regularly utilized. This situation changed rapidly during

[30] (1) See footnote 26, p. 247. (2) See footnote 27, p. 247. (3) Commission on Acute Respiratory Diseases: Study of a Foodborne Epidemic of Tonsillitis and Pharyngitis Due to β-Hemolytic Streptococcus, Type 5. Bull. Johns Hopkins Hosp. 77: 143–210, September 1945.

1943, and by 1944 most medical officers had been alerted to the fact that rheumatic fever was occurring frequently in the Army, and they were able to diagnose and treat the disease with considerable precision.

CONTROL OF STREPTOCOCCAL INFECTION

The control of hemolytic streptococcal infection was a problem which engaged the attention of all branches of the Armed Forces during World War II. Extensive studies were carried out by the Commission on Hemolytic Streptococcal Infection and the Commission on Air-Borne Infections of the Army Epidemiological Board which are described elsewhere in the records of that board. The Army Air Forces established its own rheumatic fever control program in the fall of 1943 under orders from Maj. Gen. (later General of the Army) Henry H. Arnold. Its objectives were (1) recommendations for the use of sulfonamide prophylaxis for the control of respiratory infections and rheumatic fever, (2) adoption of uniform standards for the diagnosis of rheumatic fever, (3) coordination and standardization of bacteriologic techniques in the study of the hemolytic streptococcus, (4) establishment of a uniform convalescent program and followup studies on positive and suspected cases of rheumatic fever, and (5) consideration of special projects and investigations at various Army Air Forces posts.

Only objectives 1, 3, and 5 are germane to this chapter. The other two were accomplished under the direction of Ruth Pauli Callender. Uniform techniques for the isolation and serologic classification of the hemolytic streptococci were adopted for use throughout Army Air Forces streptococcal laboratories.

Forty hospitals were members of the initial cooperating group and a chief of the streptococcal laboratory was designated in each. Later reports presenting detailed epidemiologic information were derived entirely from 10 stations: Amarillo Army Air Field, Buckley Field, Davis-Monthan Field, Drew Field, Hamilton Field, Calif., Keesler Field, Lincoln Army Air Field, Lowry Field, San Antonio Aviation Cadet Center, and Santa Ana Army Air Base. At each of these posts a comprehensive program was undertaken in January 1944 and continued until September 1945. The work was divided into three phases, proceeding concurrently: (1) Determination of incidence of hemolytic streptococcal infection by systematic study of nasopharyngeal flora of all hospital admissions with respiratory illness, (2) determination of hemolytic streptococcal carrier rates by regular bacteriologic samplings of nasopharyngeal flora of troops, and (3) determination of serologic groups and types of all isolated hemolytic streptococci.

A very large amount of information in regard to certain aspects of the epidemiology of hemolytic streptococcal respiratory infection was obtained during the 21 months in which this program was in operation.

It is probable that no other infectious disease has been studied on a nationwide basis by such a well-coordinated group applying uniform techniques.

Methods of Control

Methods applied during World War II for the control of hemolytic streptococcal infection may be considered in two categories: (1) General hygienic measures and (2) chemoprophylaxis. Any possible value of the former, which included such techniques as isolation of infected personnel, elimination of overcrowding, and the like, was largely n[illegible]y in the war by the failure to recognize the importance of strepto[illegible]on occurring without a rash. For this reason, cases of scarlet fever were isolated and their contacts intensively examined, but a huge reservoir of potential transmitters of disease in the nonrash cases was ignored. Later, this situation was rectified somewhat, but such nonspecific measures failed to terminate the serious outbreaks of streptococcal infection, and chemoprophylaxis was given very serious consideration by the Army.

Sulfonamide prophylaxis

Sulfonamide prophylaxis of hemolytic streptococcal respiratory infection was used infrequently in the Army during World War II, but a more detailed review of the development of this technique, its applications, and subsequent failures is appropriate to this history.

The first reports of the possible value of the continuous administration of sulfonamides over long periods of time for the prevention of streptococcal disease were presented from two civilian clinics in 1939. This experience was rapidly expanded by others with uniformly good results and low toxicity. A panel of experts in the summer of 1943 was able to recommend the widespread application of this method for the prevention of recrudescences in rheumatic subjects.[31]

Early in 1942, it was discovered by the Commission on Meningococcal Meningitis that the administration of 2 gm. of sulfadiazine in a single dose was highly effective in eliminating meningococci from the nasopharynx of carriers and in terminating epidemics of meningitis caused by these organisms. The earliest known deliberate use of these drugs in the Armed Forces for the prevention of streptococcal infection was that of Maj. (later Col.) Russel V. A. Lee, MC, at the Santa Ana Army Air Base in 1942. The study was not controlled. At about this time, Watson and his associates conclusively demonstrated the value of sulfonamide prophylaxis in the control of an epidemic of scarlet fever on a Navy pier in New York.[32]

In spite of this information and in the face of the gravely high incidence of streptococcal infection and rheumatic fever in the Army and the Navy, sulfonamide prophylaxis was not undertaken by either service during the winter of 1942–43. It is difficult for the author, who was in constant touch

[31] Proceedings of Conference on Rheumatic Fever, Washington, D. C., October 5–7, 1943. U. S. Department of Labor, Children's Bureau Publication 308. Washington: U. S. Government Printing Office, 1945.

[32] Watson, R. F., Schwentker, F. F., Fetherston, J. E., and Rothbard, S.: Sulfadiazine Prophylaxis in an Epidemic of Scarlet Fever. J. A. M. A. 122: 730–733, 10 July 1943.

with the situation in the Army through this period, to be certain why this delay occurred. Presumably, the constant fear of reactions to the prolonged administration to large numbers of troops of these potentially highly toxic drugs was a principal deterring factor.

At a conference on 7 September 1943, the Navy decided to employ sulfadiazine prophylactically, and the program was well established by mid-December. The results of this extraordinary experiment in preventive medicine have been described in two monographs.[33] More than 600,000 men received either 0.5 or 1.0 gm. of sulfadiazine daily for varying periods of time throughout the winter and spring of 1943–44. For the first few months, controlled studies were done and brilliant results obtained in that the incidence of streptococcal respirarory infection and rheumatic fever was strikingly reduced. By March 1943, controlled work had been abandoned, and the drug was given to all naval personnel in training in the continental United States.

During 1943, the Army was deeply interested in this technique, and plans were laid for its study during the winter of 1943–44 by the Commission on Hemolytic Streptococcal Infections and by the Army Air Forces Rheumatic Fever Control Program. Only one order from The Surgeon General permitting chemoprophylaxis in the Army during 1943 has been discovered.[34] No record of its implementation is available. On another occasion, Headquarters, New York Port of Embarkation, recommended the administration of sulfonamides to the scarlet fever contacts. It is not known whether the drug was used in this situation.[35]

The Commission on Hemolytic Streptococcal Infections failed to institute its proposed investigation of sulfonamide prophylaxis during 1943–44, but the Army Air Forces proceeded with an extensive program. Several careful studies were done at Sioux Falls Army Air Field, Truax Field, Wis., and Lowry Field. Various schedules were employed, and it was learned that a daily 0.5 gm. dose of sulfadiazine was adequate. Toxicity was amazingly uncommon. Only 47 men of 36,500 receiving the drug experienced any untoward effects, and in 33 the reactions were mild. The usual impressive reduction in streptococcal disease and rheumatic fever was obtained.[36] These controlled investigations were not completed until May 1944, at which time the streptococcal disease rates were declining rapidly. Chemoprophylaxis was not employed widely through the remainder of the Army Air Forces installations and not at all by Army Ground Forces during this season.

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[33] (1) See footnote 14, p. 241. (2) The Prevention of Respiratory Tract Bacterial Infections by Sulfadiazine Prophylaxis in the United States Navy. NAVMED 284, Bureau of Medicine and Surgery, Navy Department, Washington: U. S. Government Printing Office, 1944.

[34] Letter, Lt. Col. R. V. Lee, MC, to Air Surgeon, Headquarters, Army Air Forces, 19 Mar. 1943, subject: Prophylactic Use of Sulfadiazine in Prevention of Scarlet Fever, with endorsements thereto.

[35] Memorandum, Commanding General, Headquarters, New York Port of Embarkation, for Surgeon, Staging Area, 20 May 1943, subject: Disposition and Prophylaxis Treatment of Contacts With Scarlet Fever in Task Forces and Station Complement Units.

[36] Professional Division, Office of the Air Surgeon: Prophylactic Use of the Sulfonamides. Air Surgeon's Bull. 1 No. 9: 5–7, September 1944.

The Army Air Forces was greatly impressed with its studies during early 1944 and went ahead rapidly with the development of a program for general application during 1944–45. This culminated with the issuance of Army Air Forces Letter 25–20 from the Commanding General, Army Air Forces, authorizing chemoprophylaxis as a command function and the publication of the details of the method in the *Air Surgeon's Bulletin*, September 1944. A conference was held on 3 September 1944 attended by representatives of The Surgeon General, the Army Epidemiological Board, the Air Surgeon, and the Bureau of Medicine and Surgery (United States Navy) for the purpose of discussing this program which had already been authorized by the Air Surgeon.

Although some evidence had already been obtained that prophylaxis was breaking down in the Navy, this group endorsed the Army Air Forces program for use throughout the Army. Mass administration of the sulfadiazine was not approved. The drug was to be given only when acute respiratory disease, scarlet fever, or meningococcal meningitis reached a certain prescribed minimum rate and was to be continued for only 3 weeks unless special circumstances indicated a more prolonged use of this technique. On 1 November 1944, TB MED 112 was issued, authorizing the use of sulfonamide prophylaxis throughout the Army when proper indications were found to exist. Approval of the appropriate higher medical echelon was required.

During this same month, an epidemic of type 17 sulfonamide-resistant streptococcal infection, which continued for several weeks, began at Keesler Field. Outbreaks of disease caused by this same organism occurred later at Amarillo Army Air Field and at Lowry Field and were traced to troops moved into the two posts from Keesler Field. Several similar epidemics were observed during the winter of 1945, although the total streptococcal disease rate remained low.

Sulfonamide prophylaxis was, therefore, ineffective in terminating the more serious epidemics and not needed under other circumstances in 1945. It is believed that it was applied only rarely during that year although actual data as to the number of men treated are not available.

The appearance of sulfonamide-resistant streptococci as a cause of disease among men receiving mass chemoprophylaxis was believed to be the result of the appearance of drug-fast mutants during the rapid multiplication and transfer of these organisms in a population altered by the administration of these drugs in such a way as to favor the survival of resistant variants and the disappearance of sensitive ones.

Although sulfonamide-resistant hemolytic streptococci have not become a common cause of disease in the civilian population since the war, there is every reason to suppose that they would again appear if mass prophylaxis with these drugs were undertaken in the Armed Forces. There is no reason why the method should not be applied in more limited groups on a short-term basis in an attempt to terminate an epidemic of streptococcal infection.

Other control methods

The continuing epidemic of hemolytic streptococcal infection in many Navy installations after the breakdown of sulfonamide prophylaxis led to an exhaustive discussion of the problem at a National Research Council conference held in Washington, D. C., on 6 and 7 July 1945. The techniques of potential value that were considered are described as follows:

Control of carriers. The Commission on Air-Borne Infections demonstrated that the individual convalescent from streptococcal infection who harbored organisms in the nose disseminated them much more freely than did simple tonsillar and pharyngeal carriers. Such persons were regarded as "dangerous" carriers. Detection by bacteriologic methods and isolation of these potentially infectious men was considered but never attempted during World War II. Their number would have been great in certain camps and the administrative problem enormous. There was general agreement that detection of carriers by mass pharyngeal swabbing was not a valuable procedure since many of these men would not be "dangerous."

Oiling of floors and bedding. The Commission on Air-Borne Infections demonstrated that many hemolytic streptococci could be recovered from the air of barracks in which "dangerous" carriers were housed and that their numbers increased sharply during periods of activity such as dressing, bedmaking, and, particularly, floorsweeping. The possibility existed that these extrahuman reservoirs might be potent sources of infection.

Simple and entirely applicable methods were devised for their control including oiling of floors (fig. 4) and bedding. These techniques were shown experimentally to reduce greatly the contamination of the air of barracks by bacteria and were field tested for their ability to reduce the incidence of streptococcal disease. The results were disappointing since, at a time when these infections were not actually epidemic and in a population composed largely of seasoned men, the reduction of streptococcal illness was only about 40 percent. When applied during an epidemic of undifferentiated respiratory disease, no decrease in rate occurred. A similar test during a severe outbreak of streptococcal infection was not attempted by the Army.

The available results clearly indicate that extrahuman reservoirs are potentially important in the spread of streptococcal disease and that their control is desirable. Unfortunately, droplet infection during direct contact of carrier and susceptible seems to be the most important mode of transmission of these organisms. This is probably particularly true during the periods of intimate physical association connected with basic training in the Armed Forces.

Glycol vapors. The vaporization of glycols into the air of hospital wards containing "dangerous" streptococcal carriers was shown by the Commission on Air-Borne Infections to be effective in lowering the number of hemolytic streptococci in the air. The effect was most marked when floors and blankets had been oiled.



FIGURE 1. Soldiers of a sanitary company oiling the floor of a barrack. The soldier on the left sprinkles oil from a can while the two on the right spread the oil with brushes.

Methods were never devised by the Commission which would permit the accurate control of the concentration of glycol vapor in the air. This difficulty, in addition to other problems involving the relative humidity at which these substances are bactericidal, made it most unlikely that they would be of value in the control of streptococcal or other respiratory infection in the Armed Forces. A controlled field study of this method was never attempted by the Army.

Ultraviolet irradiation. The Army did not make use of or study extensively ultraviolet irradiation in the control of airborne streptococcal disease. It was not found to be of value in a limited investigation by the Navy and will probably not be applicable in the future by the Armed Forces.

Active immunization. Active immunization, leading to the enhancement of resistance to infection by the tissues of the susceptible host, was theoretically the most desirable way to control the spread of infectious disease when general public health measures proved to be ineffective. This technique had not been extensively explored in relation to hemolytic streptococcal disease, but certain considerations suggested that it might not be feasible. Immunity to infection

by group A *Streptococci* was believed to be type specific, and many different types were endemic in the Armed Forces. A polyvalent vaccine would have been required containing representatives of a large number of types. Probably none could have been administered in such a mixture in sufficient quantities to stimulate the production of effective quantities of antibody.

Two groups investigated streptococcal vaccination during World War II. The Navy carried out an important field study in which a monotype epidemic was not interrupted by immunization under the most favorable circumstances. The Commission on Hemolytic Streptococcal Infections demonstrated that measurable type-specific antibodies appeared in the serum only when relatively huge amounts of streptococcus were administered over a period of several weeks. Both of these studies also showed that group A streptococcal vaccines were very toxic substances, and the latter indicated that their use was associated with the development of increased sensitivity.

The use of Dick immunization for the prevention of scarlet fever was repeatedly considered during the war but, insofar as is known, never undertaken. This procedure was discarded since it conferred no antibacterial immunity and could not be expected to reduce the total incidence of streptococcal infection. It would merely have lowered the frequency of occurrence of cases with a skin rash. This result could be of no value, particularly in the light of the toxicity inherent in this form of immunization and the large number of injections required.

General sanitary measures.—No type of special sanitary measures over and above those generally recommended for the control of respiratory disease were employed by the Army to combat the spread of streptococcal infection. In 1945, the Navy did propose an intensive program including a number of such techniques because epidemics of infection caused by these organisms continued to be an important problem. These proposals included (1) maintenance of 50 square feet of floor space and 450 cubic feet of room space per man and avoidance of overcrowding of all other facilities; (2) dust-control measures, including oiling of floors and bedding, in barracks, classrooms, and all other indoor areas where men congregated; (3) airing and cleaning of barracks and bedding between each filling with recruits; (4) adequate ventilation; (5) adequate refrigeration, improvement of milk dispensers, and inspection of foodhandlers; (6) changes in dispensary practice to prevent overcrowding and spread of disease among men on sick call; and (7) changes in hospital practice designed to prevent cross-infection.

These seven proposals could not have been expected to affect materially the course of a serious outbreak of streptococcal disease, although each was desirable in its own right and all should have been part of standard military practice. Certain other phases of the program might well have been of great value although administratively exceedingly difficult to apply; namely, (1) reduction of size of training regiments, (2) segregation of each regiment, (3) placement of groups of new arrivals together rather than spread throughout

the installation, (4) screening of newly arrived and departing men for presence of respiratory infection, and (5) division of barracks into cubicles.

The effects of this program on an epidemic of streptococcal disease was probably never determined since the war ended in 1945. Because it failed to come to grips with essential problems in the epidemiology of streptococcal infection, it would probably not have been more than partially effective.

SUMMARY

The previous sections of this chapter have indicated that hemolytic streptococcal infection and rheumatic fever are most likely to become major problems to the health of the Army when large numbers of recruits are brought together for training in certain geographic areas. Among Army personnel, more than one-third of all cases of rheumatic fever in the continental United States during World War II occurred in the Sixth and Seventh Service Commands, although the total number of troops trained there was relatively small. For instance, according to a special sample tabulation of individual medical records during 1944–45, 17 percent were derived from installations in Colorado, Wyoming, and Utah, where the streptococcal disease rates were appallingly high.

A simple measure for the partial control of hemolytic streptococcal disease and its complications would be a change in policy so that basic training is not undertaken in high-incidence areas. Seasoned men will also be at risk but to a lesser degree, partly because they are seasoned and partly because their training is more likely to be accomplished in large units with relatively little replacement of fresh men in small groups.

A second goal of this program would be the prevention of seeding of many widely scattered installations of the Army by men who received their basic training in areas in which streptococcal disease is epidemic and who have become carriers of highly communicable organisms. Wide dispersement of men at the end of periods of basic training is inevitable. It is much less likely to occur among troops at later stages of their training program.

Certain evidence, notably incomplete, has been presented to show that this was an important factor in the spread of streptococcal infection throughout the Army between 1942 and 1944. Information obtained by study of the epidemiology of these diseases among naval installations convincingly demonstrates dissemination by men moving from boot camps to more advanced training centers.

Administrative problems may well make such changes in training programs difficult during periods of rapid mobilization. Other control measures will undoubtedly be necessary, and none of those suggested and investigated during the last war, with the possible exception of chemoprophylaxis, is likely to be of value.

Chemoprophylaxis with sulfonamides, although of no value in mass application, may be useful in the control of localized outbreaks caused by

sulfonamide-sensitive streptococci. Penicillin and the newer antibiotics, Aureomycin, Terramycin, and chloramphenicol, may also have a place in prophylaxis, particularly since resistance to these agents develops less readily than do sulfonamides.

Additional new techniques involve the termination of the carrier state by administration of penicillin and the prevention of rheumatic fever by treatment of established hemolytic streptococcal infection with this agent.

Evaluation of these methods and their proper application will require the accurate recognition of hemolytic streptococcal respiratory disease. It is believed that the developments in recent years have made physicians more aware of the importance of these infections and the clinical signs permitting their diagnosis.

Satisfactory bacteriologic examination of the respiratory flora is essential to the study of respiratory illness and was available in only a few Army hospitals during World War II. It is clear that this situation must be rectified. Every Army hospital should be prepared to carry out these exceedingly simple bacteriologic procedures using standardized methods and materials. Individual station hospitals need not maintain facilities for grouping and typing. Several laboratories throughout the country should be able to identify streptococci in this way and to test the sensitivity of the organisms to various antimicrobial agents. Sampling and study of strains isolated in the field should be a continuous operation.

Laboratory control of the character just described will permit the rational application of prophylactic and therapeutic regimes presently available and will permit the application of new ones. In addition, accurate information as to the incidence of hemolytic streptococcal disease would be constantly available to The Surgeon General, permitting vigorous action at the earliest sign of an epidemic. A few teams of qualified officers or civilian consultants should be available to go into the field and apply special methods when excessively large numbers of cases appear in any establishment.

It is not known whether these measures can be expected to diminish the frequency of streptococcal infection in the Army. The availability of new tools raises the hope that their rational and vigorous application may be at least partly successful in reducing the hazard of streptococcal disease and its important complications.

CHAPTER XIV

Tuberculosis

Esmond R. Long, M. D.

TUBERCULOSIS IN WORLD WAR I [1]

Tuberculosis was first recognized as a significant medicomilitary problem in World War I. Data are available in United States Army records and previous military histories on rates and disposition of cases in the Civil and Spanish-American Wars, but the known prevalence in the Army, in comparison with that of other diseases, particularly typhoid fever and malaria, was so small that no special attention was paid to tuberculosis and no analytical studies were made of its military importance.

In World War I, on the other hand, United States Army medical officers were aware of its significance at the outset, for the disease was recognized as a grave problem in the French Army before the United States was engaged in hostilities. In the light of the French experience, The Surgeon General of the United States Army determined to have rigid physical examinations for tuberculosis (roentgen examination being then in its infancy) made by experts at the time of induction to prevent acceptance of men with this disease. In order to carry out this program, the Army assembled large numbers of distinguished experts and conducted special courses of training in physical diagnosis. The experience and shortcomings in diagnosing and excluding tuberculosis were described at great length by Col. George E. Bushnell, MC, Chief Consultant in Tuberculosis, Office of the Surgeon General, in the official history of the Medical Department of the United States Army in World War I. Although some 50,000 men with diagnosed tuberculosis were excluded at induction, the imperfections of the procedure were such that many men with early tuberculosis were accepted, as was indicated by a steady discovery of cases in the rapidly mobilized Army.

Tuberculosis in the Army was exhaustively analyzed by Colonel Bushnell. He attributed it almost entirely to breakdown of cases that escaped detention at induction. It must be recalled that this was a period when virtually all adult pulmonary tuberculosis was considered endogenous, representing the flaring up and progression of latent lesions of childhood as a result of adult strains. In favor of this view was the fact that nationwide sampling by the tuberculin test and all necropsy experience indicated almost universal infection

[1] The Medical Department of the United States Army in the World War. Communicable and Other Diseases. Washington: U. S. Government Printing Office, 1928, vol. IX, pp. 171-202.

of the population by the age of 20 years. It was a decade or more before superinfection of adults was recognized as a frequent occurrence.

Before the war was over, more than 2,000 men had died of tuberculosis in the Army, and thousands more had been hospitalized. The admission rate in Army hospitals averaged 19 per 1,000 strength per year. Throughout World War I, tuberculosis was the leading cause of discharge for disability, accounting for 13.5 percent of all discharges. At the end of the war, a huge and costly problem was left for the newly organized Veterans' Administration.

TUBERCULOSIS BETWEEN THE TWO WORLD WARS

Between 1918 and 1941, great progress took place in the understanding of the pathogenesis of tuberculosis. Careful studies of its epidemiology brought out a clear relationship between exposure and subsequent development of manifest tuberculous lesions of the adult type, and in time the exogenous theory of adult tuberculosis assumed at least an equal standing with the endogenous. The disease was no longer considered invariably an exacerbation of an old childhood infection but rather, in many if not most cases, the result of an adult acquisition of new infection.

The new understanding was reflected in a radically changed approach to the public health attack on the disease. Two principal procedures became recognized as the nucleus for tuberculosis control: case finding and isolation of discovered cases. In the earlier years of the two decades between the wars, case finding was developed as a public health procedure on a contact basis. The family was recognized as the source of most exposure, and clinic and public health measures were taken accordingly. Later, commencing in the late 1930's, case finding by mass surveys was recognized as highly effective. A few surveys were made by affiliates of the National Tuberculosis Association and local health departments just before the United States entered World War II, which clearly demonstrated the possibilities of the method. These surveys, made by methods of cheap and rapid roentgenography, resulted in the discovery of cases at relatively low cost as compared with all previous efforts. Paper films were used at first; subsequently, methods of photofluorography were developed, which made possible a less expensive and even more rapid roentgen examination of large groups. The United States Public Health Service took great interest in the development of methods and cooperated closely with tuberculosis associations before developing an independent program of its own.

Coincidentally with this development, hospitalization for tuberculosis was increasing rapidly. Standards for the number of beds which communities should provide for proper care of the disease were developed and raised with the passage of time. Before the beginning of the war, some 75,000 beds were available, and it was universally accepted in the United States that at least two beds should be provided for every annual death in any community. This was a goal for which all communities with a sense of public health responsibility strove.

During these two decades, a notable decline in tuberculosis mortality occurred throughout the country. At the close of World War I, the tuberculosis mortality rate in the registration area was 150 per 100,000 population. At the opening of World War II, the figure was less than 50. There has been extensive discussion and analysis of the reasons for the striking decrease. Specific measures, including case finding and isolation of open cases, were certainly effective. Combined with this, however, was a general rise in the standard of living, which must have affected the mortality from tuberculosis indirectly in various ways.

In addition to the development of roentgenographic methods for the detection of the disease, improvements occurred in its laboratory diagnosis. The years between the two wars were fruitful in the development of new methods for examination of sputum and of other tests in the recognition of tuberculosis infection. At the same time, education of physicians with respect to the disease improved, so that the rank and file of medical officers at the opening of World War II were much better informed on the disease than were the corresponding physicians of World War I.

TUBERCULOSIS IN WORLD WAR II

Examinations for Detection of Tuberculosis at Induction

As mobilization for World War II became imminent, Army and civilian experts on tuberculosis, the National Tuberculosis Association, and other public health organizations repeatedly called attention to the high incidence of tuberculosis in troops in World War I and to the necessity of avoiding a repetition of that unfortunate experience. They pointed out that, since World War I, roentgenologic methods had been developed to the point that they could be effectively used in detecting tuberculosis and excluding it at induction.

The principle of the roentgen method of detection was accepted by The Surgeon General long before the outbreak of the war, and standards for the exclusion of men with tuberculosis were drawn in terms of roentgenography. However, when war actually began, few places in the country were in a position to furnish the required roentgen examination. In the summer of 1940, The Surgeon General of the Army had requested the aid of the Division of Medical Sciences of the National Research Council in the formulation of proper standards. A subcommittee of the division, consisting of Drs. J. Burns Amberson, Jr., Bruce H. Douglas, Herbert R. Edwards, Paul P. McCain, and James J. Waring, with the author as chairman, drew up a set of recommendations that formed the basis for the chest section of MR (Mobilization Regulations) No. 1–9 which was in effect when examination of selective service registrants began in August 1941. This committee formulated an improved and greatly extended section in MR No. 1–9 of March 1942.[2] Efforts to establish roentgeno-

[2] MR. No. 1–9, Standards of Physical Examination During Mobilization, 31 Aug. 1940 and 15 Mar. 1942.

graphy as a required routine procedure at induction were pushed vigorously by Col. (later Brig. Gen.) C. C. Hillman, MC, of the Office of the Surgeon General, and after many delays the procedure finally was universally employed.

One of the greatest difficulties in implementing the program lay in the multiplicity of stations for enlistment. Many of these were small and remote from medical centers. Equipment was not available nor were experts who could interpret roentgen films accurately. Induction stations for selective service registrants were better supplied, and the frequency of roentgen examination gradually increased throughout these stations, varying greatly, however, in speed of development in the nine corps areas or, as they were later designated, service commands. At first, chest roentgen examination was required on all registrants in whom pulmonary disease was suspected and was requested whenever local facilities made it possible.[3] At the beginning of the war, only the three corps areas on the North Atlantic Coast made roentgen examinations routinely, and at first some of the stations in these areas utilized the services and equipment of affiliates of the National Tuberculosis Association and local health departments to carry out the examinations. The volunteer service rendered by these organizations was of major value to the Army in bridging the gap until Army facilities were available and at the same time tied the Army program in closely with State public health activities, which were of immediate concern to the assisting agencies.

By 1 March 1941, it was estimated by Colonel Hillman that 51 percent of all men called to induction stations had had a chest roentgen examination. With further increase in facilities and equipment, the time finally arrived, about 1 April 1942, when all selectees were subjected to roentgen examination before acceptance. Roentgen examination for appointment of officers was already universal.[4] Nearly 100 induction stations were in operation, each of which had suitable equipment. Eight of the nine service commands used 4- by 5-inch stereoscopic photofluorograms, and one, the Fifth Service Command, used paper films. Ultimately, all stations in all service commands used 4- by 5-inch photofluorograms, supplemented as need dictated by full-size chest roentgenograms on celluloid film. A full review of the procedures for exclusio. of tuberculosis from the Army was made by the author following World War II.[5]

It is estimated that, before roentgen examination became mandatory (MR No. 1–9, 15 March 1942), one million men had been accepted without this form of examination. Where roentgen examination was practiced, it resulted in a rejection rate of about 1 percent for tuberculosis. Applying this figure, it can be estimated that some 10,000 men were accepted who would have been rejected if they had been subjected to chest roentgen-ray study. Various studies have shown that approximately one-half of these would have been cases of active

[3] Letter, The Adjutant General, to Commanding Generals of all Corps Areas and Departments, 25 Oct. 1940, subject: Chest X-rays on Induction Examinations.

[4] (1) Letter, The Adjutant General, to Each Corps Area and Department Commander; Each Chief of Arm or Service; and to the Chief, National Guard Bureau, 30 Jan. 1940, subject: Physical Standards and Physical Examinations, Par. 4b. (2) Letter, The Adjutant General, to Each Corps Area and Department Commander; Each Chief of Arm or Service; and to the Chief, National Guard Bureau, 28 Dec. 1940, subject: Physical Standards and Physical Examinations, Par. 4b.

[5] Long, E. R.: Exclusion of Tuberculosis. Physical Standards for Induction and Appointment. [Official record.]

disease. Thus, failure to employ roentgenographic methods probably resulted in the acceptance of 5,000 cases of clinically active tuberculosis. Among the large number of men accepted after roentgen examination, errors—either in interpretation or in administrative procedure—permitted the acceptance of about 1 man per 1,000, as shown by subsequent research, who should have been rejected for tuberculosis.[6] Among the 10 million men routinely examined by roentgenographic methods and accepted, it is believed that about 10,000 with active or potentially active lesions were inducted. Altogether, therefore, there is reason to believe that some 15,000 men were taken into the Army with tuberculous lesions that could have been detected on roentgen examination and recognized as a cause for exclusion.

It would be unwarranted, however, to assume that under existing circumstances X-ray diagnosis at induction stations could have been refined to a point where a greater degree of success could have been attained. Induction station roentgenologists were subjected to a variety of conditions tending to lower the level of their performance, chief of which were fatigue from long hours, required speed of operation, and repeated pressure from command sources to reduce the rate of rejection in the interest of manpower needs. In the effort to improve accuracy, the consultant in tuberculosis in the Office of the Surgeon General visited every induction station in the United States and read sample films with the station roentgenologists. Some improvement was probably brought about in this way.

It would be fully justified, on the other hand, to stress the great benefit to public health practice in the country effected through the report of cases which were properly identified. States varied in the energy with which advantage was taken of Army and Selective Service reports on rejection for tuberculosis, but it appears unquestionable that those with the more progressive health departments used these returns with great success in improving their case finding and followup programs.

Breakdown from Preexisting Tuberculosis

As indicated in the last section, it is estimated that some 15,000 men with active or potentially active tuberculosis were accepted in the Army as the result of failure to carry out roentgenologic examination or to recognize lesions in photofluorograms and roentgen films, when these were made. The majority of men so admitted had the disease in the minimal stage. In a substantial number, however, the disease was far advanced. It is believed that, in many of these cases, administrative errors in recording the lengthy selective service identification numbers were responsible for acceptance.

From the beginning of the war, all films of accepted men were filed with the Veterans' Administration. Reexamination of X-ray films at any time was thus

[6] Long, E. R., and Stearns, W. H.: Physical Examination at Induction; Standards With Respect to Tuberculosis Induction and Their Application as Illustrated by a Review of 53,400 X-ray Films of Men in the Army of the United States. Radiology 41: 144–150, August 1943.

possible, provided film filing was up to date, which was by no means invariably the case. Reexamination of the films of accepted and discharged men has shown that, in the majority of instances, men who entered the Army with advanced tuberculosis were detected within the first 6 months of acceptance, were hospitalized, and were discharged. In a few cases, remarkably, men were able to carry advanced lesions over a period of many months or even years before the disease finally was detected.

On the other hand, thousands of men with minimal lesions served months or years before breakdown occurred. In some instances, no deterioration in health or progression of the disease ever occurred in men whose initial films, on review, showed lesions that had the roentgenologic appearance of clinical activity. Also, large numbers of men with minute lesions which were considered well healed carried these lesions without harm throughout the period of their service.

Careful studies have been made of circumstances leading to the breakdown of tuberculous lesions of various types during the course of service. One study, carried out by Dr. Waring and Capt. (later Maj.) William H. Roper,[7] was organized by the Subcommittee on Tuberculosis of the Division of Medical Sciences of the National Research Council and implemented by the Office of Scientific Research and Development. This study showed that heavy physical labor and the strain of combat were particularly important circumstances favoring breakdown. Other factors, such as malnutrition and psychologic strains, played a less definite role. Waring and Roper also found that a considerable proportion of breakdowns following acceptance for military duty occurred within the first year of service. In many, the onset occurred with pleurisy with effusion. An additional observation was that assignment of "poor risks" to limited duty afforded an appreciable measure of protection against breakdown, although frankly active cases mistakenly taken into service did equally badly on limited and general duty.

During the first 2 years of the war in the Army's one special tuberculosis hospital, Fitzsimons General Hospital, Denver, Colo., the majority of admitted cases appeared to represent a breakdown of lesions present at the time of induction. Medical officers on duty in the hospital stated that during the last 2 years of the war the majority of cases occurred in men whose chests were negative by roentgen examination at induction.

Contraction of Tuberculosis

It has been pointed out that a substantial number of men with tuberculosis were admitted to the Army through failure to exclude recruits with recognizable disease at induction. Long and Jablon[8] made an extended postwar study of the induction and separation X-ray films of 3,099 men discharged from the Army with a diagnosis of tuberculosis and 3,000 discharged for other reasons

[7] Roper, W. H., and Waring, J. J.: Primary Serofibrinous Pleural Effusion in Military Personnel. Am. Rev. Tuberc. 71: 616-634, May 1955.

[8] Long, Esmond R., and Jablon, Seymour: Tuberculosis in the Army of the United States in World War II. An Epidemiological Study with an Evaluation of X-ray Screening. Washington: U. S. Government Printing Office, 1955.

during the years 1942 to 1945, inclusive. This study disclosed the fact that approximately one-half of the tuberculosis that led to discharge from the Army was already present in diagnosable form at the time of acceptance for military duty.

In addition to these men, presumably other men were inducted with latent infections undetectable by the X-ray screen. Men from each of these groups may have broken down as a result of progression of their preexisting disease. Over and above these, however, there is reason to believe that a considerable amount of tuberculosis resulted from new infections acquired during service, through contacts in the Army itself or through associations outside of military duty. Opportunity for contracting tuberculosis through civilian contact in the United States existed for soldiers just as for other citizens. They visited their families while on leave, and presumably in some of these families there were members with tuberculosis who were likely to spread their infection. Thus, soldiers may have carried back tuberculous infections when they returned to their stations. Also, as in civilian life, soldiers visit friends in the general population, some of whom may have tuberculosis. Therefore, troops presumably experienced a certain amount of exposure to tuberculosis from this source in the United States. The average intensity of exposure would depend upon the prevalence of tuberculosis in the sections of the population visited.

Overseas, the opportunity for contraction of tuberculosis through exposure to civilians was still greater. In North Africa, Italy, France, the islands of the Pacific, the Philippine Islands, and Japan, the prevalence of tuberculosis was far higher than in the United States. Rates in the British Isles and in Germany also exceeded those in the United States but not by a great margin.

Because of the multiplicity of contacts that might be significant, it is difficult to trace new cases of tuberculosis to their source under Army conditions. However, the acceptance of some 15,000 men with active or potentially active tuberculosis as a result of defects in the induction screen was apparently a factor not to be overlooked in the total development of tuberculosis in military personnel. The study by Long and Jablon indicated that, except in the case of men who had served periods as prisoners of war and men who apparently had been exposed excessively to disease through other special circumstances, a relatively even uniformity of risk of acquiring tuberculosis occurred, regardless of arm, military occupation, overseas service, theater of service, or civilian contact. In that study, differences observed in risk seemed to be no greater than those to be expected through chance sampling. It seemed reasonable, therefore, to infer that a substantial percentage, perhaps a majority, of cases of newly contracted tuberculosis in servicemen resulted from infection from fellow soldiers.

Admission Rates

The best measure of the prevalence of tuberculosis in the Army was the admission rate for this disease, although the figure may give an exaggerated picture of the actual prevalence. Cases admitted for tuberculosis included all

those so diagnosed after complete examination and, in addition, cases originally judged as probably tuberculous in nature but requiring further study to establish or disprove the diagnosis. On the other hand, incidence rates failed to take into account asymptomatic cases not brought to medical attention. Possibly the error in one direction was as great as that in the other, so that the recorded prevalence may have been fairly close to the actual.

It is useful to compare the incidence rates in the Zone of Interior with those in foreign areas, in an effort to determine where cases of tuberculosis originated. Needless to say, admission in the Zone of Interior or admission in overseas areas would not necessarily mean that infection ultimately diagnosed was acquired in the same region. A soldier might be infected in the Zone of Interior and first show evidence of tuberculosis after going overseas, and, vice versa, a soldier infected overseas might return to the Zone of Interior and pass several months in service before his tuberculosis became manifest.

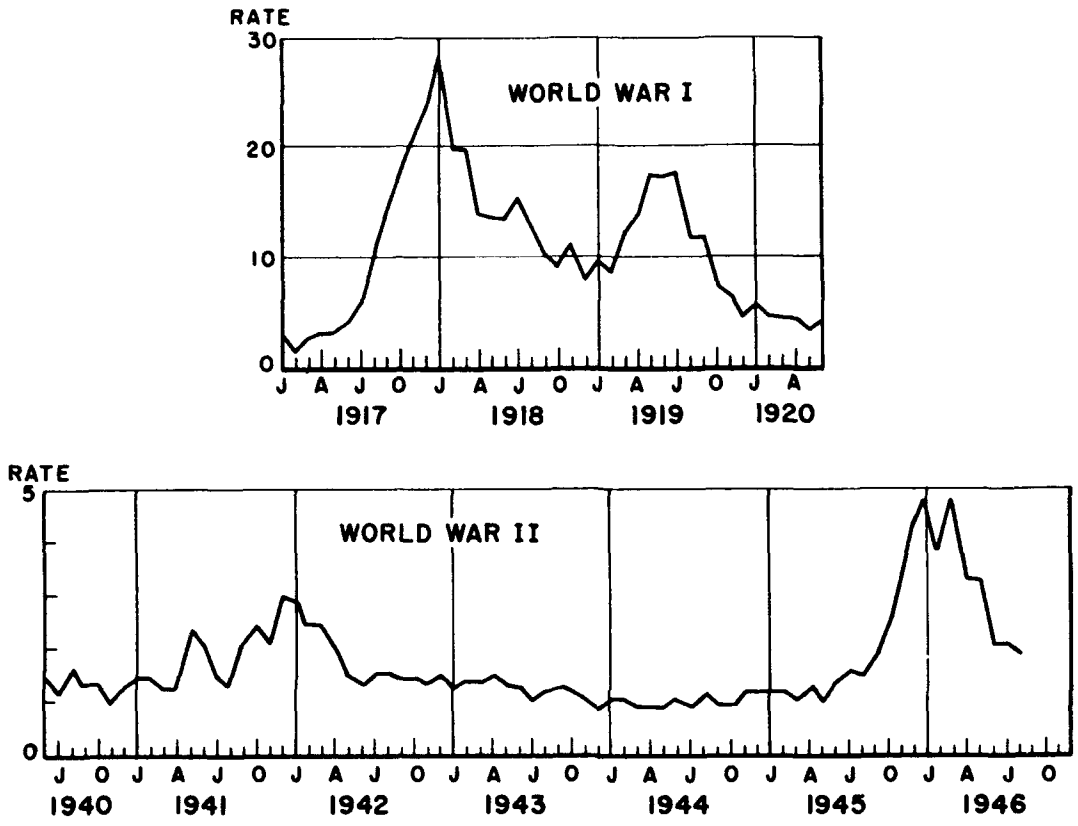
It is interesting to note that throughout the war incidence rates for the Zone of Interior as recorded in periodic statistical health reports were consistently higher than those reported in the overseas theaters. Several reasons may be given in explanation. In the first place, cases of obvious tuberculosis overlooked at induction usually came to light before transfer overseas. Training in the United States prior to overseas duty was rigorous, and cases likely to break down ultimately in normal life were eliminated by what was essentially a process of natural selection before overseas assignment. Also, it is possible that there was more leisure for examination in the Zone of Interior, and therefore more cases were detected. Whether this is a fact or not is debatable, for the examination overseas of service and combat troops was probably equal most of the time to that carried out in the Zone of Interior.

Chart 23 illustrates the admission rates for the total Army in the Zone of Interior from July 1940 to October 1946, inclusive. The admission rates among white enlisted men in the continental United States in World War I for the years 1917–20 are presented for comparison. It will be noted that, in each war, a high rate prevailed shortly after the beginning of the war. This can be explained as the result of imperfection in examination for service during the early months in each war. In World War II, there was a long period from April 1942 to the middle of 1945 when examinations were excellent and the rate of recognition of tuberculosis in the Army was relatively low and constant, representing the sum of cases that escaped recognition on induction and those that actually developed within the Army. It will be noted that a high rate occurred toward the end of 1945. This rate is, of course, artificial and represents the tuberculosis rapidly discovered at the separation centers on discharge from the Army.

A fact of interest brought out in the figures is that, at all times during World War I, the admission rate for tuberculosis was approximately 10 times that in World War II. The explanation can be found, in part, in inferior screening at induction during World War I and also in the greater prevalence of tuberculosis in the civilian population during the earlier period, when the

CHART 23.—*Incidence of tuberculosis in the U. S. Army in the continental United States, World War I and World War II*

[Rate expressed as number of cases per annum per 1,000 average strength]



mortality rate was three times as high as during World War II. Hence opportunity for chance failure of recognition at induction was much smaller during World War II, as was also the opportunity for exposure to the disease after induction. Actually, the difference in prevalence in the Army in the two wars was probably considerably greater than that recorded for admission rates, since diagnostic accuracy had improved markedly in the interim and the admission rate in World War II, presumably, was therefore closer to the true prevalence rate than it had been in World War I.

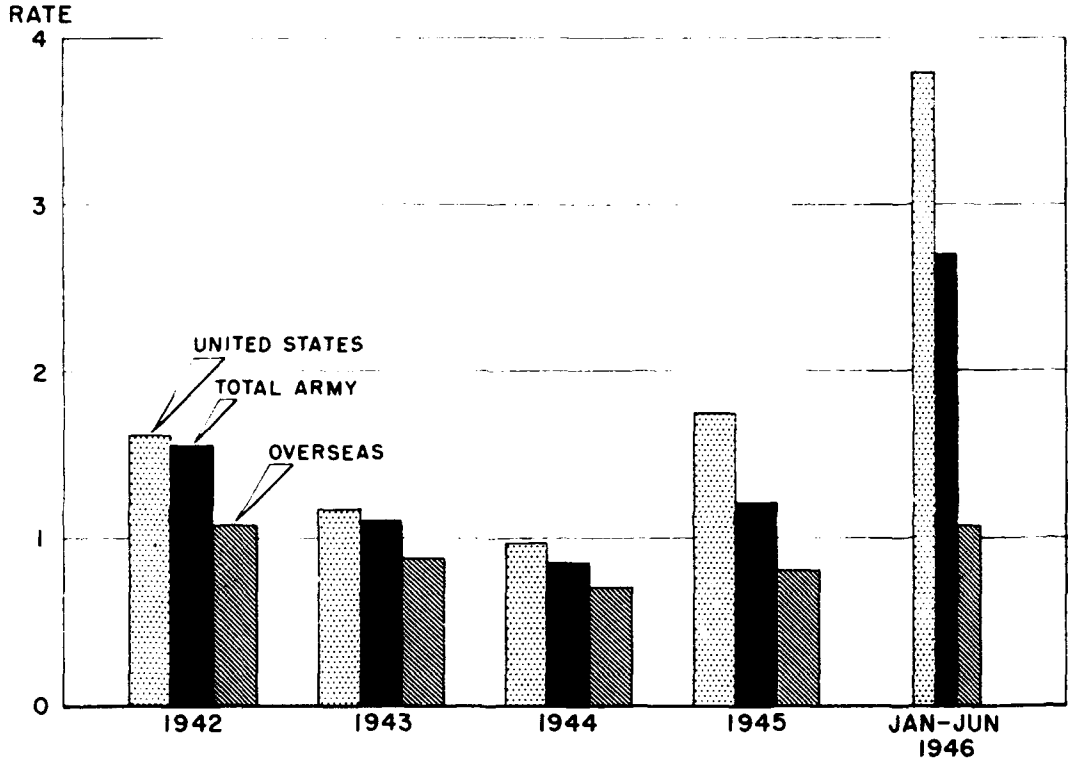
Chart 24 compares incidence rates for tuberculosis for the United States and overseas theaters for the years 1942 to June 1946, inclusive. It will be seen that the rate in the United States was at all times greater than that overseas. All overseas theaters reported relatively low rates. Annual rates per 1,000 strength recorded for the European Theater of Operations by Lt. Col. Theodore L. Badger, MC, senior consultant in tuberculosis in the theater for 3½ years of war, were as follows: 1942, 0.89; 1943, 1.27; 1944, 0.76; 1945 (1 January to 31 May), 0.69.[9] The average admission rate for the Mediter-

[9] Semiannual Report of the Senior Consultant on Tuberculosis, Office of the Chief Surgeon, European Theater, 1 Jan. 1945 to 30 June 1945.

ranean (formerly North African) Theater of Operations from November 1942 to May 1945, as recorded in the theater, was 0.58. Chart 25 shows the rates for all theaters for the years 1942–45 on the basis of returns in the statistical health reports.

CHART 24.—*Incidence of tuberculosis among U. S. Army troops in the United States and overseas, January 1942 to June 1946, inclusive*

[Rate expressed as number of cases per annum per 1,000 average strength]



As has been pointed out (chart 24), during World War II tuberculosis was more prevalent in troops in the United States than in troops in overseas theaters. The opposite was evident, however, at the time of discharge. Figures from separation examinations in the Zone of Interior, assembled separately for those who had service in the United States only and for those who had service overseas, showed a striking difference.[10] Chart 26 illustrates the difference in withdrawals for tuberculosis in the two groups. It is evident at once that diagnoses of probable tuberculosis in soldiers with overseas experience were considerably more frequent than in troops with Zone of Interior service only.

The explanation would not seem difficult. Troops overseas presumably actually did acquire new infections to a somewhat greater extent than troops in this country. Most of these infections were minimal and therefore were

[10] Long, E. R., and Hamilton, E. L.: A Review of Induction and Discharge Examinations for Tuberculosis in the Army. Am. J. Pub. Health 37: 412-420, April 1947.

not discovered on a symptomatic basis nor recorded in the normal admission rates in the theaters. When the millions of troops with and without foreign service were discharged at separation centers, roentgen examination was

CHART 25.—*Incidence of tuberculosis in the U. S. Army, by theater and year, 1942–45*

[Rate expressed as number of cases per annum per 1,000 average strength]

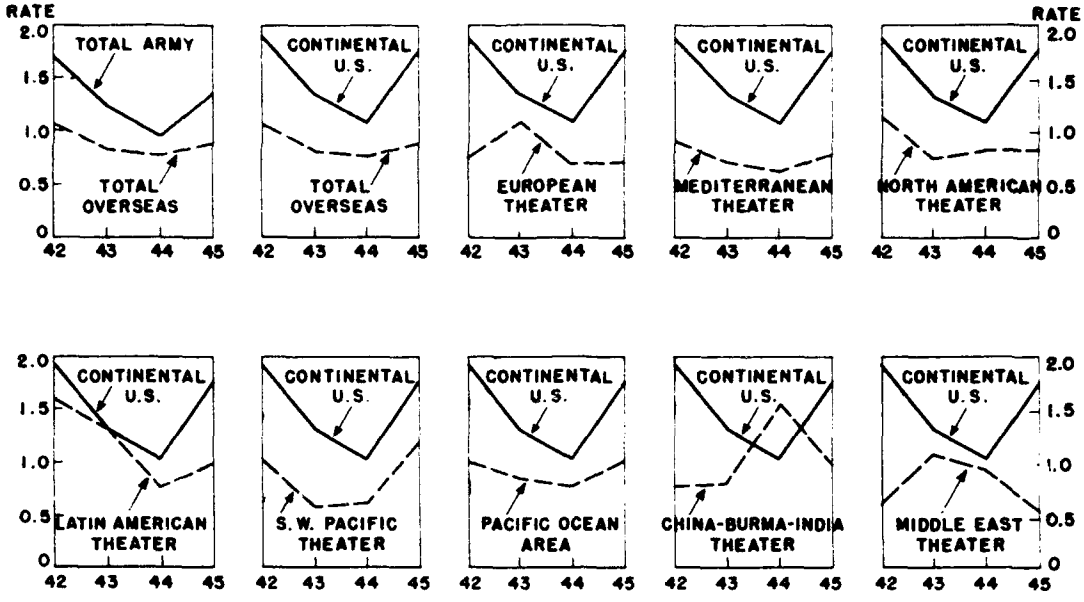
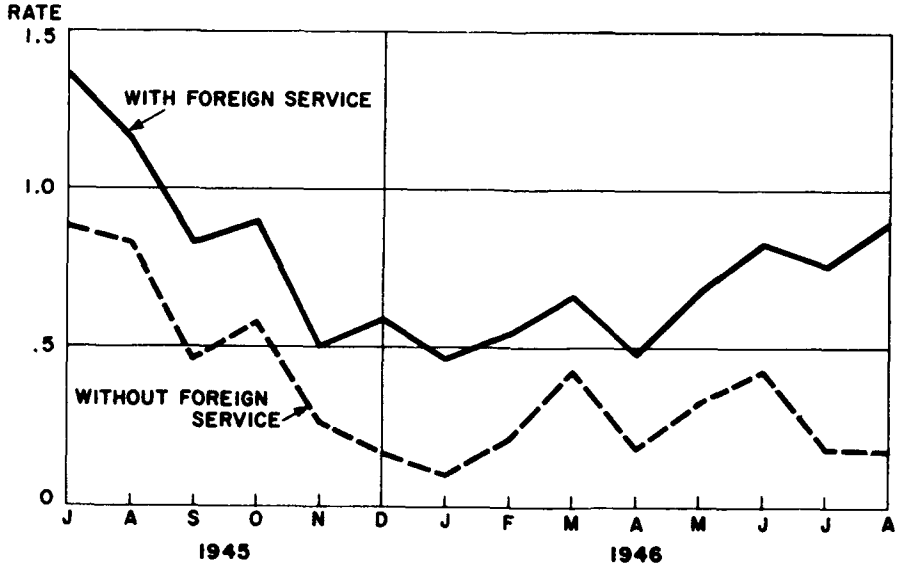


CHART 26.—*Withdrawals from separation processing for pulmonary tuberculosis*[1] *in the U. S. Army separation centers, July 1945 to August 1946*

[Rate expressed as number of withdrawals per 1,000 processed]



[1] Some cases withdrawn for suspected pulmonary tuberculosis, not later confirmed by further examination, are included. Not included are cases withdrawn as "other defects of lungs and pleura" and later found to be pulmonary tuberculosis.

universal.[11] Hundreds of small lesions, undetectable by any other means, were found. The examinations were of the same character for both groups of troops. The conclusion would seem inescapable that some significant factor operated overseas to make the rate in troops with overseas service greater. The excessive rate could not be attributed entirely to the strenuous nature of overseas service, for many overseas troops had approximately the same type of service as troops in the Zone of Interior. The simplest explanation, and the most logical, would seem to be that the excess of lesions found in troops with foreign service represented overseas infection in the numerous regions where United States troops were in close contact with populations with far higher tuberculosis rates than the rate in the United States. For such an explanation to be valid, however, it would be necessary to exclude from consideration lesions which were present at induction but which, because of their small size and apparent innocuous character, were not judged to be a cause for rejection.

Certain pitfalls in such a deduction, however, have been pointed out by Long and Jablon in the study previously cited. This study showed for white soldiers some increase in risk in service in Europe and Asia, presumably related to civilian contact. Among nonwhite troops in the Mediterranean theater, in which civilian association was known to be unusually great, the figures demonstrated an excessive rate of tuberculosis at discharge. It was impossible to establish a positive correlation between the development of tuberculosis and illness known to result from civilian contact, such as venereal disease. On the whole, the evidence did not indicate a strikingly greater risk of acquisition of tuberculosis in the Army overseas than in the Army in the United States. Certain exceptions to this conclusion were clear, however. Rates among Medical Department personnel with foreign service, among nonwhite troops stationed for long periods in port areas, and in military personnel who had been prisoners of war were found to be excessive.

The study by Long and Jablon should be consulted for detailed information concerning the development of tuberculosis with respect to age, race, home environment, and length of Army service. Space does not permit extensive review here. A definite correlation with youth was evident, and rates in general were higher for nonwhite than for white troops. The most significant factors, however, were those listed above.

Before the subject of prevalence of tuberculosis in the Army is concluded, some reference should be made to tuberculosis mortality in the Army. A proper analysis of Army mortality should take into account mortality after discharge as well as deaths in the Army, as the majority of persons destined to die of the disease were transferred from the Army to the Veterans' Administration. In an Army of approximately 4 million men in 1942, the tuberculosis mortality was less than 4 per 100,000 persons. The combined figure for personnel on duty and discharged personnel in later years, when the Army was much larger, increased to about 6 per 100,000 in 1943, 10 in 1944, and 12 in 1945. The

[11] War Department Technical Manual 8-255, Terminal Physical Examination on Separation From Military Service, 10 Sept. 1945.

higher rates of the later years reflected the time required for mortality to succeed onset of disease, as well as the cumulative increase of cases in a population originally almost free from tuberculosis. Army mortality figures have been analyzed in some detail by Aronson,[12] who called attention to a rising mortality in Army personnel with rise in age and to the fact that, despite similarity of living conditions, income, and military duties, the Negro race, representing about 10 percent of the Army population, contributed 43.4 percent of the deaths from tuberculosis.

Preventive Measures

Theater measures.—No specific screening to exclude tuberculosis was practiced immediately prior to departure of troops for overseas. The magnitude of the task of making new roentgen examinations on such a large body of troops prohibited the procedure. On the other hand, there is every reason to believe that many soldiers with tuberculosis were automatically eliminated from troops going overseas. The rigors of basic training probably brought to light many of the cases which, in spite of the presence of small active lesions, were overlooked at induction. A few, but not many, cases were detected by the physical examination made routinely just before departure for overseas service.

Cases that escaped discovery prior to departure from the United States were detected from time to time in overseas areas in the course of numerous roentgen examinations for respiratory disease or for special survey purposes. Surveys were made from time to time in Army Air Force personnel, and roentgen examination was required before assignment to officer candidate schools.

On the whole, roentgen examination of troops was a frequent procedure overseas and not a few asymptomatic cases was found. Such widespread roentgen examination resulted in the discovery of many inactive cases that had been knowingly inducted because the lesions seen were believed healed and within acceptable limits in size. These cases constituted a problem of disposition. Medical officers overseas, in ignorance of the fact that the small lesions were previously studied and considered acceptable, returned many such cases to the Zone of Interior rather than continue them as medical risks where responsibilities were so great.

Facilities for the detection of tuberculosis were first class, even up to the frontline. Not a few cases were discovered and worked up thoroughly by roentgenographic examination and laboratory tests for tubercle bacilli in evacuation hospitals within a few miles of the front.

Altogether, such measures probably detected most cases of open tuberculosis within a relatively short period of their development as open, contagious cases and prevented much dissemination within the Army.

Beyond this, relatively little precaution was taken to prevent tubercu-

[12] Aronson, J. D.: The Occurrence and Anatomic Characteristics of Fatal Tuberculosis in the U. S. Army During World War II. Mil. Surgeon 99: 491-503, November 1946.

losis. Specific measures that might have been used are discussed in following paragraphs. Official directives were considered necessary only in the case of one type of tuberculous infection in which the hazard was recognized and the method of prevention was clear. This infection was milkborne tuberculosis. In most countries of the world, bovine tuberculosis is far more prevalent than in the United States. The hazard from bovine infection in this country is virtually negligible. It is well known that much of the milk for general consumption in the British Isles during the war contained tubercle bacilli. The Army Veterinary Service was well aware of this situation and issued several directives requiring discontinuance of purchase of milk unless the source could be approved.[13] These regulations sharply restricted the purchase of milk and imposed rigid standards with respect to its source. After the early months, very little British milk was consumed in the official ration. As a matter of fact, most of the milk consumed everywhere in the Army overseas was dried milk from the United States, in which full precautions had been taken to prevent contamination. There is little reason to believe that any significant amount of bovine tuberculosis was acquired either overseas or in the United States.

The evacuation of tuberculous soldiers presented a special problem in preventive medicine. Tuberculous patients were congregated in relatively small space for transfer back to the Zone of Interior. Theater surgeons recognized the danger of contagion, and efforts were made on hospital ships and airplanes to provide facilities minimizing transmission of infection. Unfortunately, a considerable number of tuberculous patients had to come back in troop-class quarters, and in these cases, proper precautions could not be taken. It is impossible to estimate how much transmission of tuberculosis took place in this way.

Quarters on hospital ships were usually fully adequate for tuberculous patients. Fortunately, relatively few cases of acute contagious diseases were evacuated from overseas, and, accordingly, the space originally reserved for such patients was turned over to tuberculous patients.

Insofar as possible, when troops were returned by airplane, tuberculous patients were held until a planeload accumulated. They were then transferred to the Zone of Interior in a single group, with a noncommissioned officer of the Medical Department and a nurse in charge.

Specific measures.— Measures employed for the prevention of tuberculosis in theaters of operations were general rather than specific. All medical officers were aware of the possibility of development of tuberculosis, and frequently, although by no means invariably, when cases were discovered in quarters, soldiers in the same barracks or billet were subjected to roentgen examination to determine if infection had been transmitted. In all probability, in the majority of cases, this was a gesture rather than an effective procedure because the examination so ordered was usually made too soon to discover

[13] (1) Circular No. 40, Headquarters, European Theater of Operations, U. S. Army, 5 Sept. 1942. (2) Circular No. 72, Headquarters, European Theater of Operations, U. S. Army, 10 Nov. 1942.

active cases and because adequate followup under the conditions of overseas service was impossible. Actually, such surveys not infrequently disclosed cases of inactive tuberculosis which had been admitted at induction stations knowingly or unknowingly; these, as pointed out above, at once constituted a problem of disposition.

Overseas medical officers had inadequate nutrition in mind as a possible factor predisposing to tuberculosis. Each of the several histories prepared by officers assigned the task of analyzing records with respect to tuberculosis in the specific theaters mentioned malnutrition as a possible predisposing influence. Actually, no correlation was discovered between the occasional forced periods of impaired nutrition in frontline positions and subsequent development of tuberculosis. As already pointed out, a study by Waring and Roper showed that other factors, particularly physical strain, predisposed more than any other factor, including nutrition, to breakdown from the disease.

Unfortunately, no sustained educational program with respect to tuberculosis was possible. The circumstances of military service were not conducive to such education. Occasionally, under unusual circumstances, an educational campaign was carried out. Troops in Manila, Philippine Islands, after the city was recovered, were warned against the danger of tuberculosis. The extremely high rate in the population of the Islands impressed medical officers at the time, and the Surgeon General's Office was asked to provide educational material to indicate to the troops the danger and the way to avoid it.

Early in the war, the advisability of BCG (bacille Calmette Guérin) vaccination was discussed. Considerable pressure was brought upon the Office of the Surgeon General to make it routine in the Army. The Surgeon General decided that its value was not fully established and that, with so many other inoculations of fully demonstrated efficacy, it was impractical to add one the value of which was uncertain.

Thus, in the last analysis, the program for prevention of tuberculosis rested on the two procedures of most value in the country as a whole: early diagnosis through case finding and hospitalization for isolation and treatment.

Special Problems

Prisoners of war and displaced persons.—The recovery of American prisoners of war, captured by the Germans and Japanese in various phases of the conflict and liberated in the late months of the war, created a new problem in preventive medicine for the Medical Department of the United States Army. A still greater problem resulted from the assumption by the Medical Department of responsibility for the medical care of thousands of aliens who had been held prisoners by the Germans and Japanese and who were liberated by the advancing armies of the United States in the spring of 1945.

Admission rates for tuberculosis in recovered United States military personnel were higher than in the Army as a whole. The increase was not

alarming nor such as to suggest that excessive exposure had taken place during the period of confinement. However, the conditions of confinement were rigorous, and malnutrition was particularly severe. There is reason to believe that these conditions caused breakdown in many men who were held prisoners over a period of several months.

In their study of the medical records and induction and separation X-ray films of 6,099 men, Long and Jablon found that among white men with overseas experience, for whom their figures were statistically significant, the prevalence of tuberculosis at the time of discharge from the Army was approximately three and one-half times as high among men who had been prisoners as among those who had not (figures chiefly for men captured by the Germans). In another investigation, Cohen and Cooper [14] found that the prevelance of active tuberculosis among United States prisoners of war at the time of their liberation was 6 per 1,000 for those captured by the Germans and 37 per 1,000 for those captured by the Japanese. The latter had experienced more rigorous prison conditions. These figures, of course, do not take into account the mortality from tuberculosis in prison camps, which is believed to have been high in Japan.

Most of the recovered prisoners were routed back to the United States through an established chain of hospitals and ports of embarkation. The problem was the same as that of ordinary personnel, except in degree. Recovered prisoners with tuberculosis returned to the United States in the normal manner, chiefly in hospital ships, and on arrival were sent to one of the special tuberculosis hospitals. The largest number of enlisted men with tuberculosis went to Bruns General Hospital, Santa Fe, N. Mex. The load was at no time severe enough to cause an undue strain on Zone of Interior hospital facilities or to constitute an excessive danger through contagion for nurses and other military personnel.

In marked contrast, the problem of care for liberated displaced personnel held by the Germans and Japanese was a severe one. At times, in eastern France, it was critical. Thousands of Russians, Poles, Hungarians, Yugoslavs, Italians, and persons of other nationality were found in hospitals or workcamps following the German retreat in eastern France. A high percentage of these inmates had tuberculosis, commonly in a far-advanced stage. It was necessary for the Army to establish several hospitals for the care of these patients. The 46th General Hospital at Besançon, with more than 1,000 tuberculosis patients of foreign nationality, was the largest tuberculosis center of the United States Army, exceeding Fitzsimons General Hospital in its census of tuberculous patients. The 50th General Hospital in Commercy also had a large number of such patients, and smaller numbers were scattered through various general hospitals throughout eastern France. The condition in which these patients were received was deplorable. Many were moribund on admission. Language difficulties and long years of abuse had destroyed all sense of discipline,

[14] Cohen, Bernard M., and Cooper, Maurice Z.: A Follow-up Study of World War II Prisoners of War. Washington: U. S. Government Printing Office, 1954.

so that the problem of medical care was exceedingly difficult. The exposure of medical personnel in these hospitals was high.

Fortunately, the senior consultant in tuberculosis in the area, Colonel Badger, was aware of the possibility of contagion. At his instigation, a series of important circulars with reference to the gravity of the problem were promulgated from the Office of the Theater Chief Surgeon, and directives were issued establishing a sanitary code in these hospitals that reduced contagion to the minimum possible.[15] Ultimately, tuberculosis aliens of this type were concentrated in a small number of hospitals, particularly the 46th General, and those surviving were repatriated through displaced-personnel channels.

The problem of care of enemy prisoners of war was much less complicated, as the primary burden was thrown upon captured medical officers of the enemy nations. Almost no exposure of United States personnel was involved, and the preventive medicine problem was minor.

Military Government.—After V–E Day and V–J Day, the medical departments of the occupying forces were concerned with the public health problems of the occupied countries. The postwar tuberculosis rate was high in Italy, Germany, and Japan. Prior to the war, the tuberculosis mortality rate in Germany was little more than that in the United States. The vicissitudes of war, the hardships of the laboring population, the breakdown of tuberculosis control measures, the importation of foreign labor unscreened for tuberculosis and other factors combined to double the mortality rate from tuberculosis in Germany. Most of the tuberculosis sanatoriums were in use for other purposes, particularly for the care of wounded and sick prisoners of war. Hence, tuberculosis patients who normally would have been in tuberculosis hospitals and sanatoriums remained at home, where they served as foci for infection of others. Conditions were similar in Italy and Japan.

Immediately after the war in Germany, a tuberculosis section was established in the Public Health Branch of Military Government, and the chief consultant in tuberculosis in the Office of the Surgeon General was asked to serve in this office during the early postwar months. The first objective was to restore reporting to a normal state, so that the magnitude of the problem could be learned. Fortunately, basic laws were in effect that made this relatively easy, and the primary public health organization at the Land level had been preserved. Trustworthy figures were soon obtained, and with the passage of time both case finding and hospitalization of discovered cases improved, so that less exposure of the population took place.

In each Land in Germany, the peak of mortality occurred in 1945 or 1946, after which a steady decline in tuberculosis mortality occurred.[16] The role of the medical departments of the occupying forces included such aid as could

[15] (1) Circular Letter No. 41, Office of the Chief Surgeon, Headquarters, European Theater of Operations, U. S. Army, sec. II, 11 May 1945. (2) Letter, Col. E. R. Long, MC, Consultant in Tuberculosis, Office of the Surgeon General, to The Surgeon General, 28 May 1945, subject: Visit of Tuberculosis Consultant in European Theater of Operations, United States Army.

[16] Sartwell, P. E., Moseley, C. H., and Long, E. R.: Tuberculosis in the German Population, United States Zone of Germany. Am. Rev. Tubere. 59: 481 493, May 1949.

be given the population through surplus Army supplies and food and steady insistence on accurate reporting by the German public health authorities to the statistical service of military government.

In Japan, extraordinarily high tuberculosis rates prevailed before the war, and these became exaggerated in the late years of the conflict. Progress in control by military government was similar to that in Germany, and ultimately remarkable success in lowering the death rate occurred. In this achievement, the Japanese themselves and medical officers from the occupying forces gave great credit to BCG and related vaccines used by the Japanese. It appears doubtful, however, that vaccination could have had such a striking rate so early. Presumably, various forces, as in Germany and other countries, were concerned.

Progress in Prevention During the War

The Army's experience in the control of tuberculosis during World War II, combined with that gained in World War I, resulted in a good understanding of organization in screening for tuberculosis. This experience found application on a large scale in later case-finding programs throughout the Nation. It should be equally valuable in military orientation whenever the need for similar operations arises again. The vast program of roentgenologic examination for tuberculosis carried out in induction stations was repeated on a mammoth scale in much more rapid fashion at discharge. Not long after the separation procedure was concluded, however, induction and separation centers were closed, and within a few months after the war roentgenologic examination at stations of enlistment was no longer an established procedure. The methods in operation before the war prevailed, and roentgen examination was not made at ordinary recruiting stations. Also, unfortunately, the requirement for roentgen examination at the first station following enlistment often was not fulfilled.

The experience of the induction stations showed the need for greater efficiency in roentgenologic screening to prevent acceptance of men with active or potentially active lesions. The cost to the United States Government of long-term care of patients with tuberculosis in the Army or in the Veterans' Administration is so great that the relatively minor expense of extreme care in acceptance of men at induction should not be opposed.

The hospitalization system carried out by the Army for tuberculosis was excellent. Army hospitals improved coincidentally with civilian hospitals in the medical and surgical treatment of tuberculosis during the years of the war. The isolation of tuberculous soldiers in Army hospitals, supplementing that of civilian hospitals and governmental hospitals of municipal, county, State, and Federal operation, was an important factor in removal of sources of contagion in the country and corresponding reduction in the spread and prevalence of tuberculosis.

Careful analysis of cases of active tuberculosis discovered during the war showed conclusively that certain types of strain paved the way for breakdown

of latent lesions. Heavy physical labor and combat conditions were notable in this respect.[17]

The Army, which had had little experience, except for World War I, with the problem of tuberculosis in foreign countries, learned much about opportunity for contact and sources of contagion. If the United States continues to maintain occupation forces in foreign countries, the problem will remain. The experience gained through overwhelming exposure in the care of destitute displaced personnel was excellent, though severe, training for adverse conditions that might be encountered again.

Army medical officers took advantage of current advances in the diagnosis and therapy of tuberculosis, and improvement in technique in the care of patients took place steadily. Progress in bronchoscopy and surgery was notable. As the war closed, new antibiotic methods of therapy were coming into practice, but antibiotic therapy in tuberculosis in general was a postwar rather than a war development.

Army medical officers shared with civilian tuberculosis specialists in developing new understanding of the recognition of small lesions, of the relation of pleurisy with effusion to tuberculous infection, and of the need for prompt care and isolation of cases if the progress of the disease was to be prevented and spread of infection checked. The remarkably good clinical progress made by men whose disease was detected in a minimal stage, a frequent finding on discharge, was of high value in bringing home the excellent prognosis of small tuberculous lesions when discovered and treated in time.

SUMMARY

1. Tuberculosis was first recognized clearly as a military problem in World War I. In spite of specific efforts to prevent the acceptance of recruits with tuberculosis, a considerable number of men entered the Army with this disease, and a large and costly burden in medical care and compensation was left as a Federal responsibility.

2. Between World War I and World War II, great progress was made in the diagnosis of tuberculosis. Rapid methods of examination, including the use of paper films and photofluorography, led to significant advance in case finding. In addition, the number of beds available for tuberculosis was greatly increased, and opportunities for spread of the disease correspondingly decreased. During this period, a continuing decline in tuberculosis mortality occurred in the United States, as well as in the rest of the Western World.

3. During World War II, in the light of the experience of World War I, an intensive effort was made to exclude tuberculosis at induction. An X-ray program was instituted at the outset. This program was imperfect at the start, but after March 1942 all men entering the Army had a chest roentgen examination before acceptance for service. The examination itself had its

[17] See footnotes 7 and 8, p. 264.

imperfections, however, and it is estimated that approximately 15,000 men with active tuberculosis were admitted to the Army either as a result of omission of roentgen examination in the early months or failure to detect tuberculosis in films made after their use became routine.

4. Subsequent studies showed that a considerable number of these men broke down with clinical disease during service in the Army. The experience of Army tuberculosis hospitals indicated that, during the first 2 years of the war, a majority of the men developing tuberculosis in service were men who entered the Army with the disease already present. Postwar investigation of induction and separation X-ray films on file in the Veterans' Administration demonstrated that approximately one-half of the men ultimately discharged by reason of tuberculosis entered the Army with the disease.

5. In addition to those who developed tuberculosis as a result of the flaring up of lesions undetected on entrance into the Army, a significant number acquired tuberculosis during Army service, presumably as a result of exposure to the disease. There is reason to believe that this resulted from contact with tuberculosis both in the United States and abroad and from military as well as civilian contacts.

6. A comparison of admission rates for tuberculosis in the Army in the Zone of Interior and in overseas theaters shows that throughout the war the rate was higher for Zone of Interior than for overseas troops. However, there is reason to believe that a significant amount of early tuberculosis was overlooked overseas, because roentgen examination at time of separation disclosed a somewhat higher rate of tuberculosis in men with overseas service than in those with Zone of Interior service only.

7. The principal measures for control of tuberculosis in overseas theaters were early diagnosis and evacuation to the United States. Many examinations were made in the course of routine studies, as for promotion to officer status, and in the diagnosis of acute chest disease. Men with active tuberculosis and men in whom activity was suspected were evacuated to the Zone of Interior for further observation and care. Thus, the theaters depended upon diagnosis, isolation, and care for the control of tuberculosis.

8. Tuberculosis in recovered United States prisoners of war was a problem of some concern to the Army. The tuberculosis rate in recovered prisoners was considerably higher than in the Army as a whole, although far short of that prevailing in recovered prisoners in other armies.

9. The greatest amount of tuberculosis encountered by the Medical Department of the United States Army during the war was in displaced personnel of other nationalities recovered by the Army during the occupation of areas previously in the hands of the German Army. Huge numbers of patients with tuberculosis were found in displaced personnel camps abandoned by the Germans, and their care became a difficult problem for the hospitals of the Army.

10. Military government assumed the control of tuberculosis as one of its public health functions during the period of occupation after the war. The principal responsibilities of medical officers of military government with respect to tuberculosis were the restoration of normal practice in reporting and the provision of an adequate number of beds for the care of tuberculous patients.

11. During the war, notable advances took place in the control of tuberculosis, in which the Army shared. Perhaps the most important of these was improvement in methods of case finding, through mass roentgen examination. In addition, provision for isolation was much improved, and advances were made in surgical therapy. At the end of the war, the antibiotic treatment of tuberculosis was under consideration. Actually, no cases of tuberculosis were so treated in the Army prior to the end of 1945, although subsequently Army hospitals contributed greatly to the development of chemotherapy in tuberculosis.

12. Research in the Army advanced the knowledge of the causes of breakdown from tuberculosis and indicated what types of strain are particularly likely to lead to relapse and what strains individuals withstand without breakdown of latent lesions. Light was thrown upon the relation of pleurisy with effusion to tuberculosis, and renewed emphasis was placed on the danger of small lesions whose activity is difficult to determine.

13. In general, Army experience indicated the great importance of means for exclusion of tuberculosis. It was clearly evident that measures for this purpose should be continued without fail and improved with the advance of technical methods. Inasmuch as numerous lesions believed to be inactive are seen and become a cause for later inquiry, it is desirable that in the future such lesions be recorded on each soldier's immunization or other record.

CHAPTER

Whooping Cough

Joseph Stokes, Jr., M. D.

The history of the Medical Department of the Army reports only 119 admissions for whooping cough in the total Army during World War I.[1] There were no deaths.

In adults, the disease is often difficult to diagnose and paroxysms of coughing are usually attributed to other causes so that *Hemophilus pert* . . rarely considered as a causative agent. It has been estimated by Collins . . . roximately 75 percent of adults have had recognizable attacks of whooping ough in childhood, while about 95 to 98 percent are immune to the disease. Such data suggest that a considerable number of unrecognized cases without cough, with mild upper respiratory symptoms, or with a cough, not recognized as whooping cough, may well have occurred in childhood. Thus, the number of susceptibles arriving at induction centers would be extremely few and would account for the low rates in World Wars I and II.

The incidence in World War II may well have been higher than is indicated by table 47, which includes the total number of cases recorded and the rates.

TABLE 47.—*Incidence of whooping cough in the U. S. Army, 1940–45*

[Rate expressed as number of cases per annum per 1,000 average strength]

Year	Total Army		United States		Overseas	
	Number of cases [1]	Rate	Number of cases [1]	Rate	Number of cases [1]	Rate
1940	[2] 2	0.01	2	0.01	.....	0
1941	[2] 12	.01	7	.01	.....	0
1942	30	.01	29	.01	1	.00
1943	71	.01	60	.01	11	.01
1944	38	.01	30	.01	8	.00
1945	35	.01	25	.01	10	.00

[1] Excludes cases treated in a duty status.

[2] Total includes all admissions, whereas data for United States include Alaska and cover enlisted men only and data for overseas are for white enlisted men and native troops only.

[1] The Medical Department of the United States Army in the World War. Statistics. Washington: Government Printing Office, 1925, vol. XV, pt. 2, p. 86.

[2] Collins, S. D.: Age Incidence of the Common Communicable Diseases of Children; a Study of Case Rates Among All Children and Among Children Not Previously Attacked and of Death Rates and the Estimated Case Fatality. Pub. Health Rep. 44: 763-826, 5 Apr. 1929.

Studies of the best methods of immunization have been extensive since World War I. Inasmuch as whooping cough has not been a military problem, it would not seem appropriate to enter here into a discussion of the newer methods which have been developed nor will reference be made to their application or usefulness.

Disease Caused by Fungus

CHAPTER XVI

Coccidioidomycosis

Charles Edward Smith, M. D.

HISTORICAL NOTE

Before World War II, there was little thought that coccidioidomycosis, an almost unknown disease, could ever become a military problem. The infection was first reported from Argentina in 1892 [1] and was described 2 years later in California.[2] At that time, the etiologic agent was believed to be a protozoon related to *Coccidium*, and it was therefore named *Coccidioides immitis*.[3]

The mycotic nature of the organism was established in 1900.[4] Further study revealed the diphasic form—the "parasitic" endosporulating spherules in man and the "saprophytic" mycelial form in cultivation. *Coccidioides immitis* was believed to cause serious illness or death in at least half of those who were infected. The disseminated infection, notable for its mimicry of tuberculosis and termed "coccidioidal granuloma," was thought to occur only rarely. In fact, before World War I, most of the recognized cases were reported individually in the literature as they occurred. Apart from Argentina, the disease seemed to be restricted to California, whence the name "California disease" and, even more specifically, "San Joaquin Valley disease." In short, coccidioidal granuloma was a medical curiosity and presented no military problems before World War II.

Between World War I and World War II, the knowledge concerning coccidioidal infections greatly increased. An important advance was the recognition that the disease occurred very frequently in a benign form, coccidioidal erythema nodosum, with negligible mortality.[5] Moreover, the fungus

[1] (1) Posada, A.: Un Nuevo Caso de Micosis Fungoidea con Psorospermias. An. d. Circ. Med. Argent. 15: 585–597, 1892. (2) Wernicke, R.: Ueber einen Protozoenbefund bei Mycosis fungoides. Centralbl. f. Bakt. 12: 859–861, 28 Dec. 1892.

[2] Rixford, E.: A Case of Protozoic Dermatitis. Occidental M. Times 8: 704–707, December 1894.

[3] Rixford, E., and Gilchrist, T. C.: Two Cases of Protozoan (Coccidioidal) Infection of the Skin and Other Organs. Johns Hopkins Hosp. Rep. 1: 209–267, 1896.

[4] Ophüls, W., and Moffitt, H. C.: A New Pathogenic Mould (Formerly Described as a Protozoon; *Coccidioides immitis* Piogenes); Preliminary Report. Phila. M. J. 5: 1471–1472, 30 June 1900.

[5] (1) Gifford, M. A.: San Joaquin Fever. *In* Annual Report Kern County Health Department for Fiscal Year July 1, 1935 to June 30, 1936, pp. 22–23. (2) Gifford, M. A., Buss, W. C., and Douds, R. J.: Data on Coccidioides Fungus Infection, Kern County, 1901–1936. *In* Annual Report Kern County Health Department for Fiscal Year July 1, 1936 to June 30, 1937, pp. 39–54. (3) Gifford, M. A.: Coccidioidomycosis in Kern County, California. Proc. Sixth Pacific Science Congress 5: 791–796, 1942. (4) Dickson, E. C.: "Valley Fever" of San Joaquin Valley and Fungus Coccidioides. California & West. Med. 47: 151–155, September 1937. (5) Dickson, E. C.: Coccidioidomycosis; Preliminary Acute Infection With Fungus Coccidioides. J. A. M. A. 111: 1362–1365, 8 Oct. 1938. (6) Dickson, E. C., and Gifford, M. A.: Coccidioides Infection (Coccidioidomycosis); II. The Primary Type of Infection. Arch. Int. Med. 62: 853–871, November 1938.

was recovered from the soil and was shown to cause natural infection in cattle and sheep as well as in man.[6] Thus, it came to be appreciated that coccidioidomycosis is an exceedingly common infection, rather than a rare one, and that its disseminated form, coccidioidal granuloma, is the exception rather than the rule.

Studies completed just before World War II indicated that a very high infection rate occurs among newcomers to endemic areas.[7] This fact suggested that a significant medical problem could arise if large military installations were to be established in these areas and susceptible adults were to be stationed there. The studies indicated that the endemic areas, while still ill defined, probably extended into some arid regions of southern California, Arizona, and west Texas. However, no methods of preventing acquisition of coccidioidal infections or of minimizing the disseminating form were even suggested. Indeed, the idea that a health problem even existed was largely a conjecture, since the newly suspected, extended endemic area enjoyed high favor for its salubrious climate.

INCIDENCE OF COCCIDIOIDOMYCOSIS

It has not been possible to obtain accurate information as to the incidence of clinical coccidioidal infection in military personnel during World War II. The Medical Statistics Division of the Surgeon General's Office has furnished provisional estimates based on sample tabulations of individual medical records. Table 48 shows that there was a total of 3,809 cases for the 4 years, 1942–45, and table 49 shows 39 deaths attributed to coccidioidomycosis for the same period. Unfortunately, there could be no breakdown by Army Ground Forces as compared with Army Air Forces. Provisional data based on summaries of the statistical health report provide a distribution of cases in the United States by service command. While this source is subject to underenumeration for this diagnosis (coccidioidomycosis was a writein entry only), the over-all statistics are of interest. Of the 2,889 cases reported from 1942 through 1945 in the United States, 2,717 (94 percent) were in the Ninth Service Command, 86 (3 percent) were in the Eighth Service Command, and the remainder was distributed among all the other service commands, except the Sixth, for which no cases were reported. The fact that more than 700 cases of coccidioidomycosis, or 18 percent of the reported Army total, were hospitalized in only 4 station hospitals in the San Joaquin Valley (author's personal records) indicates that 3,809 cases is a gross understatement of actual incidence of the disease.

[6] (1) Stewart, R. A., and Meyer, K. F.: Isolation of *Coccidioides immitis* (Stiles) From the Soil. Proc. Soc. Exper. Biol. & Med. 29: 937–938, May 1932. (2) Giltner, L. T.: Occurrence of Coccidioidal Granuloma (Oidiomycosis) in Cattle. J. Agric. Research 14: 533–541, 16 Sept. 1918. (3) Beck, M. D.: Occurrence of *Coccidioides immitis* in Lesions of Slaughtered Animals. Proc. Soc. Exper. Biol. & Med. 26: 534–536, March 1929. (4) Beck, M. D., Traum, J., and Harrington, E. S.: Coccidioidal Granuloma; Occurrence in Animals—Reference to Skin Tests. J. Am. Vet. M. A. 78: 490–499, April 1931. (5) Beck, M. D., Dickson, E. C., and Rixford, E.: Coccidioidal Granuloma. California State Health Dept. Spec. Bull. 57, June 1931. (6) Davis, C. L., Stiles, G. W., and McGregor, A. N.: Coccidioidal Granuloma in Calves. J. Am. Vet. M. A. 92: 562–563, April 1938.

[7] Smith, C. E.: The Epidemiology of Acute Coccidioidomycosis with Erythema Nodosum ("San Joaquin" or "Valley Fever"). Am. J. Pub. Health 30: 600–611, June 1940.

It will be seen from table 48 that an insignificant number of cases of coccidioidomycosis occurred overseas. Apparently, the disease appeared only in troops who had acquired their infections in the arid southwestern United States before transportation overseas. The number of admissions was too small to permit any deductions as to the endemicity of the overseas areas in which they occurred.

TABLE 48. *Total cases and incidence rate of coccidioidomycosis in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records of primary and secondary diagnoses]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate
United States	3,626	0.25	599	0.26	1,310	0.25	977	0.25	640	0.22
Overseas	183	.02	2	0	13	.01	88	.02	80	.02
Total	3,809	.15	701	.22	1,323	.19	1,065	.14	720	.10

TABLE 49.—*Deaths from coccidioidomycosis, in the U. S. Army, by area and year, 1942–45*

[Rate expressed as number of deaths per annum per 100,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate
United States	32	0.22	6	0.23	12	0.23	9	0.23	5	0.17
Overseas	7	.07	------	0	------	0	3	.08	4	.09
Total	39	.15	6	.18	12	.17	12	.15	9	.12

COCCIDIOIDOMYCOSIS IN THE ARMY AIR FORCES

Military attention to the problem of coccidioidomycosis in World War II converged simultaneously from two directions and almost immediately united. In 1940–41, the Army Air Forces began development of an extensive year-round aviation-training program to be located at airfields in the climatically advantageous Southwest, specifically in the San Joaquin Valley. Recognizing the potential hazard of coccidioidomycosis at these proposed airfields, Dr. Walter T. Harrison, United States Public Health Service, liaison officer for the Ninth Corps Area, warned the corps area surgeon and the headquarters staff of the West Coast Training Center. Dr. Harrison brought the author, who had carried on epidemiologic investigations of the disease in the selected areas, to

meet the commanding officer of the West Coast Training Center and the senior flight surgeon. The members of this conference, appreciating the danger of serious infection, agreed that further detailed clinical and epidemiologic studies were desirable. The problem was further considered in correspondence between the surgeon of the Ninth Corps Area and the Surgeon General's Office during February and March 1941, and a policy of minimizing use of the endemic areas was established.[8] A deviation from this policy in the summer of 1942 resulted in a severe epidemic of coccidioidomycosis (p. 293).

While the basic training fields in the San Joaquin Valley rapidly increased, the Commission on Epidemiological Survey of the Army Epidemiological Board, Preventive Medicine Division, Office of the Surgeon General, developed plans for a study of coccidioidomycosis, including research into its epidemiology. A Ninth Corps Area consultant group, with Dr. Edwin W. Schultz as director and Dr. Edward B. Shaw and the author as members, was established in February 1941 to help accomplish this study. Dr. Francis Blake, president of the Army Epidemiological Board, and Dr. Stanhope Bayne-Jones, chairman of the Commission on Epidemiological Survey, worked closely with the author in preparing plans for this study of coccidioidomycosis which were approved by the Army Epidemiological Board in June 1941. Rapid and direct communication between the Office of the Surgeon General and the Commanding General, West Coast Training Center, was greatly facilitated by the interest taken by Col. (later Brig. Gen.) James S. Simmons, MC, Chief, Preventive Medicine Division, Office of the Surgeon General.

The study was centered in the Department of Public Health and Preventive Medicine, Stanford University School of Medicine, where laboratory and epidemiologic facilities were provided. Dr. Rodney R. Beard of that department was appointed to the Ninth Corps Area consultant group and participated in the initial coccidioidin testing and in the subsequent semiannual retesting. Funds for the first 4 months of the study were supplied by the Rosenberg Foundation. By July 1941, the project was in active operation when all the permanent party personnel of Minter Army Air Field, Bakersfield, Calif., and Gardner Army Air Field, Taft, Calif., were coccidioidin tested.

Headquarters of Minter Field was at Bakersfield Junior College. The men lived in tents pitched at the Kern County Airport. The dispensary where coccidioidin testing was performed was a large tent equipped with an electric hotplate and an empty vegetable can for a sterilizer. The Gardner Field infirmary was an unfinished building shared by the Medical Department, post exchange, and barber shop and partitioned by cases of beer and soft drinks. Dust was ankle deep and swirled in clouds over the fields. Clinical cases of coccidioidomycosis were appearing; indeed, with dust completely uncontrolled, infections were maximal and never occurred again at such a rate. At Minter Field, one-fifth of all susceptibles were infected during the summer and fall of

[8] (1) Letter, Col. H. R. Beery, Surgeon, Ninth Corps Area, to Col. C. C. Hillman, Assistant to the Surgeon General, 24 Feb. 1941. (2) Letter, Col. C. C. Hillman, Office of the Surgeon General, to Col. H. R. Beery, Ninth Corps Area, 11 Mar. 1941.

1941. The personnel of the two fields were interested in cooperating for their own protection, and the surgeons at both fields set a pattern of collaboration which their successors emulated.

New arrivals at both fields were coccidioidin tested, and results were entered on the immunization records.[9] Immediate interest focused on whether or not clinical infections occurred in personnel who were sensitive to coccidioidin on arrival at the fields. It was soon apparent that clinical infections never occurred in those people and that the skin testing with coccidioidin differentiated susceptibility from immunity. Thus, the medical officers were provided with a most sensitive method of diagnosis.[10] As further aids in diagnosis and prognosis, recently discovered serologic tests and mycologic facilities were made available as a result of studies supported by the Commission.[11]

A death from coccidioidal meningitis at one of the fields served to emphasize the importance of careful diagnosis and medical surveillance of the disease. An enlisted man had failed to react to coccidioidin when initially tested in July 1941, but he was found to have a positive reaction when retested in October. At that time, he reported that he had experienced a "flu-like" illness associated with pleural pain in late September, but he stated that he was feeling much better. No sedimentation or serologic tests were performed, and the soldier was permitted to continue strenuous activities. Meningitis developed in this soldier in December. After this occurrence, it became a requirement that histories of all personnel previously negative to coccidioidin who converted to positive at retest be recorded on clinicoepidemiologic forms and that blood from these individuals be drawn for serologic and sedimentation tests. A number of soldiers were hospitalized as a result of this procedure. Criteria of recovery from coccidioidomycosis were established: these emphasized that patients should not only be clinically recovered but should also have normal sedimentation rates, receding complement fixation titers, and regressing roentgenographic densities before being discharged from the hospital to active duty. Accurate diagnosis of the disease was also emphasized.

When the author visited the field at monthly intervals to test new personnel, he saw the patients with the medical officers. Some of the clinicoepidemiologic forms were filled out by local medical officers, some by the author. Those filled out by local medical officers were reviewed by the author in the presence of the patient to insure uniformity both at the two fields and, since turnover of medical officers was rapid, on the individual fields. A fundamental objective of these activities was the provision of maximal medical service for persons who were infected, which in turn minimized the risk of disseminated

[9] (1) Smith, C. E., Beard, R. R., Rosenberger, H. G., and Whiting, E. G.: Effect of Season and Dust Control on Coccidioidomycosis. J. A. M. A. 132: 833-838, 7 Dec. 1946. (2) Smith, C. E., Beard, R. R., Whiting, E. G., and Rosenberger, H. G.: Varieties of Coccidioidal Infection in Relation to the Epidemiology and Control of the Diseases. Am. J. Pub. Health 36: 1394-1402, December 1946.

[10] Smith, C. E., Whiting, E. G., Baker, E. E., Rosenberger, H. G., Beard, R. R., and Saito, M. T.: The Use of Coccidioidin. Am. Rev. Tubere. 57: 330-360, April 1948.

[11] Smith, C. E., Saito, M. T., Beard, R. R., Kepp, R. M., Clark, R. W., and Eddie, B. U.: Serological Tests in the Diagnosis and Prognosis of Coccidioidomycosis. Am. J. Hyg. 52: 1-21, July 1950.

infection. The byproduct of these activities was increased knowledge of the pathogenesis and clinical aspects of coccidioidomycosis.[3]

As has been indicated, periodic retesting of personnel at the fields was necessary. The results of the testing and retesting, together with clinical observation, provided information on the proportion of clinical cases to inapparent infections and served as the bases for calculating monthly and annual infection rates.[3] Annual rates were essential in evaluating control measures which, as will be presented later, focused on dust control.

Late in 1941, two more basic training fields were established in the San Joaquin Valley, one at Lemoore, Calif., and the other at Merced, Calif. With the approval of the Commission on Epidemiological Survey and the Western Flying Training Command, the study was extended to those fields. Testing of the Merced personnel began in February 1942, and testing of the Lemoore personnel began in March. The same outstanding cooperation that had been shown at Minter and Gardner was shown by the personnel at Merced and Lemoore.

The intensive studies were continued at the four fields as long as they remained under the Western Flying Training Command. On 1 March 1945, Gardner Field became inactive. On 1 May 1944, Lemoore Army Airfield was transferred to the Fourth Air Force to serve as an inprocessing center. The study of coccidioidomycosis continued at Lemoore until 30 September 1945 when the field became inactive and at Minter until the field was closed in February 1946. On 1 July 1945, Merced Army Airfield was transferred from the Training Command, and, because the 3 years of close study had proved it was not an area where coccidioidomycosis was endemic, the intensive field work of the Commission on Epidemiological Survey was terminated. Merced had served as an unexpected but fortunate control area and also had enabled studies of the duration of coccidioidin sensitivity in a nonendemic area.[4] Furthermore, studies at Merced had proved that high "inapparent" infection rates manifested by asymptomatic "conversions" at the other three fields were valid.

Other Control Programs

The Commission on Epidemiological Survey's intensive clinical and epidemiologic investigations served as a pilot program for two more extensive control programs established by the Air Forces. The first was established by the West Coast Army Air Forces Training Center. In the summer of 1942, the Western Flying Training Command was confronted with a wave of coccidioidomycosis in its Arizona flying fields. The training command surgeon requested the surgeon of the West Coast Army Air Forces Training Center to organize a control program. Flight surgeons of Santa Ana Army Airbase,

[3] Smith, C. E., Beard, R. R., and Saito, M. T.: Pathogenesis of Coccidioidomycosis With Special Reference to Pulmonary Cavitation. Ann. Int. Med. 29: 623-635, October 1948.

[3] See footnote 9, p. 289.

[4] See footnote 10, p. 289.

Calif., were assigned the responsibilities of developing this program, and the author was called upon as consultant.

The ultimate coccidioidomycosis-control program of the Western Flying Training Command, expanded from the Commission on Epidemiological Survey's pilot program on the four fields, was inaugurated on 16 October 1942. This new program consisted of testing new permanent party personnel on arrival at stations and of retesting them on transfer and semiannually in January and July. Cadets were coccidioidin tested during their preflight training at Santa Ana and again when taking their final "64" examination at advanced fields. To insure a uniformly high quality of medical care, all seriously ill patients and patients whose illness lasted 3 months were transferred to the Santa Ana Army Airbase Regional Hospital.

A coccidioidomycosis-control officer for the West Coast Army Air Forces Training Center was designated, and a local coccidioidomycosis-control officer was appointed at each installation. An outstanding syllabus was prepared, bringing together the orders setting up the program and essential information regarding coccidioidomycosis.[15] A second edition was issued on 15 September 1943 and a third on 15 March 1944. Excellent pulmonary roentgenograms were included. The Western Flying Training Command maintained the policy of having the Commission on Epidemiological Survey's investigative group operate the coccidioidin-testing programs in the four San Joaquin Valley airfields in order to continue the detailed epidemiologic studies. Standardized coccidioidin and containers for serum specimens were sent to the West Coast Army Air Forces Training Center at Santa Ana Army Airbase to be distributed to the various fields. The serologic tests were all performed by the group at Stanford University.

A high caliber of medical care of patients with coccidioidomycosis resulted from the program, and important epidemiologic data were assembled. The value of the latter was somewhat impaired by the fact that rapidly changing personnel and varying enthusiasms at the local fields caused the quality of records to be uneven. At some of the fields in nonendemic areas, the amount of work imposed by the repeated testing, especially in light of the frequent transfer of personnel, was open to some criticism.

The second Army Air Forces control program came into being when Lemoore Army Airfield was transferred from the Western Flying Training Command to the Fourth Air Force. In consultation with the author, the Surgeon, Fourth Air Force, established a coccidioidomycosis-control program based on local needs. The testing and retesting program of the permanent party personnel at Lemoore was continued. Since the most important problem would be the infection of personnel processed at Lemoore and then assigned to other stations while in the incubation period of coccidioidomycosis, a summary of the control program was prepared and sent to all stations of the Fourth Air Force. From a depot established by the Commission on Epidemiological

[15] Coccidioidomycosis Control Program for the West Coast Army Air Forces Training Center, 16 Oct. 1942.

Survey at Lemoore, the coccidioidomycosis-control officer sent coccidioidin and sterile containers for blood to each installation.

Coccidioidomycosis was also of importance in two airfields outside the Western Flying Training Command: Davis-Monthan Field, Tucson, Ariz., and March Field, near Riverside, Calif. There was close liaison between the Commission on Epidemiological Survey and Davis-Monthan Field, and the medical officers at the field were provided with coccidioidin and serologic and mycologic diagnostic facilities. The extensive experiences of the field's regional hospital were summarized by Lt. Col. Charles F. Sweigert, MC, and his colleagues.[16] Although March Field itself was not in an endemic area, occasional cases of coccidioidomycosis in personnel who had acquired infections in the nearby endemic areas were detected with the diagnostic facilities provided by the Commission on Epidemiological Survey. In the spring of 1943, many positive specimens were received from men who had been on bivouacs around Banning, Calif., for a period appropriate for the incubation of coccidioidomycosis. On 8 May 1943, the chief of medicine at March Field Regional Hospital, Maj. Forrest M. Willett, MC, was advised to scrutinize the situation carefully and, if it appeared to justify the suspicion that this area was endemic, to test personnel going to Banning from March Field and to retest them on their return. The importance of the possible endemic area not only to March Field but also to the Desert Training Center of the Army Ground Forces was under consideration. Major Willett and Capt. Alvin Weiss then carried out a very extensive study. Their report reviewed the spotty endemicity of coccidioidomycosis in southeastern California and their experiences with 100 coccidioidal infections.[17] This study was brought to the attention of Army Ground Forces.

COCCIDIOIDOMYCOSIS IN THE ARMY GROUND FORCES

As has been indicated, in February and March 1941, a policy was established placing the San Joaquin Valley out of bounds as a location for camps and maneuvers. Only a few months later, the wisdom of this policy was supported when a new "fringe" endemic area over the Coast Range Mountains at Camp Roberts, Calif., was authenticated. In May 1941, just as the work of the Commission on Epidemiological Survey was being instituted at Stanford, Lt. Robert M. Shelton had requested coccidioidin for testing some suspicious cases of erythema nodosum. The positive coccidioidin tests were followed by serologic and mycologic confirmation.[18] Apparently, the infections at Camp Roberts were acquired not at the main campsites along United States Highway 101 but on bivouacs and maneuvers in the eastern sections of the camp. As time went on, Camp Roberts provided a significant proportion

[16] Sweigert, C. F., Turner, J. W., and Gillespie, J. B.: Clinical and Roentgenological Aspects of Coccidioidomycosis. Am. J. M. Sc. 212: 652-673, December 1946.

[17] Willett, F. M., and Weiss, A.: Coccidioidomycosis in Southern California; Report of a New Endemic Area With a Review of 100 Cases. Ann. Int. Med. 23: 349-375, September 1945.

[18] Shelton, R. M.: Survey of Coccidioidomycosis at Camp Roberts, California. J. A. M. A. 118: 1186-1190, 4 Apr. 1942.

of belatedly recognized coccidioidal residual as well as of fatal disseminating infections. Had *Coccidioides* been any more prevalent, the value of the campsite might have been in jeopardy. No criticism can be made of locating the camp in that region for, before Lieutenant Shelton's investigations, no one realized that the endemic area extended across the Coast Ranges.

Maneuvers in San Joaquin Valley

Ground Force personnel did experience one epidemic of coccidioidomycosis as the price for violating the "no-trespassing" warning for the San Joaquin Valley. Recognition of this epidemic came as a direct result of the close liaison between the chief of the laboratory service at Camp San Luis Obispo, Calif., and personnel of the Commission on Epidemiological Survey's coccidioidomycosis-control program. During the winter and spring of 1941 and 1942, a number of serologic specimens were sent to the Commission's facilities. Only one of these specimens was positive; it came from a soldier who had acquired an infection by travel through the San Joaquin Valley. In July 1942, a large number of specimens sent by the Camp San Luis Obispo laboratory were positive. The 7th Motorized Division had maneuvered in the northwest corner of Kern County (Antelope Valley, an arm of the San Joaquin Valley) during June and July. An epidemic of severe respiratory illnesses ensued which was proved to be coccidioidal. An excellent description of the epidemic was prepared by Maj. David M. Goldstein, MC, and Capt. Stanley Louie, MC.[19] Seventy-five cases of clinical coccidioidomycosis were diagnosed; on the ratio of erythema nodosum to infections, it is calculated that there were between 300 and 400 infections. On 3 August, the author reported the situation to the Surgeon General's Office, and on 5 August a memorandum was sent to the Surgeon, Army Ground Forces, informing him of the situation and reemphasizing the hazards of maneuvers in endemic areas.[20] Furthermore, on 15 August 1942, The Surgeon General prepared a brief outline of the coccidioidomycosis problem which was sent to the Commanding General, Services of Supply, calling attention to the unfortunate consequences of the recent maneuvers and recommending that maneuvers in the valley be kept to a minimum.

On 18 August 1942, Army Ground Forces endorsed the July sanitary report of the 7th Motorized Division and approved the recommendation it contained: "That the Western Portion of the San Joaquin Valley south of Fresno, California, and especially that portion lying in Kern County not again be used for a maneuver or training area for Army personnel." When endorsing the sanitary report to Services of Supply, the Surgeon General's Office concurred in the recommendation and invited attention to its letter of 15 August which had discussed the problem.

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[19] Goldstein, D. M., and Louie, S.: Primary Pulmonary Coccidioidomycosis; Report of an Epidemic of 75 Cases. War Med. 4: 299-317, September 1943.

[20] Memorandum, Col. J. S. Simmons, Office of the Surgeon General, for Col. F. A. Blesse, Army Ground Forces, 5 Aug. 1942, subject: Coccidioidomycosis (San Joaquin Valley Fever) in California.

The constant admonitions of the Preventive Medicine Service together with the finger-burning experience of the 7th Motorized Division sufficed to keep other ground troops out of the San Joaquin Valley.

California-Arizona Maneuver Area

A serious problem developed in the California-Arizona Maneuver Area, the Desert Training Center, which was located in the desert areas of southeastern California and western Arizona. Roughly triangular in shape, it was 300 miles long and 200 miles wide at its base. It consisted of series of dry lakes, sandy valleys, and mountain ranges of rock or shale. The maneuver area was used to harden troops physically, to train them mentally for the shock of battle, and to enable them to operate under realistic battle conditions; it was also used to test and develop equipment. Coccidioidomycosis constituted the chief health hazard peculiar to the area, although this fact was not recognized during the early months of the war.

Recognition of the problem began in 1943. In January, Lt. Col. Roswell Brown of the Desert Warfare Board visited the author and discussed the possible hazard of coccidioidomycosis in the Desert Training Center. Sample skin-testing surveys were advised, and it was suggested that medical officers be alerted to the danger of this infection, particularly in the spectacular and easily recognized form of erythema nodosum. While this plan was under consideration, the Desert Training Center received the following information from the 54th Station Hospital near Yuma, Ariz.[21]

> * * * We were out on "grand maneuvers" for three weeks, returning to our base a week ago. Very suddenly we got a number of men with influenza-like symptoms, and a bizarre lung finding, on physical and on x-ray. Today we have three positives out of five tests, as well as an outbreak of "Epidermo phytid" [doubtless erythema multiforme] and erythema nodosum in these same patients. (One of these is a man from the Royal Dutch Army, who had been in this country only one month, three weeks of which were out on the desert, and one week in the hospital.)

Subsequent serologic examinations confirmed the epidemic as coccidioidomycosis. The site of the infections was specifically located in an area near Pallen Pass, 20 miles west of Blythe, Calif. This was in the maneuver area where personnel received final polishing. The information was sent at once to the Surgeon General's Office which immediately notified the Surgeon, Army Ground Forces, that a previously undetermined area was heavily infected with *Coccidioides*.[22]

Medical officers of the California-Arizona Maneuver Area initiated a coccidioidomycosis control program by arranging for the commanding officer of the 5th Medical Laboratory to visit the Commission on Epidemiological

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[21] Letter, Dr. C. E. Smith, to Maj. R. K. Brown, MC, Desert Training Center, 19 Mar. 1943

[22] (1) Letter, Lt. Col. K. R. Lundeberg, Office of the Surgeon General, to Surgeon, Army Ground Forces, 26 Mar. 1943, subject: Coccidioidomycosis in the Desert Training Center. (2) Goldstein, D. M., and McDonald, J. R.: Primary Pulmonary Coccidioidomycosis: Follow-up of 75 Cases, with 10 More Cases from New Endemic Area. J. A. M. A. 124: 557-561, 26 Feb. 1944.

Survey's Stanford facilities the first week in May 1943. At this meeting, it was recognized that a testing and retesting program, such as that followed in the Air Forces Western Flying Training Command, was impractical in this area. The Commission consultants recommended a twofold program: (1) To educate the medical officers in recognition of coccidioidomycosis and provide them with diagnostic facilities, and (2) to delimit endemic areas by epidemiologic investigations including selected skin-testing surveys and a continuous search for "sentinel" erythema nodosum. It was agreed that coccidioidin and, after precipitin screening by the 5th Medical Laboratory, serologic tests would be provided by the Commission on Epidemiological Survey. However, the 5th Medical Laboratory was succeeded by the 7th before these procedures could be instituted.

Meanwhile, a greater appreciation of the problems of coccidioidomycosis was stimulated among the medical staff of the headquarters of the communications zone of the California-Arizona Maneuver Area at Banning. It was here that Major Willett was conducting an intensive investigation as a result of the epidemic described on p. 292.[23] Possible procedures were discussed by the staff with Major Willett during June and July (1943). Later, the author offered to them the same suggestions as had been made to the commanding officer of the 5th Medical Laboratory. In August, the commanding officer of the 7th Medical Laboratory visited Stanford to institute the necessary diagnostic facilities. However, the work had only begun when the 7th Medical Laboratory was succeeded by the 9th. The commanding officer of the latter developed an efficient method for the distribution of skin-testing coccidioidin and for the collection and screening of serum specimens. Consequently, there was a great increase in confirmed diagnoses of coccidioidomycosis.

On 23 August 1943, the office of the surgeon of the communications zone proposed a systematic control program under an established control officer together with recognition and avoidance of highly endemic areas, but the proposal was rejected by the commanding general on 31 August because of shortage of personnel and in the belief that there seemed to be no urgent necessity for such a program.[24]

Although no systematic coccidioidomycosis study was made by the medical staff of the Desert Training Center Headquarters, an analysis of the area was made by one officer. His survey showed that in the Desert Training Center coccidioidomycosis was highly endemic in the Pallen Pass Maneuver Area and in the southern Arizona strip from Yuma to Camp Hyder, whereas it appeared not to be endemic in much of the northern section of Arizona and in California localities including Pomona, San Bernardino, Camp Young, and most of the area of Imperial, Riverside, and San Bernardino Counties.[25]

That a very significant hazard persisted is borne out by the history of

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[23] See footnote 17, p. 292.

[24] Letter, Lt. Col. H. A. Furlong, MC, to Surgeon, Desert Training Center, Camp Young, Calif., 23 Aug. 1943, subject: Coccidioidomycosis.

[25] (1) Letter, Maj. G. A. Young, Jr., MC, to Dr. C. E. Smith, 18 Jan. 1944. (2) Letter, Capt. G. A. Young, MC, to Surgeon, Headquarters, Communications Zone Desert Training Center, 18 Aug. 1943, subject: Comprehensive Survey of Coccidioidomycosis in the Desert Training Center, with 2d endorsement.

coccidioidomycosis in the 77th Infantry Division, which was at Camp Hyder, Ariz., from 1 April to 4 October 1943. Infections in personnel from this division were reported from Indiantown Gap Military Reservation, Pa., Camp Pickett, Va., and the 219th General Hospital on Oahu, T. H. The monthly statistical report of the division for September 1943 refers to an outbreak of 106 cases of epidemic pleurodynia, all of which occurred at Camp Hyder, while even in 1944 roentgenograms of the personnel in the Central Pacific revealed 80 suspicious cases, of which 20 were established as coccidioidal in etiology.[26]

A patient who had cavitation due to coccidioidomycosis was discovered at Fort Bragg Station Hospital, N. C. The patient was one of a group of field artillery trainees from the California-Arizona Maneuver Area.[27] Subsequent tests of 1,522 cavalry personnel trained at Camp Hyder revealed that 22 percent reacted positively to coccidioidin.[28] However, they were sent overseas before adequate histories or roentgenograms could be taken.

An excellent memorandum reviewing the coccidioidomycosis problem was prepared for The Surgeon General by the Epidemiology Division, Preventive Medicine Service, Office of the Surgeon General.[29] Capt. Philip E. Sartwell of that division visited the Surgeon, Army Ground Forces, in an attempt to improve the control measures of the California-Arizona Maneuver Area.[30] Before any realistic measures could be taken, however, the maneuver area began to disband, so the problem ceased to exist.

Perhaps the policy of virtually ignoring the problem of coccidioidomycosis was wise. However, if there should be future need for reestablishing such a training program and if advantages of climate and terrain do not outweigh the hazards of coccidioidomycosis, the highly endemic southern rim and central portion of Arizona and the Pallen Pass area of California as well as the southern and western San Joaquin Valley should be avoided.

COCCIDIOIDOMYCOSIS AMONG PRISONERS OF WAR

Another Army problem due to coccidioidomycosis concerned prisoners of war. A major camp for prisoners was established at Florence, Ariz. Col. Verne R. Mason, MC, medical consultant to the Surgeon, Ninth Service Command, visited the Florence Station Hospital in September 1943. Recognizing that there were a number of probable coccidioidal infections, he alerted the medical officers, reported his findings, and also informed the Commission on Epidemiological Survey. On 7 October, a letter of inquiry was sent from

[26] (1) Letter, The Surgeon General to Commanding Officer, Desert Training Center, 23 Oct. 1943, subject: Reported Epidemic of Pleurodynia, with endorsements and enclosure. (2) Letter, The Surgeon General to Commanding Officer, Indiantown Gap Military Reservation, 13 Nov. 1943, subject: Reported Cases of Coccidioidomycosis, with 1st endorsement.

[27] Letter, Dr. T. J. Abernethy to Dr. C. E. Smith, 8 Jan. 1944.

[28] Letter, Dr. T. J. Abernethy to Col. Stanhope Bayne-Jones, 16 Mar. 1944.

[29] Memorandum, Col. K. R. Lundeberg, MC, for The Surgeon General, 3 Jan. 1944, subject: Review of Coccidioidomycosis Problem in the Army.

[30] Memorandum for file, Capt. P. E. Sartwell, Office of the Surgeon General, [23 Dec. 1943], subject: Conference With Ground Surgeon on Coccidioidomycosis.

Florence Station Hospital to the Commission at Stanford. A detailed reply outlined a control program and offered the Commission's facilities.[31] Florence, the Arizona prisoner-of-war headquarters from which a number of side camps for field work radiated, was in a known endemic area. Already, the Commission on Epidemiological Survey had provided coccidioidin, serologic facilities, and consultative advice to the Japanese Relocation Center on the Gila River not far away because of outbreaks of cases there. From that experience and from the information garnered from Williams Field, Ariz., in the Western Flying Training Command, it was realized that endemicity was high.

Despite the Commission's offer of facilities, personnel at Florence made no use of them until December 1943, when the first serologic specimens were sent for testing. The results of these tests, together with recognition of two deaths due to coccidioidomycosis, and a return visit by Colonel Mason initiated action. An additional complication at Florence was the fact that, because of the reputation of the area for its salubrious climate, Florence Station Hospital was being used to hospitalize all tuberculous prisoners of war. On 4 February 1944, Colonel Mason transmitted to Professional Services and, in turn, to Preventive Medicine Service of the Surgeon General's Office a report on the coccidioidomycosis hazard in the prisoner-of-war area. Colonel Bayne-Jones, aware of recent German complaints that prisoners of war had been placed in unhealthy areas, decided upon an immediate investigation.[32] Accordingly, the author was dispatched to Florence on 23 February. A review of hospital records using erythema nodosum as an index of infection showed a rapidly increasing problem. A sampling coccidioidin survey of hospital patients and personnel substantiated the more extended series made previously at Florence and the extensive coccidioidin-testing figures from nearby Williams Field. In that region, 50 percent of susceptibles were infected within 6 months' time. Indeed, 10 tuberculous prisoners of war were found to have been infected while they were hospitalized in the wards. One patient who had a tuberculous effusion on admission to hospital developed a coccidioidal effusion in his other lung. The superimposed coccidioidal infections did not appear to affect the tuberculous infections adversely.

A systematic testing and retesting program for detecting coccidioidomycosis was suggested by the author, with the realization that very soon everyone in the area would be infected and, as a result, immune. Since prompt recognition of the onset of the infection was important in order that appropriate medical attention might be given and since patients hospitalized because of tuberculosis were already undergoing bed rest and apparently were not harmed by the superimposed coccidioidomycosis, the author recommended that a control program be inaugurated and that operation of the hospital be continued.[33]

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[31] Letter, Dr. C. E. Smith to Capt. T. Kendig, MC, 12 Oct. 1943.

[32] Memorandum, Col. S. Bayne-Jones, MC, for General Simmons, 18-19 Feb. 1944, subject: Coccidioidomycosis in Prisoners of War at Florence, Arizona.

[33] Letter, Dr. C. E. Smith to The Surgeon General through Col. S. Bayne-Jones, Preventive Medicine Service, 3 Mar. 1944, subject: Coccidioidomycosis at the Prisoner of War Camp, Florence, Arizona.

However, when the report was reviewed in Washington, the decision was made to avoid any criticism of violation of policies governing hospitalization of prisoners of war,[34] and tuberculous patients were moved to other hospitals. Florence was maintained as headquarters for Italian and later German prisoners of war working out of the side camps. Because of the small number of medical officers and the extreme rapidity with which prisoners as well as medical personnel were transferred, the suggested systematic coccidioidin-testing program could not be carried through. However, the hospital's personnel continued to make use of the Commission on Epidemiological Survey's serologic diagnostic facilities.

Another outbreak of coccidioidomycosis in prisoners of war occurred the following year (1945) in the San Joaquin Valley. From the prisoner-of-war base at Camp Cooke, Calif., several work camps were established near Minter and Lemoore Fields. The station hospitals of these fields were used for temporary hospitalization of the prisoners. In an exchange of correspondence with the surgeon of Camp Cooke Station Hospital, the author indicated the probability that coccidioidomycosis would become a serious problem in these camps after June.[35] When General Bayne-Jones received copies of this correspondence, he informed the Provost Marshal General, who indicated that the endemic areas would be avoided. However, nothing was done, and the incidence of the disease rose during that summer.

On 7 August 1945, the chief of the Epidemiology Division, Preventive Medicine Service, pointed out to the commanding officer of Camp Cooke that the incidence of coccidioidomycosis in prisoners at that camp was greater than the total for all of the United States Army troops. Further information was requested.[36] During 2 months and 10 days, June to August, 150 German prisoners of war with coccidioidomycosis were hospitalized at Minter Field.[37] These represented approximately 10 percent of the local prisoner-of-war population. During the same period, there were only 22 clinical cases in the 3,400 Minter Field personnel. Even if one-third of the Minter personnel were immune, the rate was still less than one-tenth that of the prisoners. These data were strong testimony to the value of the dust-control program provided at Minter Field. Indeed, the rate in the prisoners of war was comparable to that which had occurred in 1941 at Minter itself. This rate attested the problems posed by coccidioidomycosis every time recommendations concerning operations in endemic areas were ignored despite the vigilance of the Preventive Medicine Service.

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[34] Memorandum, Brig. Gen. H. J. Morgan, Director, Medical Division, Office of the Surgeon General, for Hospital Division, Office of the Surgeon General, 24 Mar. 1944, subject: Tuberculosis Center for Prisoners of War at Florence, Arizona.

[35] Letter, Dr. C. E. Smith to Col. W. C. von Kessler, Surgeon, Camp Cooke, 19 Feb. 1945.

[36] Letter, The Surgeon General, to Commanding Officer, Camp Cooke, Calif., through Commanding General, Ninth Service Command, 7 Aug. 1945, subject: Coccidioidomycosis.

[37] See footnote 9 (1), p. 289.

SERVICES PROVIDED BY THE COMMISSION ON EPIDEMIOLOGICAL SURVEY

One of the the objectives of the Preventive Medicine Service was provision of facilities for accurate diagnosis of coccidioidomycosis in order that patients would have the benefit of care which would minimize risk of disseminated infection. Therefore, the Commission on Epidemiological Survey at the Stanford University Medical School prepared coccidioidin, standardized it, and distributed it freely on request to all the armed services. The Commission also performed serologic tests. In 1941, it performed 500 serologic tests; in 1942, 2,000 tests; and, in the succeeding years, it performed over 3,000 tests annually.

Since it was required that brief histories accompany specimens and that interpretations be included with reports, a considerable amount of consultative service developed. The data obtained also aided greatly in determining the values and limitations of the tests. These diagnostic aids made unnecessary most of the culturing of *Coccidioides*, which is known to be hazardous from the point of view of laboratory infections. However, in seven military installations, coccidioidal infections are known to have occurred in laboratories.

The Commission on Epidemiological Survey's facilities were always available for examination of suspicious cultures and were used freely by installations in the West. This arrangement minimized risk of infections in the laboratories of the Armed Forces, although it probably contributed to the continuation of laboratory infections at the Stanford University School of Medicine. Moreover, one of the most valuable aspects of the provision of diagnostic materials and facilities together with consultative service was the "listening post" it provided for the Preventive Medicine Service which took immediate and appropriate action as the need arose.

COLLABORATION OF ARMED FORCES ORGANIZATIONS WITH THE PREVENTIVE MEDICINE SERVICE

The Deputy Air Surgeon had maintained a special interest in coccidioidomycosis since the inception of investigations in 1941, and close liaison with the Preventive Medicine Service, Office of the Surgeon General, provided a continuity of interest and collaboration even after the Office of the Air Surgeon was organized as a unit independent of the Office of the Surgeon General.

Special mention should also be made of the extraordinary interest and contributions of a large number of medical officers, many of whose studies have been cited in this chapter, in effecting the objectives of the Preventive Medicine Service in the prevention of complications of coccidioidomycosis, in minimizing the movement of troops in endemic areas through necessary delimiting of such areas, and in achieving dust control for installations in

known endemic areas. Moreover, data which they assembled also added significantly to knowledge of the pathogenesis, epidemiology, and clinical aspects of coccidioidomycosis.

An interesting study initiated by an officer at the Marine Corps Air Station, Mohave, Calif., established Mohave as an endemic area.[38] The endemicity of this desert region was confirmed by positive serologic specimens sent from adjacent Muroc Army Airfield.

Another outstanding instance of Navy medical interest and cooperation occurred at the United States Naval Hospital, Corona, Calif., where an officer of the Medical Service Corps of the United States Navy provided specimens and histories which showed that the endemic area in the San Joaquin Valley extended to include the Naval Air Station, Vernalis, Calif. Subsequent careful studies revealed that the Corona region itself was mildly endemic.

In the endemic area of Texas, investigations at William Beaumont General Hospital and Fort Bliss confirmed the fact that coccidioidomycosis had been locally acquired in the El Paso area. Investigation at Pyote Regional Hospital established the Pyote area of Texas as one of high endemicity. In discussing the surgical manifestations of coccidioidomycosis, Quill and Burch reviewed endemicity of coccidioidomycosis in Texas military installations.[39]

A study at Regional Hospital, Fort Ord, Calif., aided in delimiting adjacent endemic areas and in identifying coexisting tuberculosis and coccidioidomycosis.[40] Working closely with the Commission on Epidemiological Survey, staff members at Hammond General Hospital also carried on coccidioidin studies, sampling various personnel returned from overseas, including a considerable number from Australia. However, these investigations could locate no one who reacted positively to coccidioidin.[41] Members of the staff at Letterman General Hospital, San Francisco, Calif., took an active role in the investigation of cases, and staffs at the various service command laboratories, especially the Ninth at Monterey, Calif., the Second at New York, N. Y., and the Fourth at Fort McPherson, Ga., took a significant interest in the subject. The staffs of Rhoads General Hospital, N. Y., Percy Jones General Hospital, Mich., Moore General Hospital, N. C., and Baxter General Hospital, Wash., were very much alert to their problems of coccidioidomycosis.[42] Very often one could trace the location of medical officers aware of coccidioidomycosis by the reporting of specimens from patients having recognized coccidioidal

[38] Pfanner, E. F.: Coccidioidomycosis at the U. S. M. C. Air Station, Mohave, California. U. S. Nav. M. Bull. 46: 229–236, February 1946.

[39] Quill, L. M., and Burch, J. C.: Surgical Manifestations of Coccidioidomycosis. Ann. Surg. 120: 670–679, October 1944.

[40] Cherry, C. B., and Bartlett, A. G.: The Diagnosis of Acute *Coccidioides immitis* Infections. Bull. U. S. Army M. Dept. 5: 190–193, February 1946.

[41] (1) Denenholz, E. J., and Cheney, G.: Diagnosis and Treatment of Chronic Coccidioidomycosis. Arch. Int. Med. 74: 311–330, November 1944. (2) Cheney, G., and Denenholz, E. J.: Observations on the Coccidioidin Skin Test. Mil. Surgeon 96: 148–156, February 1945.

[42] (1) Rifkin, H., Feldman, D. J., Hawes, L. E., and Gordon, L. E.: Coexisting Tuberculosis and Coccidioidomycosis. Arch. Int. Med. 79: 381–390, April 1947. (2) Groover, M. E., Jr., Cleve, E. A., Bornstein, S., Rice, A. G., Galloway, A. F., and Macaluso, C. P.: Sensitivity of Skin to Histoplasmin in Differential Diagnosis of Pulmonary Disease. Arch. Int. Med. 80: 496–513, October 1947.

infections. This was evident in the assignments of Maj. Lewis T. Bullock, a pioneer worker on coccidioidomycosis at Lake Field, Ariz., in the Western Flying Training Command, as he moved back and forth across the country. Another example is in the assignments of Lt. Col. J. Murray Kinsman first coccidioidomycosis-control officer at Lemoore Army Airfield who moved first to Florida, then to the Schick General Hospital, Clinton, Iowa, to Walter Reed General Hospital, Washington, D. C., and finally to Fort Bragg, N. C., where at each station many cases of coccidioidomycosis were recognized coincidentally with his arrival.

A number of interesting diagnostic and epidemiologic problems, some of which were reported by Maj. Dumont Clark and J. H. Gilmore, were worked out by the staff of McCloskey General Hospital, Temple, Tex.[43] At LaGarde General Hospital, New Orleans, La., several cases of coccidioidomycosis in Negro troops from the Desert Training Center were recognized. The primary infections in these cases had not been recognized in California or in Arizona. Several cases of primary coccidioidomycosis in troops formerly in the Desert Training Center were discovered at Thayer General Hospital, Nashville, Tenn., and proved serologically.

While a complete listing of all the helpful investigations is not possible, special mention should be made of the contributions of staffs at three tuberculosis hospitals for Army personnel Fitzsimons, Bruns, and Moore General Hospitals. The histories accompanying specimens from Fitzsimons General Hospital, Denver, Colo., contributed to knowledge of distribution and pathogenesis of infection, especially of coccidioidal cavitation, as well as to laboratory methods.[44] The staff of Bruns General Hospital, Santa Fe, N. Mex., also took an active part in investigations. There, Capt. H. E. Bass focused attention on the problem of differentiation from tuberculosis,[45] and Capt. S. R. Rosenthal developed a theory of contagiousness of coccidioidomycosis based upon instilling pus or sputum into tracheas of guinea pigs.[46] The importance of coccidioidal residual pulmonary lesions in differentiation from tuberculosis was apparent also at Moore General Hospital. As an outgrowth of this interest, the group at Moore wrote an extensive paper on the results of skin testing.[47]

Both in the Office of the Surgeon General and in the field, there was a close liaison between clinical and preventive medicine personnel. Medical consultants for the Eighth and Ninth Service Commands were alert to problems of coccidioidomycosis and frequently stimulated the interest of medical groups unfamiliar with the infection. Col. Verne R. Mason, medical consultant to the

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[43] Clark, D., and Gilmore, J. H.: A Study of 100 Cases With A Positive Coccidioidin Skin Test. Ann. Int. Med. 24: 40–59, January 1946; correction Ibid. 24: 519, March 1946.

[44] Wilhelm, S.: Isolation of *Coccidioides immitis* from Sputum. Bull. U. S. Army M. Dept. 5: 468–473, April 1946.

[45] (1) Bass, H. E.: Coccidioidomycosis and Tuberculosis Diagnostic Problem. Tuberculology 7: 72–75, January 1945. (2) Bass, H. E., Schomer, A., and Berke, R.: Coccidioidomycosis: Persistence of Residual Pulmonary Lesions. Arch. Int. Med. 82: 519–528, December 1948. (3) Bass, H. E., Schomer, A., and Berke, R.: Question of Contagion in Coccidioidomycosis, a Study of Contacts. Am. Rev. Tuberc. 59: 632–635, June 1949.

[46] (1) Rosenthal, S. R., and Routien, J. B.: The Infectiousness of Coccidioidomycosis. Science 104: 479, 22 Nov. 1946. (2) Rosenthal, S. R., and Routien, J. B.: Contagiousness of Coccidioidomycosis, An Experimental Study. Arch. Int. Med. 80: 343–357, September 1947.

[47] See footnote 42 (2), p. 300.

Surgeon, Ninth Service Command, made an extensive review of the problem of coccidioidal pulmonary cavitation.[48]

Even from overseas, requests for consultative service and diagnostic facilities were received by the Commission. It was hoped that evidence of other endemic areas would be obtained, but this was never realized. Coccidioidin was distributed widely to Europe, North Africa, the Near East, India, Australia, China, the South Pacific, the Philippines, Hawaii, and North and South America, and, while some serum specimens were sent in from these scattered areas, the only positive specimens found were in the military personnel who had been infested in the arid Southwestern United States prior to overseas assignment. Since World War II, the continued activities of the Armed Forces Epidemiological Board have disclosed two new arid endemic areas: Lara, Venezuela, and the Paraguayan Chaco.[49]

While widespread collaboration continued throughout all medical branches of the services, the greatest amount of epidemiologic and practical data was assembled through the integrated studies of the Western Flying Training Command. In addition to the studies of Sweigert, Clark, and others, Maj. H. W. Jamison and Maj. J. R. Colburn were among those who made outstanding studies of roentgenographic lesions.[50] In the Commission on Epidemiological Survey studies of coccidioidomycosis at the four basic training fields of the San Joaquin Valley, everyone from the succession of surgeons to the enlisted personnel participated enthusiastically. These participants are indicated in the Commission's first published article on seasonal distribution and dust control.[51]

CONTROL MEASURES

The control of coccidioidomycosis by means of recognition of the primary infection and proper care of the patient has been emphasized throughout this discussion. Some of the lessons learned with respect to the significance and limitations of the skin test and serologic tests will be indicated later (p. 303). Certainly, in any evaluation of control measures, one must be certain of diagnosis, which can be achieved only by laboratory methods, including the skin test.

Control through more accurate knowledge of the geographic distribution of endemic areas aids in diagnosis by raising the level of suspicion. Mere travel through an endemic area may provide the exposure. From the military standpoint, knowledge of endemic areas is even more important in providing

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[48] Letter, Lt. Col. V. R. Mason, MC, to Commanding General, Army Service Forces. Attention The Surgeon General, 7 Oct. 1943, subject: Report on Patients With Pulmonary Cavities Following Coccidioidomycosis.

[49] (1) Campins, H., Schary, M., and Gluck, V.: Coccidioidomicosis (Enfermedad de Posadas) Su Comprobacion en Venezuela. Arch. venezol. patol. trop. y parasit. med. 1: 1-20, October 1949. (2) Gomez, R. F.: Endemism of Coccidioidomycosis in the Paraguayan Chaco. California Med. 73: 35-38, July 1950.

[50] (1) Jamison, H. W.: A Roentgen Study of Chronic Pulmonary Coccidioidomycosis. Am. J. Roentgenol. 55: 396-412, April 1946. (2) Jamison, H. W., and Carter, R. A.: The Roentgen Findings in Early Coccidioidomycosis. Radiology 48: 323-332, April 1947. (3) Colburn, J. R.: Roentgenological Types of Pulmonary Lesions in Primary Coccidioidomycosis. Am. J. Roentgenol. 51: 1-8, January 1944. (4) See footnote 16, p. 292.

[51] See footnote 9 (1), p. 289.

information on where not to establish large camps or conduct maneuvers unless climatic and topographic advantages outweigh the hazards of coccidioidomycosis. Here, however, additional special knowledge with respect to the infection spectrum, racial susceptibility, and infection rate is of importance. For instance, an installation which can be stabilized and which contains white personnel would not be a serious problem even in an area of high incidence because everyone would soon be infected and, if properly cared for, would recover without many serious complications and would subsequently be immune. However, transient personnel in such an area could present serious problems during the coccidioidomycosis season. Serious consideration should be given to bringing Negro troops into an area of high endemicity because, as it will be pointed out (p. 307), dissemination occurs in 10 percent of those clinically ill and in over 2 percent of all those infected.

The importance of dust control became increasingly apparent during the course of World War II, although application of dust-control measures was limited. Of no value during maneuvers or with ground forces in training, dust control proved very valuable in reducing rates of coccidioidomycosis at airfields. There, in permanent installations, the requisite steps could be undertaken. Dust control itself constituted a research problem because it was necessary to determine monthly infection rates in order to evaluate the effect of dust control. This control method will be discussed, therefore, in the following section on research.

RESEARCH DURING WORLD WAR II

Methods of Diagnosis

Coccidioidin.—First in importance in research accomplishments are the combined field and laboratory studies which permitted the widespread use of coccidioidin. The use of coccidioidin is the first step in diagnosis, vital to the clinical care of the patient; in epidemiologic investigations; and indirectly in control of the environment. Coccidioidin was prepared by the group at Stanford University as an activity of the Commission on Epidemiological Survey and was distributed as a service. The data accumulated from reports on its use, however, were ample reward for this effort. The active principle of coccidioidin was shown to be very stable, withstanding autoclaving and remaining potent for years even when diluted. The reaction was of the bacterial type, not a passive transfer of sensitivity. As prepared on asparagin synthetic medium, coccidioidin was not significantly antigenic. Since it did not evoke humoral antibodies, it could be used without complicating subsequent serologic tests. It did not aggravate a quiescent infection, and, even in very sensitive patients, except for local discomfort and slight fever and malaise, the only complication encountered was occasional erythema nodosum. Sensitivity was always demonstrable in patients with uncomplicated primary infection. Although dermal sensitivity appeared earlier than diagnostic precipitins or

complement-fixing antibodies, it was absent in one-sixth of all patients during the first week of illness. However, with the use of 1 : 100 dilution of coccidioidin, dermal sensitivity always appeared by the end of the third week. Sweigert and Willett found that sensitivity to 1 : 1 000 coccidioidin was sometimes slightly slower.[52] The degree of sensitivity was shown both by Beare and by the Commission on Epidemiological Survey to increase during the first 3 weeks of illness.[53] These two studies also confirmed previously observed association of erythema nodosum with maximal sensitivity; the sensitivity to coccidioidin was found to be quite durable, even in one group tested outside the endemic area. However, Cheney and Denenholz, as a result of tests in an adjacent nonendemic area, believed that sensitivity was less tenacious.[54] Certainly sensitivity was frequently lost during dissemination of the infection, and a definite association was noted between sensitivity and favorable prognosis.

When stronger coccidioidin was used, cross-reactions were observed. Indeed, care had to be taken to standardize coccidioidin both for specificity and potency. The coccidioidin did not cross-react with bacteria or viruses, but several investigators found definite positive reactions to it occurring in nonendemic areas in the Middle West.[55] Emmons and Ashburn called attention to apparent cross-reaction with *Haplosporangium parvum*.[56] As the result of positive reactions seen in new arrivals at the San Joaquin Valley airfields, an association was suggested in 1943 between these cross-reactions, histoplasmosis, and pulmonary calcification in those persons negative to tuberculin. Two years later, as the result of the reports of Christie and Peterson and of Palmer, extensive studies of histoplasmin sensitivity and pulmonary calcifications were inaugurated and were continued after World War II.[57] The Commission on Epidemiological Survey summarized their experiences in the *American Journal of Public Health*.[58] It was found that histoplasmin produced a much higher proportion of cross-reactions in those persons who had had coccidioidomycosis than coccidioidin produced in those with dominant sensitivity to histoplasmin. Indeed, if histoplasmin was too concentrated, it evoked larger reactions than did coccidioidin during a course of clinical coccidioidomycosis. Used in "balance," however, the tests could be interpreted without difficulty.

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[52] See footnotes 16 and 17, p. 292.

[53] (1) Beare, W. K.: Primary Pulmonary Coccidioidomycosis. Air Surgeon's Bull. 2: 397-399, November 1945. (2) See footnote 10, p. 292.

[54] See footnote 41 (2), p. 282.

[55] Smith, C. E.: Coccidioidomycosis. M. Clin. North America 27: 790-807, May 1943.

[56] Emmons, C. W., and Ashburn, L. L.: The Isolation of *Haplosporangium parvum* n. sp. and *Coccidioides immitis* from Wild Rodents; Their Relationship to Coccidioidomycosis. Pub. Health Rep. 57: 1715-1727, 1 Nov. 1942.

[57] (1) Christie, A., and Peterson, J. C.: Pulmonary Calcification in Negative Reactors to Tuberculin. Am. J. Pub. Health 35: 1131-1147, November 1945. (2) Christie, A., and Peterson, J. C.: Pulmonary Calcification and Sensitivity to Histoplasmin, Tuberculin and Haplosporangium. J. A. M. A. 131: 658-660, 22 June 1946. (3) Christie, A., and Peterson, J. C.: Histoplasmin Sensitivity. J. Pediat. 29: 417-432, October 1946. (4) Palmer, C. E.: Nontuberculous Pulmonary Calcification and Sensitivity to Histoplasmin. Pub. Health Rep. 60: 513-520, 11 May 1945. (5) Palmer, C. E.: Geographic Differences In Sensitivity to Histoplasmin Among Student Nurses. Pub. Health Rep. 61: 475-487, 5 Apr. 1946.

[58] Smith, C. E., Saito, M. T., Beard, R. R., Rosenberger, H. G., and Whiting, E. G.: Histoplasmin Sensitivity and Coccidioidal Infection. I. Occurrence of Cross-Reactions. Am. J. Pub. Health 39: 722-736, June 1949.

Serologic tests. Simultaneous precipitin and quantitative complement-fixation tests provided as service functions by the Commission on Epidemiological Survey were extremely important in the diagnosis and treatment of coccidioidal infections. Again, the data obtained from reports by investigators permitted an evaluation of the procedures. These tests indicated specificity with respect to viral, rickettsial, spirochetal, bacterial, and most other mycotic infections.[9] There was evidence of irregular cross-reaction in histoplasmosis. The combination of the two tests detected over 90 percent of clinically manifest, nondisseminating primary infection but less than 10 percent of asymptomatic infections. It confirmed three-fifths of coccidioidal pulmonary cavities and 99 percent of disseminating infections. Precipitins only were demonstrated in 44 percent, and complement fixation alone in 22 percent of positive serums from those with uncomplicated primary infections. The tests were positive only after allergy was established unless the infection was disseminating and the patient was anergic. Thus, coccidioidin was shown to be a useful screen. Precipitin tests were more useful early in the course of infection; complement-fixation tests were more useful later. Precipitins were established within the first month of illness, although within the first week only one-half of them had converted from negative to positive; in most of the serums, antibody content had reverted from positive to negative within 3 months. Complement-fixation tests converted for 3 months, while reversions were slower. Even after uncomplicated primary infections, positive tests sometimes persisted for years. *Regarding prognosis*, it was observed that the titer of complement fixation rose with severity of infection. Less than 1 in 40 patients with nondisseminating coccidioidal disease had maximal complement-fixing titers in excess of 1 in 16, but more than one-half of the patients with disseminating disease exceeded that level. While patients with solitary extrapulmonary lesions had findings comparable to those with nondisseminating disease, titers exceeded 1 in 16 in 95 percent of the patients with extensive fatal lesions. Precipitins and complement-fixing antibodies were demonstrable in ascitic and pleural fluid due to coccidioidomycosis. Complement was fixed in three-quarters of the patients with coccidioidal meningitis and, if present, it was diagnostic of coccidioidal etiology.

Recognition of Endemic Areas

Knowledge of the endemic areas is of great importance in recognizing clinical coccidioidal infection and in planning its control. Prior to World War II, the endemic area in the United States was not clearly defined. It was believed to center in the southern San Joaquin Valley of California, while its northern limit was uncertain. On the basis of some cases reported from southern California, this area was suspect.[60] Just before the war, isolated

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[9] See footnote 11, p. 280.

[60] Kessel, J. F. Recent Observations on Coccidioides Infection. Am. J. Trop. Med. 21: 447–453, May 1941.

cases were reported from Tucson [61] and Phoenix, Ariz.,[62] while the regions around El Paso and San Antonio, Tex., had been established as other endemic areas by a few reported cases.[63] Northern Mexico was suspect, while the Chaco region of Argentina was the only other such area known.[64]

By means of extensive coccidioidin tests and from records of clinical cases (proved serologically or by recovery of the fungus) in which the known incubation period fixed the site where the infection was acquired, the endemic areas became much more accurately delimited. The studies at the San Joaquin Valley airfields showed that the incidence in that valley was maximal in the south. Further north, at Merced and Modesto, it was very spotty. Along the west side of the valley, incidence extended further north, nearly to Tracy, and over the Coast Range, as Shelton demonstrated at Camp Roberts, into Monterey and San Luis Obispo counties. Skin tests and recognized cases also implicated contiguous Santa Barbara, Ventura, and Los Angeles counties. Spotty incidence in Riverside and San Bernardino counties was also recognized, and the region around San Diego was reconfirmed as mildly endemic. Imperial County was uncertain but "probable." The experiences of Army Air Forces Western Flying Training Command,[65] the Army Ground Forces in the California-Arizona Maneuver Area, and the prisoner-of-war camp at Florence proved that southern and central Arizona were the most highly infected of all the areas in the United States. The studies in the Western Flying Training Command also indicated that there were infected areas in the southern tip of Nevada and southwest Utah, as well as in southern New Mexico. The region of Albuquerque and Sante Fe, N. M., was free of infection. In Texas, the endemic area was not proved to extend as far north as Wichita Falls, but it did extend south along the Rio Grande to the Mission-McAllen region. No other endemic areas were found in the continental United States.

Coccidioidin tests of Italian and German prisoners of war captured in North Africa were uniformly negative, as were tests of natives in Arabia. However, shortly after the war, the work of the Commission on Epidemiological Survey aided in discovering two other endemic areas in the arid province of Lara, Venezuela, and the Paraguayan Chaco. Thus, all known endemic areas are arid.

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[61] (1) Woolley, M. T.: Mycological Findings in Sputum. J. Lab. & Clin. Med. 23: 553-565, March 1938. (2) Farness, O. J., and Mills, C. W.: Coccidioides Infection: A Case of Primary Infection in the Lung with Cavity Formation and Healing. Am. Rev. Tuberc. 39: 266-273, February 1939. (3) Storts, B. P.: Coccidiodal Granuloma Simulating Brain Tumor in a Child of Four Years. J. A. M. A. 112: 1334-1335, 8 Apr. 1939.

[62] (1) Phillips, E. W.: Presence of Coccidioidal Infection in Phoenix. Southwestern Med. 23: 48-51, February 1939. (2) Brown, O. H.: Coccidioides Infection in Arizona—Allergic Factors in Nodules? Southwestern Med. 23: 131-132, April 1939.

[63] (1) Caldwell, G. T.: Coccidioidal Granuloma, A Report of Three Cases Recognized in Texas. Texas State J. Med. 28: 327-333, September 1932. (2) Smith, L. M.: Coccidioidal Granuloma; Report of a Case Originating in Western Texas. Arch. Dermat. & Syph. 28: 175-181, August 1933. (3) Smith, L. M., and Waite, W. W.: Coccidioidal Granuloma; Report of a Fatal Case. Southwestern Med. 18: 305, September 1934. (4) Lehmann, C. F., and Pipkin, J. L.: Coccidioidal Granuloma (Chronic Hypertrophic). Arch. Dermat. & Syph. 31: 586-589, April 1935. (5) Foley, M. P., Love, T. F., Broders, A. C., and Heilman, F. R.: Coccidioidal Granuloma; Report of a Case Originating in Texas. West. J. Surg. 48: 738-741, December 1940.

[64] See footnote 1 (1) and (2), p. 285.

[65] Lee, R. V.: Coccidioidomycosis in the Western Flying Training Command. California & West. Med. 61: 113-134, September 1944.

Pathogenesis of Coccidioidomycosis

One of the primary purposes of the intensive study of the San Joaquin Valley Army airfields was to ascertain the frequency of inapparent infection, clinical disease, and especially dissemination. All permanent party personnel were tested with coccidioidin soon after arrival at a field. Initially, there was great interest in ascertaining the number of coccidioidin reactors who might develop manifestations of the disease. None did, nor were any such cases reported in the entire Western Flying Training Command. Apparently, second attacks of primary infection are very rare. Every 6 months, those personnel previously negative to coccidioidin were retested. The proportions of various types of coccidioidomycosis were revealed.[66] Of 1,351 infections, 60 percent were asymptomatic and only 25 percent were manifest clinically and had been diagnosed (table 50). Erythema nodosum occurred in 24 percent of infected white females, in 4.3 percent of infected white males, but very rarely in Negroes (table 51). This form of the disease can serve as a useful index of infection. Dissemination of the organism occurred in about 1 percent of clinically manifest infections in white males and in 0.25 percent of all their infections; whereas disseminated infections occurred over 10 times as frequently in Negroes despite the fact that Negroes had the same housing, food, and medical care as the white males.

TABLE 50.—*Distribution of primary coccidioidal infections at Minter, Gardner, and Lemoore Fields*

Field	Diagnosed cases			Missed and subclinical infections			Inapparent infections		Total number of infections
	Number	Percent of total infections		Number	Percent of total infections		Number	Percent of total infections	
		Total	With symptoms		Total	With symptoms			
Minter [1]	171	26.5	59.6	116	18.0	40.4	359	55.5	646
Gardner [2]	108	24.9	64.3	60	13.9	35.7	265	61.2	433
Lemoore [3]	53	19.5	58.2	38	14.0	41.8	181	66.5	272
Total	332	24.6	60.8	214	15.8	39.2	805	59.6	1,351

[1] For period 1941–45.
[2] For period 1941–44.
[3] For period 1942–45.

The study by Willett and Weiss (p. 292) of the infections at Banning indicated 8 percent disseminations among Negroes with clinical illness. For the entire Western Flying Training Command, Lee and Jamison suspected an even higher risk of dissemination in the Negro.[67]

[66] See footnote 9 (2), p. 289.
[67] See footnotes 65, p. 306, and 50 (1), p. 302.

TABLE 51.—*Distribution of primary coccidioidal infections in white personnel at Minter, Gardner, and Lemoore Fields, 1 July 1941 to 31 December 1945*

Sex	Diagnosed cases			Missed and subclinical infections			Inapparent infections		Total number of infections	Coccidioidal erythema nodosum			
	Number	Percent of total infections		Number	Percent of total infections		Number	Percent of total infections		Number	Percent of total infections		
		Total	With symptoms		Total	With symptoms					Total	Diagnosed cases	With symptoms
Females	20	48. 8	80. 0	5	12. 2	20. 0	16	39. 0	41	10	24. 4	50. 0	40. 0
Males	285	23. 4	58. 5	202	16. 6	41. 5	731	60. 0	1, 218	52	4. 3	18. 2	10. 7
Total	305	24. 2	59. 6	207	16. 4	40. 4	747	59. 4	1, 259	62	4. 9	20. 3	12. 1

These findings reveal a grave hazard in exposing Negroes to coccidioidal environment, a point to be borne in mind in future military planning. However, these studies also indicate that among white troops it should be possible to maintain a stabilized command in a coccidioidal endemic area without too grave a risk. The value of periodic coccidioidin testing is apparent, since a conversion constitutes the most sensitive diagnostic procedure. Moreover, once the person is positive, the doctors need not worry that an attack of influenza or pneumonia is coccidioidomycosis.

Special mention may be made of coccidioidal pulmonary cavitation. In the Army experience, it occurred as follows: In 2 percent of 753 hospitalized cases in the San Joaquin Valley airfields, in 4 percent of Goldstein and Louie's cases and in 4 percent of Colburn's 75 cases from Camp San Luis Obispo, in 6 percent of Willett and Weiss' 100 March Field cases, and in 8 percent of Sweigert, Turner, and Gillespie's 77 cases from Davis-Monthan Field. These cavities were seen to be a complication of the primary infection and not comparable to disseminating coccidioidal disease. The relatively benign nature of these cavities was discussed in an article by Smith, Beard, and Saito who pointed out that among 153 military patients with such cavities, three-fifths were diagnosed incidentally and two-fifths had symptoms; among civilians, only one-quarter were asymptomatic.[68] This study on the pathogenesis of coccidioidal cavities and Bass' studies also were evidence against the theory of contagion which Rosenthal advanced after he infected guinea pigs by instilling coccidioidal pus down their tracheas (p. 301).

Three additional military papers dealt with pathogenesis of coccidioidomycosis. Kunstadter and Pendergrass, at Ashford General Hospital, White Sulphur Springs, W. Va., presented evidence of asymptomatic pulmonary lesions, including observations of an anatomical specimen from a soldier on whom an autopsy was performed for accidental death.[69] Schlumberger summarized 13 cases of coccidioidal meningitis from material of the Army Institute of Pathology.[70] The most ambitious study and one supported by the Commission on Epidemiological Survey was that of Forbus and Bestebreurtje.[71] It contains a comprehensive illustrated discussion of the pathogenesis based on specimens from 95 patients with disseminated coccidioidomycosis. Certain of the conclusions with respect to geographic distribution were taken from protocols which were incomplete; thus, Oregon and Missouri were listed as endemic areas. Also, based upon the continuing endogenous reinfections of immunologic defectives, these investigators deduced that disseminations would continue to develop in veterans for 10 years and would cause a number of deaths. Fortunately, these apprehensions are not being borne out.

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[68] See footnote 12 (1), p. 290.

[69] Kunstadter, R. H., and Pendergrass, R. C.: Primary Coccidioidomycosis; A Possible Pediatric Problem. J. A. M. A. 127: 624–627, 17 Mar. 1945.

[70] Schlumberger, H. G.: A Fatal Case of Cerebral Coccidioidomycosis With Cultural Studies. Am. J. M. Sc. 209: 483–495, April 1945.

[71] Forbus, W. D., and Bestebreurtje, A. M.: Coccidioidomycosis: A Study of 95 Cases of the Disseminated Type With Special Reference to the Pathogenesis of the Disease. Mil. Surgeon 99: 653–719, November 1946.

Infection Rates and Seasonal Distribution

The intensive study of the San Joaquin Valley airfields provided an opportunity for determining monthly infection rates at those stations. Clinically recognizable infections were listed according to the months in which they occurred, and inapparent infections were distributed proportionately. The susceptibles exposed each month were known, and infection rates were calculated. The sum of the monthly infection rates gave the annual infection rate.[72] Throughout the study, Merced remained remarkably free of infection and virtually served as a control.

Monthly infection rates of Minter, Gardner, and Lemoore fields which were located in the endemic areas are presented in table 52. Annual rates for these three fields as well as for Merced are presented in table 53. At each of the three fields in the endemic area, the initial season was the worst. As will be noted (p. 313), the presence of excessive dust was associated with each of the new fields during the initial seasons. Tables 52 and 53 also show that

TABLE 52.—*Monthly incidence of coccidioidomycosis at San Joaquin Valley Army airfields, and monthly precipitation, 1941–45*

Date	Precipitation [1] (inches)	Disease rate [2]	Infection rate [3]		
			Minter	Gardner	Lemoore
1941					
July	0	0. 10	3. 17	1. 08	0
August	Trace	. 41	5. 12	1. 89	0
September	0	. 55	4. 47	2. 39	0
October	. 53	. 45	3. 75	4. 60	0
November	. 49	. 17	2. 47	3. 44	0
December	1. 54	0	. 38	2. 22	0
1942					
January	. 47	. 03	. 56	2. 06	0
February	. 19	0	. 38	0	0
March	. 60	. 07	. 88	. 63	. 40
April	1. 03	. 06	1. 17	. 29	. 23
May	. 19	. 15	1. 04	. 37	. 53
June	0	. 17	1. 71	2. 02	1. 27
July	0	. 34	2. 32	2. 06	1. 24
August	. 01	. 42	2. 93	2. 35	1. 55
September	0	. 41	2. 84	1. 85	2. 24
October	. 24	. 29	1. 14	1. 03	1. 70
November	. 20	. 11	. 58	1. 38	. 73
December	1. 33	. 10	1. 39	1. 00	. 84

See footnotes at end of table.

[72] This method of computing the annual rate is not the usual procedure followed by the Medical Statistics Division, Office of the Surgeon General, since equal weight is given to each month regardless of the amount of variation in the number of susceptibles from month to month. The following method is generally used: Annual rate per 100 susceptibles equals the total infections during the year multiplied by 100 divided by the average number of susceptibles during the year. In this chapter, the data are presented as they were originally calculated by the author.—E. C. H.

TABLE 52.—*Monthly incidence of coccidioidomycosis at San Joaquin Valley Army airfields, and monthly precipitation, 1941–45*—Continued

Date	Precipitation [1] (inches)	Disease rate [2]	Infection rate [3]		
			Minter	Gardner	Lemoore
1943					
January	2. 87	0. 02	0. 22	0. 87	1. 12
February	1. 55	. 04	. 07	. 39	. 58
March	. 80	0	. 13	. 15	0
April	2. 39	0	. 19	0	. 08
May	. 25	0	. 18	. 37	. 41
June	0	. 23	1. 52	. 12	. 16
July	Trace	. 35	. 81	. 57	. 47
August	0	. 43	1. 45	3. 09	. 39
September	0	. 87	4. 10	1. 86	. 69
October	. 05	. 81	2. 85	3. 04	. 75
November	. 09	. 43	1. 49	1. 46	1. 04
December	1. 58	. 28	. 53	. 62	1. 17
1944					
January	. 71	. 05	. 34	. 31	. 50
February	1. 18	. 06	. 22	. 20	. 55
March	. 76	. 10	. 86	. 65	0
April	. 63	. 10	. 80	. 46	. 75
May	. 23	. 21	. 88	. 65	. 59
June	. 13	. 15	1. 28	. 67	. 21
July	0	. 24	. 89	1. 60	. 35
August	0	. 22	1. 15	1. 69	. 16
September	0	. 20	1. 05	1. 82	. 14
October	. 14	. 39	1. 96	1. 27	. 28
November	1. 70	. 15	. 54	. 64	. 27
December	. 60	. 03	. 08	0	. 13
1945					
January	. 82	0	0	0	0
February	1. 91	0	0	0	. 24
March	1. 15	0	0	0	0
April	. 64	. 09	. 77	0	0
May	. 26	. 22	1. 29	0	0
June	. 14	. 12	. 60	0	0
July	0	. 29	1. 62	0	0
August	Trace	. 41	3. 20	0	0
September	. 07	. 25	1. 65	0	0
October	. 58	. 14	. 71	0	0
November	. 28	. 18	. 79	0	0
December	1. 48	. 07	. 38	0	0

[1] Precipitation data from United States Department of Commerce Weather Station, Kern County Airport, Bakersfield, Calif.

[2] Expressed as number of cases per month per 1,000 average strength (Minter).

[3] Expressed as number infected per month per 100 susceptible men.

greatest incidence of the disease occurred in seasons preceded by heavy rainfall in the winter and spring. Lemoore had much lower rates than Minter and Gardner fields to its south. From the infection rates, one can readily understand why nearly all longtime residents of the San Joaquin Valley react to coccidioidin. However, the incidence, even during the initial year at Minter when one-quarter of the susceptibles were infected, was far below that in Arizona where, in the region of Florence and Williams Field, one-half of the susceptibles were infected within 6 months.

The monthly infection rates rose and declined, according to the rainfall (table 52). The peak occurred in the dusty summer and fall, and the rate continued to be high until the winter rains appeared. While the effect of heavy winter rainfall in providing abundant infective chlamydospores during the ensuing summer is hypothetic, the immediate beneficial effect of the rain in keeping the chlamydospores from blowing around in the air is obvious.

TABLE 53. *Annual coccidioidal rates at Army airfields in the San Joaquin Valley and precipitation at Bakersfield, Calif., 1941–45*

[Disease rate expressed as number of cases per annum per 1,000 field strength. Infection rate expressed as number infected per 100 susceptible men]

Year	Precipitation during previous rainy season (inches)	Disease rate [1] at Minter	Infection rate [1] at Minter	Infection rate at		
				Gardner [2]	Lemoore [3]	Merced [4]
1941–42	10.96	2.16	25.10	20.99		
1942–43	5.04	1.81	13.51	11.57	12.43	0.63
1943–44	9.64	3.89	15.61	13.58	6.21	.36
1944–45	5.36	1.68	8.43	7.02	2.86	1.40
1945	7.36	1.34	8.35	----	1.43	----

[1] Disease and infection rates were for the period 1 July–30 June in 1941–42, 1942–43, 1943–44, and 1944–45; in 1945, they were for the period July–December.

[2] Rates were for the period 1 July–30 June in 1941–42, 1942–43, 1943–44; in 1944–45, the rate was for the period July–November.

[3] Rates were for the period 1 March–28 February in 1942–43, 1943–44, and 1944–45; in 1945, the rate was for the period March–September.

[4] Rates were for the period 1 February–31 January, 1942 to 1945, inclusive.

Dust Control

Appreciation of the importance of dust control at the permanent installations in the endemic areas came from all directions. At the 1944 annual meeting of the Army Epidemiological Board, the advisability of environmental (dust) control of coccidioidomycosis was discussed. Dr. Blake, as chairman, and General Bayne-Jones, representing The Surgeon General, requested the Commission on Epidemiological Survey to direct attention to the problem.[73]

[73] (1) Letter, Brig. Gen. S. Bayne-Jones to Dr. C. E. Smith, 7 May 1944. (2) Letter, Dr. C. E. Smith to Brig. Gen. S. Bayne-Jones, 22 May 1944.

As a matter of fact, this type of control was already being planned, but the directive from the Preventive Medicine Service, Office of the Surgeon General, greatly strengthened the Commission's efforts. Dust control was also supported by the Air Surgeon. On the individual fields, the commandants, post surgeons, post engineers, and coccidioidomycosis-control officers were well aware of its advisability. Originally considering dust control only as a procedure to protect engines from being ruined, the Pacific Division, United States Army Engineers, located at San Francisco, became very much interested in dust control in preventing coccidioidomycosis. Maj. Howard B. Sprague, Army Air Forces liaison officer to that Division, made a very complete review of the dust problem at Gardner, Lemoore, Davis-Monthan, Luke, and Marana Fields.[74] Particularly striking was his comparison of coccidioidomycosis at Davis-Monthan and Marana. Both fields were located near Tucson, Ariz., and their incidence of coccidioidomycosis was parallel until Davis-Monthan organized systematic dust control. Then the incidence of coccidioidomycosis at Davis-Monthan fell sharply while that of Marana continued unchanged.

In the fall of 1941, there were vast earth scars where Minter and Gardner Fields were being built. As there was no dust control in operation, the locally generated dust billowed in clouds over the areas. The highest coccidioidal infection rates of the study occurred during this season of maximal dust at these two fields (table 53). The following year (1942–43), during which Lemoore Field was opened, was a season of relatively low incidence rates at all three fields. However, in the second year at Lemoore (1943–44), the incidence rate was only one-half that of the preceding years while it had increased at both Minter and Gardner. The improvement in local dust control at Lemoore had more than offset the more favorable conditions for *Coccidioides*.

The measures largely responsible for local dust control up to the 1944 season had been grassing and construction of surfaced roads. After the relatively bad year of 1943–44, the surgeons, coccidioidomycosis control officers, commanding officers, and post engineers at Minter and Gardner Fields worked diligently to effect additional improvements. The principal areas left without turf, and those which therefore presented problems, were the areas used for calisthenics, baseball, volleyball, and other forms of physical training. Some of these areas were surfaced with asphalt which was not completely satisfactory as it is both expensive and hard on the feet in hot weather. A crushed rock or gravel surface causes injuries and was therefore not used. The decision was made to try oiling these areas. Crude oil was considered but was rejected as it forms a crust and breaks into lumps as well as stains clothing and irritates the skin. Highly refined oils were finally selected for use in dust control because of their availability and because it was found that they are nonirritating, that they penetrate the soil, and that, even if the treated soil breaks

[74] Memorandum, Maj. H. B. Sprague, Army Air Forces Liaison Office, Pacific Division, United States Army Engineers, for Lt. Col. L. B. McCloud, Army Air Force Headquarters, 1 Oct. 1945, subject: Summary of the Relation Between Occurrence of Dust and Incidence of Coccidioidomycosis (Valley Fever) at Army Air Fields in California and Arizona.

into dust, the oil-impregnated particles fall back to the ground instead of being blown into clouds and dispersed.

In the spring of 1944, a highly refined oil was applied to the athletic areas at both Minter and Gardner Fields. Despite two applications, the procedure was a failure at Gardner. This field had been built on the very fine silt of an old lake bottom and because of the character of the soil the oil merely formed a crust. When the crust broke, the dust was exposed. At Minter Field, however, the soil was heavier and contained more clay, so that the application of the oil was more successful. Preliminary tests conducted on volleyball courts determined optimal dosage to be one quart per square yard. When oil was applied in late May and early June, dust was fairly well controlled until the fall. In October, the effect of the oil was diminished, but fortunately late October rains put an end to the dusty season. In 1945, two applications were necessary at Minter Field. Just after the first application in May, unseasonable rains appeared and drove in the oil. By August, the areas became dusty, and, upon a sudden increase in number of cases of coccidioidomycosis, it was decided to reapply oil in September, after which there was a prompt decline in infections.

A comparison of the infection rates of Minter and Gardner Fields in the fall of 1944 indicated that Minter had benefited by the oiling. Infection rates for the 6 months from 1 June to 30 November 1944 were 6.87 per 100 susceptibles at Minter and 7.62 at Gardner. While the difference between these rates is not great, this was the first time that the Minter rate had been less than the Gardner rate. Rates for infections for the same period during the preceding year were 12.22 at Minter and 10.13 at Gardner. In 1943, the Minter infection rate exceeded the Gardner rate in 4 of the 6 months. In 1944, the Gardner rate exceeded the Minter rate in 4 of the 6 months. Moreover, the only months when the 1944 Minter rates exceeded the Gardner rates were June and October when the effectiv ness of the oiling had been diminished at the former field. Rates during July, August, and September at Gardner Field all exceeded rates for October at that field, while the October Minter rate was almost as high as any two of the preceding months combined.

Another item of evidence supports the argument that dust control is effective in reducing the incidence of coccidioidomycosis. In the summer and fall of 1943, enlisted personnel of the medical detachment at Minter Field suffered 15 coccidioidal infections during a 6-month period. The number of susceptibles was an even 100, so the infection rate was 15 per 100 men. Athletic fields were behind the station hospital, and the barracks of the enlisted personnel were directly at the edge of the athletic areas. In the summer and fall of 1944 and after initiation of dust-control procedures, in the same period of time, only two infections were acquired. The comparable 1944 infection rate was 5 per 100 men. Admittedly, the number are small, but the trend coincided with the rest of the evidence favoring dust control.

Interesting comparison in rates can be made for 1945. The Minter Field strength was three times that of a prisoner-of-war group which was performing agricultural labor in the vicinity, yet as many prisoners were being hospitalized

for coccidioidomycosis per day as were Minter Field personnel per month. Within 2 months and 10 days, June to August, 22 Minter Field personnel were hospitalized for coccidioidomycosis while 150 prisoners of war were hospitalized for the same disease. Dust was well controlled at Minter Field, where the airfield personnel were stationed, but the prisoners were harvesting potatoes and sugar beets in maximal dust nearby.

In summary, local dust-control measures certainly seem warranted in preventing coccidioidomycosis. Of course, definite knowledge as to where the infections have been acquired is necessary. For example, local dust control at the base area of Camp Roberts or at March Field proper was not indicated, since infections of personnel at these two installations were acquired on bivouacs. Where the installation itself is probably infected, dust control should be employed to the fullest extent possible. Whenever feasible, grassing should be used. Surfacing of roads and, to a limited degree, athletic areas is important. Many athletic areas cannot be surfaced because of excessive heat and the danger of injuries after falls. If turf cannot be maintained on such areas, a highly refined oil may be tried. However, the character of the soil should first be examined. If the soil is a fine, loose silt—like that of Gardner Field—success is unlikely. If it is a heavy, adobe type of soil, the outlook is favorable. In any event, preliminary tests should be run to prove effectiveness, to decide the product to be used, and to fix the quantity to be applied. Volleyball courts are especially suitable for such tests. In carrying out these dust-control procedures, a close liaison between the post engineer, the surgeon and his staff, and the commanding officer is very important. Also, the engineers responsible for dust control in the respective service commands should be consulted, and their advice should be heeded. In addition, experience has shown that in order to avoid exposure of personnel to dust in endemic areas, every effort should be made to develop an aquatic physical training program during the hot months which correspond to the season of high incidence of coccidioidomycosis.

SUMMARY

The leadership that was undertaken by The Surgeon General of the Army and his preventive medicine representatives in safeguarding the health of United States troops was well portrayed in the experience of the armed services with coccidioidomycosis in World War II. The Preventive Medicine Service of the Medical Department aided in the provision of vital diagnostic and service facilities; through the Army Epidemiological Board, it fostered both fundamental and applied research, including environmental control, and throughout the war it maintained constant vigilance in order to minimize exposures in endemic areas. The Army Air Force demonstrated a kindred interest. Thus, there resulted a fine collaboration which developed a successful control program and performed extensive and rewarding research. The Army Ground Force was not as alert or responsive; its control programs and

research were minimal and very largely dependent upon the unsupported enthusiasm and initiative of individual medical officers. The increase in medical knowledge as the result of the influx of the physicians of the Army of the United States into endemic areas of coccidioidomycosis is especially notable. Intrigued by this "new" infection, most medical officers contributed wholeheartedly to programs of control and research.

Part III

DISEASES TRANSMITTED CHIEFLY THROUGH ALIMENTARY TRACT

CHAPTER XVII

Diarrhea and Dysentery

Frank R. Philbrook, M. D., and John E. Gordon, M. D.

ARMY EXPERIENCE WITH DIARRHEAL DISORDERS BEFORE WORLD WAR II

> Montgomery says the Eighth Army won, but Rommel claimed the victory for dysentery * * *. But, as the Germans learned at El Alamein, dysentery can still win battles, when hygiene discipline on one side is slack.—SIR SHELDON F. DUDLEY.

General Considerations

Throughout history, military populations have experienced great morbidity by reason of the diarrheas and dysenteries. As causes of illness and noneffectiveness, these diseases have plagued the United States Army since it was first organized. Until the time of World War I, they were important causes of mortality.

Diarrheal disease was rampant among troops during the Revolution and apparently was responsible for more deaths than were caused by enemy action. Available figures for the Civil War indicate the military significance of this disease in combat troops; diarrheas and dysenteries "occurred with more frequency and produced more sickness and mortality than any other form of disease." More than 1,755,889 patients were admitted to field medical installations and hospitals. For diarrheas and dysenteries, including gastroenteritis, the case rate was 741.2 per annum per 1,000 average strength. Deaths were 46,277, representing a mortality rate of 18 per 1,000; 2.6 percent of the men admitted for these causes died. During the Civil War, more than 1 death in every 4 caused by disease was ascribed to diarrheas and dysenteries. In the Union Army, of every 1,000 men the following numbers succumbed to dysentery or diarrhea during each year of the war; Negro troops fared worse than white troops: [1]

Year	*White troops*	*Negro troops*
1861–62	4. 17	0
1862–63	15. 99	0
1863–64	15. 78	43. 54
1864–65	21. 29	36. 29
1865–66	16. 00	26. 97

[1] Medical and Surgical History of the War of the Rebellion. Medical History. Washington: Government Printing Office, 1879, vol. I, pt. II, pp. 1, 6, passim.

The dysenteric diseases are at their worst in prisoner-of-war camps. The stockade for Union prisoners of war at Andersonville, Ga., is an outstanding historical example, to such extent that it became known as the most fatal field in the War Between the States. At least 16,772 cases of diarrhea and dysentery occurred at this prison and represented an estimated annual rate of 1,724 per 1,000 strength; 4,529 men died. More than one-half of all fatalities were attributed to diarrhea and dysentery.[2]

During the Spanish-American War, at the turn of the 20th century, the incidence of diarrheas and dysenteries among United States Army troops was slightly lower than that among troops during the Revolutionary War and the Civil War. However, typhoid fever was distinguished as a disease problem because of a mortality far exceeding the more frequent but less deadly related dysenteric illnesses. Indeed, diarrhea and dysentery seemed in this war to have lost much of their killing power. During the war years of 1898–1901, 204,040 admissions were reported representing a rate of 426 admissions per 1,000 troops. The number of deaths was 1,595, a rate of 3.3 per 1,000. Case fatality dropped to 0.78 percent in this period. Diarrheal disease occurred in severe epidemic form in United States Army troops in the Philippines shortly after the American occupation in 1899–1900. Much of the excess incidence was doubtless due to bacillary dysentery, but typhoid fever was the more serious problem in terms of resulting deaths. At the time of the Spanish-American War, attention was focused upon typhoid fever with a resultant *improvement in* diagnosis of this enteric condition. Examination of data of this and other wars suggests that many fatal intestinal infections of earlier wars were in reality typhoid fever, erroneously diagnosed as infections within the group of diarrheas and dysenteries (including gastroenteritis). Comparison of experience in World War II with that of former wars must take this into account.

During World War I, the Army benefited from the great improvements in environmental sanitation of the preceding two decades. During the period from April 1917 to December 1919, there occurred in the overseas Army 48,202 admissions for diarrheal disease, including gastroenteritis, a rate of 28.9 admissions per 1,000 troops. There were only 208 deaths (0.13 deaths per 1,000 troops) which represents the best record up to that time in any American war. Case fatality was 0.43 percent. The improvement in respect to typhoid fever was even more startling (table 82). This favorable downward trend of cases and deaths of typhoid fever continued throughout World War II, but the rate of 40 cases of reported dysenteries and diarrheas (excluding gastroenteritis) per 1,000 strength in total troops overseas certainly shows no appreciable improvement over World War I experience. However, a comparison of the Army in continental United States and in the European Theater

[2] Medical and Surgical History of the War of the Rebellion. Medical History. Washington: Government Printing Office, 1888, vol. I, pt. III, p. 35.

of Operations, with troops of World War I, is more justifiable and reveals that deaths from diarrheas and dysenteries followed a downward trend in World War II, reasonably reflecting the improved sanitation of America and Europe. During World War II, as contrasted with World War I, a greater proportion of combat troops were sent to tropical or semitropical regions with high endemicity for dysenteric diseases and a low standard of sanitation. The rate of 18 cases per 1,000 strength for the Zone of Interior in World War I is to be compared with 9 cases per 1,000 for World War II (table 54). The enteric disease rate among overseas troops in World War I, approximately 29 cases per 1,000 troops per annum, may properly be compared with the rate among troops in the European theater in World War II, 14 cases per 1,000 troops per annum. The rates for World War II are about one-half those of World War I. By the same standards, the case rates for typhoid fever dropped in World War II to less than one-tenth of the rates for this disease in World War I for the total Army, and to approximately one-twentieth of the World War I rates for this disease among United States Army troops in Europe. Typhoid case rates among the United States Army at home became an almost insignificant 0.006 case per 1,000 troops per annum. Marked improvement also occurred in respect to the paratyphoid fevers.

During World War II, deaths ascribed to diarrheas and dysenteries (including also gastroenteritis, ileitis, and ulcerative colitis) totaled 116. Using the 523,331 cases reported on WD MD Form 86ab, Statistical Health Report, (not including cases diagnosed as gastroenteritis) the deaths represent a case fatality of 0.022 percent. The death rate of 0.005 per 1,000 strength is considerably less than the rate in World War I.

In comparing current experience with past Army history of these diseases, due consideration must be given to the fact that earlier data often were based on hospital admissions alone, whereas World War II morbidity statistics include all patients, whether sick in quarters or admitted to hospital. Although reporting is still far from complete, the general level of reporting is better than in former wars. World War II data for common diarrheas are considerably increased by these factors, while the more severe illnesses such as the dysenteries (when diagnosed as such) and typhoid fever are probably little affected, since most patients with these diseases would have been admitted to hospital even in former wars. As a result, more mild cases of diarrheal disease probably have crept into statistical records of recent wars as compared with earlier wars. This factor must be considered in evaluating both incidence of disease and effectiveness of medical care. With the reporting of a larger number of mild cases and better medical care, case fatality rates fell to almost insignificant levels, despite the high morbidity reported in several theaters of operations in World War II. Nevertheless, control of the diarrheas and dysenteries has not kept pace with that accomplished for typhoid fever.

TABLE 54.—*Incidence of diarrhea and dysentery in the U. S. Army, by area and year, 1940–45 and total for the years 1942–45* [1]

[Preliminary data based on periodic summary reports]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1940		1941		1942		1943		1944		1945		1942–45	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	1,874	7	16,564	15	19,074	8	59,511	12	36,543	9	18,492	6	133,620	9
Overseas:														
Europe [2]					1,240	17	3,082	12	18,720	13	31,154	14	54,196	14
Mediterranean					719	34	57,006	132	35,798	54	7,126	22	100,649	70
Africa-Middle East					1,124	196	9,069	170	5,427	115	3,210	79	18,830	128
Middle East					(983)	224	(8,621)	179	(2,197)	114	(2,503)	89	(14,304)	143
Central Africa					(141)	104	(448)	86	(3)	(3)	(3)	(3)	(3)	(3)
Persian Gulf Command					(4)	(4)	(4)	(4)	(3,230)	115	(707)	56	(3)	(3)
China-Burma-India					699	123	6,706	146	28,850	181	20,696	93	56,951	131
China					(3)	(3)	(3)	(3)	[5] (297)	[5] 68	(5,284)	122	(3)	(3)
India-Burma					(3)	(3)	(3)	(3)	[5] (3,317)	[5] 107	(15,412)	86	(3)	(3)
Southwest Pacific	323	27	601	28	3,965	59	13,627	70	30,710	55	66,607	74	114,909	67
Australian mainland					(3)	(3)	(3)	(3)	(1,442)	23	[6] 53	[6] 5	(3)	(3)
Philippine Islands	(323)	27	(601)	28					(5,033)	114	59,229	104	(64,262)	105
Other					(3)	(3)	(3)	(3)	(24,235)	54	[6] 4,619	[6] 32	(3)	(3)
Pacific Ocean	36	1	686	18	4,968	34	12,580	43	10,949	28	6,269	19	34,697	30
Central Pacific	(36)	1	(686)	18	(1,526)	15	(1,741)	11	[7] 2,794	[7] 21	(3)	(3)	(3)	(3)
South Pacific					(3,442)	77	(10,839)	76	[7] 4,552	[7] 38	(3)	(3)	(3)	(3)
North America			4	1	145	4	158	2	293	7	136	6	732	4

Latin America	221	8	667	13	2, 145	21	2, 557	21	1, 477	17	1, 141	16	7, 320	19
Panama	(63)	3	(103)	3	(257)	6	(184)	4	(135)	3	(201)	6	(777)	5
Antilles Department	(158)	34	(564)	26	(1, 770)	29	(1, 665)	26	(865)	24	(694)	20	(4, 994)	25
South Atlantic					(118)	112	(708)	114	(477)	65	(246)	40	(1, 549)	75
Alaska	(8)	(8)	(8)	(8)	240	5	890	8	248	3	49	1	1, 427	5
Total overseas	580	9	1, 958	17	15, 245	30	105, 675	66	132, 472	38	136, 319	33	389, 711	40
Total Army	2, 454	7	18, 522	15	34, 319	11	165, 186	25	169, 015	22	154, 811	22	523, 331	21

[1] In terms of data based on diagnoses reported on individual medical records, totals for 1940–42 appear to be understated approximately as follows: One-half in 1940, one-quarter in 1941, and one-third in 1942. It is not known whether these proportions apply equally to each geographic area.

[2] Includes United Kingdom and continental Europe.

[3] Data are not available.

[4] Included in Middle East.

[5] Data for November and December only.

[6] Data for first 8 months only.

[7] Data for first 7 months only.

[8] Included with continental United States.

NOTE.—Figures in parentheses are subtotals.

Classification

For the purposes of this analysis, the diarrheas and dysenteries are classified as common diarrheas and dysenteries (protozoal, bacillary, and unclassified) according to the diagnostic titles set forth in statistical health reports.

The common diarrheas (as defined in Army Regulations No. 40-1080) include a variety of infections of the gastrointestinal tract of unknown cause, such as colitis, ileocolitis, enteritis, diarrhea, mucous colitis, intestinal indigestion, sigmoiditis, and intestinal toxemia (when associated with diarrhea). Grouping these infections under this single inclusive title has an advantage over the practice of former years when each clinical entity had individual consideration irrespective of admittedly inexact anatomic or symptomatic criteria for diagnosis. All these infections are now grouped within the single category of the common diarrheas. Too little is known about these several conditions, and the criteria for differentiation are too inexact to warrant continued separation in the course of attempts to describe the diarrheal disorders of an army or another population.

Diagnostic and microbiologic interrelationship.—One or another of these terms, and also that of gastroenteritis, is often used provisionally for infections suspected to be of specific bacterial origin. Bacteriologic diagnosis ultimately determines a number of such infections as being bacillary dysentery, amebic dysentery, or instances of salmonellosis. As for the remainder, much of what is now grouped under the common diarrheas is in all probability dysentery of unknown etiology, unknown because a search for an etiologic agent either was not made or was reported negative.

The proportion of common diarrheas actually representative of protozoal or bacillary dysentery can only be estimated from limited data in a few theaters where laboratory surveys were made. The important consideration is that no practical analysis of dysenteric disease nor specific consideration of bacillary dysentery is either reasonable or feasible unless the common diarrheas are included. This opinion is held despite recognition that diagnostic titles included in the common diarrheas do not in their entirety represent enteric infections or intoxications due to known specific microbial agents. Through unofficial statements of former Army medical officers, it is known, for example, that acute alcoholism was recorded occasionally under one or another of the diagnostic titles included in the common diarrheas, or as gastroenteritis, because of the stigma attached to the straightforward diagnosis. The important fact remains that, when careful laboratory studies were made, a major part of the common diarrheas as seen in World War II could be demonstrated as due to *Shigella*, *Salmonella*, or *Endamoeba histolytica*. Many epidemic peaks of common diarrhea coincide in time with recognized outbreaks of shigellosis.

Clinical interrelationship.—The entities included in the mixed group of anatomic and symptomatic diagnostic titles, known collectively as the common diarrheas, seemingly have much in common in their clinical manifestations with

diseases distinguished etiologically as either bacillary or amebic dysentery. Diarrhea is the typical symptom, often of sudden onset and brief duration, and is accompanied by varying degrees of tenesmus and abdominal pain and followed by rapid recovery. The common diarrheas are characterized by an extremely low fatality. The few deaths attributed to common diarrhea are presumably the result of failure to recognize a more serious infection such as bacillary or amebic dysentery. Specific etiologic agents are by definition undetermined.

Epidemiologic interrelationships. Epidemiologically, the behavior of common diarrhea is suggestive of an infection, and transmission in the great preponderance of cases is by the same general means, through ingestion of food or drink. The occurrence of most of them reflects directly or indirectly the general level of environmental sanitation under which different army units existed or of the countries or specific locations in which troops were deployed. Occurrence in close correlation with known specific infectious diarrheal diseases suggests similar epidemiologic mechanisms. Many epidemic peaks coincide in time with recognized outbreaks of shigellosis. Incidence was high in theaters of operations when the environment was poorly sanitated and bacillary and amebic dysenteries were common diseases. Incidence was low in theaters or environmental situations where known specific agents of diarrheal diseases were infrequent. The rigid application of sanitary principles, especially in relation to food and drink, to feces disposal, and to fly control reduced the risk of the common diarrheas in equal degree as it lowered the incidence of the dysenteries.

Titles not included.—Some 10 other diagnostic titles or diseases are variously included in classifications of diarrheal disease, none of which are represented in this analysis. Brief consideration is given to possible effects of their exclusion on comparisons of the present data with diarrheal disease in former wars and in some instances on comparisons between theaters of World War II.

The diagnostic title of gastroenteritis is not included among diarrheas and dysenteries as presented in table 54. Statistics of former wars often included gastroenteritis, an addition materially affecting comparisons. Separate data on the incidence of gastroenteritis are not available for the first 2 years of World War II, but it is estimated that approximately 350,000 cases occurred during the 4 war years. If these cases had been included among diarrheas and dysenteries of World War II, the total would be more than 850,000, and incidence would exceed 34 per 1,000 per annum, a rate somewhat greater than the 29 for the United States Army in Europe during World War I.

Typhoid and paratyphoid fevers and bacterial food poisoning if they were reported as such are not here included. They are considered elsewhere in this volume. Army units were instructed in October 1943 to record bacterial food poisoning as a separate item, and the diagnostic title was added to the Statistical Health Report (WD MD Form 86ab of 22 August 1940) when the report was revised in 1944. Before October 1943, bacterial food poisoning had been included under common diarrhea, and undoubtedly a considerable pro-

portion of cases continued to be so reported after this date.[3] The extent to which the new system of reporting was followed in the several theaters of operations and the promptness with which it was adopted were doubtless subject to much variation. Better results were undoubtedly obtained in areas where epidemiologic investigation and laboratory diagnosis were readily available services. Current statistical information on typhoid and paratyphoid fevers can be accepted as reliable, since diagnosis depends almost universally upon demonstration of the etiologic agent. That this reliability factor was not always present must be taken into consideration when comparing incidence (of either these specific diseases or the diarrheas and dysenteries) in World War II with that of earlier wars.

Also excluded from specific and separate consideration here are such entities as diarrhea and dysentery of presumed viral etiology such as epidemic viral gastroenteritis or acute infectious gastroenteritis or winter vomiting disease or vomiting and diarrhea syndrome, as described by numerous authors during and since World War II.

There conditions doubtless played an important role in the World War II Army and will in the Army of the future. As more becomes known about them, they may become subject to separate statistical analysis, but during World War II their recognition and reporting was not specifically accomplished. Viral etiology was suspected in several theaters, and some attempts that were made to recover these agents are described in this chapter. Such entities during World War II were merely given one or another of the common diarrhea diagnoses or were considered to be cases of gastroenteritis or unclassified dysentery.

Factors Affecting Reporting of Specific Dysenteries

By regulation, the diagnosis of bacillary and amebic dysentery during World War II was contingent on recovery or identification of the bacillus or ameba, a practice that did not hold in former wars. Instances of clinical dysentery associated with pus, mucus, and possibly blood in the stools were designated "dysentery, unclassified," in the absence of microbiologic confirmation. In general, the frequency of etiologic diagnosis of bacillary or protozoal dysentery was a reflection of the amount and quality of laboratory work.

The techniques necessary for identification of *E. histolytica* are relatively simple, compared with those of determinative bacteriology. As a consequence, the search for protozoal agents of diarrheal disease was usually more exhaustive than that for bacillary agents and probably accounted for the greater frequency with which amebic dysentery was recognized. A significant proportion of many populations are carriers of *E. histolytica*, estimated by Mackie

[3] Gordon, J. E.: A History of Preventive Medicine in the European Theater of Operations, U. S. Army, 1941–45. [Official record.]

and others [4] as 9.8 percent for the United States and in excess of 50 percent for poorly sanitated regions of the Tropics and subtropics. An even larger number are carriers of protozoa closely resembling *E. histolytica*. The diagnosis of amebic dysentery was in all probability frequently made when the true infectious agent was an unsought *Shigella*. Persons without clinical symptoms are known to have been treated with a view to eliminating the *Endamoeba*, and some were given the diagnosis of amebic dysentery. For these reasons, the incorrect diagnosis of common diarrhea was made less frequently for cases of amebic dysentery than for cases of bacillary dysentery. Small numbers of patients with common diarrheal diseases and shigellosis were in all likelihood erroneously designated as suffering from amebic dysentery. The tendency was to overreport amebic dysentery and to underreport bacillary infections.

The biologic gradient of clinical bacillary dysentery ranges from severe infections (in the diagnosis of which the physician is prompted to seek laboratory [illegible] or which he may diagnose as unclassified dysentery, enteritis, gastro[illegible]s, or enterocolitis) to instances of lesser severity, with symptoms largely absent or attended by no more than mild diarrhea. The tendency is to give the diagnosis of common diarrhea to these mild infections or, because of vague symptomatology, to call them gastroenteritis, enteritis, or colitis. For reasons already presented, this probably applies to amebic dysentery in lesser degree. Other features presumably account for varying frequencies of confirmed dysentery.

Adequate laboratory facilities and competent personnel were not uniformly available in all theaters of operations. Furthermore, the interests of medical officers varied. A specific concern about amebiasis by medical officers in one location was followed by a relatively high incidence of that type of infection. In another theater, an epidemiologic team had a specific interest in shigellosis, with the result that a relatively high incidence of bacillary dysentery was uncovered. Each of these circumstances led to the separation of a higher percentage of the entity on which interest was focused from the common diarrheas, gastroenteritis, and unclassified dysentery classifications. For example, one factor accounting for the reported high incidence of amebic dysentery in a part of the Mediterranean theater was said to have been the result of interest of certain medical officers in amebiasis.[5] The arrival of several groups studying diarrheal disease and the development of special health record forms for amebiasis in the China-Burma-India theater doubtless stimulated search for cases and carriers.

The true incidence of bacillary dysentery represents a sizeable proportion of the common diarrheas, of unclassified dysentery, and of gastroenteritis together with those cases reported as bacillary dysenteries; hence the practicality and feasibility of presenting combined information for diarrhea and

[4] Mackie, Thomas T., Hunter, George W., III. and Worth. C. Brooke: Manual of Tropical Medicine. Philadelphia: W. B. Saunders Co., 1945, p. 191.

[5] Gilmore, H. R.: Final Report of the Preventive Medicine Officer, Office of the Surgeon, Mediterranean Theater of Operations, U. S. Army, 1 January-31 October 1945. [Official record.]

dysentery as a class or group. Justification for this procedure is found in the situation in the Mediterranean theater in 1943. The annual rate for diarrheas and dysenteries was 132 cases per 1,000 troops (table 54). The rate for bacillary dysentery was only 6.22 cases (table 59), despite the presence that year of a theaterwide epidemic ascribed to bacillary dysentery. Surveys revealed that *Shigella* could be recovered from a high percentage of the cases of common diarrhea in the Fifth U. S. Army.[6] During the same year, three other theaters with no recognized general epidemic of bacillary dysentery actually reported higher rates for that disease. Two had laboratories especially active in recovering dysentery bacilli from suspected cases. In the China-Burma-India theater, several hundred consecutive admissions for diarrhea were examined in a general hospital in Assam and in two dispensaries. Dysentery bacilli were demonstrated by culture of material obtained by a single rectal swab in 24 percent of the hospital cases and in 16 percent of the dispensary infections. Multiple cultures doubtless would have increased those percentages. "As would be expected, the number of positive findings in the series of dispensary cases is less than in the hospital series. These cases were mild, and had cultures not been made they would have been classed as common diarrheas." [7]

Because of the differences in diagnostic practice just described and because facilities for laboratory work were so uneven, the various clinical entities among intestinal infections cannot be compared individually with what happened in former wars nor, in some instances, can any reasonable comparison be made between theaters of operations of World War II.

Method of Presentation of Data

The most useful approach appears to be a study of diarrheas and dysenteries as a single group of diseases and as they occurred in the several theaters of operations. In the following presentation of analyses, the practical purpose of the study—to provide a basis for future experience in the event that United States troops should again be deployed in the areas involved—is kept constantly in view. Bacillary dysentery is given special consideration in this presentation; other specific diarrheal diseases are presented in separate chapters. Individual attention is given the dysenteries so far as the data permit. Proved cases of bacillary dysentery are a part of this report; amebic dysentery, although given some general consideration here, is the subject of a separate chapter.

Grouping the diarrheas and dysenteries in a combined statistical presentation is judged a practical and feasible approach to the descriptive epidemiology of dysenteric disease. A compilation of the Medical Statistics Division, Office of the Surgeon General, entitled "Morbidity and Mortality in the United States Army, 1940–45," is the source of most of the data now presented (table 54 and charts 28, 30, 35, 39, 40, 42, 45, 46, and 47). These statistics enumerate the diarrheas and dysenteries as they were diagnosed and reported currently and

[6] Fifth Army Medical Service History, 1944.

[7] Progress and Final Report, Sub-Commission on Dysentery, Army Epidemiological Board, 20 Nov. 1944.

regularly on the statistical health report. They may not always reflect the changes in diagnosis which occurred during the course of hospitalization. Such data therefore do not tally identically with those derived from statistical tabulations of individual medical records. They do provide an excellent basis for epidemiologic description of the picture as it unfolded in the several theaters of operations during World War II. For such separate consideration as will be given to individual diagnostic entities comprising the group, especially bacillary dysentery (table 59), the major source of statistical data is various tabulations of individual medical records as compiled by the Office of the Surgeon General. In general, admissions data are used for the common diarrheas and incidence rates for the specific and unclassified dysenteries.

The epidemiologic description of the diarrheas and dysenteries in each major theater of operations or area will be followed by a consideration of available information on bacillary dysentery.

Experience From 1935 to 1941

During the years from 1935 to 1939 inclusive, Army troops stationed within the United States were a relatively stable population, living under sanitary conditions reasonably comparable to those of better sanitated civilian communities. The incidence of diarrheal disease was low, and rates were remarkably uniform from year to year (table 55). For the decade of the 1930's, incidence rates were within the range of 5 or fewer cases per 1,000 men as illustrated in chart 27. During the latter half of the period, annual

TABLE 55.—*Incidence of intestinal infections including common diarrhea and dysentery (all types), peacetime Army in continental United States, by month, 1935–39*

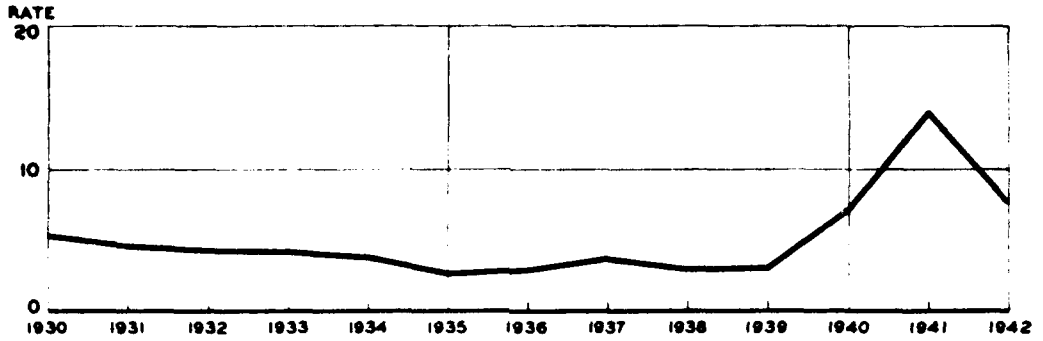
[Rate expressed as number of cases per annum per 1,000 average strength]

Month	1935–39		1935		1936		1937		1938		1939	
	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate	Cases	Rate
January	102	2.0	12	1.6	18	2.0	19	1.5	23	2.1	30	2.9
February	63	1.3	7	.9	9	.8	12	1.2	20	1.9	15	1.5
March	130	2.5	22	2.4	14	1.5	15	1.5	52	5.0	27	2.1
April	81	1.6	17	2.3	9	1.0	8	.8	28	2.2	19	1.8
May	90	1.7	16	2.1	14	1.3	14	1.1	22	2.1	24	2.3
June	136	2.6	18	2.0	16	1.7	38	3.7	27	2.6	37	2.8
July	200	3.7	14	1.8	16	1.7	51	4.1	39	3.0	80	7.3
August	334	6.3	52	5.1	81	7.1	119	11.6	40	3.8	42	3.9
September	254	4.6	56	6.6	54	5.7	41	4.0	50	3.8	53	3.9
October	188	3.4	19	2.2	84	7.1	17	1.3	33	3.2	35	3.0
November	186	3.5	16	1.5	10	1.0	104	10.3	23	2.2	33	2.7
December	121	2.0	13	1.5	11	1.1	51	4.0	17	1.3	29	1.8
Total	1,885	3.0	262	2.5	336	2.8	489	3.6	374	2.8	424	2.9

incidence varied from 2.5 to 3.6 cases per 1,000 per annum and the average was 3 cases. The highest rates were in summer, August being the peak month; winter rates were lowest, with a usual seasonal increase in late spring. Diarrheal disease presented a favorable and stabilized situation.

CHART 27.—*Incidence of diarrhea and dysentery in the Army in the continental United States, 1930–42*

[Rate expressed as number of cases per annum per 1,000 average strength]



Source: Monthly Progress Report, Army Service Forces, War Department, 31 Aug. 1943, Section 7: Health.

The incidence of diarrheal disease in troops in the continental United States rose rather sharply to 13 cases per 1,000 per annum for the 2 years 1940 and 1941 (chart 27). These were the years of preparation for war, with more than fourfold expansion of the Army. Raw recruits and untrained men were brought together from all parts of the Nation, and they engaged in active field training and maneuvers. In 1940 and 1941, diarrheal disease rates were 7 cases and 15 cases, respectively, per 1,000 troops (table 54). For the same years, rates for the total Army (both overseas and in continental United States) were also 7 and 15, which shows that the bulk of the problem was in the Zone of Interior rather than overseas. Overseas troops had slightly higher rates of 9 and 17, but less than 12 percent of the average strength of this expanding Army was stationed overseas, chiefly in the Philippines with rates of 27 and 28 cases, respectively, per 1,000 per annum and in the Antilles Department (Latin American area) with rates of 34 and 26 cases, respectively, per 1,000 per annum. The well-established program of sanitation and military preventive medicine in the Panama Canal Department proved its worth, for during these 2 years the rate was 3 cases per 1,000 per annum.

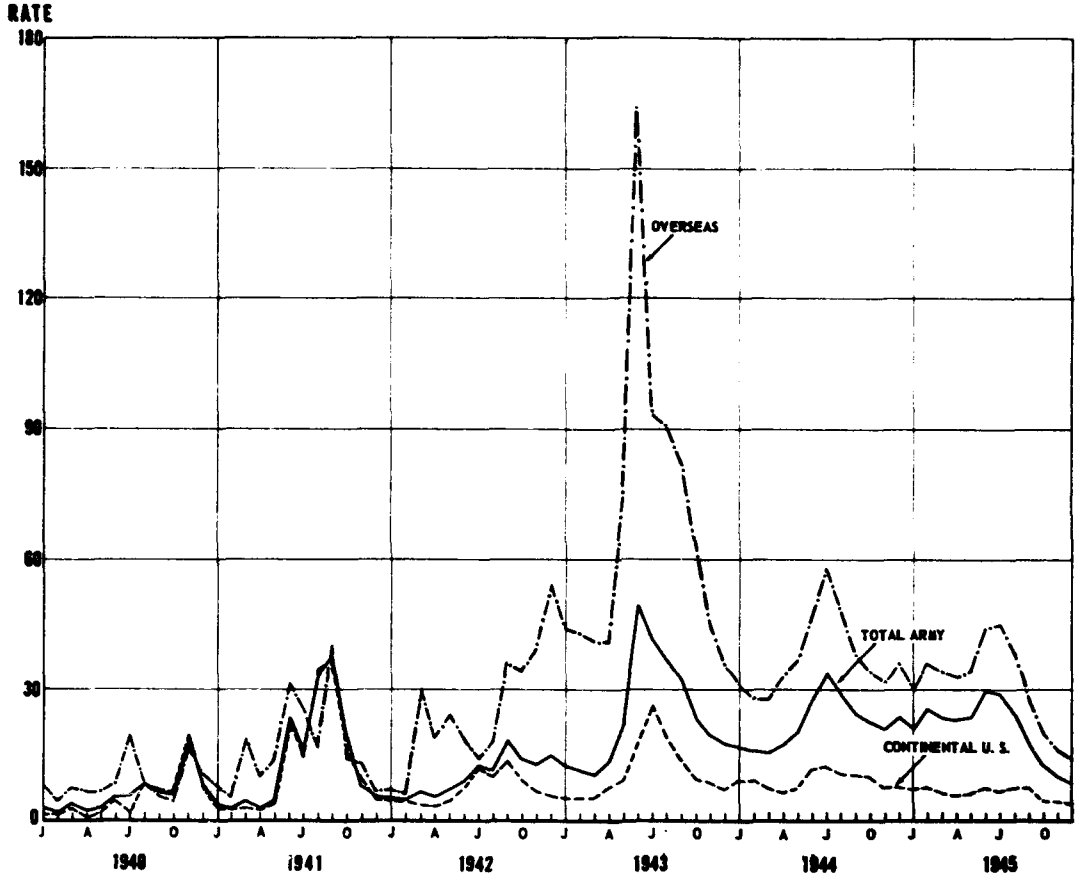
That field training contributed heavily to the incidence of diarrheal disorders in the United States is evidenced by the North Carolina maneuvers of August and September 1941. The entire Army had 8,890 cases in those 2 months; a total of 8,297 cases were in the continental United States; a large proportion was related to an outbreak of bacillary dysentery in the course of these maneuvers. Continental rates for these 2 months were 35 and 37 per 1,000 per annum (chart 28), rates higher by far than for any month in the European theater or for the Panama Canal Zone, the North American area, or the Alaskan Department at any time in World War II. However, these

monthly rates were exceeded at one time or another in all tropical or subtropical areas of military operations, with the single exception of the Panama Canal Zone.

CHART 28.—*Incidence of diarrhea and dysentery in the U. S. Army, 1940–45*

[Preliminary data based on periodic summary reports]

[Rate expressed as number of cases per annum per 1,000 average strength]



ARMY EXPERIENCE WITH DIARRHEAL DISORDERS DURING WORLD WAR II

Incidence

The average strength of the Army for the years 1942–45 was approximately 6,076,135 men. Within this population and time, 523,331 cases of diarrhea and dysentery were reported (table 54), a crude rate of 21 per annum per 1,000 average strength. Cases among troops in continental United States numbered 133,620, or 25 percent of the total, but 60 percent of the average strength of the Army during the war years was stationed in the Zone of Interior. The rate of

9 per 1,000 average strength was low; that for troops overseas was more than 4 times as great.

When the United States entered the war, combat troops were sent overseas in progressively increasing numbers and into numerous poorly sanitated areas with highly endemic diarrheal disease. However, the 1942 rate for the total Army, 11 cases per 1,000, was somewhat less than the 1941 rate of 15. Scarcely 17 percent of the average strength was overseas in 1942, and one-half the troops were favorably located in the British Isles, Hawaii, the Panama Canal Zone, and North American bases, areas that never contributed heavily to diarrheal disease during these military operations. Rates were less satisfactory in the Southwest Pacific, in the Antilles Department, and especially in Asia. The rate for all overseas troops in 1942 was 30 cases per 1,000. The continental United States rate of 8 was acceptable for an Army in training under wartime conditions.

In late 1942 and in 1943, increasing numbers of troops entered North Africa and the Mediterranean, the Middle East, and the China-Burma-India theaters, all north of the Equator. Rates for the total Army rose to 25 per 1,000 and for troops overseas to 66 per 1,000, the 1943 annual rate for all overseas troops being the highest of the war. June 1943 marked the high point in monthly rates for troops overseas, the figure being 164 (chart 28). Large numbers of unseasoned young men made a first entry into poorly sanitated areas of highly endemic diarrheal disease. Conditions were different in subsequent years for smaller numbers of men were rotated into already established bases where preventive medicine practices were established and the bulk of troops had become accustomed to conditions. For overseas troops in 1943, 7.4 percent of all disease and 3.5 percent of all noneffectiveness was due to the diarrheas and dysenteries (table 67).

Rates for the total Army were better in 1944 and 1945, 22 and 22, respectively. Rates overseas fell from 66 in 1943, to 38 in 1944, and to 33 in 1945 (table 54). All overseas theaters participated, except the Southwest Pacific area where return to the Philippines, combat conditions, and other circumstances of environmental sanitation resulted in higher morbidity. Beginning with 1943, sharp seasonal peaks characterized June and July of each war year (chart 28).

The rate of cases per 1,000 strength per annum for troops within continental United States during the 4 war years (table 54) compares favorably with the 13 cases per 1,000 strength per annum of the 1940–41 period of preparation (chart 27). Reception and training of recruits was accelerated, but large-scale maneuvers were not conducted. A rate of 26 per 1,000 in July 1943 represents the highest seasonal peak (chart 28). Gordon [8] points out that these high rates for troops in continental United States "suggests a relation to the unprecedent concentration in August and September of that year of a number of major outbreaks of common diarrhea on board transports bringing troops to Europe," the inference being that cases in these outbreaks may have been allotted to the

[8] See footnote 3, p. 326.

Zone of Interior. Carriers in the crowded populations aboard troop transports doubtless provided the necessary etiologic agents, and breaks in sanitation resulted in epidemics which further reflected in the experience in the European theater.

Annual rates for continental United States troops for 1942–45 were respectively 8, 12, 9, and 6 (table 54). The highest rate of 12 was at that time when great numbers of troops were in training and when transport of personnel to overseas theaters was frequent. In connection with previous discussion of the total problem, 1943 also was the peak year for bacillary dysentery, for the Army as a whole and for troops in the continental United States (table 59).

The principal difficulty was in the Fourth, Fifth, and Eighth Service Commands, especially in the Tennessee and Louisiana maneuver areas (chart 29). "Rates of the order of those shown for June and July [1943] * * * are compatible only with interpretation that sanitary discipline among troops is deficient. This deficiency is the more serious because of the increased hazards to which these men are likely to be exposed if they are sent overseas."[9] The validity of this opinion finds support in the subsequent records of dysentery and diarrheal disease in the several overseas theaters.

TenBroeck, Kuhns, and others, studying the incidence of clinical dysentery in several commands within the continental United States, observed that "the problem of dysentery was in direct relation to the presence or absence of waterborne sewage facilities."[10]

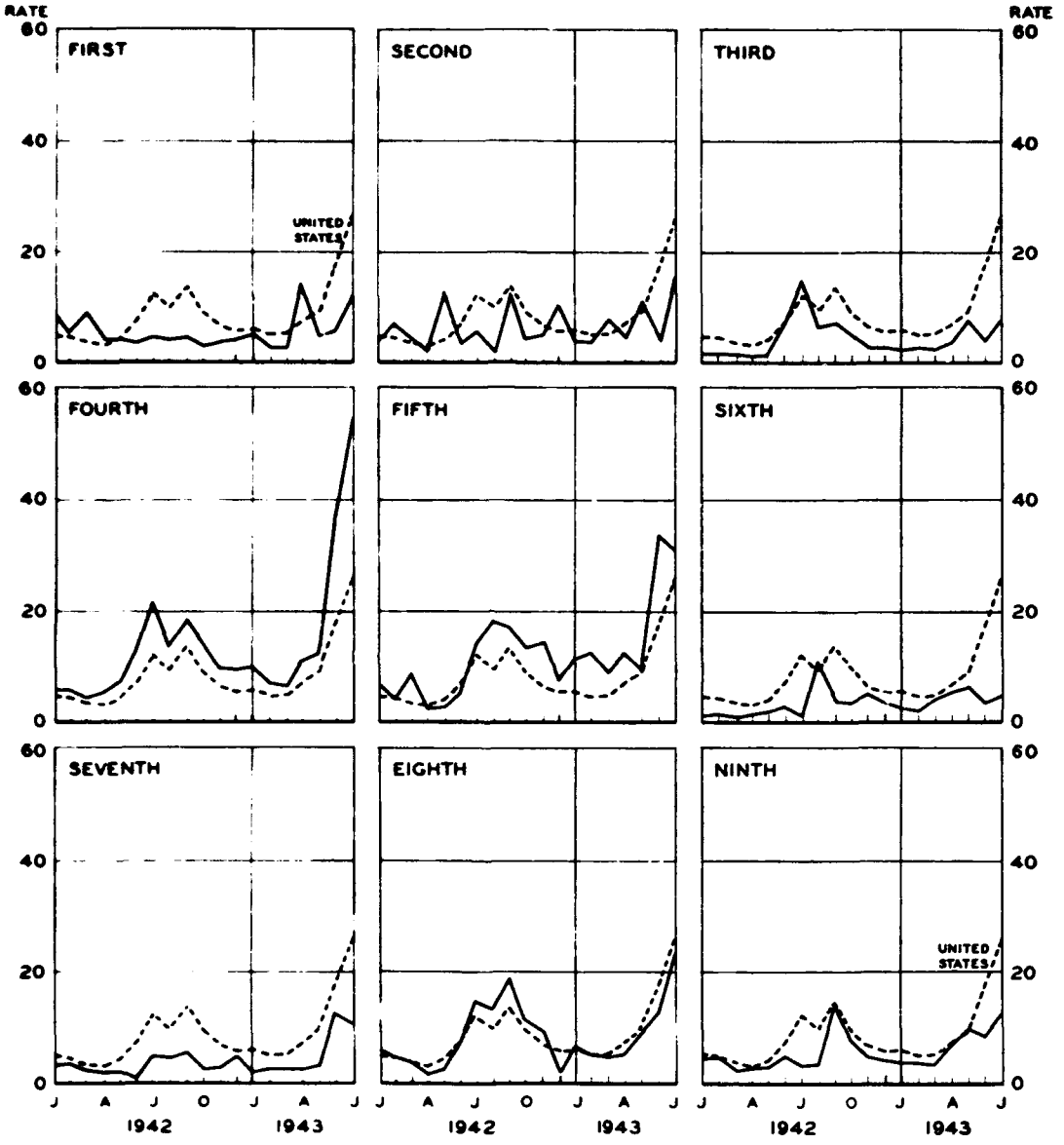
Deaths and Noneffectiveness Resulting From Diarrheal Disorders

Deaths.—Diarrheal disorder in military populations has become established in recent years as a notorious cause of morbidity and noneffectiveness and of relatively little significance in respect to mortality. These conditions held for United States troops in World War II. Only 64 deaths were ascribed to the dysenteries, all forms, including bacillary, unclassified, and protozoal (table 56), as follows: 16 to bacillary dysentery, 16 to unclassified dysentery, and 32 to amebic dysentery. Estimated case fatality rates for bacillary dysentery were in the order of 0.06 percent. Case fatality for unclassified dysentery was approximately 0.05 percent, and for amebic dysentery, 0.09 percent. Case fatality for dysentery, all forms, was 0.07 percent. The common diarrheas—to include all of ulcerative colitis, ileitis, and gastroenteritis along with enterocolitis, colitis, enteritis, and diarrhea, cause not specified—were responsible for only 52 deaths, a case fatality less than 0.01 percent. Total deaths ascribed to combined diarrheas and dysenteries were 116. As more than 500,000 cases were recorded on the morbidity reports and additional cases were recognized after admission to hospital for other causes, this group of diseases produced approximately 1 death for each 4,500. If gastroenteritis is included, as in former wars, 1 death occurred among each 7,400 cases. An

[9] Monthly Progress Report, Army Service Forces, War Department, 31 Aug. 1943, Section 7: Health.
[10] Personal communication to author.

CHART 29.—*Incidence of diarrhea and dysentery in the Army in continental United States, by service commands, January 1942 to July 1943*

[Rate expressed as number of cases per annum per 1,000 average strength]



Source: Monthly Progress Report, Army Service Forces, War Department, 31 Aug. 1943, Section 7: Health.

estimated case fatality for total diarrheas and dysenteries plus gastroenteritis is approximately 0.014 percent.

Deaths due to dysenteries and diarrheas in the total Army during the war years are presented in table 56, on the basis of data derived from complete files of individual medical records, a most reliable source. The condition specified as the cause of death is not necessarily that for which the patient was admitted. Table 56 probably includes deaths other than the total among

TABLE 56.—*Deaths due to dysentery and diarrhea, including gastroenteritis, ulcerative colitis, and ileitis, by diagnosis and year, 1942–45*

Diagnosis	Total Army					Continental United States					Overseas				
	1942–45	1942	1943	1944	1945	1942–45	1942	1943	1944	1945	1942–45	1942	1943	1944	1945
Dysentery:															
Amebic	32	6	4	4	18	9	3	1	2	3	23	3	3	2	15
Bacillary	16	3	6	3	4	2	-----	2	-----	-----	14	3	4	3	4
Unclassified	16	-----	1	4	11	1	-----	-----	-----	1	15	-----	1	4	10
Total	64	9	11	11	33	12	3	3	2	4	52	6	8	9	29
Diarrhea:															
Enteritis [1]	27	3	10	3	3	18	3	7	1	1	9	-----	3	2	2
Gastroenteritis [1]				1	1				1	1				-----	-----
Ileitis [1]				1	5				1	3				-----	2
Enterocolitis	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----
Ulcerative colitis [1]	24	-----	7	9	6	12	-----	5	7	-----	12	-----	2	2	6
Colitis, not elsewhere classified [1]				1	1				-----	-----				1	1
Diarrhea, cause unknown	1	-----	1	-----	-----	-----	-----	-----	-----	-----	1	-----	1	-----	-----
Total	52	3	18	15	16	30	3	12	10	5	22	-----	6	5	11
Grand total	116	12	29	26	49	42	6	15	12	9	74	6	14	14	40

[1] Not coded separately for 1942 and 1943.

patients admitted as having dysentery or diarrhea; and likewise some deaths in the group, due to such causes as amebiasis, amebic abscess, liver abscess, or drug reactions may be included under other titles. In addition to deaths from the diseases included in the data for table 54 (as defined in AR 40–1080, p. 324), deaths due to gastroenteritis, ileitis, and ulcerative colitis, which were not separately coded during 1942 and 1943, are also included. The infectious agents of ulcerative colitis and ileitis may or may not be related to conditions under consideration in this section, but nonetheless deaths from these causes are included despite the fact that the diseases themselves are not here included in the category of common diarrheas.

The crude annual death rate of 0.46 per 100,000 average strength of the Army during World War II for diarrhea and dysentery, gastroenteritis, ileitis, and ulcerative colitis combined is remarkable when compared with rates in former wars. The lesson to be learned from this trend is that deaths from these causes are indeed preventable.

Noneffectiveness.—Information on the average time spent in medical treatment facilities by patients with several types of diarrhea and dysentery are available for 1942, 1943, and 1945 (table 57).

TABLE 57.—*Time lost in treatment of various types of diarrhea and dysentery in selected years*

Diagnosis	Days lost per admission		
	1942	1943	1945
Dysentery:			
Amebic	43	40	40
Bacillary	16	16	16
Unclassified	6	7	12
Amebic dysentery carrier	19	(1)	26
Bacillary dysentery carrier	18	(1)	25
Diarrhea:			
Enteritis [2]	5	5	7
Enterocolitis	6	(1)	9
Colitis [3]	22	(1)	56
Diarrhea, cause undetermined	4	4	7

[1] Data not available.

[2] Includes gastroenteritis and ileitis. In 1945, the 3 conditions resulted in an average duration of 7 days, distributed as follows: Gastroenteritis, 6; ileitis, 128; and enteritis, 7.

[3] Includes ulcerative colitis. In 1945, the average duration of ulcerative colitis was 110 days; the duration of other colitis (nonfunctional) was 25 days.

Based on weighted averages of the 1942, 1943, and 1945 durations, and total admissions for 1942–45, except where indicated otherwise below, the following estimated average daily noneffectiveness (per 1,000 average strength) due to the listed diseases during 1942–45 are presented for the total Army:

Diagnosis	*Rate*	
Dysentery:		
Amebic	0.14	
Bacillary	.04	
Unclassified	.03	
Total		0.21
Dysentery carriers:		
Amebic [1]	.01	
Bacillary [1]	.00	
Total [1]		.01
Diarrhea:		
Enteritis [2]	.10	
Enterocolitis [1]	.05	
Colitis [1 3]	.06	
Diarrhea, cause undetermined	.08	
Total		.29

[1] Based on 1942 and 1945 average durations, and admissions for 1942–45.
[2] Based on 1945 average durations, and admissions for 1944–45.
[3] Includes ulcerative colitis.

Common Diarrheas

Preliminary admission data based on sample tabulations of individual medical records for the war years 1942–45 are presented in table 58. All separate diagnostic titles of the common diarrhea group of diseases from morbidity report data of table 54 are not included, but admissions data (number of cases and rates for instances in which these conditions were primary causes of admission for medical treatment) are presented for the numerically more important titles of enteritis, enterocolitis, colitis, and diarrhea of undetermined cause. The inclusion of gastroenteritis, ileitis, and ulcerative colitis in table 58 is to be noted, since these diseases were not specifically included in the morbidity report data.

Coding procedures were such that enteritis, gastroenteritis, and ileitis during 1942 and 1943 comprised a single category; individual data for enteritis are available for 1944 and 1945 only. Similarly, for 1942 and 1943 ulcerative colitis was included with colitis, not elsewhere classified. With these reservations, the data are suited to the immediate purpose of determining occurrence of certain of the common diarrheas in the several theaters of operations.

Of the group, gastroenteritis had numerically the greatest incidence. Diarrhea of undetermined cause (table 58) was next, with approximately 153,000 admissions tabulated during the war years, representing an admission rate of 6 per annum per 1,000 average strength. Approximately 80 percent of patients were overseas. The year of greatest frequency was 1943, with troops outside the continental United States having a rate of 24.3, with appreciable improvement in the two succeeding war years. For individual theaters

TABLE 58.—*Admissions for certain diseases of the gastrointestinal tract, included in the category of common diarrhea, plus gastroenteritis, ileitis, and ulcerative colitis in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records]

[Rate expressed as number of admissions per annum per 1,000 average strength]

Diagnosis	1942–45		1942		1943		1944		1945	
	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate
Continental United States										
Enterocolitis and colitis, not elsewhere classified	[1] 32, 528	2. 21	[1] 7, 128	2. 68	[1] 15, 370	2. 97	6, 825	1. 72	3, 205	1. 09
Ulcerative colitis	[2]	------	[2]	------	[2]	------	419	. 11	405	. 14
Enteritis, gastroenteritis, and ileitis	218, 447	14. 82	41, 690	15. 69	86, 645	16. 72	56, 687	14. 27	33, 425	11. 40
Diarrhea, cause undetermined	32, 382	2. 20	7, 908	2. 98	14, 185	2. 74	6, 744	1. 70	3, 545	1. 21
Total	284, 181	19. 28	56, 726	21. 35	116, 200	22. 43	70, 675	17. 80	40, 580	13. 84
Overseas										
Enterocolitis and colitis, not elsewhere classified	[1] 39, 162	3. 65	[1] 3, 298	5. 63	[1] 14, 270	8. 45	11, 174	2. 93	10. 420	2. 24
Ulcerative colitis	[2]	------	[2]	------	[2]	------	300	. 08	400	. 09
Enteritis, gastroenteritis, and ileitis	248, 931	23. 19	13, 587	23. 20	55, 918	33. 13	79, 361	20. 78	100, 065	21. 55
Diarrhea, cause undetermined	120, 563	11. 23	5, 351	9. 14	40, 936	24. 25	44, 236	11. 58	30, 040	6. 47
Total	409, 356	38. 13	22, 236	37. 97	111, 124	65. 83	135, 071	35. 37	140, 925	30. 35

	Total Army									
Enterocolitis and colitis, not elsewhere classified	[1] 71, 690	2. 81	[1] 10, 426	3. 22	[1] 29, 640	4. 31	17, 999	2. 31	13, 625	1. 80
Ulcerative colitis	([2])	-------	([2])	-------	([2])	-------	719	. 09	805	. 11
Enteritis, gastroenteritis, and ileitis	467, 378	18. 34	55, 277	17. 05	142, 563	20. 75	136, 048	17. 46	133, 490	17. 62
Diarrhea, cause undetermined	152, 945	6. 00	13, 259	4. 09	55, 121	8. 02	50, 980	6. 54	33, 585	4. 43
Total	693, 537	27. 22	78, 962	24. 36	227, 324	33. 08	205, 746	26. 40	181, 505	23. 96

[1] Includes ulcerative colitis in 1942 and 1943.
[2] Not available separately in 1942 and 1943, included with colitis, not elsewhere classified.

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of operations, however, the general pattern was a decline in rates each successive year, starting with highest rates in 1942.

Inspection of data not here presented revealed similar trends but fewer cases and lower rates for the other common diarrheal diseases. The Mediterranean theater was an outstanding exception. The rate during 1942 of 11 for diarrhea of undetermined cause was followed by an epidemic rate of 49 during 1943 and a decline to rates of 17 and 7 for the 2 subsequent years. During 1943, an outbreak of bacillary dysentery also occurred among Allied troops, spreading across all of North Africa. The association of the two epidemics is subject to differing interpretation, one of which is that the diagnosis of common diarrhea was made extensively for cases of unrecognized bacillary dysentery or for those cases from which *Shigella* was not recovered. Another interpretation is that the same faulty sanitation which contributed to bacillary dysentery also was responsible for epidemics of diarrhea of unknown etiology. In the Middle East where the incidence of bacillary dysentery was higher than for any other theater, rates for diarrhea of undetermined cause also exceeded those of any other theater or area. The converse is also true; in theaters or areas, such as the Canal Zone, North American area, and Alaska, where rates for dysentery were extremely low, so also were rates for the common diarrheas.

Dysentery, All Forms

The cases of dysentery, all forms, in tables 59, 60, and 61 include admissions and also instances in which dysentery existed concurrently with some other disease or occurred after admission to hospital for other reasons. Outpatient cases are excluded. The data derived from sample tabulations of individual medical records therefore include both primary and secondary diagnoses among excused-from-duty patients and are not strictly comparable to the combined data for diarrheas and dysenteries accumulated from current morbidity reports. Some 91,626 patients, about 1.5 percent of the average strength for the Army as a whole, had one or another of the three forms of dysentery—amebic, bacillary, or unclassified—during the war years. The group includes most of the more seriously ill of the individuals with diarrheas and dysenteries of World War II and therefore warrants separate consideration.

The crude rate for dysentery of all forms for the war years was 3.59 cases per 1,000 average strength per annum. Troops stationed overseas (40 percent of the total Army average strength) accounted for 81,462 cases or 88.9 percent of the total number of cases. Morbidity reports reveal that of the total number of cases of diarrheas and dysenteries as a group that occurred in the Army during the war years 75 percent occurred in troops located overseas. In other words, among troops overseas, there was more diarrheal disease and also a relatively higher proportion of the more serious forms than among troops stationed in the United States. This relation is reflected also by deaths, for 52 (81 percent) of the 64 deaths ascribed to dysentery (bacillary, amebic, and unclassified) occurred among admissions in overseas theaters (table 56).

Presumably, some deaths from amebic dysentery in continental United States would be allocated properly to an overseas theater on the basis of place of exposure rather than place of admission.

Four men in every 1,000 were afflicted with recognized bacillary dysentery some time during the 4 war years, for incidence was 1 per 1,000 average strength per year. Similarly, 5 men per 1,000 had unclassified dysentery and 5 per 1,000, amebic dysentery. Incidence for unclassified dysentery was 1.25 per 1,000 average strength per annum (table 61) and for amebic dysentery 1.33 (table 60). The frequency of amebic dysentery increased each successive war year in every theater population except the Middle East. On the other hand, the incidence of bacillary dysentery reached a peak early in the history of each theater, commonly 1943, in general coinciding with maximum numbers of unacclimated troops (table 59). Thereafter, rates were regularly less except in theaters with annual rates less than 1 per 1,000 per annum, where a slight increase was the rule. Unclassified dysentery tended to follow the same pattern except that highest rates were at the inception of the theater for the Pacific and Middle East, with a lesser frequency each succeeding year. Troops when first introduced into hyperendemic environments tended to contract bacillary dysentery or unclassified dysentery promptly, a feature also true of the common diarrheas. Amebic dysentery differed in that increasing incidence was associated with prolonged exposure.

Cases of dysentery, all forms, approximated 92,000 for the total Army in World War II (tables 59, 60, and 61). In continental United States, bacillary dysentery had the highest incidence of the three dysentery groups followed in order by amebic dysentery and unclassified dysentery, the latter two showing little difference. By contrast, cases overseas and for the total Army were in reversed order with respect to bacillary dysentery, so that amebic, unclassified, and bacillary dysentery represented, in that order, 37, 35, and 28 percent of dysentery cases in the total Army and 38, 36, and 26 percent of the dysentery cases overseas.

Because recognition of amebic and bacillary dysentery required identification of the specific agents, the percentage of unclassified dysenteries would be expected to exceed the other two except in areas where dysentery was infrequent or where good laboratory facilities were available. These conditions were met in the continental United States with the result that 43 percent of the dysenteries were diagnosed as bacillary and only 24 percent as unclassified. Conversely, where incidence was high and laboratory facilities deficient, the proportion of unclassified dysentery exceeded the other two, as in several overseas theaters; 36 percent of dysenteries occurring overseas were unclassified, and only 26 percent were determined as bacillary despite the presence of several large epidemics.

The greater simplicity of stool examinations for ameba, in contrast to determinative bacteriologic techniques, would suggest that more amebic dysentery would be recognized than bacillary under conditions of less than average laboratory service. However, this did not hold true. The first 2 war years saw

TABLE 59.—*Incidence of bacillary dysentery in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records of primary and secondary diagnoses among excused-from-duty patients]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942-45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	4, 330	0. 29	770	0. 29	2, 250	0. 43	685	0. 17	625	0. 21
Overseas:										
Europe	1, 948	. 44	36	. 43	32	. 12	610	. 36	1, 270	. 54
Mediterranean [1]	4, 173	2. 81	21	. 92	2, 842	6. 22	1, 035	1. 59	275	. 78
Middle East	2, 123	14. 52	124	20. 51	926	17. 46	738	15. 97	355	8. 19
China-Burma-India	3, 906	8. 91	35	4. 00	612	15. 45	1, 914	11. 35	1, 345	6. 07
Southwest Pacific	6, 065	3. 30	174	2. 44	1, 367	7. 20	1, 714	3. 18	2, 810	2. 71
Central and South Pacific	2, 803	2. 23	676	4. 48	664	2. 28	838	1. 91	625	1. 66
North America [2]	39	. 08	10	. 10	13	. 07	6	. 05	10	. 15
Latin America	316	. 83	98	. 96	75	. 62	83	. 97	60	. 82
Total overseas [3]	21, 485	2. 00	1, 174	2. 00	6, 559	3. 89	6, 977	1. 83	6, 775	1. 46
Total Army	25, 815	1. 01	1, 944	. 60	8, 809	1. 28	7, 662	. 98	7, 400	. 98

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

TABLE 60.—*Incidence of amebic dysentery in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records of primary and secondary diagnoses among excused-from-duty patients]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	3, 400	0. 23	289	0. 11	460	0. 09	621	0. 16	2, 030	0. 71
Overseas:										
Europe	1, 622	. 37	-------	0	22	. 08	610	. 36	990	. 42
Mediterranean [1]	1, 844	1. 24	11	. 40	335	. 73	1, 008	1. 55	490	1. 38
Middle East	1, 176	8. 04	29	4. 80	606	11. 42	391	8. 46	150	3. 67
China-Burma-India	10, 353	23. 61	66	7. 55	628	15. 85	4, 809	28. 50	4, 850	21. 91
Southwest Pacific	12, 048	6. 56	25	. 35	83	. 44	645	1. 20	11, 295	10. 90
Central and South Pacific	3, 098	2. 46	17	. 11	208	. 71	1, 118	2. 55	1, 755	4. 67
North America [2]	24	. 05	2	. 02	5	. 03	7	. 05	10	. 15
Latin America	269	. 71	78	. 77	32	. 26	84	. 98	75	1. 03
Total overseas [3]	30, 534	2. 84	230	. 39	1, 922	1. 14	8, 677	2. 27	19, 705	4. 24
Total Army	33, 934	1. 33	519	. 16	2, 382	. 35	9, 298	1. 19	21, 735	2. 87

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

TABLE 61. *Incidence of unclassified dysentery in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records of primary and secondary diagnoses among excused-from-duty patients]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	2,434	0.17	969	0.36	890	0.17	415	0.10	160	0.05
Overseas:										
Europe	1,007	.23	14	.17	48	.18	440	.26	505	.21
Mediterranean [1]	5,364	3.62	101	4.41	3,714	8.13	1,364	2.10	185	.52
Middle East	1,059	7.24	134	22.16	493	9.29	352	7.62	80	1.96
China-Burma-India	5,385	12.28	91	10.40	703	17.74	3,051	18.08	1,540	6.96
Southwest Pacific	11,818	6.43	760	10.67	1,352	7.12	3,261	6.05	6,445	6.22
Central and South Pacific	4,206	3.35	962	6.37	1,548	5.31	1,031	2.35	665	1.77
North America [2]	115	.23	12	.12	97	.50	6	.05		0
Latin America	338	.89	124	1.22	159	1.32	20	.23	35	.48
Total overseas [3]	29,443	2.74	2,202	3.76	8,156	3.83	9,555	2.50	9,530	2.05
Total Army	31,877	1.25	3,171	.98	9,046	1.32	9,970	1.28	9,690	1.28

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

widespread epidemics of bacillary dysentery overseas, years in which 33 and 39 percent, respectively, of dysenteries were diagnosed as bacillary and only 6 and 12 percent as amebic. With acclimatization or seasoning, rates and percentage distribution of bacillary dysentery decreased progressively. The proportion of amebic dysentery among total dysenteries increased in each successive war year, both overseas and in the United States (because most military personnel were overseas at one time or another). In 1945, 72 percent of dysenteries among troops in the United States and 55 percent in troops overseas were noted as amebic dysentery. There is reason to believe that these relations are to an extent artificial because of increased emphasis upon laboratory diagnosis of amebic dysentery. A preventive medicine officer in the Philippines stated [11] that laboratories were giving undue emphasis to recovery of helminths and ameba and insufficient attention to the bacterial causes of diarrheal disease then epidemic among troops. Stool surveys [12] were made among troops returning to the United States from overseas. Many essentially symptom-free individuals, with organisms resembling *E. histolytica*, were considered cases of amebic dysentery.

As measured by incidence rates for amebic, bacillary, and unclassified dysentery, officers fared worse than enlisted personnel, and female personnel fared worse than male. (Information of this sort is available for 1942 and 1943.) The reasons for these differences doubtless relate to many factors, but the most probable explanation is that more mild cases among officers and women were admitted to hospitals. It is probable also that more effort was expended in establishing an etiologic diagnosis in officers and women.

Race.—In the continental United States, higher rates for amebic dysentery obtained among Negro troops than among white troops. The reverse was true overseas. On the other hand, both in the Zone of Interior and overseas, Negro troops had lower rates than white troops for both bacillary dysentery and unclassified dysentery. Foodhandler examinations, which included stool examination for amebas and helminths but not for bacilli, perhaps account in some degree for this finding. Throughout the war, Negro troops had lower rates for dysenteries and diarrheas than white troops in both the European and Mediterranean theaters. Explanation of these differences is probably to be sought not in any genetic difference in racial susceptibility but rather in differences in the social environment. During the Civil War, Negro troops were more seriously affected with diarrheal diseases than were white troops. Greater exposure of Negro troops to enteric pathogens before World War II Army service and a consequently higher level of immunity in Negro troops than in white troops has been suggested in explanation of these observations. Another possibility is that many Negro troops served within relatively well-sanitated environments.

[11] Memorandum, Preventive Medicine, Southwest Pacific Area, for Deputy Chief Surgeon, 25 Feb. 1945, subject: Diarrheal Diseases of the Southwest Pacific Area.

[12] Survey of Intestinal Parasites in Soldiers Being Separated from Service. Bull. U. S. Army M. Dept. 6: 259–262, Sept. 1946.

DIARRHEAL DISORDERS IN THEATERS OF OPERATIONS AND OTHER OVERSEAS AREAS

Approximately 75 percent of reported cases of diarrhea and dysentery were among the troops located overseas (which comprised 40 percent of the average strength of the United States Army). The crude rate for overseas troops was 40 cases per 1,000 troops per annum (table 54). Table 62 lists the rates in the respective theaters and areas.

All overseas theaters exceeded the rate of 9 for diarrheas and dysenteries, achieved by troops in the continental United States, except the colder Alaskan and North American areas and the tropical but well-sanitated Panama Canal Zone, a subdivision of the Latin American area.

European Theater of Operations, 1942–45 [13]

During the war years, the European theater contained 16.4 percent of the average strength of the total United States Army, and it provided 10.4 percent of the reported cases of diarrhea and dysentery—considerably less than its expected share. The average annual rate for the United States Army in Europe was 14 per annum per 1,000 strength, with 54,196 cases reported (table 54). The rate was lower than for any other overseas theater except Alaska and the North American areas. It exceeded by about one-half the rate for troops in the continental United States, 9 per 1,000 per annum.

TABLE 62.—*Incidence rate of diarrhea and dysentery in the several theaters and areas, 1942–45*

[Rate expressed as number of cases per annum per 1,000 average strength]

Theater or area	Total	Bacillary dysentery	Amebic dysentery	Unclassified dysentery	Dysentery, all forms
China-Burma-India	131.0	8.91	23.61	12.28	44.8
Africa-Middle East	128.0	14.52	8.04	7.24	29.8
Mediterranean	70.0	2.81	1.24	3.62	7.7
Southwest Pacific	67.0	3.30	6.56	6.43	16.3
Pacific Ocean	29.9	2.23	2.46	3.35	8.0
Latin America	19.0	.83	.71	.89	2.4
Europe	13.6	.44	.37	.23	1.0
Alaska	4.9	.08	.05	.23	.4
North America	4.1				

Rates for these diseases in the European theater for the successive war years beginning with 1942 were 17, 12, 13, and 14, respectively, per 1,000 average strength. The rate of 17, the highest for the theater, is related to outbreaks in transports bringing troops to the United Kingdom and to the unsettled conditions attendant on the development of sanitary facilities at new bases in

[13] All material in this section, except when otherwise noted, is taken from Gordon, J. E.: A History of Preventive Medicine in the European Theater of Operations, U. S. Army, 1941–45. [Official record.]

England. The rates for the 2 subsequent years were not far different from those of troops in the continental United States; in fact, the rates for 1943 were the same. The increase which occurred following the invasion of the Continent was not nearly as great as might have been anticipated for troops in combat and engaged in an active war of movement. The rate of 13.6 per annum per 1,000 average strength for American troops in Europe during the interval from February 1942 to December 1945 compares favorably with the rate of 28.9 for these diseases in the American Expeditionary Forces in World War I. The wide difference in the two rates becomes even more significant in the light of the fact that data for World War II include all cases excused from duty (thus excluding outpatients but including quarters as well as hospital cases), whereas the World War I data include only the more seriously ill actually sent to hospital.

Mortality. The theater had one death ascribed to bacillary dysentery and a second to unclassified dysentery. Amebic dysentery caused two deaths (table 63). All four were in 1945. Two deaths were recorded among cases of diarrhea. These six deaths give a mortality rate per 1,000 for all diarrheal

TABLE 63. *Deaths from bacillary, amebic, and unclassified dysentery in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on tabulations of individual medical records]

[Rate expressed as number of death, per annum per 100,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate
	Bacillary dysentery									
Continental United States	2	0.01	—	0	2	0.04	—	0	—	0
Overseas:										
Europe	1	.02	—	0	—	0	—	0	1	.04
Mediterranean [1]	—	0	—	0	—	0	—	0	—	0
Middle East	1	.68	1	16.54	—	0	—	0	—	0
China-Burma-India	2	.46	—	0	2	5.05	—	0	—	0
Pacific Ocean area	2	.16	2	1.32	—	0	—	0	—	0
Southwest Pacific	8	.44	—	0	2	1.05	3	.56	3	.29
North America [2]	—	0	—	0	—	0	—	0	—	0
Latin America	—	0	—	0	—	0	—	0	—	0
Total overseas	14	.13	3	.51	4	.24	3	.08	4	.09
Total Army	16	.06	3	.09	6	.09	3	.04	4	.05

See footnotes at end of table.

TABLE 63. *Deaths from bacillary, amebic, and unclassified dysentery in the U. S. Army, by area and year, 1942–45—Continued*

Area	1942–45		1942		1943		1944		1945	
	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate
Amebic dysentery										
Continental United States	9	0.06	3	0.11	1	0.02	2	0.05	3	0.10
Overseas										
Europe	2	.05	0		0		0		2	.08
Mediterranean [1]	2	.13	0		2	.44	0		0	
Middle East	0		0		0		0		0	
China-Burma-India	2	.46	0		1	2.52	0		1	.45
Pacific Ocean area	1	.08	0		0		1	.23	0	
Southwest Pacific	15	.82	2	2.81	0		1	.19	12	1.16
North America [2]	0		0		0		0		0	
Latin America	1	.26	1	.98	0		0		0	
Total overseas	23	.21	3	.51	3	.13	2	.05	15	.32
Total Army	32	.13	6	.19	4	.06	4	.05	18	.24
Unclassified dysentery										
Continental United States	1	0.01	0		0		0		1	0.03
Overseas:										
Europe	1	.02	0		0		0		1	.04
Mediterranean [1]	1	.07	0		0		1	.15	0	
Middle East	0		0		0		0		0	
China-Burma-India	0		0		0		0		0	
Pacific Ocean area	1	.08	0		0		0		1	.27
Southwest Pacific	11	.60	0		0		3	.56	8	.77
North America [2]	0		0		0		0		0	
Latin America	0		0		0		0		0	
Total overseas [3]	15	.14	0		1	.06	4	.10	10	.22
Total Army	16	.06	0		1	.01	4	.05	11	.15

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes 1 death in 1943 among transport admissions.

diseases of about 0.002; this rate is amazingly low as compared with experience of former wars and is equally surprising when contrasted with the experience of German prisoners of war (table 64). Modern sanitary practice has its reward, not only in man-hours but in fewer deaths.

TABLE 64.—*Incidence and rates of diarrheas and dysenteries, enemy prisoners of war, European Theater of Operations, Continent only, September 1944 to June 1945*

[Rate expressed as number of cases per month per 1,000 strength]

Month and year	Cases	Rate
1944		
September	360	145. 3
October	1, 070	184. 5
November	2, 215	142. 3
December	2, 367	185. 4
1945		
January	1, 923	120. 6
February	2, 198	123. 3
March	3, 066	101. 8
April	4, 713	74. 6
May	80, 952	734. 6
June	60, 978	326. 9
Total	159, 842	349. 2

Monthly and seasonal incidence.—Examination of the successive monthly rates for the European theater (chart 30) reveals no excess rates regularly associated with any particular season. Peaks of incidence seem to be associated more with other factors than with those strictly related to climate and season; such factors as movement of troops and interruption of sanitation discipline by field and combat conditions seemed to be the main influence.

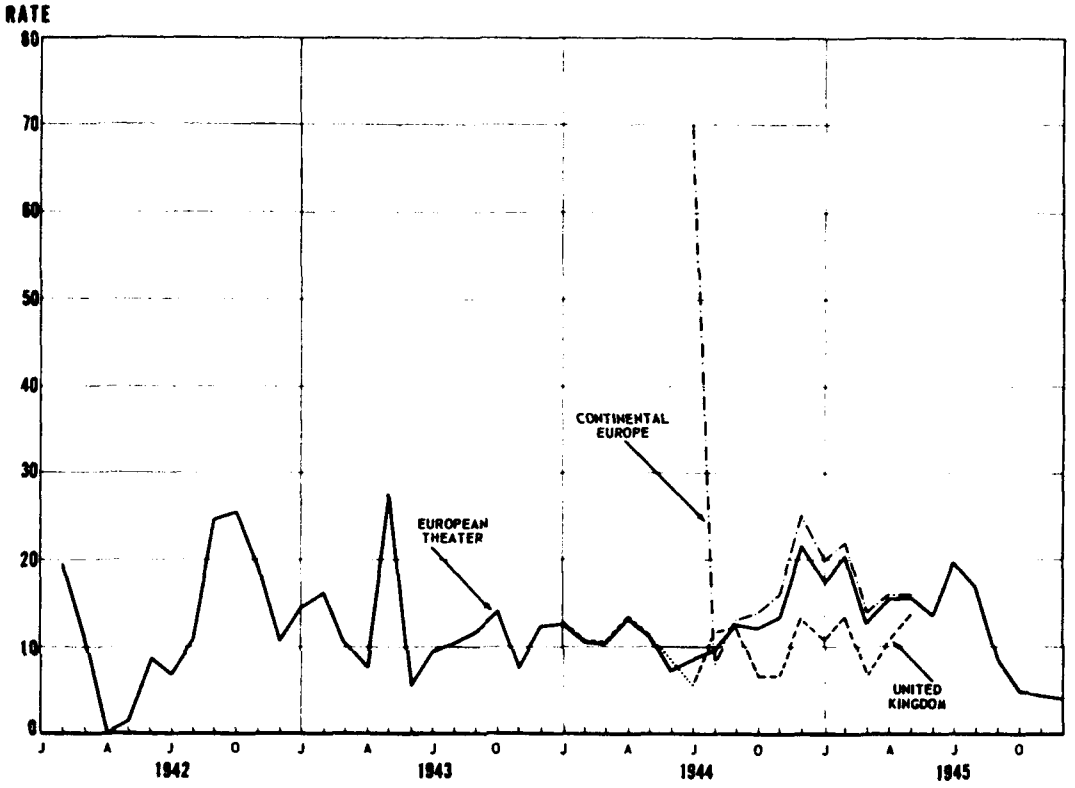
As in other theaters, sporadic cases occurring more or less continuously comprise a considerable proportion of the diarrhea and dysentery cases. Peaks in curves of incidence were usually related to groupings of cases to give epidemics of varying size. The frequency and size of the latter are important in determining whether the total experience of a theater is good or bad. The European theater was particularly fortunate with relation to both frequency and size of such occurrences in that, during the 4 years, there was only a total of 5 months in which the rates slightly exceeded 20 per 1,000 per annum (chart 30).

Intestinal infections during operations in the United Kingdom.—In Great Britain, the sporadic occurrence of diarrheas and dysenteries was related largely to eating and drinking in civilian establishments, whereas actual outbreaks or small epidemics of diarrhea were most frequently related to faulty sanitation or careless messing practice within the affected unit. Most of the unit outbreaks were foodborne.

CHART 30.—*Incidence of diarrhea and dysentery in U. S. Army troops in total European Theater of Operations, and United Kingdom,[1] and continental Europe,[2] 1942–45*

[Preliminary data based on periodic summary reports]

[Rate expressed as number of cases per annum per 1,000 average strength]



[1] Data are not available for United Kingdom after May 1945.

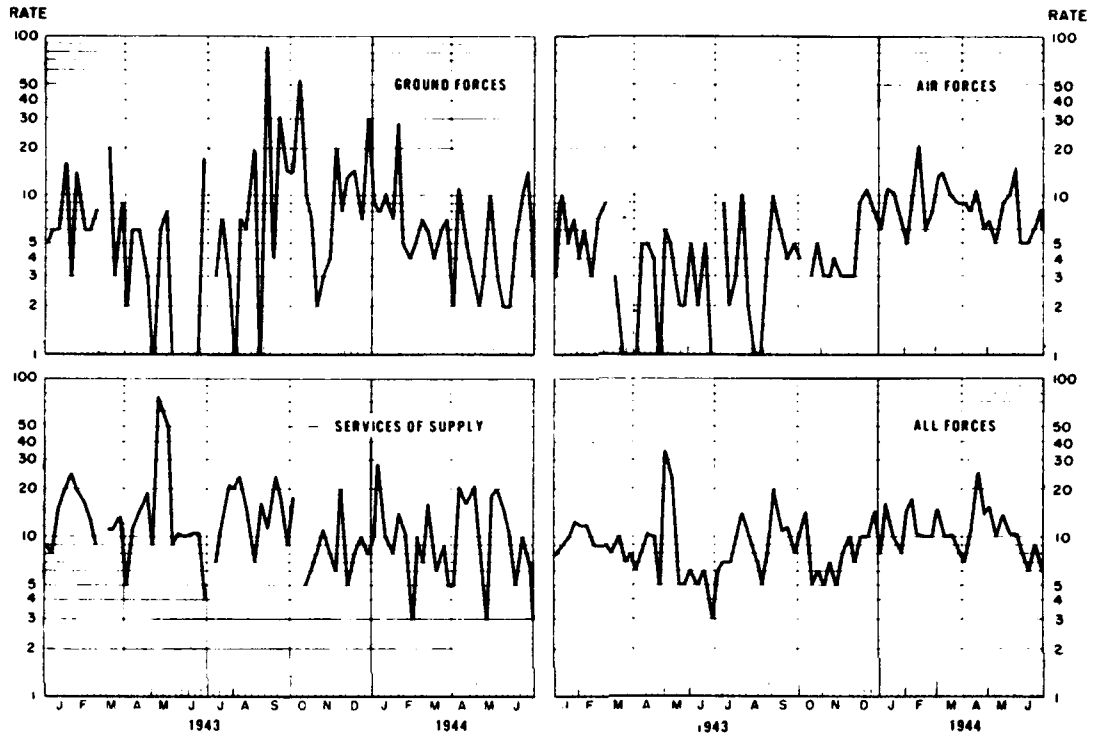
[2] Data are available for continental Europe only from July 1944 through May 1945.

The three major forces of the United States Army present in the United Kingdom experienced somewhat different frequencies of diarrheal disease. The Services of Supply experienced rates consistently higher than those of Ground Forces, and the Air Forces had the best record (chart 31). The Services of Supply conducted an intensive food-conservation program which resulted in foodborne outbreaks through use of leftover foods; this, coupled with other factors such as lack of refrigeration and somewhat lower standards of mess sanitation, accounted for their higher rates.

In chart 31, which shows the incidence of diarrheal disease, the graph pertaining to the Air Forces contains the fewest sharp peaks of weekly or monthly incidence. The mess sanitation and personnel of the Air Forces were more adequate in general, and there was less inclination on the part of Air Force personnel to conserve food. There was also less sporadic diarrheal disease among this personnel, which is accounted for by the fact that their stations were relatively isolated, and there was less opportunity to patronize civilian eating establishments.

CHART 31.—*Intestinal infections, including common diarrhea and dysentery in the U. S. Army in the United Kingdom, Ground Forces, Air Forces, Services of Supply, and all forces, by weeks, 1 January 1943 to 30 June 1944*

[Rate expressed as number of cases per annum per 1,000 average strength]



The Ground Forces during 1943 experienced a number of foodborne outbreaks, some large, especially in August, September, and October. Early 1944 saw an improvement; concentration of troops in mobilization areas took place, and special emphasis was laid upon sanitary mess management. The result was virtual elimination of major foodborne epidemics, and the incidence rapidly declined to a level even lower than that for the Air Forces.

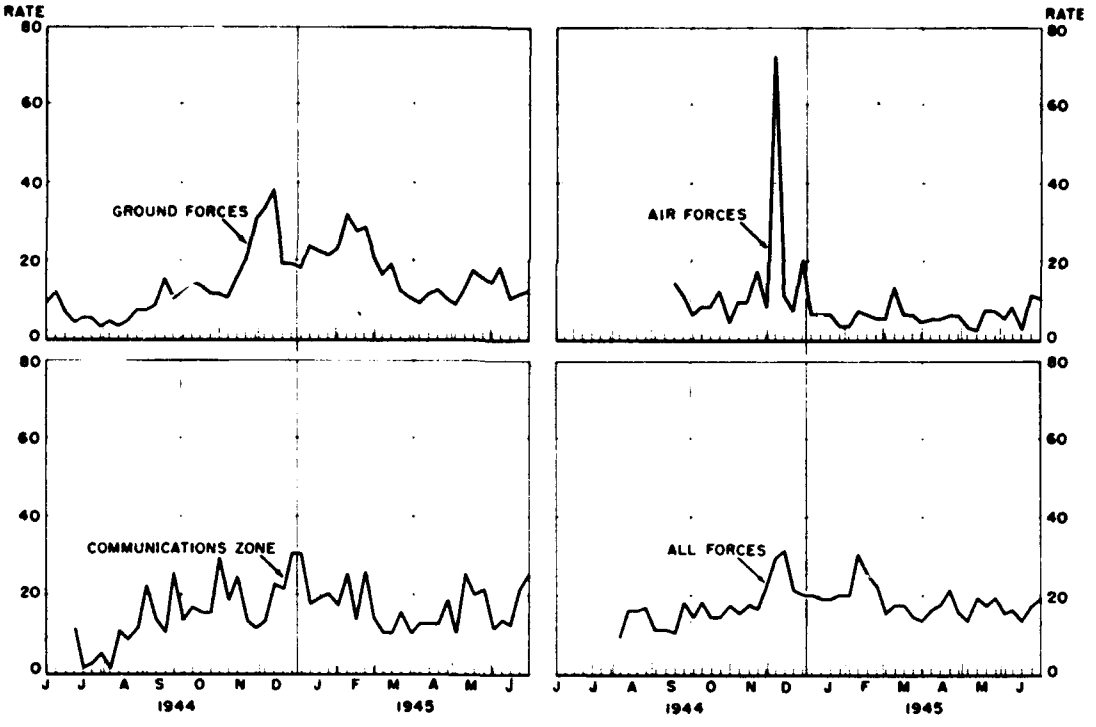
Within the Services of Supply, the Central Base Section experienced consistently higher rates than other sections, presumably because of greater numbers of sporadic cases which were attributed to the opportunity for personnel to patronize eating and drinking establishments in the London area.

Intestinal infections during continental operations.—After the invasion of Europe, theater rates for diarrhea and dysentery rose to a peak of over 20 by December 1944, the increase being largely related to troops in combat under field conditions. Nevertheless, the rates were exceptionally low for troops in active combat. The peak monthly rate (following the artificially high rate of 70 in July 1944) for troops on the Continent (chart 30) was in December 1944, only 25 per 1,000 strength. Troops remaining in the United Kingdom continued to maintain low rates comparable with those of the previous year.

The Ninth Air Force, which was engaged in tactical missions and operated under field conditions, was the principal unit of the Army Air Forces on the Continent. The rates for this organization compare favorably with those of the Eighth Air Force which remained in England on fixed bases and under relatively static living conditions. Ninth Air Force personnel experienced three fair-sized epidemics of diarrheal disease, the sharpest of which occurred in the first week of December 1944. This naturally influenced over-all Air Force rates on the Continent. Notwithstanding, Army Air Force personnel had less diarrhea and dysentery than did personnel of the Ground Forces or Communications Zone (chart 32).

CHART 32.—*Intestinal infections, including common diarrhea and dysentery, in the U. S. Army in continental Europe, Ground Forces, Air Forces, Communications Zone, and all forces, by weeks, 23 June 1944 to 29 June 1945*

[Rate expressed as number of cases per annum per 1,000 average strength]



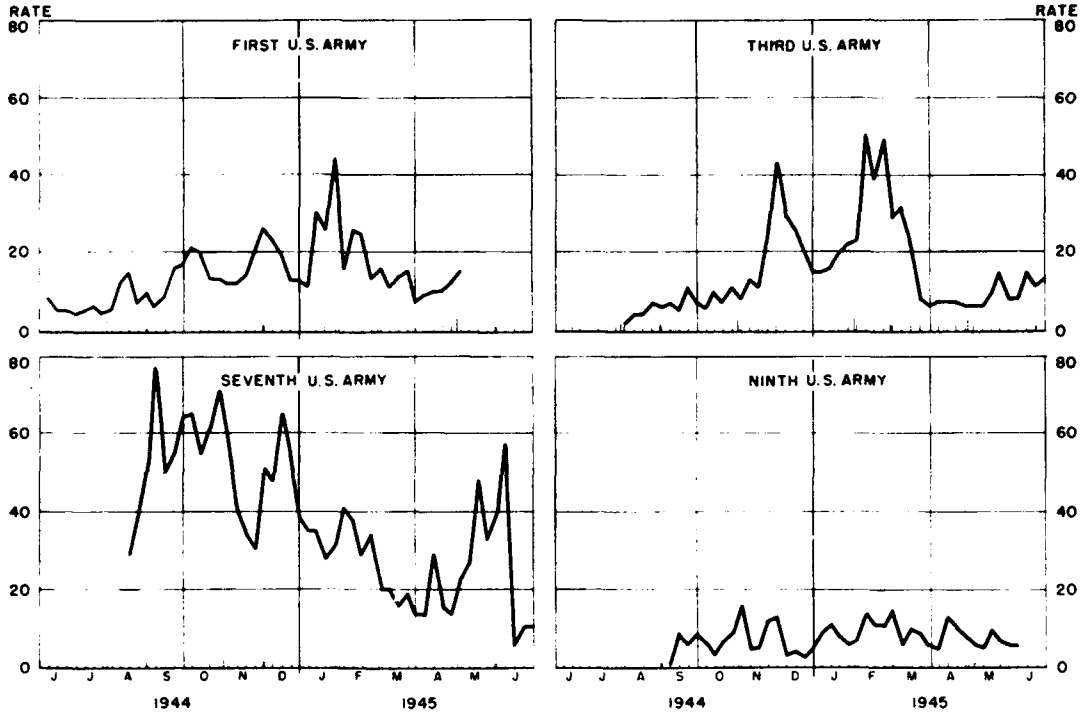
"Ground Forces went through some of the most difficult days of the European campaign with rates for the diarrheas and dysenteries that were almost unbelievably good. The diarrheal disorders were certainly not frequent; in fact, their general absence was a matter of comment." Gordon attributed this good record early in the campaign to the troops' being on C and K field rations which he considers were a first-class preventive against foodborne disease. As diets became more complicated and A and B field rations were used, the rates increased.

Communications Zone personnel maintained a good record, but monthly rates for diarrheas and dysenteries were most irregular, with frequent small outbreaks (chart 32). This is considerably different from the experience of the Ground Forces whose rates increased during the hard fighting in the last quarter of 1944 and the early part of 1945 and then decreased. The increase in rates for the Ground Forces, which started in September and attained its high point of 38 cases per 1,000 strength in the week ending 15 December 1944, reflects the general epidemic wave which involved all the field armies in an extensive and progressive manner. The second peak of about 25 per 1,000 which occurred late in February 1945 reflects heavy fighting in a war of movement in that and preceding months.

Epidemiologic case report 1.—During the history of the European theater, there was only one widespread outbreak of diarrheal disease. It involved large numbers of troops and persisted for several weeks. During the first week of October 1944, the epidemic began among troops of the XIX Corps then located near Maastricth, Holland. At this same time and place, there existed an epidemic of a similar diarrheal disease among the civilian population. The disease spread southward and was reported from several units of the XIII Corps in the middle of October. It passed to the First U. S. Army during November and later in that month invaded the Third U. S. Army (chart 33). During December, it became widespread in the Seventh U. S. Army. The Seventh U. S. Army already

CHART 33.—*Intestinal infections, including common diarrhea, in First, Third, Seventh, and Ninth U. S. Armies in the European Theater of Operations, by weeks, from approximate date units entered combat to date of redeployment* [1]

[Rate expressed as number of cases per annum per 1,000 average strength]



[1] The Third and Seventh U. S. Armies remained in Europe after V-E Day as occupation forces.

had experienced the highest rates for diarrheal disease of any of the armies in the theater, but a marked decrease in rates had occurred during successive weeks in November. The disease causing the December epidemic in the Seventh U. S. Army was recognized to be different in clinical character from diarrheal diseases which hitherto had affected this army.

The epidemiologic observation was made that officers and men were involved alike and that when a unit became involved the peak of incidence built up rapidly within 4 to 8 days and then gradually tapered off during the next 3 or 4 weeks. This was not suggestive of foodborne or waterborne diarrheas. Spread seemed to occur by steady progression or extension. In each affected unit, the incidence was not extremely high at any one time, although before the epidemic had run its course the majority of members were afflicted. As an example, at the general dispensary in Luxembourg there were 187 patients treated during November and the first 2 weeks of December. Yet 30 was the greatest number treated on any 1 day. It was known that prevalence was much greater than reported figures indicate, since many did not report to sick call and only a few of these were admitted to hospital.

The clinical observation was made that the typical case was definitely mild. There was sudden onset with abdominal cramps and from 4 to 8 watery stools within 24 hours. Symptoms persisted for 1 or 2 days, rarely longer. Nausea and vomiting were not typical, but anorexia was, and it persisted for a few days. Blood, pus, and mucus were not present in the stools. Fever was seldom a symptom and rarely exceeded 100° F. There was no leukocytosis. In all units except those of the Third U. S. Army, coryza typically preceded the diarrhea by about 2 days, occurred concomitantly, or developed shortly after the diarrhea.

Laboratory observations were made during the October outbreak in the XIX Corps and later at Luxembourg. No enteric pathogens were recovered. However, laboratory studies on the more seriously ill patients of at least one unit of the Third U. S. Army revealed many positive cultures for Flexner bacilli. This particular outbreak was definitely shigellosis. In other units of this army, *Shigella* were recovered with increasing frequency as the epidemic progressed, and were considered to be responsible for many of the mild cases during this widespread episode. In the Seventh U. S. Army, *Shigella* were recovered from about 15 percent of stools examined by a laboratory which had had much experience with dysentery in North Africa. The serologic type distribution was as follows:

Shigella paradysenteriae:	*Number*
Flexner I	1
Flexner II	4
Flexner III	2
Flexner V	9
Flexner VI	1
Boyd IV (P 274)	1
Shigella ambigua (schmitzii)	4
Shigella sonnei	2

In none of the laboratories was *Salmonella* recovery significa[illegible]

That epidemiologic history repeats itself is suggested by the resemblance of this epidemic to the World War I epidemic of diarrhea and mild dysenteries among combat troops during the period from 1 July through 30 September 1918. The two were similar clinically and in apparent mode of spread. Both occurred during a peak of military endeavor under conditions requiring marked emphasis on field sanitary discipline.

This epidemic, although traceable from one unit or army to another in a contiguous manner, probably represented a collection of epidemics of diarrheas and mild dysenteries rather than a single entity as usually thought of in terms of a single etiologic agent. Causes of the southward spread of this epidemic were inherent in the numerous opportunities for breaks in field sanitation discipline in armies under combat conditions.

Outbreaks on troop transports.—Within a period of a few weeks, during August and September 1943, several sharp outbreaks of diarrheal disease

occurred on transports carrying troops to Europe. At the time, crowding of troops on transports was extreme because of the rapid buildup in strength necessary for the coming operations in France. During one crossing, the transport *Argentina* had approximately 3,000 cases. Other transports on which excess diarrheal disease occurred were the *Shawnee*, the *Cristobal*, and the *Capetown Castle*.

Troops had not been specifically trained for the shipboard conditions they encountered and had difficulty accommodating themselves to this new restricted environment. Medical and line officers were frequently inexperienced, as were some of the transport commanders. Messing and toilet facilities were inadequate for the numbers of troops carried. Opportunities for contact infection and messhall spread of diarrheal disease were obvious.

Laboratory facilities aboard these vessels were so inadequate that the specific etiologic agents responsible for these outbreaks were not determined in any instance. However, at least one was suggestive of bacillary dysentery because laboratory studies in England after troops disembarked resulted in recovery of *Sh. sonnei* from a few residual cases of diarrhea.

Most of the diagnoses were within the common diarrhea category, and only one death occurred among the thousands of cases recorded.

Control of this epidemic situation depended upon alleviating the overcrowded conditions of troop transports. The measures to be applied had to originate in the Zone of Interior, at ports of embarkation, and at staging areas. Special reports from the European theater supplemented the routine reports of transport medical officers. Appropriate measures were instituted at ports of embarkation; furthermore, the military situation no longer required overcrowding. The result was that only eight instances of excessive rates for diarrhea occurred in 378 troop movements overseas to the European theater during the first 5½ months of 1944. None were serious.

In June 1944, the *Nieuw Amsterdam* experienced a severe outbreak. Because of the high attack rate on this vessel and of the fact that the summer season was at hand with the possibility of a seasonal upswing in incidence, and because the military situation required increasing numbers of troops, the aid of authorities in the Zone of Interior was again sought. Control measures instituted gave definite results because only one other epidemic of serious proportions occurred thereafter, in August 1944 on board the *Mariposa* which was seriously overcrowded with 9,326 troops among whom over 2,000 were reported to have had a diarrheal disorder.

Epidemiologic case report 2.—The largest single outbreak of diarrheal disease during the war years in the European theater occurred aboard the *Argentina* during the crossing which ended at Glasgow, Scotland, on 4 September 1943, after sailing from New York about the middle of August. Approximately 3,000 patients reported to sick call, among the 6,153 Negro troops, ship's company, and naval gun crew. Many other milder cases occurred. One soldier died of acute diarrheal disease. The microbiologic agent involved was never determined. Some of the more acutely ill who were taken ashore at Glasgow were examined, and stool cultures were made. Cultures were reported negative for common enteric pathogens.

The ship was grossly overcrowded, soldiers were required to sleep in shifts, and the messes were constantly serving meals from galleys which at the outset had been reported by the transport surgeon to have numerous sanitary deficiencies. Facilities for washing and sterilizing messgear were inadequate. Furthermore, toilet and bathing facilities were inadequate for such a large number of passengers.

The description by the port physician at Glasgow is quoted as follows: [14]

The first impression was of gross overcrowding; the second, of filth and dirt everywhere. A long queue of soldiers waited outside the door of the dispensary, some so weak they had to sit or lie on the deck. The epidemic of dysentery had begun 4 days out of New York. All galleys, including that of the Navy gun crew and the civilian crew, were in a deplorable state of sanitation. The main troop galley was the worst of the lot. The supposedly clean kitchen utensils had dirt, grease, and decaying food stuck to them. The stoves and tables were very dirty with old burnt grease and decayed food particles. The garbage disposal was extremely unsanitary. Garbage cans were coated with decaying organic matter and had not been washed for a long time. They were piled helter-skelter in a corner of the kitchen, and their contents were spilled all over the deck. The troops were messing in the main messhall. Half-eaten food was thrown over the tables and floor. In fact, the floor was so covered with waste food that one had to be careful not to slip and fall. The garbage cans just outside the main messhall were the most nauseating scene I have seen on board a ship. Half a dozen soldiers were standing in garbage 2 to 3 inches deep around 5 or 6 garbage cans filled to overflowing with uneaten food. Sterilization and washing of trays was unsatisfactory. There was not enough soap in the water and the water was not hot enough to really clean the trays in the very brief period that they were immersed. Inspection of the rest of the ship troop quarters, decks, and latrines revealed conditions just as unsanitary as in the kitchens. The troop quarters reek with vomitus and diarrheal fecal matter from those who were ill and could not find a place in the latrines to relieve themselves. The latrines themselves were beyond description. I can truly say that I have never seen a United States Transport in such deplorable sanitary condition.

Epidemiologic case report 3. An epidemic occurred on the transport *Shawnee* on 10 September 1943 that lasted for 2 days and subsided by the time the ship docked at Bristol, England, on 15 September. Of the 1,843 military personnel aboard, 362 cases of diarrhea were reported. The transport surgeon observed that foodhandlers of the ship's permanent personnel were unaffected and that they ate their food in the galley. Although inspection revealed no obvious contamination of food, he believed that it had become contaminated somewhere between the galley and the messhall. He further observed that kitchen police of the 2190 S Task Force had been relieved by men of the 2190 K Task Force on 9 September, the day before the epidemic started. He believed that carriers existed in the 2190 K Task Force kitchen police and returned the other group to duty. Whether or not his hypothesis was correct, the fact remains that no additional cases occurred after the change in foodhandlers.

Epidemiologic case report 4. An outbreak of diarrheal disease on the transport *Cristobal* occurred in association with the reported fact that there were only 200 compartment steel trays available to serve 3,000 troops at the rate of from 700 to 900 servings per hour. Facilities for washing and sterilizing even this small number of trays at such a rate of speed were unsatisfactory.[15]

Epidemiologic case report 5. An epidemic of 170 cases of diarrheal disease occurred among troops on the transport *Capetown Castle* on the third day of a transatlantic crossing, and 20 additional cases occurred on the fourth day. The ship's surgeon believe the out-

[14] Letter, Port Medical Inspector, to the Port Surgeon, 5 Sept. 1943, subject: Sanitary Report of USAT Argentina.

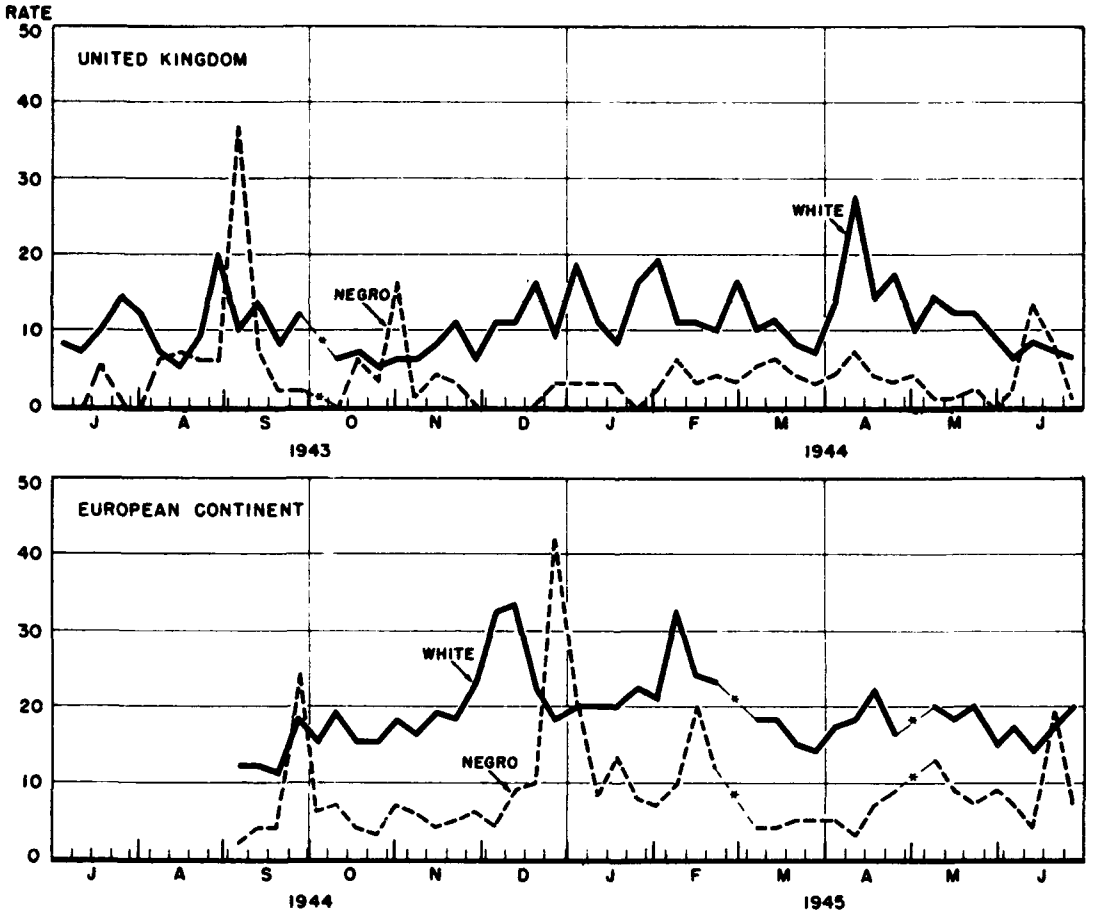
[15] Letter, Deputy Base Surgeon, Eastern Base Section, to Chief Surgeon, European Theater of Operations, U. S. Army, 13 Sept. 1943, subject: Outbreak of Diarrhea Aboard the U. S. Army Transport "Cristobal."

break was related to inadequacies in the methods of washing messgear. Accordingly, chlorine was added to the final rinse water. No further difficulty occurred during the voyage.

Diarrheal disease by racial groups.—Negro troops in World War I had less reported diarrheal disease than did white troops. In World War II, in the European theater, the rates (chart 34) among Negro troops were generally lower than those for white troops. This was true both in the United Kingdom and on the Continent. The rates were lower too among Negro troops in the continental United States and in at least one other theater of operations where separate data are available.

CHART 34.—*Intestinal infections, including common diarrhea, in Negro and white U. S. troops in the United Kingdom and on the European Continent, by weeks, 9 July 1943 to 29 June 1945*

[Rate expressed as number of cases per annum per 1,000 average strength]



*Data are not available.

Diarrheal diseases among enemy prisoners of war.—In the European theater, the most serious problem with the diarrheas and dysenteries occurred not among American troops but among enemy prisoners of war.

Rates for diarrheas and dysenteries for prisoners during the last quarter

of 1944 and the first few months of 1945 were roughly 10 times as great as those for United States troops. Extensive crowding into cages under the emergency conditions associated with the often sudden reception of a large number of prisoners favored spread of the infectious agents of intestinal disease. Deficiencies of sanitation and inadequacies of messing facilities, which seem inevitably to occur during such operations, provided additional impetus to the diarrheas and dysenteries. That the rates were not higher is due largely to the policy of not retaining prisoners on the Continent any longer than necessary. They were removed to Great Britain or to the Americas, where diarrheal disease did not constitute a major problem among them.

The main problem existed among the prisoners in the enclosures on the continent of Europe. When the number of prisoners taken was great, overcrowding was increased, and sanitation breakdowns were frequent. Under these conditions the rates went up. The high rate for December 1944, 185.4 per 1,000 prisoners per annum (table 64), was associated with the reception of large numbers taken at the Battle of the Bulge.

Improvement in rates occurred thereafter until April 1945 when the rate was 74.6. Then came the end of the war in the first week of May. Tremendous numbers of prisoners had to be accommodated in hastily prepared facilities because those already provided were hopelessly insufficient. Men were crowded into enclosures which at first were little more than cages. Water supplies were safe but were not sufficient at many enclosures; messing and kitchen equipment was inadequate as were methods for sterilizing messgear. In some enclosures, men slept in pup tents, foxholes, improvised shelters, or in the open. Flies were prevalent, although DDT, when available, was used to suppress them.

Unprecedented rains fell in several of the enclosures, which led to much mud and thoroughly chilled and soaked the prisoners. In this setting, an epidemic of dysentery broke out but was confined to the prisoners. In some of the larger camps, two or three thousand persons were sick at the same time.

In May 1945, 80,952 cases of diarrhea and dysentery occurred, and rates soared to 734.6 per 1,000; the majority of cases were in the first 2 weeks of that month. In June, 60,978 cases occurred, and the rate dropped to 326.9 per 1,000.

Hospitals had been established to take care of seriously ill patients. As rapidly as possible, water supplies were increased, and messing and sanitary facilities were improved. Before the end of May, remarkable accomplishments in prison-enclosure sanitation had been made. But dysentery had taken over with a speed greater than that necessary for the institution of proper preventive measures.

The clinical severity of the disease in this epidemic is illustrated by the 833 deaths among that particular group of 403,142 prisoners who were under the jurisdiction of the Advance Section, Communications Zone, during the 6-week period from 1 May to 15 June 1945.

Clinical, epidemiologic, and laboratory evidence suggest that a great

proportion of these cases were bacillary dysentery. Several strains of the Flexner type of dysentery bacilli were isolated.

Common diarrheas.—Over 90 percent of intestinal infections in the European theater were reported under one or another of the diagnostic titles included in the group of common diarrheas. During the 4 war years, there were approximately 50,000 cases, or 12 cases per 1,000 men per annum. The higher rates (over 20 per 1,000 per annum) in the last quarter of 1942 were related to Operation TORCH, and the increase in the spring of 1944 was related to the concentration of troops in southern England in preparation for the Normandy invasion. Contrary to expectation, the Normandy invasion itself did not result in a reported increase, but the Battle of the Bulge and operations thereafter in the winter of 1944–45 did result in some of the highest rates of the theater experience.

Analysis of sources of infections in 154 outbreaks of common diarrhea, occurring in the theater during the war years and recorded in sanitary reports or through special epidemiologic studies, revealed that 67 outbreaks had been ascribed to contaminated foods, 13 to impure water supplies, and 28 to unsanitary conditions in messes. For 46 of these reported outbreaks, the causative factor was undetermined. Thus, in the European theater, the causes of outbreaks were most frequently related to food, and only 12 percent of the outbreaks of determined cause were related to water.

As mentioned elsewhere, food poisoning became separately reportable in the United States Army in 1944. The distinction between foodborne common diarrhea and food poisoning is more or less artificial, and there is little doubt that many foodborne outbreaks continued to be reported as common diarrhea. The terminology perhaps implied to the reporting medical officers a more careful consideration of cause. If laboratory studies revealed either an actual pathogen or a suspected pathogen, the outbreak was more likely to be reported as food poisoning. If no laboratory studies were made or if studies made were negative, the outbreak often was reported as common diarrhea. Nevertheless, epidemiologic considerations are the same in both instances. Transmission of infection by food was the most common mechanism in the common diarrheas.

Another chapter of this history is devoted to food poisoning, so that attention is now directed to waterborne common diarrhea. True waterborne outbreaks of common diarrhea typically involved a large proportion of the exposed military population. The onsets were sudden, and the outbreaks terminated promptly. They usually involved engineer construction units, combat troops in the field, or other units using temporary or untested emergency water supplies.

Three epidemiologic case reports illustrate this relatively rare type of outbreak:

Epidemiologic case report 6.—On 27 September 1943, the 2d Battalion, 347th Engineer General Service Regiment, with a strength of approximately 900 men, experienced the beginning of an epidemic when 31 men were seized with nausea or vomiting, malaise, intestinal cramps, or diarrhea. All were afebrile. On 28 September, 102 others reported at

sick call, and on the following day 29 new cases occurred. More than half of the men of the battalion were mildly ill, and only the more severely ill reported to sick call. Most of the patients recovered within 24 to 36 hours.

At that time, the battalion was constructing a large camp in Great Britain, and the permanent water supply was to be drawn from a river. A chlorination and filtration plant had just been completed but was not yet working properly. As a result of a misunderstanding, the water from this new source was turned on at a time when raw water actually passed into the distribution system for only a few hours in the morning of 26 September. Lemonade made from this water was served in the messhalls.

Samples of water from the distribution system taken on 27 September were subsequently shown to be heavily contaminated with colon bacilli. The use of the water was permitted but only after collection in Lyster bags and chlorination; otherwise the outbreak might have been more extensive.

Epidemiologic case report 7.— In September 1943, troops of Site B of the 127th Army Air Force Station experienced a small outbreak entirely limited to troops at that site and not extending to other parts of the station. The site became contaminated by the overflow of sewage which was caused by the failure of a sewage pump. Drinking water was subsequently found to be nonpotable, whereas the supply to other sites on the station was safe. Epidemiologic investigation eliminated food as a likely cause of the diarrhea. Emergency measures were taken with reference to the water, and the outbreak subsided promptly.[16]

Epidemiologic case report 8.—The 2d Chemical Mortar Battalion in France moved to a new location and utilized a French water point as their source of supply. The water used was found to be nonpotable and nonchlorinated. Chlorination was instituted and the outbreak, which already had involved more than one-fourth of the battalion, ceased.[17]

The dysenteries.—Of the 54,196 cases of diarrheas and dysenteries (table 54) which were reported during the war years, approximately 8 percent were reported as dysentery of one form or another. Army regulations required that a specific infectious agent be recovered before a case received either a diagnosis of bacillary dysentery or amebic dysentery. The diagnosis of dysentery, unclassified, was used if clinical criteria alone were used. Examination of morbidity report data revealed that this category comprised the greater proportion of cases. Bacillary dysentery was next most frequent, and amebic dysentery was of lowest reported incidence. The rate in this theater for dysenteries, all forms, was slightly over 1 per 1,000 strength per year. For unclassified dysentery, the rate was 0.5; for bacillary dysentery, 0.4; and amebic dysentery, 0.3.

When incidence figures (tables 59, 60, and 61) based upon sample tabulations of individual medical records for both primary and secondary diagnoses among excused-from-duty patients are used, the order is changed. Bacillary dysentery takes the lead with 1,948 cases tabulated, representing a rate of 0.44 per 1,000 strength per annum. Amebic dysentery was next most frequent among these more severely ill patients with 1,622 tabulated cases, or 0.37 case per 1,000 per annum. Unclassified dysentery was least frequent with 1,007 tabulated cases, or 0.23 case per 1,000 per annum.

Thus, it is apparent that diagnoses were changed with considerable

[16] Epidemiological Report, Army Air Force Station 127, 18 Sept. 1943, subject: Epidemic of Gastrointestinal Disease.

[17] Letter, Office of the Surgeon, 2d Chemical Battalion, to Commanding General, European Theater of Operations, U. S. Army, 1 Dec. 1944, subject: Monthly Sanitary Report.

frequency after admission and laboratory study and that in this process cases were added to the bacillary dysentery and amebic dysentery categories and were subtracted from the unclassified dysentery category.

Since amebic dysentery is discussed elsewhere in this volume, it is sufficient to state here that a large proportion of the cases diagnosed as amebic dysentery were in patients who previously had been stationed in the Mediterranean theater and that this portion of the cases represented relapses of previous infections. It was believed that relatively little endemic infection existed in the civilian populations of Great Britain and France and that primary infection in this theater was not frequent.

Bacillary dysentery tended to occur under combat conditions. This tendency is typical of experience in many wars. Under such conditions, laboratory diagnosis becomes difficult, and the result is that many cases fall into the unclassified group or are diagnosed with the common diarrheas, the latter diagnosis being used especially for the milder cases.

During the period from January 1944 to June 1945, 1,054 cases of bacillary dysentery were reported. This represented approximately 30 percent of all forms of dysentery reported on morbidity reports during that period. It is almost certain that a large proportion of unclassified dysentery was of bacillary etiology. Together these forms represent about 75 percent of the dysenteries. Incidence data (table 59) based on sample tabulations of individual medical records reveal that there were 1,948 cases of bacillary during the 1942–45 period, representing an incidence rate of 0.44. The highest annual incidence rates occurred during the last year in association with continental operations.

There was little clear-cut evidence of a typical seasonal distribution. The monthly rates were higher in association with troop movements or combat interruptions in sanitary disciplines.

No extensive outbreak is included in the list of confirmed epidemics of bacillary dysentery which came to the attention of the theater epidemiologic service. The largest epidemic involved 183 patients at Camp Northway in the Ash Church garrison area in England and was caused by a Flexner dysentery bacillus.

Among the outbreaks of diarrheal disease which occurred on troop transports, several of which were suggestive of bacillary dysentery outbreaks, there is some evidence to confirm the suggestion and also to confirm the belief that transport outbreaks resulted in epidemic spread which continued to affect the incidence in the European theater.

Epidemiologic case report 9.- On 24 January 1943, at a staging area in England, a suspected case of bacillary dysentery was confirmed by the finding of *Sh. sonnei* in the feces. During the next 3 weeks, 54 similar cases occurred of which 19 were confirmed as Sonne dysentery by microbiologic methods. These cases occurred among a shipment of 1,000 men who arrived at the staging area directly from a troop transport which had brought them to England. Dysentery had not been previously recognized at this area nor had any outbreaks of diarrheal disease occurred. Furthermore, the men had been restricted to the camp area since their arrival on 12 January. Questioning revealed that one man, at least, had had acute diarrhea and was mildly ill for 3 days aboard the transport but had not

reported his illness. Thirty-six foodhandlers among the group were examined soon after the outbreak began. One was found to be a carrier of the Sonne dysentery bacillus.

The outbreak did not spread at this time to other units in the staging area. It was considered to have started on the troop transport, and the foodhandler carrier was considered to be the immediate source of the infection at the staging area.

Most of the recorded outbreaks of confirmed bacillary dysentery involved small numbers of cases, usually fewer than 50. Many confirmed cases seemed to occur in small groupings of three or four at a time.

The following is illustrative of a foodborne outbreak of this disease:

Epidemiologic case report 10.—The headquarters mess of Services of Supply at Cheltenham, England, housed about 40 senior officers of the theater. The attendants were mainly British civilians. An outbreak of diarrheal disease among the domestic staff began about 1 February 1943 and ultimately involved 15 persons, 3 of whom were resident officers. Bacteriologic examination of the stools of 11 foodhandlers revealed 2 carriers of *Sh. sonnei*. Removal of the carriers served to terminate the outbreak.

Another example of a clear-cut foodborne outbreak is that of 23 cases among officers and nurses of the First Medical General Laboratory, caused by contaminated tomato juice served at breakfast on 10 March 1945. *Sh. paradysenteriae* of the Boyd 103 type were recovered.

In one instance in this theater, an outbreak was clearly attributed to water. The same Shigella organism was reported to have been recovered from patients and from the water itself.

Sh. sonnei was most frequently encountered in this theater, and *Sh. paradysenteriae* (Boyd 103) and *Sh. ambigua* were also frequently identified. Most laboratories limited their examination to species differentiation.

Mediterranean Theater of Operations

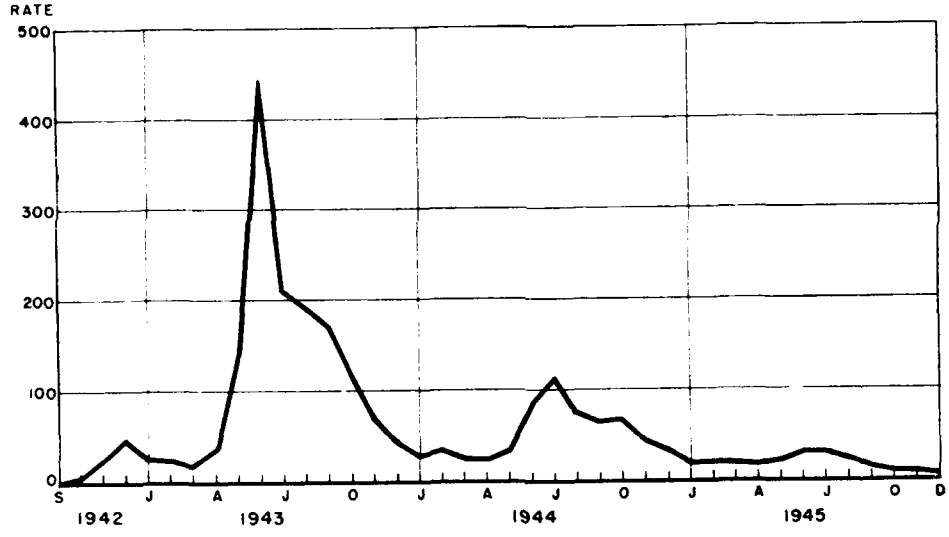
The "filth diseases of man" have been prevalent for generations in most of the area which came to be known during World War II as the Mediterranean Theater of Operations. The destruction of war added to sanitary problems in those parts of the theater which had been better sanitated. The absence of sanitary facilities among civilian populations, especially in North Africa, was general. The habit among natives of North Africa of promiscuously depositing feces upon the ground, coupled with swarms of flies present during the summer, provided ideal conditions for the dysenteries. A little less than 6 percent of the total average strength of the Army was concentrated in this hyperendemic environment, and this theater contributed almost 20 percent of the total cases of diarrhea and dysentery during the war years. One-fourth of the cases in overseas troops were in this theater. Among the overseas theaters of operations, the Mediterranean ranked third in the reported incidence of these diseases, China-Burma-India and Africa-Middle East theaters ranking first and second (table 62). The average strength during 1942–45 was 360,891, and the total number of reported cases was 100,649, a rate of 70 cases per 1,000 strength per annum (table 54).

The preventive medicine officer of the Mediterranean theater observed that the troops landing in North Africa at the opening of the theater in late 1942 and early 1943 were not adequately trained in details of housekeeping and sanitation which must extend down to the lowest echelons to be effective. He stated that "once the forces saw the results of neglect in the diarrhea outbreak which was almost universal in 1943 * * * it was no longer necessary to convince anyone of the importance of sanitation." [18] During June 1943, the rate attained the epidemic high of 445 cases per 1,000 per annum (chart 35), and the outbreak stretched across all of North Africa, from Tunis to Casablanca. This early experience focused attention upon sanitation and preventive measures which resulted in the lowering of rates in subsequent years.

CHART 35.—*Incidence of diarrhea and dysentery in the U. S. Army in the Mediterranean Theater of Operations, September 1942 through December 1945*

[Preliminary data based on periodic summary reports]

[Rate expressed as number of cases per annum per 1,000 average strength]



Annual rates.—The rate for 1943 was 132 cases per 1,000 strength and for the subsequent 2 years 54 and 22 cases, respectively (table 54). The downward trend was attributed to many factors, including seasoning of troops and frequent sanitary inspections to ensure food and water sanitation, proper disposal of waste, fly control, and examination of foodhandlers. The shift of large numbers of troops from North Africa to Italy was also a contributing factor. The downward trend also emphasizes the value of preventive medicine. From the opening of the theater to the wave of the 1943 epidemic in September, more than 500,000 men were exposed to the hyperendemic environment for the first time and during the dysentery season; whereas something like half that number of new men had entered the theater during the interval from the end of the

[18] See footnote 5, p. 327.

1943 epidemic to the end of the 1944 dysentery season (to increase the average strength to about 700,000 in August 1944). A reduction in the incidence of diarrhea and dysentery was to some extent related to the proportion of unseasoned troops. The rate was exceedingly high when all troops were newcomers to the theater, and as the proportion of unseasoned troops declined the rate declined. The decline in 1945 may also be explained similarly. However, it is difficult in retrospect to determine the actual percentage of cases in 1944 contributed by new men entering the theater. Although not too well substantiated epidemiologically, there exists the documented general opinion that during 1944 and 1945 new troops entering the theater were badly affected, whereas diarrheal diseases did not tend to recur in severe form among troops who had been in the theater for several months. In the annual report for 1943 [19] the statement is made that, "* * * outbreaks were particularly prevalent in newly arrived organizations and in replacement depots * * *. Those outbreaks which occurred late in the summer were largely confined to units new in the theater or to those involved in operations that made it difficult to control the sanitation of their environments." Hurevitz states: [20] "Another factor may have been the large number of new arrivals * * *. Troops who have been in North Africa may have developed a partial immunity to some of the more common varieties of *Shigella*, but definite proof of this is lacking."

Seasonal occurrence and monthly rates.—Chart 35 shows monthly rates for diarrhea and dysentery. The seasonal fluctuation is marked by increased incidence about May, reaching a peak in June or July, and tapering off in September or October. The peak rates were 445 in June during the epidemic of 1943 and 114 in July 1944.

The 1943 epidemic.—The 1943 epidemic in North Africa coincided with the movement of prisoners of war (among whom bacillary dysentery was common) from the Tunisian battlefields back to Casablanca, with the beginning of warm weather, and with an abundant fly population. Poor sanitary conditions in prisoner-of-war trains gave ample opportunity for the disease to spread to military personnel establishments along the rail routes. These circumstances, coupled with the fact that there was an early shortage of medical supplies, helped to set the stage. Few messes had screens, and a shortage of insect-control sprays and sprayers was particularly evident in the Eastern Base Section. Furthermore, troops recently arrived from the United States were accustomed to high levels of sanitation and did not appreciate the details entailed in maintaining high levels of field sanitation or the personal hygiene necessary during warm months in this new environment.

Dysentery became epidemic in the Eastern Base Section and in the 34th Infantry Division about 17 May 1943, in the Atlantic Base Section about 19 May 1943, and in the Mediterranean Base Section by 24 May 1943. Flies became abundant in all areas about this time, and the epidemic was soon present across the entire North African occupied area. Units of the 34th Infantry Di-

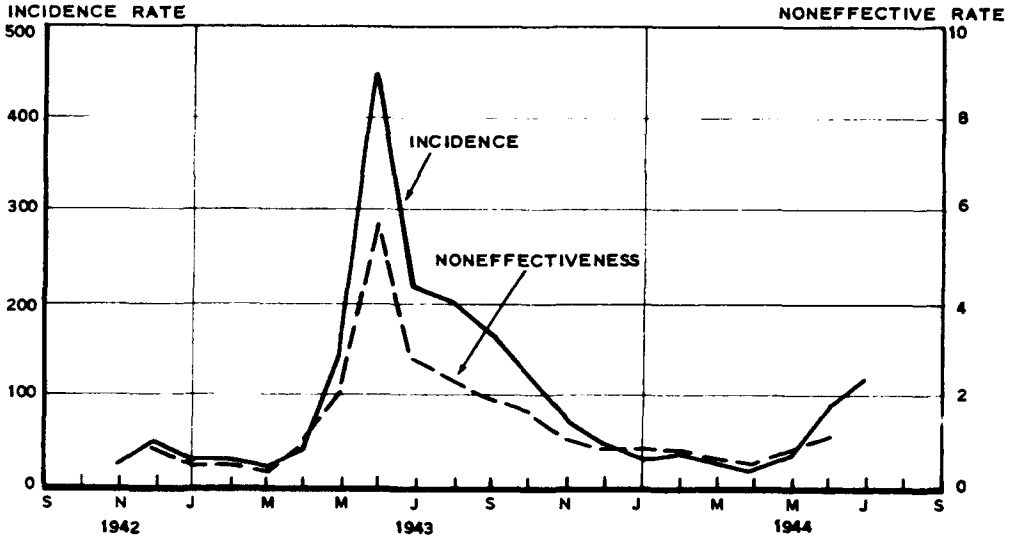
[19] Annual Report, Medical Section, Mediterranean Theater of Operations, United States Army, 1943.
[20] Hurevitz, H. M.: Dysentery and Common Diarrheas in MTOUSA. [Official record.]

vision with a rating of "good sanitation" had low dysentery rates, while units with a rating of "fair sanitation" had high rates.[21] Directives which stressed the unit command responsibility for sanitation resulted in improved sanitation and fly control, and the rates for the theater dropped from 445 cases per 1,000 troops in June to slightly over 200 in July despite the invasion of Sicily, which was under way during the last 3 weeks of July.

The 1943 epidemic was one of the most extreme outbreaks of diarrhea and dysentery experienced by the United States Army over a large area. During June 1943, when the rate was 445, illnesses from diarrheal disease accounted for 36 percent of all admissions and for 14 percent of all noneffectiveness.[22] The noneffective rate rose to 5.5 (chart 36). During the year of 1943, diarrheal conditions caused 14 percent of all admissions for disease and 6 percent of noneffectiveness charged to disease (table 67).

CHART 36. *Diarrhea and dysentery in the U. S. Army in North Africa, by month, November 1942 to July 1944*

[Incidence expressed as number of cases per annum per 1,000 average strength. Noneffective rate expressed as average daily number noneffective per 1,000 average strength]



Source: Monthly Progress Report, Army Service Forces, War Department, 31 Aug. 1944, Section 7: Health.

Laboratory facilities sufficient to make stool cultures on any appreciable part of cases were clearly not at hand. However, more cases of bacillary dysentery were identified in this theater during this epidemic year than in any other theater during any war year, although three other theaters during this same year had higher rates. Whenever diarrheal infections were studied bacteriologically, one or another type of *Shigella* was usually recovered; one laboratory in the theater was able to confirm the presence of Shigella organisms in over 50 percent of stool specimens. The theaterwide epidemic of recognized

[21] Long, P. H.: A Historical Survey of the Activities of the Section of Preventive Medicine, Office of the Surgeon, Mediterranean Theater of Operations, U. S. Army, 3 Jan. 1943 to 15 Aug. 1943. [Official record.]

[22] See footnote 20, p. 364.

bacillary dysentery occurred simultaneously with an epidemic of diarrheas and dysenteries, and a large proportion of the latter doubtless was unrecognized bacillary dysentery.

The German Army both in North Africa and later in Italy suffered diarrheal disease presumably with higher rates and with more severe illness. It is amazing that the Germans, who went to such detail in training for the North African environment woefully neglected training in the rudiments of field sanitation. Promiscuous deposition of feces in areas previously held by the Nazis was noted repeatedly by Allied troops. Dudley claims that Rommel blamed dysentery as one of the chief reasons for his North African defeat (p. 319).

Intestinal infections under combat conditions.—Actual combat conditions do not lessen the need for good field sanitation, but application is often difficult and sometimes impossible. Foodhandling for combat troops is always a problem, but foodborne disease can be prevented in considerable measure. The observation was made in this theater (as in the European theater) that certain units had a most favorable experience with dysentery while in combat and existing on C and K field rations; other units not using these individual packaged rations were hard hit.

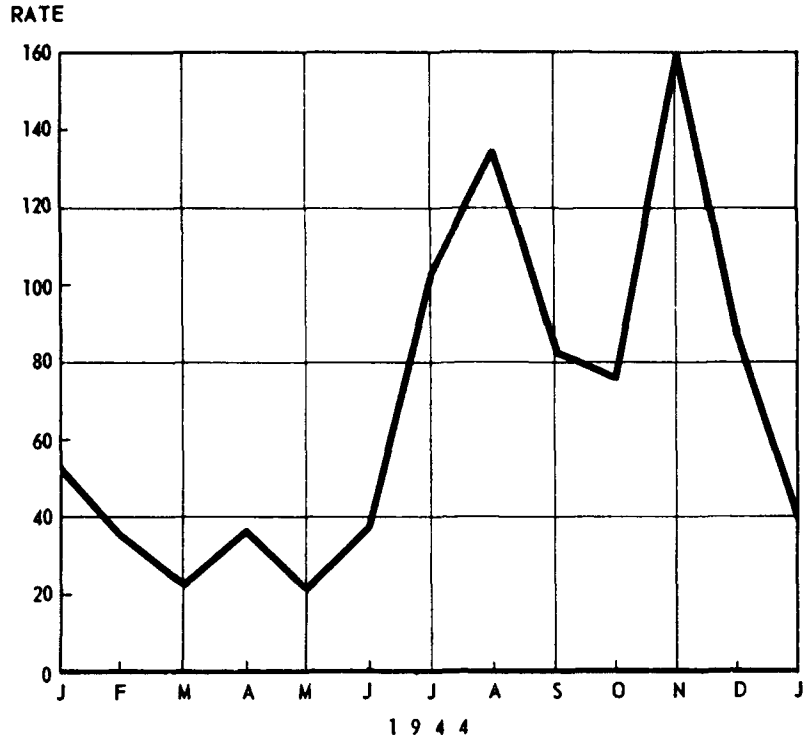
The Italian campaign and the landings at Salerno came during the wane of the dysentery season. Combat troops continued to experience a high incidence of dysentery throughout October and November 1943. Combat conditions created by the assault of the Gothic Line 1 year later, in 1944, again contributed to prolongation of the dysentery season into November. The experience of the Fifth U. S. Army in Italy provides an interesting epidemiologic case history:

Epidemiologic case report 11.—The Fifth U. S. Army, after landing in Salerno, during the latter part of 1943 experienced a jump in the rate for diarrheas and dysenteries to 72 cases per 1,000 troops in October. As the military situation became somewhat more stabilized and as cold weather set in, the monthly rate declined to 36 in January and 37 in May, 1944. The expected seasonal rise in incidence occurred thereafter; rates of 103 and 135 were reached during June and July, respectively. This rise coincided with the onset of warm weather, the fly season, the fresh-fruit season, and sustained combat activity. During August and September, the rates fell to 83 and 76, respectively (chart 37). This drop was ascribed to changes in the weather and stabilization of the front. Unit messes were utilized, and sanitary provisions were enforced among combat troops. On 12 September, an offensive was launched against the Gothic Line. Thereafter, an unexpected nonseasonal rise in rates occurred which was in contrast to the Fifth U. S. Army experience of the previous year. The monthly rate rose to 160 in October, during the height of the epidemic.

Thereafter, the monthly rate declined and was 83 in November and 41 cases in December. The rates during the October epidemic reflect only the hospital and quarters cases, and it is known that many cases of diarrhea were treated on a duty status under circumstances which did not enable good reporting. This epidemic occurred primarily among combat troops and not in service troops. Occurrence was associated with combat conditions. The fall rains and cold weather caused troops to seek shelter in areas where sanitary conditions were poor and where flies were often numerous. Water discipline was lax, and typhoid fever in civilians of the area indicated contamination of wells. Much of this contamination was probably related to the heavy rainfall and to backwater of sewage systems which had been broken by enemy demolitions. Facilities for washing messgear at the front were scant. Latrines became waterlogged, and feces sometimes floated over unit areas. As soon as the

CHART 37.—*Diarrhea and dysentery in the Fifth U. S. Army in Italy, 1944*

[Rate expressed as number of cases per annum per 1,000 average strength]



Source: Fifth Army Medical Service History, 1944.

offensive stopped and positions were stabilized, the level of field sanitation was raised, and the diarrhea rate began to decline from its unseasonable October peak.

This epidemic further illustrates the strong association of diarrheal disease with combat conditions, and emphasizes that preventive measures must be focused upon frontline troops and their environment.

Racial differences.—The interesting epidemiologic observation was made in this theater that Negro troops experienced consistently lower rates for intestinal disease than did white troops. No explanation was offered, but two possibilities were suggested: (1) That a much higher percentage of Negro than of white troops are engaged in service activities where their living establishments are reasonably stable and therefore better sanitated; and (2) that, because of greater previous exposure to enteric infection, there is greater immunity among Negroes.

Common diarrheas.—Common diarrhea represented approximately 85 percent of the intestinal disease noted on morbidity reports (Form 86ab) for this theater of operations. Some 85,000 cases of common diarrhea were reported (table 65). The diarrheas are described as often severe, with blood and pus in many cases, and as having been commonly identified, when examined bacteriologically, with Flexner dysentery bacilli.

In the Office of the Surgeon, the opinion was expressed, though it was not supported by reported data, that the great majority of common diarrheas were actually bacillary dysentery. Gilmore [23] stated: "When it has been possible to do careful bacteriology most of these outbreaks have been found to be due to bacillary dysentery infections, so that to the relatively small number of bacillary dysenteries reported as such should be added a large percentage of the common diarrheas." For example, Gowen reported [24] that 55 percent of 250 consecutive specimens from cases of acute enteritis in North Africa were positive for *Shigella*.

TABLE 65.– *Number of cases and percent distribution of intestinal infections in the U. S. Army in the Mediterranean theater, January 1943 through August 1945*

Diagnosis	1943–45		1943		1944		1945	
	Number of cases	Percent	Number of cases	Percent	Number of cases	Percent	Number of cases	Percent
Common diarrhea	85, 300	84. 3	48, 090	84. 4	31, 270	83. 8	5, 940	85. 8
Unclassified dysentery	8, 957	8. 8	5, 923	10. 4	2, 766	7. 4	268	3. 9
Bacillary dysentery	4, 088	4. 0	2, 713	4. 8	1, 181	3. 2	194	2. 8
Amebic dysentery	1, 284	1. 3	156	. 3	851	2. 3	277	4. 0
Bacterial food poisoning	1, 292	1. 3	----	----	1, 075	2. 9	217	3. 1
Typhoid and paratyphoid fevers	279	. 3	107	. 1	146	. 4	26	. 4
Total	101, 200	100. 0	56, 989	100. 0	37, 289	100. 0	6, 922	100. 0

Source: Compiled from data in final report of the Preventive Medicine Officer, Office of the Surgeon, Headquarters, Mediterranean Theater of Operations, U. S. Army.

Protozoal dysentery.—Gilmore stated further: "It is believed that many of the so-called protozoal dysenteries are actually bacillary cases in which protozoa were also found. It is felt that the increased number of cases of protozoal dysentery reported in 1944 and 1945 as compared with 1943 cases is largely a reflection of the greater interest in the problem of etiology of [this class of the] intestinal diseases."

The hospitals receiving about 40 percent of the cases in 1944 were staffed by physicians and others especially interested in protozoologic methods. The same medical facilities were not staffed with individuals of equal training and interest in bacteriologic techniques. In contrast to most other theaters, peak rates for protozoal dysentery for this theater were in 1944 rather than in 1945, although the rates were high in 1945 (table 60). In 1945, amebic dysentery represented 4 percent of all intestinal infections, and in the previous year, 2.3 percent (table 65). It is reasonable that prolonged exposure in the theater accounts for some of the increase. By contrast, bacillary dysentery,

[23] See footnote 5, p. 327.

[24] Gowen, G. H.: Acute Enteritis in North Africa. Bull. U. S. Army M. Dept. No. 71: 55-58, December 1943.

unclassified dysentery, and the common diarrheas affect troops soon after entry, and rates tend to decrease with time. In all, only about 1.3 percent of dysenteries and diarrheas (plus bacterial food poisoning, paratyphoid, and typhoid) in this theater were reported as amebic dysentery. Amebiasis therefore was not a major problem, although two deaths, both in 1943, were attributed to this disease (table 63).

Unclassified dysentery.—Approximately 9 percent of diarrheas and dysenteries were unclassified dysentery. Almost 9,000 cases were reported by the preventive medicine officer of the theater (table 65), but data in table 61 reflect only 5,364. The incidence rate for the war years was 3.62 cases per 1,000 per annum, and in the epidemic year of 1943 it was 8.13 cases per 1,000 per annum, approximately two-thirds of the cases having occurred in this year. The majority were believed to be actually bacillary dysentery in which rectal swabs or stool specimens were not examined for lack of laboratory facilities. However, that the diagnosis was not frequently changed to bacillary dysentery after admission is evidenced by the fact that reported cases for bacillary dysentery (table 65) approximate incidence figures based on tabulations of individual medical records. Either the number of cases of unclassified dysentery reported on WD MD Form 86ab included about 3,000 cases that were not admitted to hospital or the diagnosis for these 3,000 cases was changed to something other than dysentery.

One death in 1944 was ascribed to this cause (table 63).

Bacillary dysentery.—Over 4,000 cases of bacillary dysentery (tables 59 and 65) were recognized in the Mediterranean theater and represented about 4 percent of the total number of diarrheas and dysenteries reported in the theater; the incidence rate was 2.81 cases per annum per 1,000 average strength. During the epidemic year of 1943, almost 5 percent of all cases were reported as bacillary dysentery, and the incidence rate was over 6 cases per 1,000 per annum. Although bacillary dysentery caused severe illness in many patients and, together with unclassified dysentery, contributed to considerable noneffectiveness, it is remarkable that no deaths were reported from bacillary dysentery (table 63).

Of 778 cultures submitted by outlying laboratories to the 15th Medical General Laboratory for confirmation, 539 were Shigellae and 239 coliform and paracolon bacilli. The indication is that the smaller laboratories in the theater were doing good determinative enteric bacteriologic examinations.

Hurevitz stated that during the epidemic year of 1943, between 50 and 60 percent of stool specimens from patients with diarrheal disease submitted to laboratories of the theater yielded *Shigella*. The proportion in Italy during 1944 was smaller (10 to 15 percent), but during the fall outbreak in the Fifth U. S. Army about 30 percent of the specimens were positive.

A previously unidentified strain of *Shigella*, responsible for an outbreak in the 2d Replacement Depot, was isolated by the 2d Medical Laboratory at Casablanca, North Africa; 209 (53 percent) of 392 cultures gave this organism. Maj. Aaron H. Stock of the 2d Medical Laboratory designated

the organism *Sh. paradysenteriae*, type T. During the outbreak, this strain was recovered from a healthy Italian prisoner of war serving as foodhandler for the 2d Replacement Depot. Much significance was placed upon identifying the carrier because the strain was not among common strains in the theater. The carrier had a history of dysentery while with the Italian Army in Ethiopia during 1939. The strain was isolated again in 1945 by the 15th Medical General Laboratory.

Most recognized members of the *Shigella* group were isolated by one or another of the laboratories in this theater. *Sh. dysenteriae* (Shiga) was relatively uncommon, being reported from various laboratories with a frequency up to 8 percent. *Sh. sonnei* had about the same frequency, except that the 15th Medical General Laboratory in Naples during 1944 encountered it in 118 (20 percent) of 588 positive cultures (table 66). Some laboratories found *Sh. ambigua* in from 5 to 20 percent of cultures; other laboratories did not find it at all.

TABLE 66.—*Dysentery organisms isolated at the 15th Medical General Laboratory during 1944*

Organism	Number of isolations	Percent
Shigella paradysenteriae:		
Flexner I, V	11	1.9
Flexner II, W	62	10.5
Flexner III, Z	40	6.8
Flexner IV, 103	17	2.9
Flexner V, P 119	16	2.7
Flexner VI, 88 (Newcastle-Manchester group)	122	20.7
Boyd I, 170	10	1.7
Boyd II, P 288	1	.2
Boyd IV, P 274	40	6.8
Boyd V, P 143	1	.2
Shigella species, 2–193	5	.8
Shigella species (strains related to the Boyd types)	10	1.7
Sh. sonnei	118	20.1
Sh. madampensis	35	6.0
Sh. dysenteriae (Shiga)	3	.5
Sh. ambigua	23	3.9
Shigella species, Sachs Q 771	15	2.6
Shigella species, Sachs Q 1167	3	.5
Shigella species, Sachs Q 1030	3	.5
Shigella species, Sachs Q 902	2	.3
Shigella species (strains similar to the Sachs type)	6	1.0
Shigella species (strains related to the Flexner group)	24	4.1
Sh. alkalescens	13	2.2
Coliforms with *Sh. alkalescens* antigens	8	1.4
Total	588	100.0

Africa-Middle East Theater

The Africa-Middle East theater had an average strength of 36,838 during the interval between June 1942 and December 1945 (0.6 percent of the total average strength of the Army) but experienced, during the 3½-year period, no less than 18,830 reported cases of dysentery and diarrhea. This small population contributed 3.6 percent of total Army cases for a rate during the combined war years of 128. This theater ranks a close second to the Asiatic (China-Burma-India) theater which had the top rate of 131 (table 54). High carrier rates for dysentery bacilli were encountered regularly in native populations of these two areas whenever laboratory examinations were made, with rates varying from 6 to 10 percent; there was also a high incidence of both amebiasis and helminth carriers.

Environmental circumstances associated with a subtropical climate, poor facilities for feces disposal and promiscuous deposition of feces by natives, polluted-water supplies, abundance of flies, and a military situation which favored contact with natives and contamination of soldiers' food, all contributed to the high rates in this theater.

As in several other theaters and areas (notable exceptions are the China-Burma-India and Southwest Pacific), the annual rates declined each successive year; thus, the rates for the years 1942 to 1945, inclusive, were 196, 170, 115, and 79, respectively. The rare occurrence of acute diarrhea for troops in the second summer was a subject of comment, as was the fact that new troops in the area were badly affected. Bulmer,[25] describing the situation which the British Army encountered in the Middle East theater at this same time, says: "Probably every soldier in the M. E. F. had at least one attack of acute diarrhoea, usually soon after arrival * * *. Not all cases of diarrhoea were admitted to hospital * * * only about 6 percent of those reporting sick were sent to hospital." This could well have been said of United States troops. There is little doubt that the many mild unreported cases contributed to lowered military efficiency, especially among new troops entering the theater.

German troops in various portions of this theater had excessively high rates of intestinal diseases, and their field sanitary discipline was lax. That the otherwise exceptionally well-disciplined *Afrika Korps* neglected basic preventive procedures is illustrated in the following report of Col. H. S. Gear, British Assistant Director of Hygiene in the Middle East: [26]

> Enemy defensive localities are obvious from the amount of faeces lying on the surface of the ground * * *. This contempt for hygiene became such a menace to the enemy as to affect from 40 to 50% of his front-line troops, as interrogation of captured medical officers revealed * * *. The enemy appears to have no conception of the most elementary sanitary measures, and has a dysentery rate so very much higher than ours that it is believed that the poor physical condition of his troops played a great part in the recent victory at El Alamein.

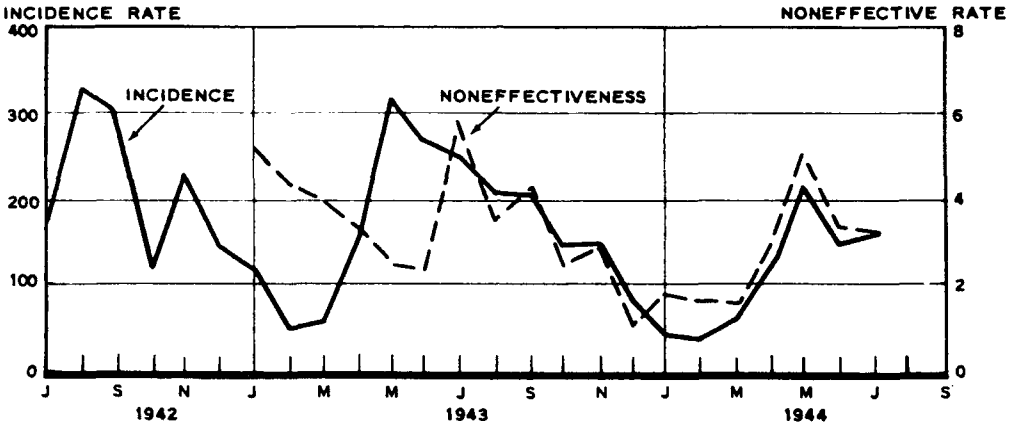
[25] Bulmer, E.: A Survey of Tropical Diseases as Seen in the Middle East. Tr. Roy. Soc. Trop. Med. & Hyg. 37: 225-242, February 1944.

[26] Gear, H. S.: Hygiene Aspects of the El Alamein Victory, 1942. Brit. M. J. 1: 383-387, March 1944.

Deaths and noneffectiveness resulting from diarrheal disorders.—Chart 38 shows incidence rates and noneffective rates from diarrheas and dysenteries as a class, from the inception of activities in the theater to the middle of 1944. One writer of a history of the Middle East component of the theater compares noneffectiveness due to these causes with that due to malaria. He states: [27] "A few months of the year, malaria would cause more man-days lost, but on the calendar-year average, gastro-intestinal infections were well ahead."

CHART 38.—*Diarrhea and dysentery in the U. S. Army in the Middle East (including the Persian Gulf Command), by month, July 1942 to July 1944*

[Incidence rate expressed as number of cases per annum per 1,000 average strength. Noneffective rate expressed as average daily number noneffective per 1,000 average strength]



Source: Monthly Progress Report, Army Service Forces, War Department, 31 Aug. 1944, Section 7: Health.

In the Persian Gulf Command (Iran) during 1942, 1 of every 7 patients admitted had an intestinal infection.[28] In the Africa-Middle East theater during the war years, diarrheas and dysenteries accounted for 14.3 percent of all admissions for disease. During 1943 (a year for which this type of data is at hand), over 15 percent of admissions and 7 percent of all noneffectiveness from disease was of this origin (table 67). Of the 22 deaths ascribed to diarrhea (including gastroenteritis, ileitis, and ulcerative colitis) among overseas admissions in 1942–45 (table 56), 1 was reported among admissions in the Middle East; only 1 death was due to dysentery, a case of bacillary dysentery in 1942 (table 63).

Seasonal and monthly incidence.— Chart 39 reveals that the lowest rates for the theater were in February and March 1943, January and February 1944, and February and December 1945. In 1943, 1944, and 1945, peak rates developed in May and incidence continued high through October and November, except in 1945. This seasonal pattern was punctuated with numerous epidemics in summer months consistent with the occurrence of a considerable amount of

[27] Ward, Thomas G.: History of Preventive Medicine, U. S. Army Forces in the Middle East, 19 Oct. 1941 to 23 June 1944, vol. III. [Official record.]

[28] McNaughton, L. M.: History of Preventive Medicine, Persian Gulf Command. [Official record.]

TABLE 67.—*Admission and noneffective rates due to diarrhea and dysentery in the U. S. Army compared with respective rates for all disease in several theaters or areas, 1943*

[Rate expressed as number of admissions per annum per 1,000 men and average daily noneffectiveness per 1,000 average strength]

Theater or area	Admission rate for all diseases	Diarrhea and dysentery		Total disease noneffective rate	Diarrhea and dysentery	
		Admission rate	Percent of total disease admission rate		Noneffective rate	Percent of total disease noneffective rate
Total overseas	891	66	7. 4	31. 2	1. 08	3. 5
Middle East [1]	1, 105	170	15. 4	39. 7	2. 81	7. 1
North Africa	946	132	14. 0	31. 6	1. 88	6. 0
South Pacific	1, 623	75	4. 6	56. 4	1. 32	2. 3
Southwest Pacific	1, 042	70	6. 7	42. 7	1. 30	3. 0
Asia	991	146	14. 7	35. 1	3. 28	9. 3

[1] Including Persian Gulf Command.

Source: Monthly Progress Report, Army Service Forces, War Department, 31 Aug. 1944, Section 7: Health.

bacillary dysentery. In fact, the Africa-Middle East theater had higher incidence rates for bacillary dysentery than any other, and the incidence rates for unclassified dysentery were the second highest. Although the dysentery season was ushered in by appearance of flies, the disease did not always decline with the end of the fly season. In November 1944, the case rate was 129 per 1,000 per annum despite the reduction in flies prior to that time.

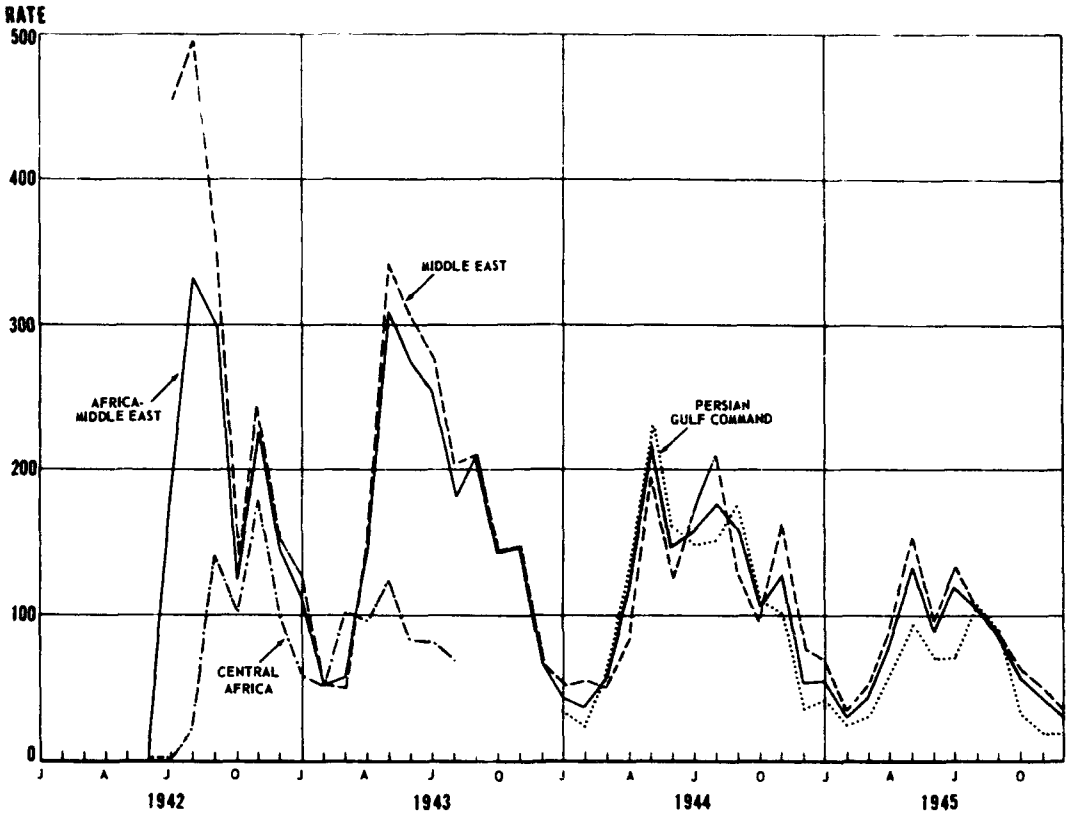
Comparable seasonal fluctuations occurred in the several component subdivisions of the theater: Central Africa, Persian Gulf Command, and the Middle East.

Somewhat less dysentery and diarrhea was reported during 1942 and 1943 among troops in Central Africa than in the Middle East component, although peak case rates of 180 per 1,000 in November 1942 and 126 in May 1943 were anything but low; these peak case rates are to be compared with the rates of 243 cases per 1,000 in November 1942 and 343 cases per 1,000 in May of 1943 in that portion of the theater designated as the Middle East. During 1944, the Persian Gulf Command and Middle East experienced strikingly similar rates, but during 1945 the Persian Gulf Command showed slightly more improvement than in the latter component. The average strength of the Persian Gulf Command steadily decreased during the period of improvement, whereas the average strength of the Middle East continued to build up through July 1945. This suggests that new unseasoned troops entering the theater may have contributed to the excess incidence of the Middle East or that the Persian Gulf Command maintained a relatively higher proportion of seasoned troops during 1945. The high incidence of intestinal diseases in the Middle East Service Command, particularly those troops stationed in camps near Cairo (transients passing through those stations), was due in part to the fact that these

CHART 39.—*Incidence of diarrhea and dysentery in the U. S. Army in the total Africa-Middle East theater and its components (Middle East,[1] Central Africa,[2] and Persian Gulf Command), 1942–45*

[Preliminary data based on periodic summary reports]

[Rate expressed as number of cases per annum per 1,000 average strength]



[1] No troops were present in the area prior to July 1942.

[2] Not reported as a separate area after August 1943.

soldiers frequently were able to go to the nearby city for food and recreation, and often were careless in their choice. "Military personnel had not been adequately informed of the dangers of consuming raw and uncooked fruits and vegetables from native sources, and almost invariably over 70 percent of personnel among newly arriving units suffered a gastro-intestinal upset within a few weeks of arrival." [29]

Epidemiologic case report 12.—United States Army personnel in the Belgian Congo slept in tents and used food prepared in poorly screened kitchens.[30] Pit latrines were screened at the outset. Flies were numerous. A native village and a Congoese military camp were within flyrange of Army kitchens and barracks. Native huts were scattered in the brush near the camp. Bacillary and endamebic dysentery were prevalent among natives. The nearby city had restaurants which were patronized by the troops. Kitchens of some of these restaurants were unclean by Army standards. The Army nurses were billeted in a hotel for 2½

[29] See footnote 27, p. 372.

[30] Dunham, C. D., and Gillespie, W. H.: Diarrheal Diseases in U. S. Troops in Belgian Congo. Bull. U. S. Army M. Dept. No. 78: 76–80, July 1944.

months, and many contracted febrile diarrhea. Four nurses were admitted to the hospital with bacillary dysentery, and one died of a fulminating infection. After 3 months, barracks and messhalls were ready for the nurses, and, except for one case of gastroenteritis, no more diarrheal disease occurred among them. That bacillary dysentery constituted a significant portion of the diarrheal disease problem at this location in the Belgian Congo is evidenced by the following 1943 bacteriologic findings:

	Cases
Sh. paradysenteriae (Flexner)	8
Sh. paradysenteriae (Sonne)	2
Sh. dysenteriae (Shiga)	1
Sh. dysenterine (Schmitz)	5
Shigella species (Flexner and Sonne)	1
Shigella species (Flexner and Schmitz)	1

The common diarrheas.—Numerous investigators were of the opinion that the major portion of "gyppy tummy," "basra-belly," and common diarrhea in this area were due to dysentery bacilli. Others were not able to show, with any consistency, any microbial agent. In most units, the entire biologic gradient of mild diarrhea, severe diarrhea, and clinically typical dysentery with blood, pus, and mucus in the stools appeared simultaneously. Bulmer, in a British general hospital in the Middle East asked: "* * * why ascribe the mild 'gyppy tummy' to the ingestion of sand, or a draught on the abdomen at night, or some other curious theory, and incriminate the dysentery bacillus for the severer forms? The selective media now available should establish the clinically irresistible conclusion that they are the same disease * * *." The causes of common diarrhea remain unknown, but many mild infections by Shigella bacilli probably were reported as one or another of the common diarrheas.

Epidemiologic case report 13.—During the militarily critical month of August 1942, a case rate of 335 per 1,000 was attained for diarrhea and dysentery in the Africa-Middle East theater. The case rate for the Middle East component of the theater for that month was 497 per 1,000 which is one of the highest monthly rates experienced during World War II (chart 39). The average strength of Middle East troops was only a little over 3,000 during this month, and only 123 cases contributed to produce this rate. However, it is of more than passing interest that similar high rates were observed in British troops in this theater during this same month.[31] Our allies fared as badly in this environmental situation. Powerful reinforcements of fit, tough, and well-disciplined British troops arrived in the theater during August. Although these troops subsequently proved themselves to be extremely tough, they were attacked promptly by diarrheal disease to such extent that a military campaign could not have been successfully conducted during the first few weeks after their arrival. One of the hospitals providing medical care for these British reinforcements admitted as many as 90 new cases of acute diarrhea in 1 day, and 450 beds were occupied at one time by clinical dysentery cases. Of one battalion, 350 men were in hospital for weeks, and 180 were admitted on a single day with diarrhea. Concurrently, other British troops who had been in the theater for a few months were relatively free from dysentery. Although there was some contact, it is believed that the high incidence of diarrhea and dysentery among United States troops was related more to the general environmental situation and contact with native populations than to a spread from British to United States troops. The military lesson to be learned from this epidemiologic case report is that unseasoned troops from Europe or the United States entering the Middle East for the first time, especially during the summer months, may be expected promptly to become

[31] See footnote 25, p. 371.

militarily noneffective because of diarrheal disease. Either seasoned troops must be used for military operations or a period of time must be allowed for diarrheal disease to run its course.

The dysenteries.– Incidence rates for dysentery, all forms, ranged as high as 162 cases per 1,000 troops in the Middle East Service Command, whereas the highest rate for the other forces was 40.[32]

During the period 1942–45, the incidence rate for bacillary dysentery in the entire theater was 14.52 cases per 1,000 average strength per annum. The annual rates for the 4 successive years were, respectively, 20.51, 17.46, 15.97, and 8.19 cases per 1,000 average strength. The rates are almost double those of the nearest comparable theater, China-Burma-India, which had a rate of 8.91 cases per 1,000 average strength per annum, during the same interval (table 59). Of 18,830 cases of dysentery and diarrhea reported in the Africa-Middle East theater (table 54), 212 were diagnosed as bacillary dysentery. One death in 1942 gives an annual rate of 0.68 death per 100,000 average strength during the history of the theater (table 63). In the China-Burma-India theater, less than 1.5 percent of the cases of diarrheal disease were recorded as bacillary dysentery. More protozoal dysentery was identified among common diarrheas in the China-Burma-India theater than in the Africa-Middle East theater. The reverse is true of bacillary dysentery. In the China-Burma-India theater, special directives, an amebiasis register, and other measures directed attention to amebic dysentery, whereas in the Middle East theater, the emphasis was on *Shigella* or other bacterial pathogens. The following statement was included in a report entitled "Dysenteries in Iran" which was distributed in 1943 by Headquarters, Persian Gulf Command:[33] "Any medical officer not submitting stools of a dysentery patient for laboratory examination, where facilities exist, is guilty of criminal negligence * * *. In the Middle East the bacillary type of dysentery appears to be the more common." Since both amebic and bacillary dysentery in United States Army practice requires identification of the infectious agent, such incentives probably account for much of the difference noted, and the rates tend to reflect the amount and kind of laboratory work. Among the several theaters, the Africa-Middle East had the highest rates for bacillary dysentery and the second highest rates for unclassified and amebic dysentery (table 62).

Most outbreaks of dysentery in the Middle East that were adequately investigated epidemiologically and bacteriologically were caused by a Flexner type of dysentery bacillus.

China-Burma-India Theater

This theater had an average strength of 108,362, or about 1.8 percent of the total Army, during the war years. This small population suffered 10.9 percent of all reported dysentery and diarrhea in the United States Army,

[32] Essential Technical Medical Data, Headquarters, U. S. Army Forces in the Middle East, for October 1944.
[33] Report, Maj. A. A. Carabelli, MC, 10 May 1943, subject: Dysenteries in Iran.

with 56,951 reported cases and a rate of 131 cases per 1,000 strength, the highest rate of any of the theaters of operations (table 54).

China, Burma, and India are all hyperendemic areas in respect to diarrheal disease. Many sanitary practices of these overcrowded populations, both personal and communal, are tied to habits and customs of generations past. The saving and collection of night soil for agricultural purposes and the promiscuous deposition of filth including feces and garbage favored survival and spread of the infectious agents of intestinal diseases. Food is commonly unprotected from dust, rodents, and flies. Refrigeration is almost universally lacking. Flies are generally abundant. Diarrhea and dysentery, bacillary and protozoal, are constantly present.

These diseases were quickly established among United States troops entering this environment, where standards of sanitation were so different from those at home. Close contact with the native population was often demanded by the military situation. Native help was almost universally employed in foodhandling throughout the theater.

In the China sector, arrangements existed whereby the Chinese Government provided food and lodging for United States Army personnel. A Chinese Government agency, the War Area Service Command, organized and staffed messes and living accommodations at stations and airfields. Air shipments over "the Hump" were limited to essential military materials, and personnel and sanitation materials had low priority. The United States Army in China, in certain respects, was a nonpaying guest, and severe criticism of sanitation was not considered diplomatic. However, inspections were made by medical officers, and mess sergeants were assigned to supervise. Ultimately, new equipment and sanitation supplies were provided, and a gradual improvement in sanitation was achieved. Separate data for the sectors of the theater are available for the period from November 1944 through December 1945. The China rate for diarrheas and dysenteries was 122 cases per 1,000 troops per annum, contrasted with a rate of 86 for the India-Burma sector. The latter sector had the benefit of a belatedly organized preventive medicine program and the opening of area and research laboratories.

Annual rates.—The annual incidence rate for reported dysenteries and diarrheas for 1942 was 123 cases per 1,000 troops and for the successive 3 years was 146, 181, and 93, respectively, per 1,000 (table 54). During 1943, approximately 15 percent of all admissions for disease were for diarrhea or dysentery, accounting for some 9 percent of noneffectiveness (table 67).

The average strength of the theater constantly increased until March 1945, which marked the peak monthly average strength of over 250,000 men. Unseasoned troops were sent regularly to this theater. The greatest increase was in 1944, which also was the year with the highest incidence rate for diarrheas and dysenteries. Expansion in strength often exceeded available facilities; consequently, overcrowding existed, and sanitary facilities were insufficient. The lowest yearly incidence rate was in 1945 when the fewest new

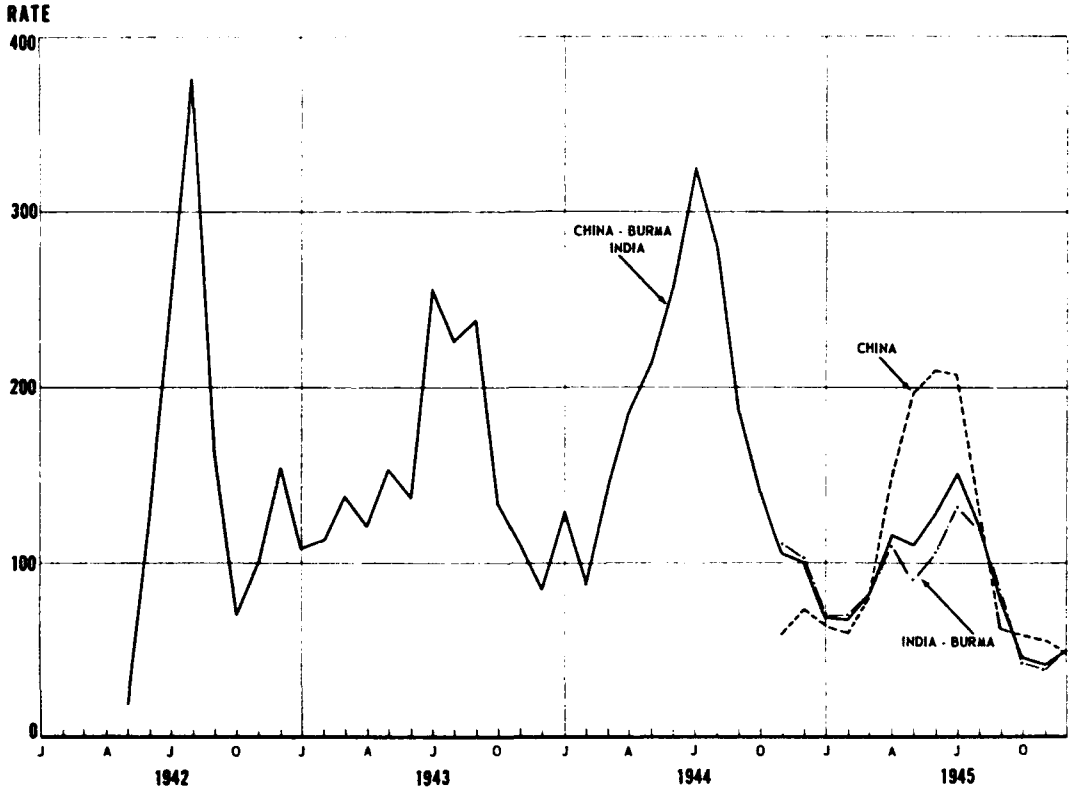
men entered the theater and when demobilization led to rapidly decreasing strength. General sanitation also improved.

Control.—According to Van Auken,[34] "there was no organized control program for the enteric infections until 1944." In 1942, a letter was sent to troop units describing sanitary procedures applicable to fresh fruits and vegetables prior to consumption, and another letter was sent describing treatment and prevention of selected tropical diseases. The latter letter included two lines pertaining to enteric diseases, a summary statement that prevention is dependent upon good sanitation. In December 1943, another letter having theaterwide distribution prescribed methods for preparing fruits and vegetables and precautions in the use of native foodhandlers. Other than these letters, no recommendations were made, and organization commanders were left to shift for themselves in spite of a diarrhea and dysentery rate which had reached 256 per 1,000 per annum in July 1943 and 326 per 1,000 per annum in July 1944 (chart 40).

CHART 40.—*Incidence of diarrhea and dysentery in the U. S. Army in the total China-Burma-India theater, 1942–45, and in the China and India-Burma theaters, November 1944 through December 1945*

[Preliminary data based on periodic summary reports]

[Rate expressed as number of cases per annum per 1,000 average strength]



[34] Van Auken, H. A.: A History of Preventive Medicine in the United States Army Forces of the India-Burma Theater, 1942 to 1945. [Official record.]

The situation received attention in June 1944 when a Sub-Commission on Dysentery of the Army Epidemiological Board, Office of the Surgeon General, United States Army, arrived to investigate diarrheal disease. Surveys were made by this Sub-Commission of troops in the Calcutta and Ledo areas. Many defects in environmental sanitation practices were noted, and the diagnosis and prevention of diarrheal disease were considered to be unsatisfactory generally. The Sub-Commission believed that widespread use of native Indians as cooks and foodhandlers was important as a factor in the spread of these diseases among United States Army troops. "Of great importance is the experience of a number of organizations that as soon as they removed Indians from the kitchens the diarrheal rates dropped." [35]

The Sub-Commission [36] recognized the inadequacy of the training of laboratory personnel for work on enteric pathogens and of laboratory supplies and equipment. Identification of protozoa was frequently inaccurate, and few laboratories were able to do good bacteriologic work. Time was devoted by this group to improving laboratory services, with emphasis on amebic dysentery, a disease judged of much importance in this theater. As a further result, the theater surgeon and the theater commander instituted a campaign to bring diarrheal disease under control. Appropriate directives were sent to subordinate commands, and special attention was given to improving mess sanitation. Attention was also paid to improving restaurant sanitation in certain eating establishments in large cities, especially Calcutta, at which United States Army personnel were permitted to dine. A special sanitary inspector was assigned to the Services of Supply surgeon who was charged with checking and improving mess and restaurant sanitation throughout the theater. A theater preventive medicine section was formed for the first time, late in 1944. Arrangements were made for better distribution of sanitation supplies, especially compound germicidal rinse; for sanitary messgear and utensils; for insecticidal spray; and for an all-purpose soap. A pamphlet on water sanitation was distributed. Furthermore, commanding officers were held strictly accountable for sanitation in their commands and were advised to reduce native labor in Army messes to a minimum and to place such labor under strict supervision. Contact, at least by letter, was made thereafter with commands reporting high incidence of diarrheal disease.

In October 1944, another observer group, referred to as the Kelser Mission, arrived in the theater. Inadequate water-heating facilities and refrigeration were recognized as additional defects to be remedied, along with the need for better laboratory facilities in clinical diagnosis and bacteriologic control of water supplies.

The 29th Medical Laboratory arrived in the theater in May 1945 with the specific assignment to investigate diarrheas. This group worked primarily on the etiology of diarrhea in the Calcutta and New Delhi areas. This laboratory

[35] See footnote 34, p. 378.

[36] (1) Report, Sub-Commission on Dysentery, Army Epidemiological Board, 15 Dec. 1944. (2) See footnote 7, p. 328.

supplemented the 9th Medical Laboratory which had been functioning as an area laboratory for the past several months.

The general reduction in diarrheal disease during 1945 was largely influenced by preventive methods instituted as a result of the missions to the theater and by the control which resulted from administrative achievement of a preventive medicine organization giving emphasis to control of intestinal diseases. The decrease in rates was more notable in the India than in the China sector; the greatest preventive effort was expended in the India sector. Furthermore, the Ledo Road building activity was nearly completed in the first quarter of 1945, and regular motor convoy service to China was inaugurated. Reasonably sanitary camps were established for stops along the road. Extensive troop movements through unsanitated areas were no longer necessary except for small numbers traveling by rail, homeward bound, from Assam to Karachi. Enforcement of sanitary regulations, more sanitation supplies, and a change in operational status of the theater thus had effect in reducing the rates for diarrheal disease.

Improvements in sanitation related to threat of cholera epidemic.—No discussion of diarrheal disease in the China-Burma-India theater would be complete without mention of the fact that there were no reported cases of cholera among United States Army troops in the Calcutta area during the 1945 cholera epidemic. Cholera assumed epidemic proportions in the native population of Calcutta in February 1945, and a high incidence continued into June. More than 3,500 cases and 1,200 deaths were reported in the Calcutta area. Fourteen cases were reported among approximately 700 British military personnel billeted at the Grand Hotel in Calcutta; 135 United States Army officers, also billeted at this hotel, were moved out on the day cholera was reported. A medical survey revealed 21 mild cases of cholera among the 1,700 hotel employees. Periodic immunization against cholera, as required for United States Army troops, was not required for British troops. Including cases at the Grand Hotel, the British Army had 27 cases with 2 deaths.

The Medical Department of the United States Army took special action to protect the 24,500 troops in the Calcutta area. The epidemic served effectively in bringing about improvements in mess sanitation, foodhandling and water purification, fly control, and supervision of native foodhandlers in Army messes. The Allied Hygiene Committee increased its activities and required that restaurants in bounds to United States military personnel should have their employees immunized against cholera. More important, emphasis was placed upon the serving and eating of hot cooked foods only, the sterilization of drinking water, and the necessity for drinking only water known to be safe. It is impossible to determine how much these measures contributed to the over-all prevention of diarrheal disease, but doubtless some portion of the improved incidence rates in the Calcutta area, at least for the year 1945, may be attributed to this situation.

By contrast, the China sector of the theater, which did not receive as much benefit from the increased impetus given to preventive medicine in the theater

late in the war, experienced two outbreaks of cholera among United States Army troops, with 13 cases and 2 deaths, comprising the sole experience of the United States Army with cholera in World War II (p. 459).

Reliability of laboratory diagnosis.—Bacteriologic diagnosis of diarrheas and dysenteries was often inaccurate or not attempted. Reliable work simply was not done during the first 2 years of the theater and thereafter was accomplished at only a few laboratories. In many laboratories, technicians examined stools for *E. histolytica*, but some diagnosed "anything that moved under the microscope" as that organism. Van Auken noted that "the faults lay in inexperienced, untrained personnel in the laboratories and lack of equipment and supplies authorized for the smaller hospitals which served much of the Theater." There is little wonder that during the peak month of July 1944 three cases of amebic dysentery were found for each case of bacillary dysentery found and almost half of the recognized dysenteries (bacillary, amebic, and unclassified) were amebic. Amebic dysentery was undoubtedly a frequent disease in this theater, more so than in most others, but the true incidence of shigellosis is certainly not reflected in recorded data.

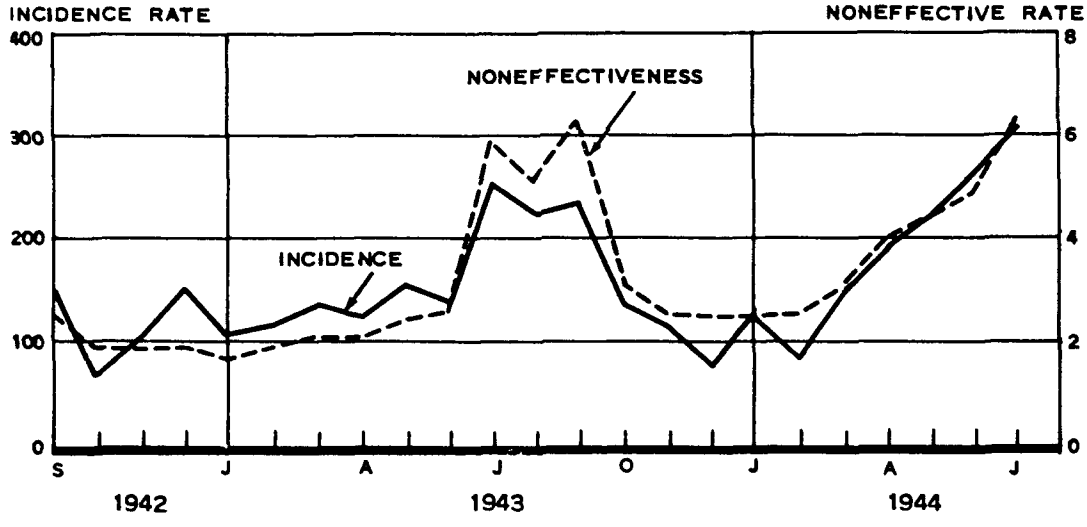
The training of laboratory technicians was begun at the 9th Medical Laboratory in December 1944. The laboratory arrived in the theater early in 1944, was opened in August as a separate organization providing theaterwide service, and became belatedly the first true public health or area laboratory in the theater. No attempt was made to train new technicians but to improve the standards of performance of technicians already working in the theater. Deficiencies were corrected, and special emphasis was placed on parasitology and recognition of *E. histolytica*. Although some attention was given to determinative bacteriology of enteric pathogens, few laboratories had much capability in enteric bacteriology upon which to build. The commanding officer of the 9th Medical Laboratory was appointed theater laboratory consultant in February 1945. He inspected numerous hospital laboratories to improve the quality of work and the liaison with his own central laboratory. Improvement in some laboratories was definite, but demobilization came before full results were manifest.

Of all cases of diarrhea and dysentery reported in June 1945, 26 percent were recorded as amebic dysentery, whereas in June 1944 only 15 percent had been so diagnosed and in June 1943 only 5 percent. The emphasis given laboratory diagnosis of amebic dysentery is illustrated by the 1,067 cases of dysentery and diarrhea reported for the India-Burma sector in January 1945; some 43 percent (454) were called amebic dysentery, an extraordinary frequency for a winter month. The Sub-Commission on Dysentery of the Army Epidemiological Board expressed the belief that amebic dysentery was a greater problem in the theater than bacillary, because of higher reported incidence and because of its chronic nature. Bacillary dysentery was stressed as being a mild illness of short duration, in contrast to the more severe and chronic course of amebic dysentery as seen in the theater.

Monthly rates and seasonal incidence.—Except for an occasional winter month, monthly rates for diarrheas and dysenteries were never under 100 cases per 1,000 strength until the fall of 1945 (chart 40). The months with the lower rates were November through April, and the peak rates occurred in late summer, July having the highest rate. Noneffectiveness per 1,000 strength was 6 or more during July and September 1943 and during July 1944 (chart 41). The frequency coincides with that of flies which are prevalent in parts of the theater (Calcutta) throughout the year, November or December being the better months and July the worst. The sharp epidemic peaks in summer are more suggestive of bacillary dysentery than of amebic dysentery. Indeed, the actual bacillary dysentery incidence of 8.91 cases per 1,000 strength per annum for the period 1942–45 is the second highest rate reported for bacillary dysentery for any theater during the war (table 59). The rate for amebic dysentery was 23.61 cases per 1,000 per year, almost three times as high (table 60).

CHART 41. *Diarrhea and dysentery in the U. S. Army in the China-Burma-India theater, by month, September 1942 to July 1944*

[Incidence rate expressed as number of cases per 1,000 average strength. Noneffective rate expressed as average daily number noneffective per 1,000 average strength]



Source: Monthly Progress Report, Army Service Forces, War Department, 31 Aug. 1944, Section 7: Health.

Rail travel and troop transport in relation to diarrheal disorders.—During the war, troops and supplies were landed at Karachi and Bombay on the west coast of India, the sea route to Calcutta having been rejected as too dangerous because of Japanese submarines. Not until late 1944 were any significant numbers of troops debarked at Calcutta. The port was, however, an extremely important terminus for operations incident to the construction of the Ledo Road. Troops were therefore shipped by rail across about 2,000 miles of the width of India, to Calcutta and thence to upper Assam, the orbit of troop concentration. Troop units crossing India had much intestinal illness, seldom failing to have many members hospitalized at the end of the journey near

Ledo, at Calcutta, or elsewhere en route. Troops held over in the Bombay area awaiting transportation were assigned to several camps. An inspection of Camp Deolali in June 1943 revealed large numbers of flies in kitchens having numerous other sanitary defects. Food was inadequately refrigerated and exposed to flies; messhalls were not screened; and latrines were of the bucket type, unprotected from flies. Many troops embarked upon the long rail journey already suffering from diarrhea.

Poor sanitation along the railroads, especially at stopping places, was general, sanitary control over the train-side vendors and station restaurants being nonexistent. The men often ate at these unauthorized places. Water carried to the coaches was potentially contaminated, for it came from unauthorized sources. During the first few months after the theater was established, sanitation supplies were short. In the beginning, troops were not adequately briefed regarding these conditions and the procedure for keeping healthy. Not until 10 August 1945 did the first theaterwide directive appear, providing for sanitary control of troops in transit. The directive, entitled "Transportation Procedures," was issued in time to be of value for homeward-bound personnel at the end of the war.

Many troops traveled from Assam to Karachi, the major port of debarkation, by airlift instead of by rail. However, more than 20,000 troops were sent by train on a 2,470-mile trip over a route not previously used by United States troops. Special preparations were made for this rail movement in the light of past experience and in compliance with special directives. During October 1945, some 7,986 troops arrived in Karachi, and only 18 men were hospitalized because of diarrheal disease. In November, 4,632 men arrived, and 12 were hospitalized. This experience compares favorably with that of earlier years of rail travel in the theater.

The common diarrheas.—For all war years, the India-Burma sector of the theater had more than 29,000 admissions for the common diarrheas representing 58 percent of the admissions for all diarrheal diseases. With each successive year of operation, the percentage of diarrheal disease diagnosed as common diarrhea decreased because of increasing separation of definite entities, mainly amebic dysentery. In late months of 1942, 77 percent of diarrheal disease was called common diarrhea; in the 3 subsequent years, the proportions for common diarrhea declined to 65, 61, and 49 percent, respectively.

Several studies of microbial agents of the common diarrheas were made late in the war by various laboratory teams. The Sub-Commission on Dysentery of the Army Epidemiological Board studied 369 hospitalized patients and 175 with diarrhea treated in two dispensaries. Dysentery bacilli were recovered from 24 percent of the hospitalized cases and 16 percent of the dispensary cases. This study and other studies of the common diarrheas undertaken late in 1944 and in 1945 led to the estimate that at least one-fifth of the common diarrheas in the theater were shigellosis.

Another laboratory team of the 29th Medical Laboratory investigated the etiology of nonspecific diarrhea in the theater during 1945. One group

worked with troops in the Calcutta area and a second at Delhi. Studying consecutive cases of diarrhea and utilizing good parasitologic and bacteriologic techniques, they obtained the following results:[37]

	Calcutta group (100 cases)	*Delhi group (164 cases)*
Bacteriologically positive instances	49	95
Shigella	(11)	(35)
Salmonella	(3)	(6)
Paracolon	(35)	(54)
Parasitologically positive instances	36	42
Endamoeba histolytica	(17)	(6)
Endamoeba coli	(8)	(10)
Endolimax nana	(1)	(7)
Giardia lamblia	(2)	(7)
Chilomastix mesnili	(1)	(4)
Iodamoeba bütschlii	(1)	(2)
Trichomonas hominis	(3)	(4)
Isospora hominis	(1)	(0)
Dientamoeba fragilis	(0)	(1)
Hookworm ova	(2)	(1)
Negative instances	34	69

Only one stool and rectal-swab examination was made in each case. As many investigators have demonstrated, the percentage of enteric pathogens recovered would have mounted if repeated specimens had been examined. The findings are not at all inconsistent with those of the Sub-Commission on Dysentery of the Army Epidemiological Board and lend further support to the belief that at least one-fifth of common diarrhea in this theater was shigellosis. That the intermediate paracolon group are potential etiologic agents of common diarrhea was suggested by findings of the 29th Medical Laboratory. The diarrhea known as Delhi belly was thought to be the clinical type from which these organisms were recovered.

The following epidemiologic case reports demonstrate some of the problems in determining etiology of the common diarrheas where the usual enteric pathogens are not found:[38]

Epidemiologic case report 14. Numerous cases of common diarrhea characterized by mild watery stools were prevalent in Base Section II and were less frequent at the 20th General Hospital and at Base Section III during October 1944. Laboratory studies by the enteric commission led it to believe that this condition was not a mild bacillary dysentery. Stools from three typical cases were used for inoculation of monkeys. One cubic centimeter of each specimen was introduced through a nasal catheter into the stomach of each of two monkeys. The animals were kept under observation for at least 1 week. All six monkeys had normal stools and showed no signs of illness. The etiology remains unknown.

[37] Essential Technical Medical Data, Headquarters, U. S. Forces, India-Burma Theater, for October 1945.
[38] See footnote 7, p. 328.

Epidemiologic case report 15.—An organization of about 100 men, over a period of 3 days, had 18 cases clinically resembling bacillary dysentery requiring hospitalization. There was fever and frequent bowel movements containing blood, mucus, and pus. Vomiting was present in a few cases. Two cultures were made from each of fifteen cases; in no instance were dysentery bacilli found. Blood agar and other media inoculated with the stools of two typical cases showed no recognizable pathogens. A carrier survey was made on the organization, and not a single carrier was found. Stools from 2 typical cases were used for monkey inoculations, 2 monkeys for each specimen, and each animal was given 1 cc. of stool through a tube into the stomach. The monkeys remained well. Questioning failed to show that any new or replated dishes had been used in the mess, and no acid drinks had been served before the outbreak. Urine from five typical cases was examined by the 9th Medical Laboratory. None contained appreciable amounts of heavy metal. All cases recovered promptly, and no more appeared in the organization. The cause of the outbreak is not explained.

Despite concerted effort in the theater in search of agents of the dysenteries, especially amebas, a large number of cases severe enough to be diagnosed as clinical dysentery remained undetermined. Not all such cases were diagnosed as dysentery, unclassified; many were called common diarrhea.

Should United States troops ever again be deployed in this theater, advance planning and provision of well-trained laboratory and epidemiologic teams would be a necessity.

The dysenteries.—According to data submitted by Van Auken, unclassified dysentery accounted for about 13 percent of all diarrheal disease, the proportion each year being essentially constant except for 1942 when it was 6.5 percent. No deaths were ascribed to this cause. The bulk of unclassified dysenteries were probably bacterial, because, in view of the effort directed toward protozoal forms, especially during the last months of the war, had they been protozoal they would have been classified. Bacillary dysentery and unclassified dysentery together accounted for about 21 percent of diarrheal disease, essentially equaling amebic dysentery. Incidence of unclassified dysentery was lowest in the colder months of January and February and highest in June, July, and August. The incidence rate for the war years as judged by sample tabulations of individual medical records, including secondary diagnoses, was 12.3 cases per annum per 1,000 average strength, almost twice as high as in any other theater.

Amebic dysentery, on the other hand, was the diagnosis for more than 20 percent of all diarrheal disease during the 1942–45 interval. During 1942 and 1943, approximately 10 percent of all diarrheal disease was determined to be amebic; this figure increased in 1944 to about 20 percent and in 1945 to 30 percent.

Much of the relative increase in amebic dysentery may be attributed to an increase in the prevalence of the disease as the period of exposure in the theater lengthened. Other and equally probable factors are the interest in amebic dysentery manifested by the Sub-Commission on Dysentery of the Army Epidemiological Board, the emphasis on parasitologic laboratory examination of feces, the meager facilities for bacteriologic diagnosis, the development of a theaterwide amebic dysentery register with special health record

forms, and various special and specific directives. Furthermore, laboratory diagnosis of *E. histolytica* was generally unreliable until late 1945, except at a few laboratories. Some medical officers also believed that the diagnosis of amebic dysentery often was used when amebic dysentery carrier or amebiasis would have been more proper; because of the existing "amebaphobia" many of the latter were treated as cases of amebic dysentery. These are factors to be considered in a retrospective appraisal of the situation as it existed in the theater. The following epidemiologic case report is illustrative:[39]

Epidemiologic case report 16. This report is based on observations made on 748 cases of amebiasis. Of these, 218 were admitted to the United States Army Hospital in Calcutta during the period May 1943 through June 1944. The remaining 530 were admitted to the United States Army Hospital at Panagarh from July 1944 to February 1945.

On the whole, sanitary control was far superior in the Panagarh area, yet the hospital admission rate for amebiasis was seven times as high there as it was in Calcutta. There were 530 cases of amebiasis in the 3,727 admissions to the Panagarh hospital and only 218 in the 10,961 admissions to the Calcutta hospital.

In striking contrast were the admission rates for bacillary dysentery and acute enteritis, presumably of infectious origin. In Calcutta, the ratio of nonamebic to amebic diarrhea was approximately 3 to 1, while in Panagarh this ratio was reversed. The greater prevalence of nonamebic diarrhea in Calcutta was to be expected in view of the poorer sanitary conditions prevailing there, but the lower incidence of amebiasis was difficult to explain.

It was believed that this paradox could be explained by several factors. The first of these was the length of service in India. At the time of this study, the majority of troops in the Calcutta area had been in India between 6 months and a year, and there were many recent arrivals, while in the Panagarh area the vast majority had been there between 18 months and 2 years. Obviously, the longer exposure to infection played a role in the higher incidence of amebiasis in Panagarh, but it was not the only factor, since the admission rate in Calcutta never approached that in Panagarh during the following year.

Another factor which was considered of great importance was the diligence with which amebiasis was hunted. Before coming to Panagarh, the medical officers on duty at the hospital there had had considerable experience with the disease in the Middle East; they soon had all medical officers in the area ameba conscious. Most of the installations had dispensaries equipped with microscopes, and all cases of diarrhea and other gastrointestinal disturbances were investigated. In addition, a number of enthusiastic flight surgeons, in an effort to control the disease, undertook to do stool examinations on all their personnel. Except in the presence of active diarrhea, the routine stool examinations at both the hospital and the dispensaries were done after the administration of a strong purge. Purgation was rarely used in Calcutta, and as a consequence many cases were probably missed.

Food and water contaminated with *E. histolytica* cysts, either directly or through the agency of carriers and flies, are generally considered responsible for the transmission of amebiasis. If food and water were the only factors involved, it is difficult to explain the high incidence of the disease in the Panagarh area, where sanitary control was excellent. That the protection of food and water was actually effective is indicated by the low incidence of nonamebic diarrheas. It seems reasonable, then, to assume that some other mode of transmission was in operation in this area.

There is good reason to believe that the environment in the Panagarh area was heavily contaminated with *E. histolytica* cysts. Most installations were in the midst of an agricultural area where human feces were used as fertilizer. Numerous natives were employed in construction work and as guards, personal bearers, laundrymen, and clerks. They

[39] Klatskin, G.: Observations on Amebiasis in American Troops Stationed in India. [Official record.]

rarely used the special latrines that were provided for them. The cysts of *E. histolytica* soon lose their viability when subjected to drying, but the warm, humid climate afforded them ample protection especially during the long monsoon. There is every reason to believe that the environment was heavily contaminated with viable cysts and that this was one of the most important factors in the spread of the disease in the Panagarh area.

The incidence of amebiasis and amebic dysentery doubtless was relatively high compared with their incidence in most other theaters, and much of the actual amebic dysentery was correctly diagnosed. However, some diarrheal disease of other nature was incorrectly diagnosed as amebic dysentery.

Despite the excessive frequency of amebic dysentery in this theater, only 2 deaths occurred, 1 in 1943 and 1 in 1945, a record which compares favorably with the experience in the Southwest Pacific where the death rate was higher and incidence lower (tables 60 and 63).

Bacillary dysentery. Approximately 8 percent of dysenteries and diarrheas were diagnosed as bacillary dysentery. Sampling tests suggest 20 percent as a more likely figure. Less than 4,000 cases in the India-Burma sector were reported during the war years.[40] Laboratory facilities and trained microbiologists were very few. Diagnosis was frequently made on the basis of pus and mucus in the stools, without the benefit of laboratory confirmation. The rates for bacillary dysentery were highest during the period from June through September.

The incidence rate of 8.9 cases per annum per 1,000 average strength during the war years is the second highest for bacillary dysentery among the several theaters, exceeded only by the incidence rate in the Africa-Middle East theater (table 62). The rates by years were 4.00, 15.45, 11.35, and 6.07, respectively (table 59). Two deaths from bacillary dysentery occurred in 1943, again representing the second highest mortality rate among theaters (table 63).

The Sub-Commission on Dysentery observed that the convalescent carrier rate was low in a series of 47 patients adequately treated with sulfadiazine; 4 carriers (less than 10 percent) were observed on the basis of 2 or 3 successive cultures started 4 days after treatment was stopped. There were no untreated controls. The following tabulation illustrates the results and the prevalent Shigella types found in followup cultures on cases of bacillary dysentery treated with sulfadiazine at the 20th General Hospital:

Type found	*Number treated*	*Number showing positive cultures*
Flexner V	11	2
Flexner W	9	1
Flexner Z	1	0
Boyd 88	2	0
Boyd 103	5	0
Shiga	5	0
Sh. ambigua	14	1

[40] See footnote 34, p. 378.

Although no controls were used, Hardy had elsewhere shown that 75 percent of untreated patients are carriers when ordinarily discharged from the hospital and 13 percent are carriers 10 weeks later. The three patients with Flexner infection who were carriers did not respond to additional sulfadiazine.

The Sub-Commission spent much time in India making carrier surveys. From 1 August to 5 November 1944, inclusive, 103 (5.3 percent) of 1,936 presumably healthy persons were found to be carriers of dysentery bacilli. Military units with the highest carrier rates were also the units with the poorest level of general sanitation; in one instance, 13 percent of personnel were carriers of dysentery bacilli. The percentage of carriers was 5 percent or more in three companies. Mass treatment was instituted to determine the value of sulfadiazine in remedying the carrier state. A 2-gm. initial dose of sulfadiazine followed by 1 gm. each morning and night thereafter to a total of 10 gm. was given to each man. By this means, the carrier rate for 726 men was reduced from 9 percent to 0.4 percent. No patients with diarrhea reported to sick call from these units for "a considerable period of time following the mass treatment." Table 68 illustrates the results.

The Sub-Commission believed one outbreak to be caused by Shiga's bacillus. The outbreak is here described because of the relative infrequency of this infection.

TABLE 68. *Mass treatment with sulfadiazine in the management of field organizations having high bacillary dysentery carrier rates*

Organization	First culture				Second culture [1]			
	Date	Number cultured	Number positive	Percent positive	Date	Number cultured	Number positive	Percent positive
Aviation Engineer Company A	6 September	246	13	5	19 September	221	1	0.5
Aviation Engineer Company B	23 September	188	[2] 25	13	9 October	182	0	0
Headquarters Company	16 October	292	[3] 29	10	30 October	226	2	.9
Total		726	67	9		[4] 629	3	.5

[1] The second culture was made 7 days after completion of treatment.
[2] Includes 23 positive for Flexner W.
[3] Includes 25 positive for Flexner V.
[4] All those positive on the first culture are included in the second culturing.

Source: Progress and Final Report, Sub-Commission on Dysentery, Army Epidemiological Board, 20 Nov. 1944.

Epidemiologic case report 17.—Early in September, a quartermaster unit of 95 men sent 4 cases of Shiga dysentery to the 20th General Hospital. This unit had arrived in India on 8 August and entrained immediately. Severe diarrhea occurred en route, and, on arrival in Assam on 17 August, four men were admitted to a medical facility as having clinical bacillary dysentery. The unit reached Burma on 21 August 1944. The number of cases of diarrhea reached a peak on 1 September. The unit was visited on 6 September. Forty-two men gave

a history of diarrhea, twelve attended sick call, and a total of ten required hospitalization. Four of the twelve men at sick call were reported positive for *E. histolytica*. Many of the patients had blood, pus, and mucus in the stool, the exudate resembling that of bacillary dysentery. Cultures were made by the rectal-swab method from all men at the unit. Since it had not been believed practical to take plated media to the unit, the swabs were placed in tubes containing saline. Plates streaked on return to the laboratory were found heavily overgrown, and Shiga's bacillus was isolated from one individual only. This method was unquestionably unsatisfactory, since one would have predicted that, in view of the large number of cases of clinical dysentery, at least 20 percent of the unit would have had cultures positive for Shiga. The entire unit was given sulfadiazine in the dosage used for carrier studies. Three days after sulfadiazine was administered, the diarrheas stopped. Cultures were made again from all men at the unit on 20 September, in this instance the rectal swab material being plated directly on "S.-S." agar. No positive cultures were found. Though laboratory evidence is lacking, it is reasonable to assume that the outbreak was one of Shiga dysentery and that sulfadiazine contributed to its control.

The distribution of serologic types of *Shigella* organisms encountered by the Army Epidemiological Board Sub-Commission in India, as compared with Boyd's isolation, is presented in table 69.

TABLE 69.—*Summary of* Shigella *species isolated during entire period of study, compared with Boyd's isolations (7,339 cultures) in India during the period 1932 through 1935*

Species	112th Station Hospital		20th General Hospital		Total		Boyd's isolations (percent)
	Number	Percent	Number	Percent	Number	Percent	
Shigella paradysenteriae:							
Flexner V	11	8.7	77	34.5	88	25.1	0
Flexner W	23	18.1	51	22.9	74	21.1	50.2
Flexner Z	6	4.7	6	2.7	12	3.4	0
Boyd 103	7	5.5	28	12.6	35	10.0	2.7
Boyd 119	1	.8	0	0	1	.3	1.8
Boyd 88	27	21.3	5	2.2	32	9.1	5.0
Boyd 170	0	0	0	0	0	0	2.6
Boyd 288	1	.8	1	.4	2	.6	.9
Boyd D 1	4	3.1	1	.4	5	1.4	.9
Shiga	2	1.6	12	5.4	14	4.0	14.3
Sh. ambigua	22	17.3	37	16.6	59	16.9	5.5
Sh. sonnei	21	16.5	1	.4	22	6.3	10.9
Sh. dispar	2	1.6	1	.4	3	.9	0
Sh. alkalescens	0	0	3	1.3	3	.9	0
Total	127		223		350		

Source: Progress and Final Report Sub-Commission on Dysentery, Army Epidemiological Board, 20 Nov. 1944.

Pacific Area

This area had an average strength corresponding to approximately 4.8 percent of the total Army. It contributed 34,697 cases of diarrhea and dysentery (table 54), equivalent to about 6.6 percent of all reported cases. The

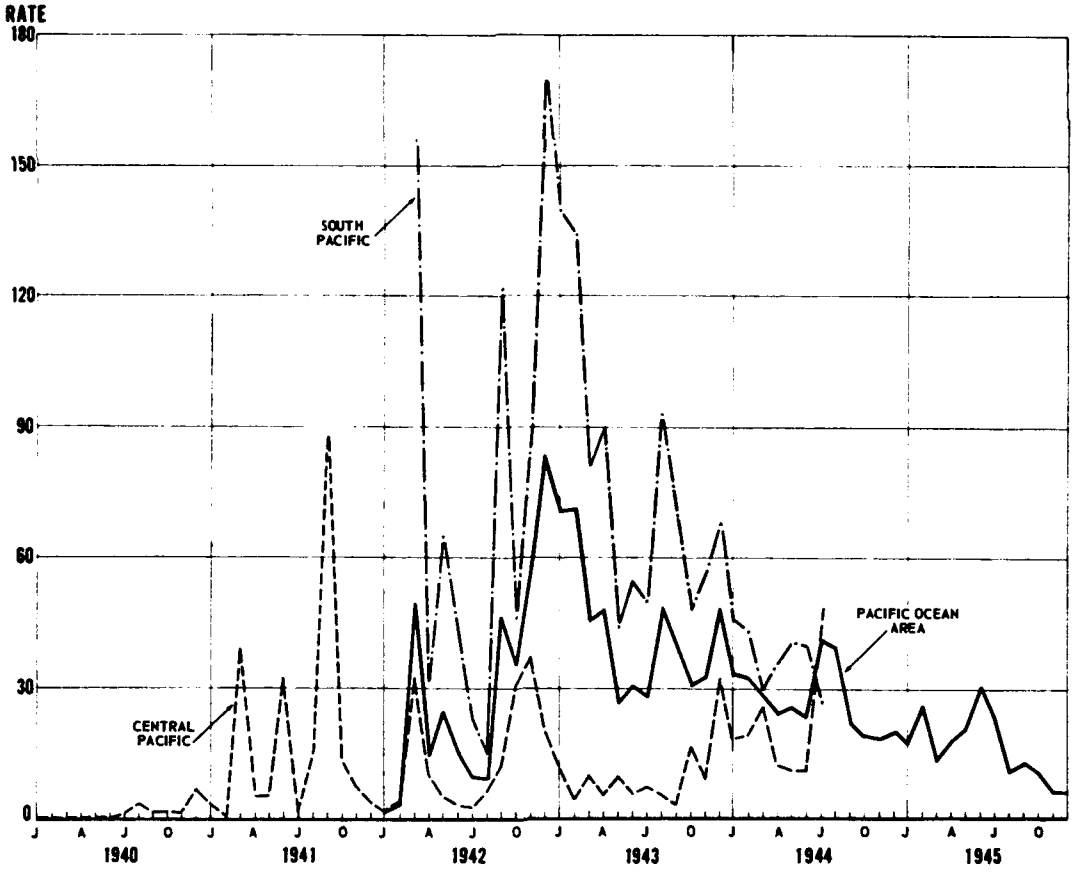
incidence rate for the area was 29.9 cases per annum per 1,000 average strength and ranked fifth among the incidence rates for overseas theaters (table 62).

Hawaiian Department, 1940–41.—The Hawaiian Department of the United States Army in the 2 years before the attack on Pearl Harbor experienced no more than occasional outbreaks of diarrheal disease.[41] Annual incidence rates were low; in 1940 the rate was 1.4. The small population was well housed, and sanitation was excellent. The annual rate for 1941 increased to 18, chiefly as the result of three outbreaks. Two resulted in rates of 39 for March and 32 for June of that year. The third outbreak was prolonged through August, September, and October, with a peak rate of 87.5 in September (chart 42). The first two epidemics were caused by bacterial food poisoning; the third included some bacillary dysentery, which is endemic in the Islands.

CHART 42.—*Incidence of diarrhea and dysentery in the U. S. Army in the total Pacific Ocean Area, Central Pacific area (including Hawaiian Department), and South Pacific area, 1940–45* [1]

[Preliminary data based on periodic summary reports]

[Rate expressed as number of cases per annum per 1,000 average strength]



[1] The Central Pacific and South Pacific areas were not reported as separate areas after July 1944.

[41] History of Preventive Medicine, U. S. Army Forces, Middle Pacific, 7 Dec. 1941 to 2 Sept. 1945. [Official record.]

Incidence.—For the Pacific Ocean Area as a whole, exclusive of the Southwest Pacific area which was a separate theater, the yearly incidence rates for diarrheas and dysenteries for the 4 war years were 34, 43, 28, and 19 (table 54). Separate rates are presented for the Central Pacific and the South Pacific divisions of the Pacific Ocean Area for the years 1942 and 1943 and for the first half of 1944. Although the South Pacific had the smaller average strength, rates for diarrheas and dysenteries were much higher than for the Central Pacific, a difference largely related to the fact that a significant proportion of the strength of the Central Pacific was concentrated in the Hawaiian Islands where sanitation was at a relatively high level. The occurrence of 172 cases of dysentery and diarrhea during March, 4 months after the attack on Pearl Harbor, resulted in a sharp increase in the rate for that particular month to 32, but this was no higher than the rates for any of the 3 peak months of the previous year (chart 42). The Central Pacific rates remained fairly low except for a peak of 37 in November 1942. This rise may be chiefly accounted for by the epidemic of bacillary dysentery on Canton Island, during the development of that base, to be later described. Central Pacific rates again fell fairly low, but fluctuated slightly, until December 1943. Thereafter, total area rates exceeded 10 until November 1945. This last and more persistent rise was associated with combat activity on islands and atolls such as Tarawa, Kwajalein, Eniwetok, Saipan, and Guam. The following epidemiologic case report is illustrative.[42]

Epidemiologic case report 18.—During the last week of November 1943, bacillary dysentery occurred in epidemic form in several regiments of an infantry division engaged in field exercises on coral wastelands on Oahu, T. H. The epidemic was brought promptly under control by emphasis on proper sanitation, especially sanitation related to improved methods of feces disposal and by isolation and treatment of cases and carriers. This outbreak served as an important training lesson in sanitation for this division which subsequently assaulted and captured the southern islands of Kwajalein atoll with a minimum of diarrhea, the first Pacific island occupation in which dysentery was not a major problem.

Logistic problems associated with the distant transport of supplies doubtless contributed to the excessive rates in the South Pacific of more than 100 in March, September, and December 1942, and January and February 1943.

Incidence declined from 171 in December 1942 to 27 in July 1944 in the South Pacific. Monthly rates for the total Pacific Ocean Area parallel those of the South Pacific, but are lower. The theater peak of 83 was in December 1942, following which the trend was a decline to 7 in November 1945. This declining trend was intermittently interrupted by sharp increases related to outbreaks during or shortly after the capture of Tarawa, Saipan, Guam, Iwo Jima, and Okinawa.

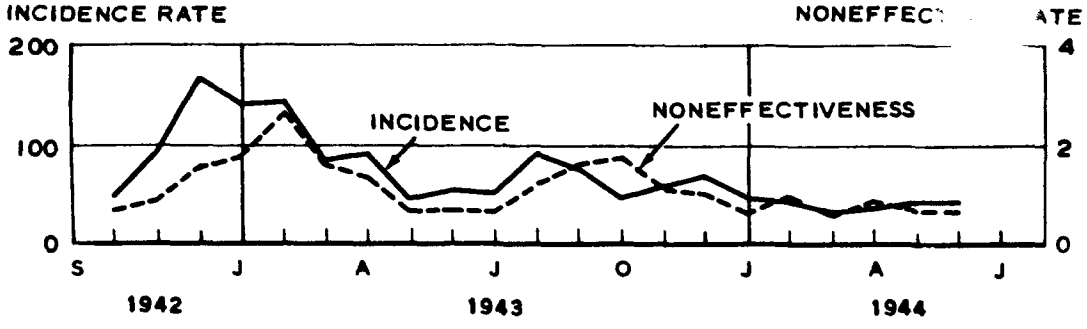
Seasonal occurrence of diarrheas and dysenteries is less clearly defined in this area than in most others. The area included vast expanses of ocean from New Zealand south of the Equator to Hawaii in the north. Outbreaks in New Zealand and other southern areas tended to occur in December, January,

[42] See footnote 41, p. 390.

and February. Peak monthly rate for noneffectiveness in the South Pacific area approached 3 per 1,000 strength and occurred in February 1943 (chart 43). The relatively stable physical environments of the islands and atolls near the Equator resulted in intermittent diarrheal disease with no regular or clear-cut seasonal variation sufficient to be reflected in the gross rates.

CHART 43.—*Diarrhea and dysentery in the U. S. Army in the South Pacific area, by month, October 1942 to June 1944*

[Incidence rate expressed as number of cases per annum per 1,000 average strength. Noneffective rate exp[illegible]verage daily number noneffective per 1,000 average strength]



Source: Monthly Progress Report, Army Service Forces, War Department, 31 Aug. 1944, Section 7: Health.

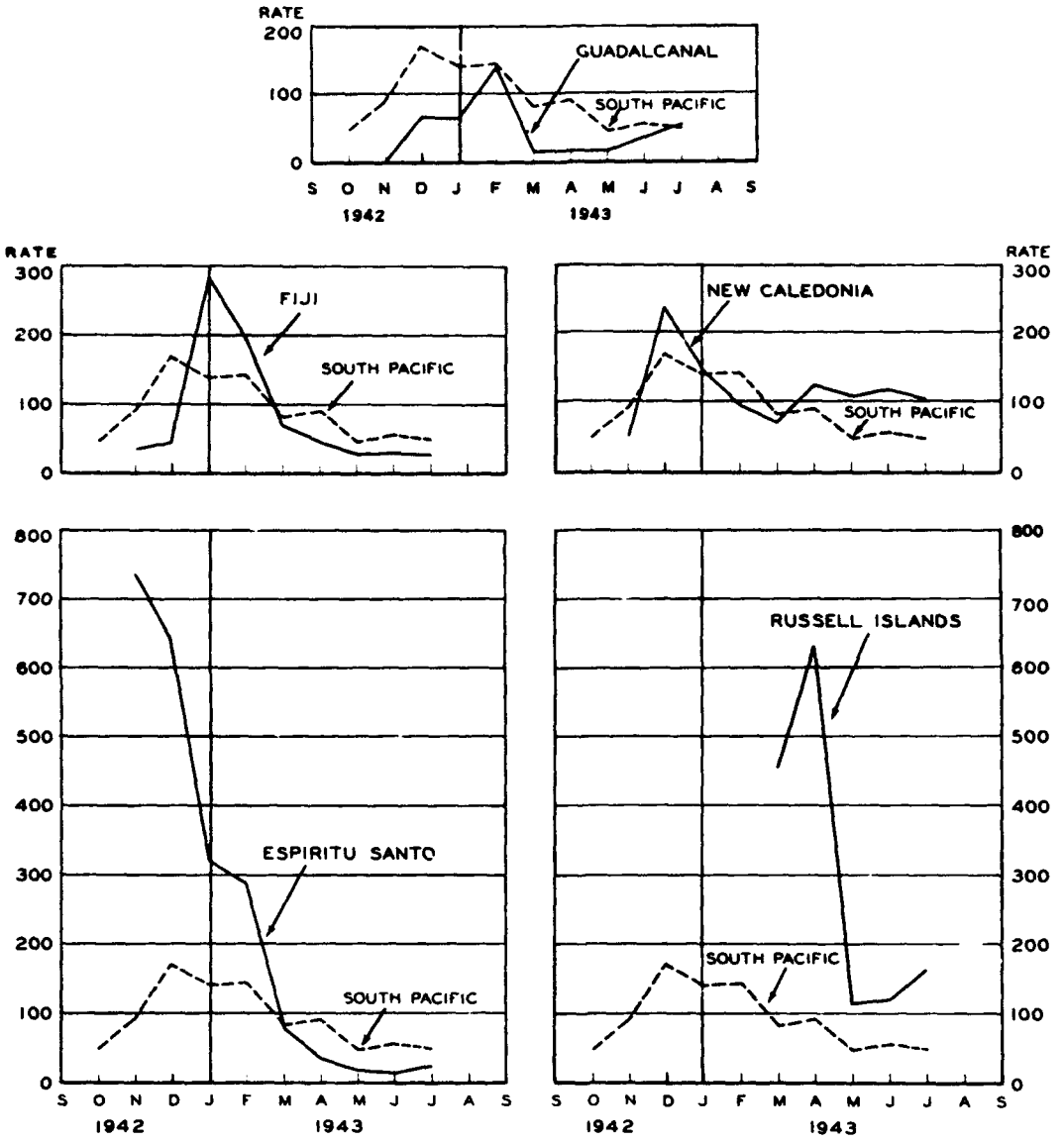
In these locations, occurrence was more related to other factors such as combat or specific lack of sanitary facilities before fixed and sanitated bases were established, than to seasonal changes. For example, initial rates were especially high for troops on Espiritu Santo and the Russell Islands and quite high for the Fiji Islands and New Caledonia (chart 44). The following epidemiologic case reports illustrate diarrheal disease on certain Pacific islands: [43]

Epidemiologic case report 19.—Approximately 1 month before Pearl Harbor day, an expedition of civilian construction workers departed with supporting troops from Oahu to set up bases on Canton and Christmas Islands. This project was designed for training and testing in problems of field sanitation in subtropical and tropical coral atolls. Beginning in September 1942 and extending through the spring of 1943, an outbreak of bacillary dysentery occurred on Canton Island. Bacilli of the Flexner group were recovered from over 400 patients and more than 225 others had diarrhea or dysentery, but positive stool cultures were not obtained. This outbreak was considered serious because of the large proportion of the command affected and because of "the strategical importance of the island as an airbase on the route to Australia, and the close proximity of strong Japanese bases in the Gilbert Islands." Before adequate control measures could be taken, a large number of carriers had been built up, as was discovered by a laboratory team that took rectal-swab cultures on all personnel. Carriers were treated with sulfaguanidine "with satisfactory results." Following this outbreak and until September 1943, all personnel arriving at and departing from the Canton Island garrison were checked by rectal-swab or stool culture for bacillary dysentery infection. Weiss and Finerman have reported the results of their studies of this outbreak.

[43] (1) See footnote 41, p. 390. (2) Finerman, W. B., and Weiss, J. E.: Control of Bacillary Dysentery in a Tropical Outpost. Report of 1,000 Cases. Bull. U. S. Army M. Dept. No. 81: 71-82, October 1944.

CHART 44.—*Incidence of diarrhea and dysentery in the U. S. Army in the South Pacific area and certain islands, by month, October 1942 to July 1943*

[Rate expressed as number of cases per annum per 1,000 average strength]



Source: Monthly Progress Report, Army Service Forces, War Department, 31 Aug. 1943, Section 7: Health.

During the course of the continuing investigation, more than 17,000 stool or rectal-swab cultures were done, and over 1,000 individuals were found infected with *Shigella* bacilli. Later study of a sample of 100 strains from this outbreak revealed that 97 percent were *Shigella* W and 3 percent were *Shigella* Flexner Z.

The reported cases from this one outbreak and study account for approximately half of the cases of bacillary dysentery reported for the entire Pacific Ocean Area during the period from 1942 through 1943.

During the outbreak, or shortly thereafter, organizations surveyed showed about 11 percent of the population at risk to be carriers. Units which had greater admission rates

for clinical disease also had the higher carrier rates. The number of carriers found in some units exceeded the number of clinical cases in those units.

Toward the end of the study, after treatment of known carriers with sulfonamide drugs, only 0.3 percent of troops leaving the island showed positive stool or rectal-swab cultures.

One death occurred among the 818 admissions during the first wave (there were two peaks of incidence) in a 24-year-old white male who was only moderately ill on admission. Symptoms did not seem sufficiently severe to be compatible with death, which occurred on the second day without obvious cause. *Shigella* were recovered at post mortem examination from the lower one-third of the ileum and from the cecum.

The procedure adopted for immediate control of this epidemic which was given greatest emphasis was the search for carriers and their elimination by treatment. In accomplishing this, over 17,000 stool or rectal-swab cultures were taken at this one small island outpost, an almost impossible or impractical method for a larger military population.

The cause of the epidemic was attributed to the difficulty which had been encountered in construction of safe pit latrines in coral sand. Ground water used for bathing was found heavily polluted. Flies were prevalent also, and latrines and messhalls had not been properly protected. The situation was remedied by construction of fly-proofed latrines with impervious concrete pits which were emptied by means of portable cesspool pumps into a truck-mounted cesspool tank, and the pit contents were then dumped into the sea in areas where there was an offshore current.

Epidemiologic case report 20.—Troops and construction personnel were sent to Baker Island in September 1943 for the purpose of building an airbase in preparation for the campaign in the Gilbert Islands. The surf made landings difficult, and the construction of sanitary facilities was delayed. Although no enemy opposition was encountered, there was a high noneffective rate because bacillary dysentery spread rapidly through the entire island garrison during October. A laboratory team was sent to Baker Island to make stool cultures and discover carriers for treatment, as had been done on Canton Island. As sanitation was improved and control of flies instituted, patients and carriers were treated with sulfaguanidine in doses of 10 gm. daily, and the epidemic was brought under control. The number of drug reactions was reported to have been high.

Perhaps the most important lesson which was learned from these two outbreaks was that diarrheal disease, especially bacillary dysentery, may be expected to occur whenever a coral island or atoll is occupied by military forces. This will be true unless adequate, safe means can be rapidly provided for sewage disposal without contamination of the usually limited water supply and unless fly breeding can be rigidly controlled.

Southwest Pacific Area

During the war years, approximately 7 percent of the average strength of the total Army was in the Southwest Pacific area. Summary of statistical health report data reveals that 22 percent of all reported cases of dysenteries and diarrheas of the entire United States Army during this interval occurred in this area. The monthly average strength attained a high of 1,160,212 men and the average mean strength for the war years was 428,223. In terms of military population, this area was second only to the European Theater of Operations. Among the several theaters and areas, it produced the greatest actual number of cases of diarrhea and dysentery with 114,909 reported cases.

The incidence rate for this interval was 67 per 1,000 (table 54), fourth highest rate (table 62) for a theater or area and a rate exceeded only by the China-Burma-India (131), the Africa-Middle East (128), and the Mediterranean theaters (70).

The annual rates of incidence of diarrheas and dysenteries for this area fluctuated considerably because the dynamic character of operations introduced great numbers of men in successive years into a variety of new environments. The progressive buildup of strength is shown by a mean strength of 3,641 in January 1942 and 1,160,212 in October 1945. During 1942 and 1943, troops were introduced into constantly expanding campsites in Australia where need for increased sanitation facilities occasionally exceeded the existing installations. The northward movement with the progress of the war caused troops to enter new environments, from Australia to New Guinea and to the Philippines. The annual rates ranged from 55 in 1944 to 74 in 1945, with 59 and 70 in 1942 and 1943, respectively (table 54). In the latter year, approximately 7 percent of admissions for disease and 3 percent of noneffectiveness were due to diarrhea and dysentery (table 67). The highest annual rate, that of 1945, marked the return to the Philippines, where a large Army was involved in combat operations under conditions which favored intermingling with a friendly native population in which diarrheal disease had been notoriously hyperendemic for many years.

During 1945, rates for diarrheas and dysenteries in Army troops in areas other than the Philippines continued a downward trend which started in 1944. Thus, for troops on the Australian mainland the 1944 rate was 23 and dropped to an estimated 5 in 1945. In areas other than Australian mainland and the Philippines, the 1944 rate was 54 and in 1945 an estimated 28 (first 8 months only). The Army returned to the Philippines in October 1944 and for 1945 had the high rate of 104 (table 54). Again the rates of diarrhea and dysentery demonstrated that when large numbers of troops enter a hyperendemic area for the first time and under combat conditions high rates may be expected; while troops of the same theater, consisting chiefly of seasoned personnel living under more or less stabilized conditions, experience declining rates.

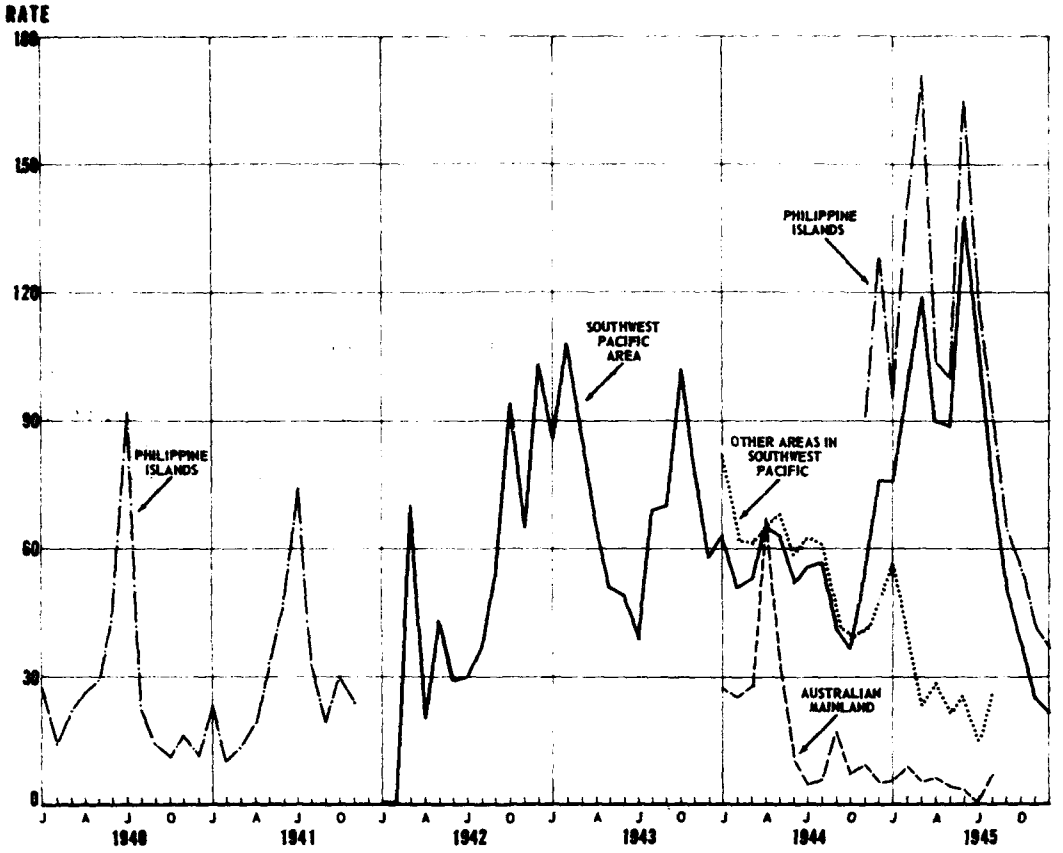
Seasonal occurrence.—Monthly distributions of diarrheal diseases varied, as might be expected, with the northward march. In Australia, peak rates were in October and December 1942 and February 1943, remaining high through March. Rates were lowest in the colder months of May, June, July, and August. For areas other than Australian mainland and the Philippines, January was the month of highest rates in 1944 and 1945 (years for which separate data for subdivisions of the theater are available). By contrast, in the more northerly Philippines seasonal peaks were in June, July, and August, a complete reversal, according to months, within a single theater of operations (chart 45).

Seasonal occurrence among troops in the Philippines before World War II, during 1940 and 1941, resulted in rates under 30 except for the peak months of May, June, July, and August. Sharp seasonal peaks occurred during these

CHART 45.—*Incidence of diarrhea and dysentery in the U. S. Army in the total Southwest Pacific Area, Australian mainland,[1] Philippine Islands,[2] and other areas in the Southwest Pacific, 1940–45*

[Preliminary data based on periodic summary reports]

[Rate expressed as number of cases per annum per 1,000 average strength]



[1] In the Surgeon General's Office, before January 1944, rates for Australia were included in those for the Southwest Pacific Area. After January 1944, rates for Australia were recorded separately.

[2] Data are not available for the period the Philippine Islands were occupied by Japanese forces.

years, reaching over 90 in 1940 and over 70 in 1941. Following the return to the Philippines under combat conditions, peak monthly rates for diarrheas and dysenteries were in March and June, 171 and 165, respectively. With demobilization and cessation of hostilities, the rates for diarrheal disease rapidly declined in succeeding months to become more nearly comparable to those of the prewar Army.

Diarrhea and dysentery deaths.—In the Southwest Pacific and Pacific Ocean Areas, deaths due to conditions included as common diarrheas in this chapter totaled 8 (p. 303). For bacillary dysentery 10 deaths occurred, another 12 were from unclassified dysentery, and 16 resulted from amebic dysentery, a total of 38 (table 63).

The entire United States Army had only 64 deaths due to dysentery (tables 56 and 63) of which 38 (59 percent) were among admissions in the

Pacific areas. Within the 12 percent of mean strength located in these areas, 24 of these 38 deaths occurred in 1945, and all but 1 occurred among admissions in the Southwest Pacific.

Australian mainland.— Troops arriving in Australia in March 1942 had an incidence rate of 70 cases per annum per 1,000 strength which dropped to 20 cases during April but increased to 43 cases in May. The rate of 29 cases in June was followed in successive months by a continuing rise to a peak of over 100 in December 1942 and February 1943 (chart 45).

This increased frequency was anticipated. In an endorsement to a letter [44] from a subordinate command, the Chief Surgeon of the United States Army Services of Supply for the area expressed the opinion that an outbreak of diarrhea or enteritis in the winter months of June and July in the Motor Transport Command was strongly suggestive, clinically and epidemiologically, of bacillary dysentery. He issued the warning that "unless every known method of prevention is instituted, the dysentery rate and non-effective rate will greatly increase * * *. It is strongly recommended that the Surgeon of your Command seriously consider the possibility of endemic dysentery and make recommendations to you for the necessary action to control * * * this disease." The warning unfortunately was not given theaterwide emphasis.

A medical officer at the 2/7th Australian General Hospital at Port Moresby recorded [45] that the majority of patients hospitalized with diarrhea or dysentery came from camps in isolated areas in small villages, often along rivers in which all drinking water was chlorinated and which utilized deep trench latrines. Flies were plentiful in these locations during the hot months. From September 1942 to February 1944, 12.4 percent of all admissions to this hospital were for diarrhea or dysentery, and 2,849 cases were observed. The experience of this hospital accounts for a major part of the bacillary and unclassified dysentery reported for the area during these 18 months as follows:

	Cases	*Percent*
Bacteriologically proved dysentery	1,026	36.0
Bacteriologically not proved dysentery	1,784	62.6
Amebic dysentery and amebiasis	39	1.4
Total	2,849	100.0

The strains of dysentery bacilli recovered in the 1,026 bacteriologically proved cases were:

	Percent
Sh. dysenteriae	13.4
Sh. paradysenteriae, Flexner types	76.0
Sh. ambigua (Schmitz)	8.0
Sh. paradysenteriae, Boyd IV, P 274	2.4
Sh. sonnei	.2

[44] Letter, Headquarters, Motor Transport Command, No. 1, U. S. Army Forces in Australia, to Chief Quartermaster, U. S. Army Forces in Australia, 8 July 1942, subject: Report Regarding Outbreak of Enteritis in Recent Convoy, with 1st endorsement thereto, dated 23 July 1942.

[45] Report, Major Fortune, 2/7th Australian General Hospital, 1944, subject: Dysentery at Port Moresby.

The majority of the bacteriologically not proved diarrheas seen at this hospital were believed to be bacillary dysentery in which the organism was not recovered, because the majority showed typical sigmoidoscopic findings and followed the same course.

No deaths occurred in 2/7th Australian General Hospital experience, although two ascribed to bacillary dysentery were included in 1943 records of the area (table 63). Table 70 shows the experience at this hospital in terms of severity of infection, based upon symptoms, fever, number of stools, and sigmoidoscopic findings of 474 patients with bacillary dysentery according to type of bacillus. Dysentery as seen at this hospital was considered mild.

TABLE 70.—*Degree of severity in 474 cases of bacillary dysentery at the 2/7th Australian General Hospital*

Organism	Mild cases		Moderate cases		Severe cases	
	Number	Percent	Number	Percent	Number	Percent
Sh. dysenteriae (Shiga)	23	41. 1	23	41. 1	10	17. 8
Sh. dysenteriae (Flexner types)	106	59. 2	63	35. 2	10	5. 6
Sh. ambigua	15	65. 2	8	34. 8		0
Bacteriologically unproved dysentery	152	71. 0	61	28. 5	1	. 5
Sh. dysenteriae, Boyd IV, P 274	1	50. 0	1	50. 0		0

Source: Report, Major Fortune, 2/7th Australian General Hospital, 1944, subject: Dysentery at Port Moresby.

A seasonal area rate exceeding 100 was seen again in October 1943 (chart 45). The sort of occurrence contributing to this high rate is illustrated by the following epidemiologic case report:[46]

Epidemiologic case report 21.—Seven explosive outbreaks of diarrhea occurred among troops of the 24th Infantry Division while being transported by railroad between Camp Caves and Toorbul Point, Queensland, Australia, during September and October 1943. Division combat teams were undergoing amphibious training at Toorbul Point. Each outbreak of diarrhea had the characteristics of a point epidemic, with explosive onset and short duration. Cultures never proved the stools to contain dysentery bacilli.

The outbreaks of 29 October are typical. Train 808 was returning troops from Toorbul Point when 89 cases of diarrhea with nausea and vomiting occurred among 339 United States Army personnel. All had eaten a meal at the railway station cafe at Gympie, and many had also eaten pies or cakes at stands adjacent to the railway stations. A subsequent inspection of the eating establishment at Gympie and Bundaberg revealed many deficiencies such as exposed food, many flies in the station and kitchen, lack of screening, inadequate refrigeration, and inadequate dishwashing facilities.

On train 812, examination of box lunches purchased by troops at Caboolture, Queensland, revealed that some meat pies in the lunches were maggot infested and others were sour.

Type "C" or other suitable rations were recommended for personnel on troop trains and eating at stations prohibited until such time as restaurants were under United States Army inspection and supervision, or improved to meet Army standards.

[46] Letter, Division Surgeon, 24th Infantry Division, to Commanding General, Sixth U. S. Army, 4 Nov. 1943, subject: Special Sanitary Report.

The conclusion to be drawn is that if diarrheal disease is to be prevented, adequate advance arrangements are necessary for feeding of troops during movements of units by train.

Figures are available after January 1944 from periodic summary reports to permit separation of experiences of troops on the Australian mainland from others in the area. Late in 1944, data became available for separate evaluation of disease in troops which had returned to the Philippines. Chart 45 shows a sharp rise in incidence of diarrheas and dysenteries for troops on the Australian mainland from 25 in February 1944 to over 65 in April, with subsequent sharp decline. An explanation of the March and April excess was sought by the surgeon of Base Section 1. The following epidemiologic case report summarizes the findings:[47]

Epidemiologic case report 22.—A survey of the several United States Army units stationed in Base Section 1, Darwin, Australia, was made late in April 1944 to determine conditions which might be responsible for the recent unusual rate of gastrointestinal diseases, principally diarrhea.

The 340th Engineer General Service Regiment (less Company A) had averaged 16 outpatients with diarrhea daily for the past 2 weeks, and 4 cases had been hospitalized.

The 49th Air Depot Group had averaged 12 outpatients with diarrhea daily at camp dispensary during 2 weeks, and 2 cases had been hospitalized.

The detachment of 119th Ordnance Company consisting of 38 men had 6 cases of diarrhea of which 4 were hospitalized.

Chinese Labor Camp detachment reported only one case with symptoms of clinical dysentery but stool cultures were not made.

Company A, 340th Engineer General Service Regiment reported one case of diarrhea.

Typical symptoms of the more than 300 cases were headache, malaise, abdominal pains, frequent watery stools, moderate tenesmus, and mild dehydration. The 12 hospitalized cases evidenced hyperpyrexia of 100° to 104° F. and generally severe symptoms. Only one case showed blood and pus in stools, diagnosed as clinical dysentery.

The majority of nonhospitalized patients returned to duty after 24 hours. Sulfaguanidine therapy was used.

Water supplies were potable at all camps.

No camp of the surveyed units had any fly proofing of kitchens and messhalls. Flies were prevalent especially in kitchens and messhalls of the 340th Engineer General Service Regiment main camp. Two camps had latrines less than 100 yards from messhalls. Flies were not numerous at the Company A camp.

Although other modes of transmission were not excluded, the outbreak was attributed to food contaminated by flies.

New Guinea sector (areas of Southwest Pacific other than Australian mainland and Philippines).—United States Army troops in New Guinea experienced a rate of 54 per 1,000 per annum for diarrheas and dysenteries in 1944 with 24,235 cases or 14 percent of all reported communicable disease in this sector (table 54).

Although the area is a hyperendemic area, rates did not attain the high levels of the Philippines, China-Burma-India, or the Middle East. Troops in New Guinea were stationed in most instances beyond the range and influence of native villages.

[47] Report, Medical Inspector, Headquarters, Base Section 1, U. S. Army Services of Supply, 28 Apr. 1944, Subject: Investigation of Gastro-intestinal Diseases in Base Section 1.

During the early weeks of 1945, when rates for diarrheal disease were soaring for the area, the rate in New Guinea was 39, one-fourth of that in the Philippines.

After January 1944 until the end of the war, the rate in this sector declined steadily from 82 to 15 except for a brief seasonal rise in January 1945.

The Japanese enemy in New Guinea experienced high rates for diarrheas and dysenteries. Dudley [48] makes the claim that sulfaguanidine, which the Japanese did not have, saved Port Moresby and that dysentery and malnutrition were in large measure responsible for the Japanese defeat in this location.

The Philippines.— When the Philippines were invaded in October 1944, Southwest Pacific area rates for these diseases were at the relatively low level of 37 (chart 45). Monthly rates for the area then soared constantly to 119 in March 1945 and to 138 in June. Rates for the area other than the Philippines were rapidly declining during this interval.

By February 1945, practically all of the Sixth U. S. Army was on Luzon. The rate for diarrhea and dysentery was 186. For Services of Supply troops in the Philippines the rate was 119.[49] Diarrheal disease with the exception of infectious hepatitis was the most important communicable disease from the standpoint of noneffectiveness.

In contrast to the situation in New Guinea, United States Army troops in the Philippines came into close contact with the native population.[50] No restrictions were placed upon fraternization with the civilian population. Troops ate large quantities of food and delicacies prepared by the friendly Filipinos under uncertain sanitary conditions, which goes far to explain the high incidence of diarrhea and dysentery. Sanitary facilities and habits of the civilian population had deteriorated during Japanese occupation and by destructive effects of war. Satisfactory sanitation was difficult to achieve, because troops were stationed frequently in or near barrios and towns. Many areas had high water tables giving a hazard heightened by the torrential rains that occurred shortly after the troops arrived. Amebic dysentery, seldom recognized in New Guinea, was encountered frequently on Leyte. Rates for diarrheal disease increased during the active phase of the Leyte campaign, but the invasion of Luzon brought real trouble. A widespread sanitation and public health program was initiated in cooperation with civilian authorities. Emphasis was placed on the building and use of latrines by civilians, and upon the collection of refuse and construction of drainage ditches. Posters and various educational signs were used. Troops were instructed in the disease problems of local areas and in measures to prevent infection. Particular emphasis was placed on avoiding native foods, drinking only from authorized water points, avoiding bathing in creeks or streams, and observing fly control.

Fighting within Manila had destroyed or damaged public utilities. The

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[48] Dudley, Sir Sheldon F.: Our National Ill Health Service; An Essay on the Preservation of Health. London: Watts & Co., 1953.

[49] See footnote 11, p. 345.

[50] Annual Report, Chief Surgeon, U. S. Army Forces, Western Pacific, 1945.

city water system was out of operation, and it was necessary for engineer water-supply companies to serve both civilian and military needs. Water sources were heavily contaminated, but potable water was obtained by heavy chlorination. Other sanitary facilities had also been destroyed. Dangers rested in the vast amounts of uncollected garbage and waste, a huge fly population, and numerous unburied dead in streets and buildings. Dwelling areas were congested, and unsanitary conditions characterized markets and food-peddlers. Damaged water and sewage systems were rehabilitated, the dead were buried, and garbage and waste disposal and fly control were begun. The rate for reported diarrheal disease in the Philippines rose steadily during early 1945, to attain 171 per 1,000 per annum for the month of March. Thereafter with improvements in general sanitation and in the military situation, rates declined except for 165 in the month of June 1945 (chart 45).

A preventive medicine officer writing to the Deputy Chief Surgeon of the Southwest Pacific Area decried the situation and felt that diarrheal disease should have more attention. He stated: [51]

> At present, much more time is being spent on venereal disease control, which is only one-eighth as important. At present, a lot of time is being spent making surveys of [helminth] parasites that are not, collectively speaking, of great importance to the Army from the standpoint of producing non-effectiveness. They are of scientific interest and important to civilians who expect to spend their lives in the locality or to individual soldiers. But, it is extremely improbable that the intestinal parasitic diseases would ever immobilize an Army or result in a non-effective rate comparable to bacillary dysentery, the paratyphoid infections or infectious hepatitis. Explosive outbreaks of intestinal parasitic diseases are uncommon and the immediate effects not nearly so disabling as in the case of intestinal diseases of bacterial origin * * *. Experience since the beginning of the invasion of the Philippines has shown that the control of gastro-intestinal infections will be one of the most important public health problems from now until the end of the war.

This preventive medicine officer requested additional personnel and facilities. He was emphasizing a cardinal principle of epidemiologic practice: to evaluate the disease situation and stress the need for attention to the more important causes of noneffectiveness and death. Laboratory effort was seemingly on helminths and ameba, for the reported rate for bacillary dysentery was only 2.71 (table 59). Nevertheless, 6 of the 16 deaths from bacillary dysentery in World War II and for the entire Army were in this area and in the last months of 1944 and early 1945. Furthermore, 11 of 16 deaths from unclassified dysentery occurred in this area and within the same time limits (table 63).

Medical care in the Philippines operation was comparable to that in other theaters. A reasonable conclusion is that case fatality was artificially high because of failure to recognize and report bacillary dysentery, and recognition and reporting is dependent on laboratory diagnosis. Of each 750 cases of bacillary dysentery reported in this area during 1942–45, 1 patient died, whereas 1 death for each 2,000 reported cases occurred in the China-Burma-India and the Africa-Middle East theaters—the other 2 theaters reporting high incidence of bacillary dysentery. There is little evidence of dysentery in a more virulent

[51] See footnote 11, p. 345.

form, although about 8 percent of strains from the 19th Medical Service Detachment (General Laboratory) in the Leyte campaign were Shiga type; more frequently encountered *Shigella* were *ambigua*, V, W, Boyd 88, and Z. Boyd 103, *Sh. sonnei*, and *Sh. alkalescens* were also encountered.

Despite the large amount and excellent quality of determinative enteric bacteriology performed by this laboratory and several others, the conclusion seems logical that many cases of bacillary dysentery infections in this area were not reported as such, but remained especially in the common diarrhea or unclassified dysentery categories.

Incomplete data from summary reports.—During the first 5 months of 1945, when rates for diarrheal disease were at their highest point, the rates for amebic dysentery rose steadily. In contrast, rates for bacillary and unclassified dysentery and common diarrhea reached a peak in March and thereafter declined.[52] Distributions by clinical form during these months were 72 percent for common diarrhea and 12 percent each for amebic and unclassified dysentery. Bacillary dysentery accounted for only 4 percent.

The area rate for bacillary dysentery was 3.30 per annum per 1,000 average strength for the years 1942–45 (table 59). The Southwest Pacific ranks third among theaters and areas in bacillary dysentery, exceeded only by the Africa-Middle East and China-Burma-India theaters (table 62).

The rates by year, 1942 to 1945, were 2.44, 7.20, 3.18, and 2.71. The high rate of 7.20 is largely related to the extensive bacteriologic work on diarrheas and dysentery by the hospital laboratory at Port Moresby. The 2.71 cases of bacillary dysentery per annum per 1,000 average strength during 1945 is low considering that the rate for all diarrheal diseases in the Philippines in the same period was over 100 cases per annum per 1,000 average strength.

Latin American Area

This area included the Panama Canal Department, the Antilles Department, and the South Atlantic Division, and 1.6 percent of the average strength of the Army during the war years. The share of reported diarrheas and dysenteries, 1.4 percent of the Army's total, was in proportion to strength.

The rate of 19 per 1,000 per annum was sixth highest rate among the overseas theaters (table 62). The average strength of 95,613 during the 4 war years contributed some 7,320 reported cases of diarrhea and dysentery (table 54), the South Atlantic Division having the highest rate among divisions of the area and the Panama Canal Department the lowest, a tribute to the existing high standards of sanitation. The Panama rate of 5 for the war years compared with the rate of 9 for troops in the continental United States, with 25 for the Antilles Department, and 75 for the South Atlantic Division. No deaths in the Latin American area either from bacillary or unclassified dysentery occurred. Amebic dysentery caused one death in 1942 (table 63).

[52] Memorandum for file, Maj. P. E. Sartwell, MC, 1 Sept. 1945, subject: Cases and Rates of Diarrheal Diseases in Philippines and Southwest Pacific Area (Including Philippines), January-May 1945.

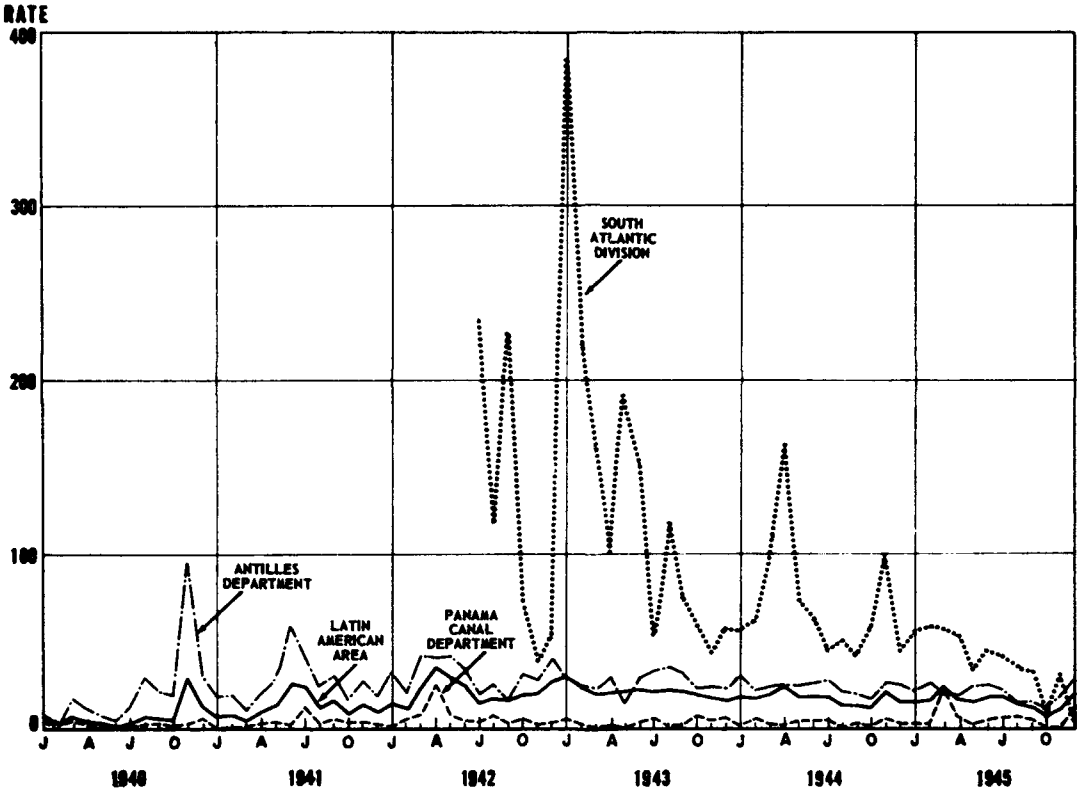
The rate of 75 for the South Atlantic Division was exceeded by China-Burma-India (131), Africa-Middle East (128), and was close to the rate of 70 for troops in the Mediterranean theater.

Seasonal incidence.—United States Army troops in the Latin American area were concentrated close to the Equator. The monthly rates (chart 46) for diarrheas and dysenteries reflect no definite seasonally recurring peaks of incidence in any subdivision of the area.

CHART 46.—*Incidence of diarrhea and dysentery in the U. S. Army in the total Latin American area, Panama Canal and Antilles Departments, and the South Atlantic Division,*[1] *1940–45*

[Preliminary data based on periodic summary reports]

[Rate expressed as number of cases per annum per 1,000 average strength]



[1] No troops were present in the South Atlantic Division of the area before July 1942.

Panama Canal Department.—The annual rate for diarrheas and dysenteries was 3 in 1940 and 1941 (table 54). Rates in the war years were slightly higher, 5 for the entire period. In only 2 months did monthly rates exceed 8 per 1,000 per annum, in April 1942 and March 1945 with 76 and 78 actual reported cases of dysentery and diarrhea. In this sanitation-conscious department,[53] there were more months with little or no diarrheal disease than months with rates over 8 (chart 46). The epidemiologic relationship between common diarrhea

[53] Cox, W. C.: The Prevention of Disease in the U. S. Army During World War II, the Panama Canal Department, 1 Jan. 1940 to Oct. 1945, vol. I. General Health Measures. [Official record.]

and the dysenteries is emphasized by experiences such as this. The common diarrheas are preventable as well as bacillary dysentery.

South Atlantic Division.—The annual rates for diarrheas and dysenteries during the 4 war years were 112, 114, 65, and 40, respectively (table 54). Initial rates were excessively high, but with seasoning of troops and general improvement in local sanitation of military establishments the situation improved.

The trend in monthly rates started with over 200 early in 1942 and ended with less than 30 in 1945, although interrupted by repeated sharp peaks, the highest being 384 in January 1943. That these were outbreaks of bacillary dysentery is suggestive, but data are not at hand to substantiate this possibility.

The Antilles Department.—The frequency of diarrheas and dysenteries for troops stationed in these semitropical islands was fairly uniform throughout the war years. Monthly fluctuations in rates were not great and averaged 25 per 1,000 per annum. Brief epidemiologic case reports illustrate the nature of diarrheas and dysenteries in widely scattered island outposts of the department.[54]

Epidemiologic case report 23.—A brief epidemic of bacillary dysentery occurred at Fort Brooke, Puerto Rico, in May 1942. Five patients with diarrhea were admitted to hospital, and two cases were due to Newcastle-type dysentery bacillus. Cultures were made from the 300 men of the detachment, and 38 were found to be carriers of the same strain. The Puerto Rican Department laboratory believed this represented person-to-person spread after infection from an outside source. Patients and carriers were treated with suflaguanidine, and all became free of the bacillus.

At Camp Tortuguero, Puerto Rico, during the war years, 502 patients with enteric disease were treated at this station, varying from mild diarrhea to severe bacillary infections. There were no deaths. A few instances of amebiasis were without serious complication.

At Borinquen Field, Puerto Rico, numbers of cases and rates for enteritis were:

Year	Cases	Rate
1942	34	7.4
1943	72	12.6
1944	50	18.3
1945 (through September)	54	17.5

Only severe cases were represented, admissions to hospital at this field being so limited.

During Christmas week of 1944, epidemic diarrhea involved approximately 2,000 persons, the difficulty due to a break in technique in reconstituting milk. Cans in which the milk was stored had not been properly cleaned and sterilized. High-pressure steam sterilization was instituted thereafter, and further trouble was avoided. No infectious agent was recovered during the outbreak.

Scattered cases of diarrhea occurred on the base seemingly related to eating in the surrounding towns. Small group outbreaks from time to time were attributed to Puerto Rican foodhandlers employed in base commissary and messes. They required constant supervision and instruction in sanitary foodhandling and personal hygiene.

About 100 persons eating at the officer's mess had diarrhea in January 1945. The origin was presumably in crushed ice. Sixteen cases of amebic dysentery were recognized from March to August 1942 with no demonstrable source of infection.

Seven cases of bacillary dysentery were seen from August to November 1942 and one other in July 1944. Sources of infection were undetermined.

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[54] History of Medical Department Activities, Antilles Department, Preventive Medicine. [Official record.]

At Fort Bundy, Puerto Rico, the few cases of diarrhea were mild and of short duration and included no dysentery.

In Jamaica, British West Indies, only 49 patients with enteric infection were sufficiently ill to be admitted to hospital; 10 bacillary dysentery, 1 amebic dysentery, and the remainder common diarrhea. No outbreaks of food poisoning were recognized.

In Curaçao, Netherlands West Indies, the rainy season and the fly season occur in late fall and early winter. About 50 percent of patients admitted with enteric disease had bacillary dysentery of the Flexner or Schmitz types.

The 359th Station Hospital, Trinidad, British West Indies, admitted 243 cases of common diarrhea during the war years: 39 enterocolitis, 95 gastroenteritis, 39 colitis, 15 diarrhea, and 55 enteritis.

One epidemic of food poisoning led to hospitalization of 50 patients on 3 October 1943. The outbreak was related to chicken pie, meat for which had been cooked 16 hours previously and thereafter held at room temperature before the pies were made. Hemolytic *Staphylococcus albus* was recovered from the meat, but no pathogens from stools of patients.

Several cases of dysentery were due to *Shigella* of undetermined types. Of 8 cases of amebic dysentery, 5 were among officers and men attending the School of Jungle Warfare during late 1943 and early 1944. They were believed due to a cook harboring the organism.

In British Guiana, South America, excluding outpatients, admissions for diarrheas and dysenteries were as follows:

Year	Number per 1,000 per annum
1942	1.3
1943	18.0
1944	14.0
1945 (first 8 months only)	3.0

This base had no serious outbreaks nor was there a death from intestinal infection. Sanitary conditions were excellent.

Bacillary dysentery.—For the area as a whole and for all war years, slightly more than 300 cases of bacillary dysentery were identified (table 59), with no deaths.

The laboratory of the 161st General Hospital was especially active in enteric bacteriology.[55] The hospital cared for 64 cases of Flexner-type dysentery, 2 Sonne and 2 Newcastle, with 9 others unclassified. The 43 cases of bacillary dysentery at this one hospital in 1943 were more than half of all those which occurred in the Latin American area that year. The Antilles Department had far less diarrhea and dysentery than the South Atlantic Division, yet one-fifth of the area total was from this single hospital. The amount of laboratory work often determines the amount of bacillary dysentery.

North American Area and Alaskan Department

The best rates for diarrheas and dysenteries during the war years, lower even than for troops in the continental United States, were attained by troops stationed in the North American area and in Alaska. The exact rates were 4 for the North American area and 5 for troops in the Alaskan Department, comparable alone with the Panama Canal Department rate of 5 (table 54), a subdivision of the Latin American area.

[55] Professional History of Internal Medicine in World War II. The Antilles Department. [Official record.]

The North American area had an average strength of 44,253, but troops were located in such widely separated places as Newfoundland, Greenland, Iceland, and Bermuda. Reported cases of dysentery and diarrhea numbered 732. Thus, 0.7 percent of the average strength of the Army contributed approximately 0.1 percent of the total intestinal infection.

The Alaskan Department with an average strength of 72,345 had 1,427 cases of dysentery and diarrhea, so that 1.2 percent of average Army strength had only 0.3 percent of the total cases.

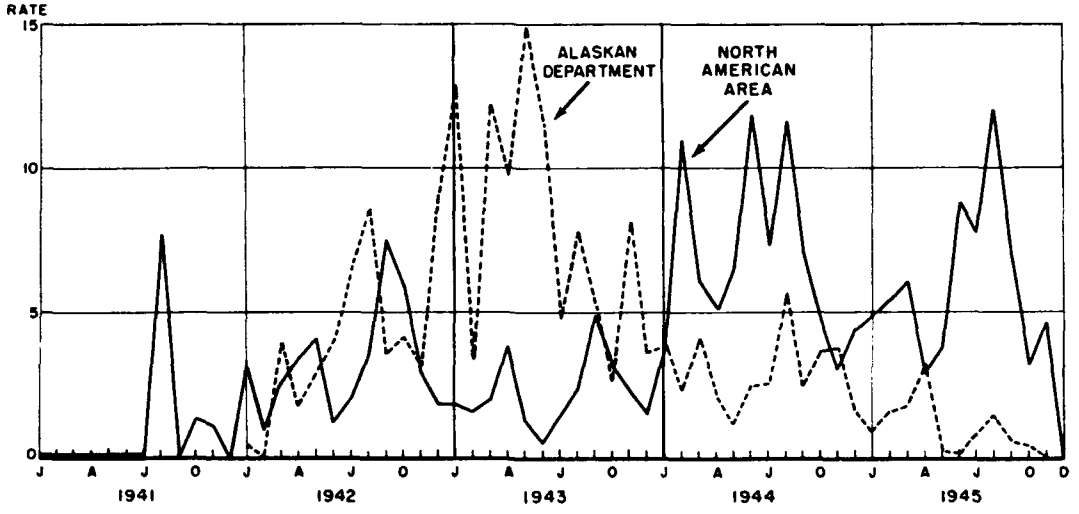
No deaths occurred in either area.

Monthly and seasonal incidence.—Monthly rates in the Alaskan Department reflect no clear-cut seasonal incidence. Rates rose from 2 cases per annum per 1,000 average strength in April 1942 to 15 in May 1943. Thereafter, and with a declining average monthly strength in the area, rates fell to 1 in August 1945 (chart 47). Dysentery rates remained low although bacillary dysentery was considered endemic in civilian and native settlements. The rate for bacillary dysentery was 0.6, with only 30 reported cases. The relatively high incidence of diarrheal disease in 1943 is partially explained by a concomitant frequency of gastrointestinal diseases ranging from gastric hyperacidity and gastric neurosis to peptic ulcer.[56] The new units coming to the command had a high proportion of older and limited service men, many reclassified by their draft boards and having an EPTI (existing prior to induction) status. Many minor gastric disturbances classed as common diarrhea were believed related to the existing monotony of the diet. The professional opinion was that low morale was also reflected in such disorders.

CHART 47.—*Incidence of diarrhea and dysentery in the U. S. Army in the North American area and Alaskan Department, 1941–45*

[Preliminary data based on periodic summary reports]

[Rate expressed as number of cases per annum per 1,000 average strength]



[56] Manning, F. G.: History of Preventive Medicine in World War II, Northwest Service Command. [Official record.]

Occasional small outbreaks of food poisoning occurred. Recognized bacillary dysentery and even unclassified dysentery were rare. Amebic dysentery was almost nonexistent.

The North American area gives suggestion of some seasonal occurrence of diarrheas and dysenteries since rates are highest in midsummer. Cases were so few that rates of over 6 per 1,000 per annum were attained in only 12 months of the 4 war years, 10 of those being summer months.

Factors contributing to low incidence.—Reasons for the low incidence of diarrheas and dysenteries in these two areas as contrasted to all others are not easily determined from the available data but may possibly include the following:

1. The type of climate and the cold environment found especially in Alaska, Iceland, and Greenland. (However, the tropical but well-sanitated Panama Canal Department had as good a record.)
2. The exercise of good control over water supplies for all bases in Alaska and most bases in other portions of these areas.
3. Low endemicity diarrheal disease in native population. Diarrheal disease, however, was prevalent and occasionally epidemic. In Newfoundland, for instance, 10,000 to 12,000 cases of diarrhea occurred in Saint John's during the summer of 1942.[57]
4. Adequate screening and protection of foodstuffs from flies; also relatively low prevalence of the common *Musca domestica* in many military units.
5. Rapid installation of water carriage sewage disposal units until, by the end of 1943 in Alaska especially, practically every unit was so provided regardless of how remote the location.
6. Decrease in average strength after summer of 1943.
7. Use of military foodhandlers.
8. Routine and frequent sanitation inspections with emphasis on mess sanitation.
9. Rigid emphasis in Alaska on mess sanitation. (This was more forcefully brought home to those with command functions whenever and wherever the diarrheal rate showed tendency to increase and also resulted, doubtless, in failure to report some few sporadic and mild outbreaks for fear of reprimand.)
10. Isolation and lack of contact with native population in many of these locations.
11. Practical public health educational programs regarding sanitation. In the history of preventive medicine in the Northwest Service Command, Manning states:

> It is surprising that the diarrheal disease rate was not appreciably higher in the early days of this Command. There were rapid changes in mess personnel in contractor camps. Education of camp managers and mess stewards * * * was a slow and difficult problem * * *. By 1945 screening of buildings, where required, had become universal; the flush toilet had replaced the pit latrine even in the most isolated camps; dishwashing procedures had become more standardized; mess personnel had become conscious of the need

[57] History of Preventive Medicine in World War II, Eastern Defense Command. [Official record.]

for mess sanitation and how to accomplish it; * * * and camp foremen, along with company commanders, were contacting medical facilities to assure that monthly examinations were conducted on schedule; our people had been educated.

Iceland Base Command.—In this command, according to the history of preventive medicine for the Eastern Defense Command, "intestinal infections have been practically nonexistent * * *. In fact, such long periods of time elapsed between the isolated cases that no special records of these diseases were kept. No epidemics of this type of disease ever occurred. Even in the early days of the command and during the period of maximum troop strength many weeks elapsed between cases and those were isolated individual cases, never groups."

Greenland Base Command.—For this command, the history just cited states: "There is no record of any serious outbreak of intestinal infections. We have never had any cases of amebic or bacillary dysentery, cholera, or protozoan infections. There have been a few isolated instances of common diarrhea resulting from spoiled or improperly prepared food but never to any serious extent."

Newfoundland Base Command.—Apparently this base command contributed a large proportion of the total diarrheal disease for the area. Diarrhea was particularly prevalent at this command throughout the war. In 1942, the incidence rates for diarrhea at Fort Pepperell and Fort McAndrew, two of the larger units in Newfoundland, were 20.27 and 25.11 cases, respectively, per 1,000 average strength; in 1943, 35.87 and 28.28, respectively; in 1944, 19.36 and 13.24; and in 1945, 18.26 and 18.10. Unclassified dysentery occurred in 1942 only at Fort Pepperell, at a rate of 2.53 cases per 1,000 average strength; bacillary dysentery occurred at this fort in only one of the war years—1944—at a rate of 0.40 case per 1,000 average strength. Although there was no dysentery at Fort McAndrew during the war, an isolated case of typhoid fever occurred there in 1944.

An epidemic estimated at 10 to 12 thousand cases of diarrhea occurred in Saint John's, Newfoundland, during August and September 1942. During this interval, soldiers were ordered not to eat or drink in any establishment in that city. This shows that diarrheal disease was prevalent in Newfoundland and might account for the relatively high incidence in contrast to other base commands of the area.

Bermuda Base Command.—Outbreaks of diarrheal disease were small, and only two occurred, with 28 cases in one instance and 33 in the other; both were considered the result of food infections and occurred in June 1944 and in April 1945. A few reported sporadic cases of diarrhea and of mild dysentery occurred.[58]

Bacillary dysentery.—Only 39 cases of bacillary dysentery were reported in these areas during 1942–45 (table 59). Over-all incidence was 0.08, and rates for individual years were 0.10, 0.07, 0.05 and 0.15, respectively, the lowest rates reported in any theater or major command area. There were no deaths,

[58] See footnote 57, p. 407.

and noneffective rates were extremely low. Unclassified dysentery was similarly infrequent. Some few were cases reported from Alaska in 1943; none in 1945. Worthy of note is that in this area with so little recognized dysentery, the incidence of common diarrhea was also extremely low.

GENERAL SUMMARY OF EXPERIENCE

Experience of the United States Army during World War II would seem to justify continued inclusion of the diarrheas and dysenteries among so-called tropical diseases. Certainly the incidence was highest in those theaters and areas in tropical and semitropical positions and was lowest in theaters, or their major subdivisions, closer to the poles. Incidence was lowest in the Alaskan and North American areas. It was lower in northern than in southern service commands among troops stationed in the continental United States. In the Latin American area, the South Atlantic Division located on the Equator experienced rates among the highest attained in the several theaters and areas. In the Southwest Pacific, incidence was lower in Australia than in New Guinea or the Philippines. The northerly situated European theater experienced low rates. In general, 40 degrees north latitude was the dividing line between relatively good and bad experience with these diseases. South of this line lay the China-Burma-India theater, the Middle East theater, the North African and Southern Italy portions of the Mediterranean theater, and the Southwest Pacific area. These all had high incidence and contributed a great preponderance of the total cases of World War II. The notable exception to this pattern is found in the Panama Canal Department, located only a few degrees north of the Equator, where the monthly rates for diarrheal diseases were among the lowest reported. The emphasis on good sanitation in this tropical, fixed base resulted in this record and demonstrates that control of these diseases can be accomplished even in the Tropics.

Severe mass problems of diarrheal disease can occur north of 40 degrees north latitude. The experience of German prisoners of war in the European theater is an example of high morbidity with quite high mortality. Diarrheal diseases occur endemically and epidemically in the civilian populations of Iceland, Greenland, and Alaska. United States Army Troops were deployed during World War II in these areas adjacent to the Arctic Circle in rapidly constructed but quite well-sanitated bases and stations. There was little diarrheal disease. Deployment of large numbers of troops under actual combat conditions in such northern latitudes conceivably could result in an altogether different situation. Little is known about field conditions in the Arctic and their relation to mass problems of diarrheal diseases. Expeditions into the Arctic during World War II have yielded information which suggests sanitary disposal of feces and provision of safe water supply could become difficult problems for large populations in permafrost or constantly frozen areas. Despite the relatively favorable experience during World War II in northern latitudes, diarrheal diseases may become special problems of an army in com-

bat in the Arctic. The history suggests that research and field tests are needed.

Highest rates were encountered when United States Army troops intermingled in densely populated areas with Eastern peoples, as in China-Burma-India, the Middle East, North Africa, and the Philippines. It was at these points of contact with Eastern civilizations that highest rates of diarrheal disease occurred. Diarrheal disease was hyperendemic in these locations into which United States troops were introduced. The criticism was often made that United States troops, accustomed to the advanced sanitary practices of Western civilization, were not sufficiently briefed regarding precautions necessary to prevent personal and mass diarrheal disease in these Eastern locations. Should United States troops again be required to contact these peoples, much diarrheal disease could be prevented by adequate advanced training of personnel, by early availability of sanitation facilities and materials, and by not permitting the use of natives as foodhandlers in military messes.

Critical Periods of Risk

The critical times and circumstances in which increased or epidemic incidence of diarrheal disease occurred during World War II among United States Army troops fell into the following major categories:

1. *Within a few days or weeks of the first entry of troops (either first arrivals or rotated personnel) into hyperendemic areas, especially under conditions which enabled contact with native populations.*—In several theaters, it did not take long, following the arrival of large numbers of United States Army susceptibles, for epidemics to occur. Troops among the early arrivals were not always able to provide ideal sanitation for themselves because of shortage of the necessary supplies. Education of these troops in personal measures to be taken to prevent diarrheal disease in these foreign environments often was lacking or inadequate.

The employment of natives as foodhandlers (as in the China-Burma-India theater) or eating with friendly natives (as reported in the Philippines) or eating in unauthorized native restaurants (as reported in North Africa and the Middle East) were frequently considered by preventive medicine authorities to be associated with high incidence. Minimizing or neutralizing these causes by training, regulation, and adequate advance planning could conceivably result in prevention of a large amount of diarrheal disease.

2. *While troops were engaged in combat, especially in war of movement.*—Under such conditions, excreta disposal, provision of safe food and water, fly control, and messgear sanitizing became difficult problems. This was particularly the case when troops were pinned down by enemy fire. There was typically a correlation between actual combat and increased incidence of diarrheal disease (as is well documented for the European theater).

The consistent observation that there was much less incidence when troops

in actual combat were subsisting on individual packaged rations indicates at least one important preventive measure.

Additional research is needed, pointing toward better field sanitation techniques and facilities for frontline troops during actual combat.

3. *During training maneuvers when troops had not yet had sufficient education in field sanitation practices.*—The obvious preventive measure is to schedule first the necessary instructions in field sanitation and in personal hygiene under field conditions.

4. *During transportation of troops by rail (for example, as in the travel across India to Assam and as in Australia from the fixed bases to training areas) or by excessively overcrowded overwater transport.*— Provision en route of adequate facilities for messing, prohibition of unauthorized use of native restaurants or food vendors, and planning for adequate safe drinking water supplies are important whenever troops must be transported by rail if outbreaks of diarrheal disease are to be prevented.

Overcrowding of troop transports, with resulting overtaxing of toilet and messing facilities, and with increased opportunity for contact spread should be avoided. If overcrowding is necessary, additional emergency sanitary facilities for excreta disposal should be provided. Research toward development of a shipboard item for this purpose would be worthwhile. Messing facilities should not be overtaxed. Instead they should be utilized for only one prepared meal daily—other meals being provided, for example, from individual packaged rations. Nor are the diarrheal diseases the only ones for which there is increased risk with overcrowding. It is better to prevent the basic cause unless a drastic military situation contraindicates.

5. *Prior to or during the construction of fixed bases.*—At such times, construction and engineer personnel especially are at risk. The troops involved are apt to be existing under difficult sanitary conditions. Primitive excreta disposal and messing facilities must be used for a prolonged period of time. Often the water supply is limited in per capita volume and requires special care because of an untreated and untested source. Special emphasis on training for and enforcement of good field sanitation practice are indicated for personnel to be assigned such duty.

6. *During the taking and holding of such terrain as coral atolls where conventional field sanitation techniques were found to be inadequate.*—Problems of excreta disposal and safe water supply development in adequate amounts, often under combat conditions, were especially difficult on the smaller atolls. The eventual development of impervious concrete sewage holding pits and disposal of the collected sewage at sea was a solution to the problem during World War II. Provision of large stills for distilling sea water enabled a safe but paucivoluminous supply in contrast to the often brackish and easily contaminated water supply from the fresh water lens of the pervious coral atolls.

The Arctic is another example of terrain where conventional techniques are inadequate.

Doubtless better methods await needed research and development for use when large numbers of troops enter such specialized environments if future unnecessary diarrheal disease in the Army is to be prevented.

7. *Among prisoners of war when overcrowding was increased in emergency enclosures or camps.*—This was true of both United States and enemy prisoners of war. Overcrowding associated with inadequacies of water supply and sanitation facilities was typical in emergency enclosures or camps. Characteristic of prisoners of war is a demoralization and lack of discipline. Sanitary disciplines must be very stringent at the very time of the demoralization if much morbidity and mortality is to be avoided. Senior officers and men captured by the enemy should insist upon the best possible sanitation discipline among their own personnel. Briefing of men prior to combat situations, in which they might become prisoners, should include information regarding the prevention of enteric diseases which so notoriously occur in such situations.

Prevention and Control

Specific immunization.—Specific preventive measures were not developed for the various diseases included in the category of diarrheas and dysenteries during World War II. Research was conducted which led to the preparation of a polyvalent dysentery vaccine, but no successful evaluation was accomplished. Further research and controlled evaluation seem indicated. The high rates for diarrheas and dysenteries in several theaters where typhoid and paratyphoid A and B fevers also were endemic in native populations but amazingly infrequent among vaccinated United States Army troops, seems to emphasize the value of the triple typhoid vaccine. Development of a successful specific prophylactic for the dysenteries could result in lowering the incidence of not only reported bacillary dysentery but also that significant proportion of unclassified dysentery and the common diarrheas which is caused by the dysentery bacilli. More could be gained by such research if successful, than a simple perusal of reported bacillary dysentery incidence would suggest.

Isolation and treatment of recognized cases and carriers.—Early diagnosis and isolation was considered important to the control of the specific dysenteries. Adequate treatment to eliminate infection was considered fundamental for both control and good medical practice. Isolation was quite regularly utilized for the recognized bacillary dysentery cases but not so regularly for the common diarrheas. A significant proportion of the latter were doubtless unrecognized cases of *Shigella* or *E. histolytica* infection. This latter fact was not invariably appreciated nor was it always realized that most of the cases of the common diarrheas are infectious and are subject to the same general control measures as are effective for the specific dysenteries.

Surveillance of recognized carriers of specific dysenteries was quite routinely accomplished. In several locations, mass attempts at bacteriologic

discovery of bacillary dysentery carriers and their treatment with the sulfonamide drugs was attempted. Such methods proved cumbersome and were impractical or impossible with the available facilities in outbreaks involving large populations.

Experimentally, in a few units with high incidence of recognized cases and carriers of bacillary dysentery, a mass prophylaxis using the sulfonamide drugs was attempted, and results seemed quite encouraging; but none of the experiments were well controlled. Sulfanilamide and its related compounds which at first seemed to be of such great value in therapy of bacillary and unclassified dysenteries later gave variable results. Doubtless the development of some resistant strains of dysentery bacilli contributed. Antibiotics, other than penicillin, which offer so much promise as therapeutic and prophylactic agents were not available during World War II.

Sanitary excreta disposal, fly control, and food and water sanitation.— The basic principles entailed in the sanitary disposal of human feces; in insect (especially common fly) control; in the sanitary supervision of the preparation, processing, and serving of foods (especially those which are moist or eaten raw); in the attainment of good personal hygiene of troops and particularly of foodhandlers; in the protection and purification of water; and in the development of adequate volumes of water supply for the literal washing away of the potentials of contact spread, were important to the prevention and control of both the specific dysenteries and the common diarrheas.

It is one thing to know that the efficient application of these basic principles can effectively prevent or control this group of diseases; it is another to apply them efficiently throughout a vast military organization dispersed in varied environments where local situations require particularized methods for rapidly achieving the desired objectives. From the history of the diarrheas and dysenteries in the United States Army in World War II, many lessons may be learned which will be of value in minimizing future incidence.

Food Poisoning

CHAPTER XVIII

Salmonellal Infections

Dwight M. Kuhns, M. D.[1]

HISTORICAL NOTE

The early history of salmonellal food poisoning is not directly related to the military; however, it is briefly presented here to orient the reader as to the status of the problem in past years. In 1888, an epidemic of food poisoning occurred in Frankenhausen, Germany, that was traced to contaminated meat.[2] From the feces of a fatal case of the disease, Gärtner isolated an organism which he called *Bacillus enteritidis*, because of the enteritis produced. The organism was renamed *Salmonella*, to honor Dr. Daniel E. Salmon in recognition of his research on hog cholera. Dr. Salmon was the first to describe a member of this group. In 1885, Dr. Salmon and Dr. Theobald Smith isolated *Bacillus cholerae-suis* from cases of hog cholera (*Salmonella choleraesuis* in modern terminology).[3] Although it was excellent work, Dorset and de Schweinitz, in 1903, demonstrated that hog cholera is actually caused by a filterable virus, the bacteria being secondary invaders and not always present.

The rod-shaped motile bacterium (implicated at Frankenhausen) was subsequently discovered in other cases of food poisoning, although many other types of food poisoning were not found to be caused by *Salmonella* organisms nor were all the newly discovered related species first found in food-poisoning cases. Staphylococci were implicated in cases of food poisoning several years before the discovery of the genus *Salmonella*. Furthermore, many of the *Salmonella* species were not first discovered in connection with food poisoning. Thus, *Salmonella aertrycke*, today known as *Salmonella typhimurium*, was first isolated from mice by Löffler in 1890, while *Sal. choleraesuis*, as has already been noted, was first found in swine. Still others, such as *Salmonella oranienburg*, were first isolated from the feces of healthy humans.

Numerous cases of food poisoning are mentioned in the older literature such as those described by van Ermengem in which sausages were suspected of having caused illness.[4] A Belgian inspector of meat, reputed to have been an expert in his field, examined the meat and declared the sausages to be fit for human consumption. The inspector developed severe diarrhea 10 hours

[1] Acknowledgment is hereby made to Martin Roth, M. A., for his faithful assistance in the research for historical material for this section and in the final preparation of the manuscript.

[2] Gärtner, A.: Ueber die Fleischvergiftung in Frankenhausen a. k. und den Erreger derselben. Cor.-Bl. d. allg. ärztl. Ver. v. Thüringen, Weimar, 1888, xvii, 573–600.

[3] Salmon, D. E.: Investigations in Swine Plague. U. S. Bur. Anim. Indus. 2d Ann. Report, 1885, pp. 184–246.

[4] Dack, G. M.: Food Poisoning. Chicago: The University of Chicago Press, 1949.

later, on 26 October 1895. This was followed by fever, high pulse, and abdominal distress. Six days later, he died. Necropsy revealed viscera laden with *Salmonella enteritidis*. This account is mentioned here not to cite the fact that some inspectors may lack competence but rather to point out that food, highly contaminated with pathogenic organisms, can appear quite satisfactory in gross appearance as well as in smell and taste.

Many organisms similar to *Sal. enteritidis* were discovered even before the First World War. However, up to and including the period of World War I, there is no substantial literature on either *Salmonella* or food poisoning. Scattered reports are available on what were then designated as the paratyphoid fevers. This group consisted of a variety of gastroenteric conditions similar, though not identical, to typhoid fever. It is generally believed that the paratyphoid fevers were by no means a major problem to the United States Army during World War I but what might be more accurately described as a nuisance. During 1916 and 1917, there were sporadic and epidemic occurrences of the disease in United States Army personnel along the Texas-Mexico border and in Mexico itself. Soon after, it was learned that outbreaks were occurring in France among British and French troops. In July 1917, the United States Army incorporated paratyphoid A and paratyphoid B organisms in its regular typhoid vaccine.

Table 71 shows the incidence and deaths for paratyphoid fever, most of which resulted from contaminated food or water, among officers and enlisted personnel from 1 April 1917 to 31 December 1919, inclusive. It is necessary to view the statistics with caution. First, a wide variety of conditions in the World War I period were consigned to the ill-defined categories paratyphoid A and paratyphoid B. They were milder than classical typhoid fever, with diarrhea a more characteristic feature. Secondly, an individual who died in an Army hospital was listed as a paratyphoid (or typhoid) death if that was the condition for which he had been admitted. It was the practice of the Surgeon General's Office, at the time, to list for statistical purposes the disease for which a patient was initially admitted to the hospital as the cause of death if the patient died.[5] Thus, while many paratyphoid deaths were, in fact, ascribed to paratyphoid fever, others were not. Further, the concept of food poisoning had not yet been clearly defined, many cases being considered examples of ptomaine poisoning, a meaningless classification.[6]

During the interval between World War I and World War II, little of note occurred in the history of salmonellal food poisoning, at least insofar as the Army was concerned. One development of great significance, however, was the classification schemata that were gradually devised through biochemical

[5] (1) The Medical Department of the United States Army in the World War. Communicable and Other Diseases. Washington: U. S. Government Printing Office, 1928, vol. IX, p. 15. (2) The Medical Department of the United States Army in the World War. Statistics. Washington: Government Printing Office, 1925, vol. XV, pt. 2, p. 12. (3) Annual Reports of The Surgeon General, U. S. Army, to the Secretary of War. Washington: Government Printing Office, 1921, pp. 20–21; 1934, pp. 5–6; 1936, p. 7.

[6] (1) Morgan, H. R.: The Salmonella. *In* Bacterial and Mycotic Infections of Man. 2d ed. Philadelphia: J. B. Lippincott Co., 1952, ch. 17, pp. 420–436. (2) Savage, W. G.: Bacterial Food Poisoning. *In* A System of Bacteriology in Relation to Medicine. London: H. M. Stationery Office, 1929, vol. III, ch. 12, pp. 407–413.

TABLE 71.—*Occurrence of paratyphoid A and paratyphoid B in World War I (absolute numbers and annual rates)*

[Rate expressed as number of cases per annum per 1,000 average strength]

Country	Total average annual strength	Paratyphoid A				Paratyphoid B			
		Admissions		Deaths		Admissions		Deaths	
		Number	Percent	Number	Percent	Number	Percent	Number	Percent
United States	2, 235, 389	32	0. 01	.....	.....	11	0. 00	1	0. 00
Europe	1, 665, 796	95	. 06	6	0. 00	56	. 03	4	0. 00
Other countries	227, 294	7	. 03	.....	.....	11	. 05	.....	.....
Total [1]	4, 128, 479	134	. 03	6	0. 00	78	. 02	5	0. 00

[1] Does not include 41 cases and 5 deaths from paratyphoid A and 17 cases and 1 death from paratyphoid B occurring among enlisted men in the United States and Europe.

Source: The Medical Department of the United States Army in the World War. Communicable and Other Diseases. Washington: U. S. Government Printing Office, 1928, vol. IX, p. 58.

study of the organisms, not only as relating to the cultural characteristics but also their antigenic composition. Progress in this area was largely due to the excellent work of Kauffmann (1937) and White (1926) who first studied the *Salmonella* species in great detail and showed how they could be classified on the basis of their antigenic structure.

TYPING AND CLASSIFICATION

At the beginning of World War II, there was at Copenhagen, Denmark, an international *Salmonella* typing center of which Dr. Fritz Kauffmann was director. In the United States, there were two large *Salmonella* centers, one at Beth Israel Hospital, New York, N. Y., under the supervision of Dr. E. Seligmann and one at the Kentucky Agricultural Experiment Station, Lexington, Ky., supervised by Dr. P. R. Edwards. Some of the work at Lexington was, as a result of the war, sponsored and paid for by the Army Medical Service Graduate School, then known as the Medical Department Professional Service School, Army Medical Center, Washington, D. C.

The main role of the Army Medical Service Graduate School was that of preparing and supplying typing serums for Army laboratories, Army service command medical laboratories, medical general laboratories, other general hospital laboratories, and station hospital laboratories. A great part of the Army's knowledge of the species of *Salmonella* causing food poisoning is due to the fine work of such laboratories as the 15th Medical General Laboratory, which was located at Naples, Italy, from 21 November 1943 to 25 October 1945. *Salmonella* typing and identification had become a new field, with new skills for the bacteriologist and technician to learn and master.[7] Military and

[7] (1) Edwards, P. R., and Bruner, D. W.: Serological Identification of Salmonella Cultures. Kentucky Agr. Exp. Sta. Cir. 54: 3-35, 1942. (2) Kauffmann, Fritz: Enterobacteriaceae: Collected Studies on Salmonella, Arizona, Ballerup-Bethesda, Escherichia, Alkalescens-Dispar, Klebsiella, Shigella, Providence and Proteus. Copenhagen: Ejnar Munksgaard, 1951.

civilian bacteriologists distinguished themselves in that field. Never before in the history of medicine and bacteriology had so much concentrated attention been centered upon the species of one bacterial genus. It should be noted that during this period of intensive research, two schools of thought on *Salmonella* classification arose. Some believed that the division of this genus into species and strains had been carried to hair-splitting extremes, where minute differences were deemed sufficient reason for such subdivision. Others maintained that the differences, regardless of how minute they might be, had taxonomic and epidemiologic significance, if they remained constant.

The following is a list of those *Salmonella* species discovered during the World War II period and is included here for reference purposes:[8]

Species:	Source
Sal. kaposvar	Human
Sal. kaapstad	Do.
Sal. hartford	Rat
Sal. manhattan	Human, turkey, chick, reptile, hog
Sal. narashino	Human
Sal. sendai	Do.
Sal. durban	Do.
Sal. rejle	Do.
Sal. meleagridis	Human, reptile
Sal. illinois	Human, hog, turkey, partridge
Sal. oregon	Human, turkey, chick, hog, reptile
Sal. pretoria	Hog
Sal. havana	Human
Sal. urbana	Human, hog, chick
Sal. salinatis	Human, rat
Sal. tennessee	Human, turkey, fowl, egg
Sal. bonariensis	Human, hog
Sal. amherstiana	Chicken
Sal. javiana	Human
Sal. weltevreden	Do.
Sal. simsbury	Human, turkey
Sal. köln	Human
Sal. infantis	Human (child)
Sal. pueris	Human
Sal. taksony	Do.
Sal. solt	Do.
Sal. mississippi	Human, hog
Sal. heves	Human
Sal. florida	Do.
Sal. madelia	Human, chick
Sal. sundsvall	Human
Sal. szentes	Do.
Sal. arechavaleta	Do.
Sal. schwarzengrund	Do.
Sal. concord	Human, turkey, chick
Sal. gorgia	Human
Sal. claibornei	Do.
Sal. loma linda	Do.
Sal. new york	Do.
Sal. butantan	Human (child)
Sal. grumpensis	Guinea pig
Sal. hormaechei	Human
Sal. adelaide	Do.
Sal. inverness	Do.
Sal. papuana	Do.
Sal. cardiff	Do.
Sal. virginia	Do.
Sal. pensacola	Do.
Sal. miami	Human, chimpanzee
Sal. canastel	Human
Sal. italiana	Do.
Sal. napoli	Do.
Sal. orion	Do.
Sal. veneziana	Do.
Sal. pomona	Human, fowl
Sal. champaign	Chicken
Sal. orientalis	Human

[8] Breed, Robert S., Murray, E. G. D., and Hitchens, A. Parker: Bergey's Manual of Determinative Bacteriology. 6th ed. Baltimore. The Williams & Wilkins Co., 1948.

The names of the species indicate, in some instances, where they were first isolated. Chickens became an important source, and rodents and hogs were often found to be infected. Spray-dried egg powder was the source of a number of *Salmonella* strains. They were found in 10 percent of over 6,000 samples of spray-dried egg powder. The commonest types were *Sal. oranienburg*, *Sal. montevidio*, *Sal. meleagridis*, *Sal. bareilly*, and *Sal. anatum*, but several others were encountered. One of the outstanding epidemiologic contributions of this experiment of infected egg powder was that it revealed the cause of a cluster of outbreaks around holiday seasons. Although dogs are not mentioned as an original source in this group, they were found to have a significant carrier role.

Table 72 is a brief summary of the outstanding salmonellal food-poisoning outbreaks during World War II. Most of the recorded outbreaks are those of the European and the Mediterranean Theaters of Operations. Salmonellal infections in these two theaters were the subject of special field study. The data which appear in table 72 represent only those cases of food poisoning which were reported on individual medical records and will grossly understate the total experience with this disease group. Several facts become apparent from a study of this table. Very often it was possible to trace the outbreak to the food responsible for it. Sometimes this was not possible as the foods had been discarded before being suspected of contamination. Certain types of foods seem to be responsible for salmonellal food-poisoning outbreaks more frequently than do others, in particular, desserts (especially puddings) and meats (especially poultry). Of the various types of poultry, turkey seems often to be responsible for outbreaks. This may be significant since turkey is not frequently served. In most outbreaks, the cause was traced to a single *Salmonella* species; however, two or three species were found to be simultaneously involved in some cases.[9] Most of the cases ran the typical course for food poisoning with nausea, diarrhea, vomiting, and cramps as the salient symptoms, and these lasted only a few days.

An analysis of national statistics made by Dr. Milton Feig, a district health officer in Wisconsin, revealed that meat and meat products were involved in 27.8 percent of salmonellal food-poisoning cases; poultry, 18.5 percent; bakery products, 14.8 percent; milk and milk products, 14.8 percent; vegetables, 13 percent; and other and unknown foods, 29.6 percent. These add to approximately 119 percent because more than one food item was responsible in some of the outbreaks.[10]

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[9] (1) Bruner, D. W., and Joyce, B. J.: Salmonella Types Encountered by the 15th Medical General Laboratory. Am. J. Hyg. 45: 19–24, January 1947. (2) Anderson, D. T., Johnson, L. M., Wetherbee, D. G., and Kuhns, D. M.: Isolation of Three Salmonella Species of Group C in One Case of Salmonellosis. U. S. Armed Forces M. J. 1: 1511–1515, December 1950. (3) Edwards, P. R., and Bruner, D. W.: The Occurrence of Multiple Types of Paratyphoid Bacilli in Infections of Fowls; With Special Reference to Two New Salmonella Species. J. Infect. Dis. 66: 218–221, May–June 1940. (4) Randall, C. C., and Marks, L. M.: A Salmonella Outbreak Involving Three Types of Genus. Mil. Surgeon 97: 144–146, August 1945.

[10] Feig, M.: Diarrhea, Dysentery, Food Poisoning, and Gastroenteritis; A Study of 926 Outbreaks and 49,879 Cases Reported to the United States Public Health Service (1945–1947). Am. J. Pub. Health 40: 1372–1394, November 1950.

TABLE 72.—*Chronological list of salmonellal outbreaks in World War II*

Date	Place	Food [1]	Organism	Number ill	Symptoms [2]	Number days	Remarks
1943							
30 January	175th Inf. Regt., United Kingdom.	Salmon	*Sal. enteritidis*	42			
2 May	825th Engr. Avn. Bn., England.	Butterscotch pudding	*Sal. sulpestifer*	67			
12–13 May	45th Gen. Hosp., MTO	Rice pudding	*Sal. oranienburg*	79	N, V, T (100° to 104° F.).	3 to 5	Serum aggl. tests of cultures of stools.
25 July			*Sal. oranienburg*, *Sal. typhimurium*, and *Sal. anatum*.	115	N, V, C, D.		
1 August	342d Engr. G. S. Regt., England.		*Sal. aertrycke*	5			Faulty washing of messgear on range.
6 October	101st A/B Div. United Kingdom.	Left-over potatoes	*Salmonella* species	61			
22 December	453d Amph. Trk. Co., United Kingdom.		*Sal. montevideo*	23			
1944							
February	605th Ord. Base, Naples, Italy.	Salmon loaf	*Sal. typhimurium*	200	N, V, D, F	2 to 3	Italian civilians also involved.
20–23 February	Camp Stoneman, Calif.	Mayonnaise	do	72	N, D, C, V	1	
March	565th Sv. Gp., Bari, Italy	Vienna sausage, vegetables.	*Sal. oranienburg*	40	N, V, D, F	2 to 3	This outbreak not connected with any one food source.
11 April	79th Gen. Hosp., North Ireland.	Rice pudding	*Sal. montevideo*	354			
17 April	314th Sta. Hosp., England		do	81			
May	58th Med. Bn., North Africa	Bread pudding	*Sal. meleagridis*, *Sal. montevideo*, and *Sal. tennessee*.	39	N, V, D, F	2 to 4	Two members of kitchen personnel were *Sal. meleagridis* carriers.
Do	52d MP Co., Naples, Italy	Meat loaf, cold canned chicken, cocoa.	*Sal. oranienburg*	100	N, V, D, T (103° F.)	2 to 4	Exact source of *Sal. oranienburg* not determined.
Do	319th Sv. Gp. Dispensary, Naples, Italy.	Ice cream	*Sal. oranienburg*	20	N, V, D	2 to 3	Small portions served may have been insufficient to produce clinical cases of salmonellosis.

15 July	15th Tk. Bn., Camp D-6, United Kingdom.		*Salmonella* species	40			
24 July	Co. A, 385th Inf. Regt., Camp McCoy, Wis.	Chicken	*Sal. typhimurium*	112	D, T (104° to 105° F.)	3	
October	125th Ord. Base, Naples, Italy	Chocolate pudding	*Sal. oranienburg*	130	N, V, D, F	3 to 5	*Sal. oranienburg* carrier in mess personnel.
9–10 October	125th Ord. Bn., MTO	do	do	170	V, D, C	1	Some rectal swabs of 10 hospitalized cases; 15th Med. Gen. Lab.
22–24 October	278th Sig. Pgn. Co., ETO		*Sal. aertrycke*	50			
5 November	96th Gen. Hosp., England	Pumpkin cobbler	*Salmonella* species	652			
26 November	58th Armd. Inf. Bn., England	Turkey and dressing	*Sal. newport*	77			
December	MP Co., Central Pacific Base Command, POA.	Roast turkey	do	53	N, D, C, T (100° to 103° F.).	3 to 7	
3 December	302d Trans. Wg., United Kingdom.	Rice pudding	*Sal. schottmülleri*	119			
7 December	Base Air Dep. #2, ETO	Canned pork sausage	*Sal. montevideo* and others.	48	V, D, C	2	Sausage cultured at 1st Med. Gen. Lab.
1945							
2 January	316th Sta. Hosp., United Kingdom.	Turkey	*Sal. montevideo*	200			
22–31 January	148th Gen. Hosp., POA	do	*Sal. typhimurium*	51	Gastroenteritis without diarrhea.		Thought to be mouse carried.
February	895th Engr. Avn. Co., South Atlantic.	Beef	*Staphylococcus albus* and *Salmonella* species.	13	N, D, C, V	3	
14 February	10th Sta. Hosp., United Kingdom.		*Salmonella* species	27			
16 February	486th Ord. Evac. Co., ETO		*Sal. suipestifer*	30			
18 February	Saipan, Western Pacific Base Command, POA.	Pork	do	62	N, D, C, V, T (103° to 109° F.).		Sulfaguanidine used in all cases.
8 April	Dep. G-45, United Kingdom	Mashed potatoes	*Salmonella* species	29			

[1] Found to contain the causative organism or suspected by circumstantial evidence.

[2] Abbreviations used are as follows: N, nausea; D, diarrhea; V, vomiting; C, cramps; T, temperature; F, fever.

Source: (1) Gordon, J. E.: A History of Preventive Medicine in the European Theater of Operations, U. S. Army, 1941–45. [Official record.] (2) Bruner, D. W.: Salmonella Infections of World War II. [Official record.] (3) Miller, E. B.: Food Poisoning and Food-borne Infections, U. S. Army, World War II. [Official record.]

CONTROL OF SALMONELLOSIS

During World War II, the approach taken to the problem of salmonellosis was largely preventive. Most of the methods employed were not new, as they had been used, though not with the same thoroughness, in previous wars.[11] Troops were thoroughly indoctrinated in the fundamentals of hygiene, with emphasis on personal cleanliness as well as cleanliness of the messgear that a soldier carries in the field.

At those installations where dishes were used, the dishes were air dried following washing and sterilization; no dish towels were used. Frequent dish culturing was carried out to determine the thoroughness of washing and sterilization.[12] Dish culturing was introduced in the Fourth Service Command early in the war and was found invaluable in maintaining good mess sanitation for a million and a half troops in training.

High standards were maintained in the processing of foods. Milk samples were sent to laboratories to determine how well pasteurization had been carried out. For meat products, cattle and poultry were inspected before being sent to slaughter as well as after slaughter, for determining fitness for consumption. Since the epidemiology of salmonellal food poisoning in animals and birds is essentially the same as for humans, the possibility of chlorinating water to be used for animals and poultry was considered. However, such measures are largely out of the control of the Army. Foods were carefully refrigerated to prevent bacterial growth during storage and in the preparation of meats; no meats were to be used for consumption if undercooked.

The need for control of fly breeding and prevention of flies from contacting excreta (for example, by screening latrines) and contacting food supplies was on the whole appreciated. Rodents, stray dogs, and stray cats were also recognized as a potential danger and were not permitted to come in contact with food supplies and human excreta.

One of the great achievements of military medicine in World War II was the accommodation of all Army installations throughout the world with laboratory facilities. Specimens of milk, foods, and feces were forwarded to the appropriate laboratories for bacteriologic examination. The fecal specimens were often those of permanent foodhandlers, and when a carrier was discovered his unit commander was to be notified and instrusted that carriers were not permitted to be permanent handlers of food.

In addition to these procedures, TAB (typhoid, paratyphoid A, and paratyphoid B) vaccine continued to be routinely administered to all Army personnel.

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[11] Dunham, George C.: Military Preventive Medicine. 3d ed. Harrisburg: Military Service Publishing Co., 1940.

[12] War Department Field Manual 21-10, Military Sanitation. Washington: U. S. Government Printing Office, July 1945.

RESEARCH

In the United States, pioneer work in the development of agglutination serums for *Salmonella* identification was done by Edwards, of the University of Kentucky, Lexington, Ky. This work, as already noted, was adopted by the Army Medical Service Graduate School where the production of typing serums was expanded for Army-wide usage (p. 419). The purpose of this was to carry out applied research and investigation whenever an outbreak of salmonellosis occurred; thus, a considerable amount of information was obtained concerning the species of *Salmonella* involved in an outbreak. Many epidemiologic investigations were carried out, and very often it was possible to trace the origins of an epidemic. If an outbreak was traceable to a sample of powdered dehydrated eggs, it was necessary to trace that egg powder to determine where and how it had become contaminated originally: whether it had become contaminated from the poultry or the foodhandlers, whether flies had contaminated it, or whether it was the fault of improperly cleaned mess equipment. Many outbreaks were never successfully traced, and often the causative *Salmonella* was not found.

EPIDEMIOLOGIC ASPECTS OF SALMONELLOSIS

Under wartime conditions, the diarrheal diseases are especially important, and many factors contribute to their occurrence.[13] For example, early in World War II, as the Armed Forces increased in size and large numbers of men from all parts of the Nation were required to live together in centers of basic military training, the various proposed methods of prevention and control were due for the acid test. The men lived together in maneuver areas and in close quarters, often at hastily constructed camps and bivouacs in which it is impossible to achieve the highest sanitary standards. Active immunity, developed from exposure to the common Salmonella organisms, had not as yet been established, and a fertile field was afforded for outbreaks of salmonellosis. There was epidemiologic significance in the fact that the Army consisted of men who had been quickly brought together from all walks of life, from all parts of the country, and from all levels of sanitary habits and education.

Among other factors of epidemiologic importance is the fact that animals and birds as well as human beings furnish an ever-present reservoir of the Salmonella organisms. Among the possible carriers of Salmonellae are rats, mice, rooks, turkeys, pigs, ducks, and chickens. Rats, flies, mosquitoes, and even ticks have been shown to be capable of acting as vectors.[14] The matter is further complicated by the fact that carriers play a vital role in the spread of

[13] Rubenstein, A. D., Feemster, R. F., and Smith, H. M.: Salmonellosis as a Public Health Problem in Wartime. Am. J. Pub. Health 34: 841-853, August 1944.

[14] (1) Ostrolenk, M., and Welch, H.: The House Fly as a Vector of Food Poisoning Organisms in Food Producing Establishments. Am. J. Pub. Health 32: 487-494, May 1942. (2) Smith, H. W., and Buxton, A.: An Outbreak of *Salmonella schwartzengrund* Infection in Poultry. J. Path. & Bact. 63: 459-463, July 1951. (3) Welch, H., Ostrolenk, and Bartram, M. T.: Role of Rats in the Spread of Food Poisoning Bacteria of the Salmonella Group. Am. J. Pub. Health 31: 332-340, April 1941.

salmonellosis. Some of these carriers are of the convalescent type since they continue to excrete the bacteria during the time they are recovering from their illness. Others are asymptomatic and therefore represent a far greater danger than those exhibiting symptoms, since the former may be the origin of many an outbreak of undetermined origin before being apprehended. Some *Salmonella* carriers are intermittent since periods of time during which Salmonella organisms are excreted alternate with periods of time in which they are apparently absent. Most individuals continue to be carriers for relatively short periods of time, while a few retain the organisms for longer periods. In civilian life, the laws and facilities in some areas are inadequate for dealing with the carrier problem, while in the Army close followups are maintained on known carriers.

A Typical Outbreak

Greifinger and Silberstein describe a completely investigated outbreak of *Salmonella* food infection in military personnel.[15] The places of occurrence perhaps for reasons of military security are not mentioned. The outbreak was first recognized on 25 July 1943 when 28 individuals, all males, between 21 and 50 years of age were hospitalized mainly for the symptoms of nausea and abdominal cramps. These symptoms subsided in 24 to 48 hours. Bowel movements numbered 8 to 15 per day and were loose, watery, and brownish green in color. Fourteen of the patients had occult blood in their stools, ten had pus, and three showed pus and mucus. Temperatures ranged from 101° to 104° F. Fried fish, tartar sauce, and rice pudding had been served at the evening mess. The fish was suspected, since it was noticed that the ice, refrigerating it, had melted. More individuals developed the usual symptoms until a total of 115 persons were incapacitated; 93 percent had diarrhea, 72.2 percent had cramps, and only 21.7 percent complained of nausea.

An interesting followup study was conducted to determine how long the individuals would continue to harbor the organisms. This is one of the greatest contributions to historical literature on salmonellosis. The results are summarized in table 73.

From the figures in table 73, it is apparent that as long as 3 months after the initial infection one may remain a carrier of *Salmonella.* Of the 115 individuals who were infected, 5.2 percent continued to harbor *Sal. oranienburg* into the 13th week. The patients were retained in the hospital until three consecutive stools were negative. The differential media employed were desoxycholate citrate, eosine methylene blue, and S.-S. (*Shigella-Salmonella* thiosulfate-citrate-bile) agar. Of these differential media, S.-S. agar was found to yield the largest incidence of positives. If the 5.2 percent infected individuals were carriers rather than reinfected individuals, it would indicate that individuals who have had salmonellosis should be potential carriers for that period of time.

[15] Greifinger, W., and Silberstein, J. K.: Salmonella Food Infection in Military Personnel; An Outbreak Caused by *S. oranienburg, S. typhimurium, S. anatum.* J. Lab. & Clin. Med. 29: 1042-1053, October 1944.

TABLE 73.—*Persistence of salmonellal infection as determined by positive fecal cultures in 115 patients*

Week following ingestion of suspected food	Frequency by type of organism						Total [1]	
	Sal. sp. Oranienburg		*Sal. typhimurium*		*Sal. anatis*			
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
First	100	86. 9	27	23. 4	0	0	109	94. 8
Second	94	81. 7	18	15. 6	0	0	104	90. 4
Third	74	64. 3	6	5. 2	14	12. 2	87	75. 7
Fourth	68	59. 1	4	3. 4	8	6. 9	73	63. 5
Fifth	52	45. 2	3	2. 6	6	5. 2	56	48. 7
Sixth	48	41. 7	0	0	9	7. 8	51	44. 3
Seventh	38	33. 0	0	0	10	8. 7	44	38. 3
Eighth	27	23. 5	0	0	14	12. 2	36	31. 3
Ninth [2]	20	17. 4	0	0	16	13. 9	28	24. 3
Tenth	16	13. 9	0	0	2	1. 7	17	14. 8
Eleventh	12	10. 4	0	0	1	. 9	12	10. 4
Twelfth	7	6. 1	0	0	0	0	7	6. 1
Thirteenth	6	5. 2	0	0	0	0	6	5. 2
Fourteenth	0	0	0	0	0	0	0	0

[1] Some cultures were positive for more than 1 type organism.
[2] Sulfasuxidine administered.

Source: Greifinger, W., and Silberstein, J. K.: Salmonella Food Infection in Military Personnel. J. Lab. Clin. Med. 29: 1042-1053, October 1944.

RELATIVE INCIDENCE OF SALMONELLA SPECIES

Several *Salmonella* species were cited as the one most frequently occurring. Thus, according to Seligmann, in 1946, *Salmonella schottmülleri* was the most common *Salmonella* in the United States. Dack, in 1949, considered *Sal. typhimurium* as the *Salmonella* species found in more outbreaks of food poisoning than any other type. Edwards and Bruner [16] studied 225 cultures isolated from the feces of patients who had gastroenteritis. In order of frequency, the first nine species were: *Sal. typhimurium*, *Sal. newport*, *Sal. paratyphi B.* var. *java*, *Sal. panama*, *Sal. oranienburg*, *Sal. san diego*, *Sal. bareilly*, *Sal. montevideo*, and *Sal. enteritidis*. Undoubtedly, the original source of some of these species was eggs. During World War II, dried eggs were shipped to United States troops in all theaters of operations. According to Dack, of 7,584 samples that were sent from the United States, Canada, and Argentina, 754 (9.9 percent) were found to contain *Salmonella*. In all, 33 species were found.

A thorough study on the relative occurrence of *Salmonella* species was made by Bruner in the Mediterranean theater during World War II.[17] It is

[16] Edwards, P. R., and Bruner, D. W.: The Occurrence and Distribution of *Salmonella* Types in the United States. J. Infect. Dis. 72: 58-67, January-February 1943.
[17] Bruner, D. W.: Salmonella Infections of World War II. [Official record.]

probably the most comprehensive analysis as to species and group occurrence on *Salmonella* conducted in any theater of operations. The study not only covers the incidence among United States Army troops in the Mediterranean theater but also includes valuable data on incidence among civilians, prisoners of war, French Army personnel, and carriers. The group C Salmonellae apparently were the most frequent cause of salmonellal food poisoning. *Salmonella sp.* (Type Oranienburg) was the most common *Salmonella* species causing gastroenteritis in United States Army personnel in the Mediterranean. Also, there were more carriers of *Salmonella sp.* (Type Oranienburg) in the United States Army than any other *Salmonella* species. *Salmonella sp.* (Type Montevideo) remained a close second. The increased number of carriers in each species were in proportion to the number of cases of gastroenteritis reported and cultured.

The occurrence of salmonellosis in the Pacific area was recorded by the 19th Medical General Laboratory, the laboratory research center for New Guinea and the Philippines. The species of *Salmonella* that were isolated on New Guinea in order of the number of bacteriologic isolations is shown in table 74. On New Guinea, as in other areas of the Pacific, the Salmonellae were not as common a cause of food poisoning as the Shigellae.

TABLE 74.—*Salmonellosis outbreaks and isolations of* Salmonella *species and types in New Guinea, September 1944 to July 1945*

Species and type	Isolations	Outbreaks
Sal. enteritidis	17	2
Sal. typhosa	13	4
Sal. paratyphi	10	3
Salmonella sp. (Type Uganda)	3	2
Sal. typhimurium	2	1
Salmonella sp. (Type Chester)	1	1
Sal. anatis	1	1
Sal. typhisuis	1	1
Total	48	15

It is to be noted that while *Sal. enteritidis* was the number one disease in occurrence, *Salmonella typhosa* was responsible for the greatest number of outbreaks. The distinction made here is of epidemiologic significance particularly in food-poisoning outbreaks. Eventually, it is presumed that bacteriologists will no longer refer to infections with *Salmonella* contracted through food as food poisoning but simply as Salmonella infections.

The species of *Salmonella* that were isolated in the Philippines, in descending number of isolations, is shown in table 75. The outstanding causes of salmonellal food poisoning in the Philippines were, according to these data, *Sal. enteritidis* and *Sal. paratyphi.*

TABLE 75.—*Salmonellosis outbreaks and isolations of* Salmonella *species and types in the Philippines, July 1945 to December 1946*

Species and type	Isolations	Outbreaks
Sal. enteritidis	17	4
Sal. paratyphi	15	10
Sal. typhosa	7	1
Sal. hirschfeldii	4	4
Sal. schottmülleri	4	3
Salmonella sp. (Type San Diego)	3	3
Salmonella sp. (Type Thompson)	1	1
Sal. typhimurium	1	1
Salmonella sp. (Type Uganda)	1	1
Salmonella sp. (Type Lexington)	1	1
Total	54	29

LABORATORY ADVANCES IN DIAGNOSIS OF SALMONELLOSIS

The laboratory diagnosis of salmonellal food poisoning is basically not different from what it has always been. It depends upon the recovery of the organism, usually from the stools, based on a principle dating back to Koch's postulates. Yet, considering the incidence of salmonellal food poisoning, it is a fact that in many cases the organism is never demonstrated. In the field, it was often impossible to transport biological specimens under proper conditions. Another and perhaps more important point is the fact that Salmonellae will often be present only during the brief period of acute symptoms. According to Verder and Sutton,[18] there is an inverse relationship between the number of ingested organisms and the incubation period. The smaller the number of organisms taken in, the more prolonged will be the period of incubation. Thus, there is a strong likelihood that a stool specimen taken even 1 day following the subsidence of symptoms will be void of Salmonellae. As for the contaminated food, it is often discarded before an examination can be conducted.

There are several differential media in use, including *Shigella-Salmonella* medium, tetrathionate broth base, and selinite-F enrichment medium. Occasionally, one encounters a *Salmonella* species that will not grow on S.-S. agar. For this reason it is a prudent policy to use a less inhibiting medium in addition. Most often in salmonellal outbreaks, only one species is found. Occasionally, two or even three species are present simultaneously, and it would be a moot question to inquire as to which species is producing which symptoms.[19] A procedure of great value in diagnosing salmonellal food

[18] Verder, E., and Sutton, C.: Is *Salmonella* Food Poisoning Caused by Living Bacilli or by Thermostabile Toxic Products? J. Infect. Dis. 53: 262-271, September-October 1933.

[19] See footnote 9 (2), p. 421.

poisoning was the anal-swab technique introduced by Hardy. Its main advantage lay in the fact that it was not necessary to wait until stool specimens were collected from those who were to be tested. Anal swabs could be taken from a number of individuals in a very short time and cultures made on a large scale.

EVALUATION OF THE ARMY'S EXPERIENCE

Carriers.—In evaluating the Army's experience with salmonellosis, several conclusions can be elicited. First, it was never among the most serious of the medicomilitary problems. The history of salmonellal food poisoning shows that the preventive aspects have always been given more attention than the therapeutic aspects. In typical cases, the symptoms are striking and soon disappear completely leaving the victim little cause for alarm. As a result, many of the patients recover, only to become carriers for a greater or shorter period of time. Although the Army's treatment of the carrier problem compares favorably with that of the best civilian health departments, it is also true that the Army especially during the time of war places major stress on returning to duty as many men as possible as soon as they appear capable of resuming work. However, in cases of salmonellal infection, it is not safe to return a man to duty in the mess merely because of the subsidence of acute symptoms. In connection with the carrier problem, it should be noted that surprisingly few outbreaks during World War II were found traceable to a carrier origin.

Foodhandlers.—The most important military consideration in the carrier problem is that of the career foodhandler as developed during World War II.[20] A foodhandler may without his or anyone else's knowledge dispense Salmonellae systematically to large numbers of troops throughout his career. Of lesser significance is the temporary foodhandler, especially in basic training units where it is accepted procedure to utilize kitchen police in the handling and distribution of food.

Vectors and reservoirs of salmonellal infection.—The role of vectors and reservoirs in salmonellal food poisoning has already been mentioned in the literature. Flies are of prime importance. The problem is more than one of using insecticides; also required is the rigorous application of the basic well-known principles of hygiene and the execution of such measures as the screening of messhalls and latrines whenever possible. Even then, there exists the problem of animal and bird reservoirs, perhaps more challenging than the flies and surely a far more difficult problem under field conditions. It must be stressed, however, that even though hygienic principles were sometimes abandoned during World War II field conditions, the sanitary level achieved in the field during the war was higher than that of any previous war.

The military problem.—Salmonellosis is of special interest to the Army for more than the theoretic reasons that have been discussed. Although it

[20] Stone, W. S.: Food Handlers in the Army and Their Relationship to *Salmonella* Food Poisoning. Am. J. Pub. Health 33: 706-708, June 1943.

rarely caused serious infection, it can with dramatic suddenness temporarily incapacitate large numbers of troops. In time of war, this can lead to catastrophic results. During World War II, as in World War I, most of the Army's outbreaks were sporadic, and real epidemics were the exception rather than the rule. There are two explanations for this. The first explanation is that over a period of half a century the Army had attained and on the whole maintained a high standard of sanitation. The second explanation is the preventive role of the Army's TAB vaccine which is routinely administered to all military personnel. The validity of the first point is generally accepted. There is, however, divided opinion as to the value of the vaccine. While some consider it effective in preventing salmonellal infection, many believe it has little value. The truth may be between the two extremes, and while TAB vaccine might not be effective in preventing salmonellosis it may have some effect on mitigating the symptoms, thereby modifying the course of the disease.

CHAPTER XIX

Staphylococcal and Streptococcal Food Poisoning and Botulism

G. M. Dack, M. D.[1]

STAPHYLOCOCCAL FOOD POISONING

Experiences Before World War II

Staphylococcal food poisoning has probably occurred for hundreds of years. From the period of 1870, when the occurrence of cocci in inflammatory processes was first observed, through the first three decades of the 20th century, the cause of these outbreaks was not generally recognized because of the ubiquitous nature of the organism and the failure to demonstrate enterotoxin in implicated foods as well as in cultures of *Staphylococci* isolated from foods. Many outbreaks of staphylococcal food poisoning have falsely been ascribed to ptomaine poisoning or to products from other bacteria which were toxic when injected parenterally into animals.

Staphylococcal food poisoning is not a reportable disease in our civilian populations, and usually only the large and spectacular outbreaks are reported. The disease has been common in armies. A good illustration of this occurred among the German troops in World War I.[2] Although this outbreak was attributed to the bacillus *Proteus vulgaris*, it was obviously an outbreak of staphylococcal food poisoning since micrococci were found in the product upon bacteriologic examination and since the symptoms and epidemiologic features were characteristic of staphylococcal food poisoning. The following excerpts were translated from this report:

* * * Therefore I am going to report in the following about an extensive sausage poisoning outbreak (approximately 2000 cases) which took place in the spring of 1918 during the siege of Verdun and which could possibly have led to catastrophical military consequences.

In the beginning of June 1918 all of a sudden mass outbreaks which had the appearance of acute gastro-enteritis and which, in some severe cases, were even increased to cholera nostras, spread among the troop surrounding Verdun; with a single blow, whole companies were disabled with the exception of just a few people, and within two days about 2000 men had been attacked. The symptoms were so severe in part of the troops that more

[1] The author is indebted to Maj. (later Lt. Col.) Everett B. Miller, VC, Office of the Surgeon General, Veterinary Division, for his painstaking work in gathering the source material used in this manuscript.

[2] Baerthlein, K.: Ueber ausgedehnte Wurstvergiftungen, bedingt durch Bacillus proteus vulgaris. München. med. Wchnschr. 69: 155–156, February 1922.

than 200 sick people had to be transferred to field hospitals. Suspicion of food poisoning arose since, according to statements by the sick people, severe illness set in 2-3 hours (with a smaller part of the sick 6-8 hours) after the ingestion of a certain sausage dish. Only those people among the front troops who had not partaken in the meal were spared, i. e. orderlies of the company who on the same day had gone back to headquarters to receive orders; or soldiers who for other reasons had not eaten the sausages, and those parts of the troops who were in rest billets and had a different diet. However, it was surprising that among the troops behind the front, i. e. the corps butchers, who had eaten from the same sausages though two days earlier, no cases of sickness were observed * * *.

Situation at the Beginning of World War II

At the beginning of World War II, staphylococcal food poisoning was recognized in public health laboratories, but it was not generally known to practicing physicians. Many large outbreaks were reported in civilian defense establishments [3] and in military establishments, including personnel in the Pentagon, as well as among troops in camps and in the field.[4]

Methods of control.—The control of staphylococcal food poisoning is concerned principally with the refrigeration of perishable foods which furnish a medium for the growth and enterotoxin production of *Staphylococci*. Where recurrent outbreaks have occurred,[5] carriers have been sought among the foodhandlers because in some outbreaks enterotoxic strains of *Staphylococci* have been demonstrated in pyogenic lesions as well as in the noses and throats of carriers. Other control measures are concerned with scrupulous cleanliness, with particular attention being given to cutting boards and all kinds of cooking utensils.

Research.—Research should be directed toward purification and isolation of the enterotoxin with the hope that this approach will lead to a better method of assaying enterotoxin. If a good assay method were available, progress could be made in processing and protecting foods from staphylococcal food poisoning. Investigation in this field is expensive, and the problem involves such a wide segment of the food industry that no one industry can carry this research load. The Armed Forces and Government agencies should cooperate in this program to hasten the solution of the problem.

Outbreaks During World War II

Staphylococcal food-poisoning outbreaks are cosmopolitan and have occurred among troops in all the war theaters, as well as on the homefronts. Since staphylococcal food poisoning is not generally reported even in the Army, there is no accurate information as to its magnitude. A survey of the outbreaks of food poisoning reported in the Army in World War II was tabulated under the following headings: Code, unit, location and date, epidemiology, case

[3] Getting, V. A., Rubenstein, A. D., and Foley, G. E.: Staphylococcus and Streptococcus Carriers: Sources of Food-borne Outbreaks in War Industry. Am. J. Pub. Health 34: 833-840, August 1944.

[4] Williams, G. C., Swift, S., Vollum, R. L., and Wilson, G. S.: Three Outbreaks of Staphylococcal Food Poisoning Due to Ice-Cream. Month. Bull. Min. Health & Emerg. Pub. Health Lab. Serv. 5: 17-25, January 1946.

[5] See footnotes 2, p. 433, and 4.

history, food, laboratory findings, remarks, and document. Under this method of tabulation, 190 outbreaks involving 22,364 cases were reported for all causes. In this tabulated summary, much of the information was not available, and in some instances not even the number of cases was reported. Of the 190 outbreaks reported, 76 were chosen in which the epidemiologic data, together with the laboratory findings, suggested staphylococcal food poisoning. These 76 outbreaks involved 14,214 men. In addition to these 76 outbreaks, many of the others may very well have been staphylococcal food poisoning, but the information available was inadequate to establish them definitely as such. From a study of the data, staphylococcal food poisoning was without doubt the most important cause of the illnesses.

These 76 outbreaks of staphylococcal food poisoning occurred during the following months of the 4-year period from 1942 through 1945:

Month	*Number of outbreaks*
January	2
February	6
March	8
April	6
May	9
June	12
July	10
August	2
September	5
October	7
November	6
December	3

More outbreaks occurred in each of the months May, June, and July than in the other months of the year. The seasonal incidence may have less meaning in view of the fact that Army personnel were scattered in many parts of the world where the prevailing temperatures differed for the separate geographic areas according to months. The inconstant and shifting Army populations in these different areas, together with the fact that the reported outbreaks probably are only a small part of the total, make this information unsuitable for statistical analysis. Furthermore, in many outbreaks epidemiologic and laboratory studies either were not done or were inadequate to permit the drawing of conclusions.

The number of outbreaks reached a peak in 1944 (41 of the 76 selected outbreaks). This number was far greater than reported in the two preceding years (5 and 8) and almost twice as great as reported for the following year (22). This yearly incidence is subject to the same variables mentioned for the seasonal incidence. Although the yearly average of the strength of the Army reached a peak in 1944, there is not a direct relationship between the yearly number of outbreaks and the number of men at risk for that period. Furthermore, information disseminated through Army regulations, training manuals,

and training courses may also have influenced recognition and reporting of outbreaks.[6] With the better understanding of the causes of staphylococcal food poisoning, more care may have been utilized in the use of leftovers and in the provision for more adequate refrigeration of perishable foods, thus influencing the yearly incidence of the disease.

A list of foods involved in the staphylococcal food-poisoning outbreaks comprises some 43 different food items, the most common of which are as follows:

Foods involved	*Number of outbreaks*
Bread pudding	11
Ham	9
Potato salad	6
Rice pudding	3
Ice cream	3
Turkey	3
Roast veal	2
Ham salad	2
Macaroni and chicken	2
Ham sandwich	2

Foods which were involved in 76 staphylococcal food-poisoning outbreaks comprising a total of 14,214 cases were as follows:

Food	*Number of times*
Meat:	
Canned pork sausage	1
Canned corned beef or tongue	1
Corned-beef stew	1
Frankfurters	1
Hamburger	1
Roast veal	2
Ham salad	2
Beef	1
Ham	9
Roast beef	1
Liver-sausage sandwich	1
Ham sandwich	2
Ham omelet	1
Hash	1
Poultry:	
Turkey	3
Chicken a la king	1
Chicken fricassee	1
Chicken salad	1
Chicken pie	1
Turkey a la king	1
Creamed chicken	1
Macaroni and chicken salad	2

Food	*Number of times*
Desserts:	
Rice pudding	3
Ice cream	3
Bread pudding	11
Tapioca pudding	1
Fruit pudding	1
Pumpkin pie	1
Cream filler of layer cake	1
Peanut-butter pie	1
Boston cream pie	1
Vegetables:	
Canned corn	1
Potato salad	6
Fried potatoes	1
Miscellaneous:	
Turkey dressing	1
Thanksgiving meal	1
Egg or roast-beef sandwiches	1
Liver cheese	1
Macaroni salad	1
Cream	1
Egg-salad sandwich	1
Vegetable soup	1
Unknown	1

[6] (1) AR 40-205, 31 Dec. 1942. (2) AR 40-210, 25 Apr. 1945. (3) TM 8-227, 11 Oct. 1946. (4) TM 8-210, 20 Mar. 1942. (5) TM 8-227, 17 Oct. 1941.

Of the 76 outbreaks, 21 were selected for which the data were most complete. Of these 21 outbreaks, involving 5,370 men, 8 occurred in the European theater, 1 in the Mediterranean theater, 9 in the Zone of Interior, 1 aboard ship, and 2 in the Caribbean Defense Command. Of the 5,370 men ill, 1,059 were hospitalized. There was one death which occurred in a patient hospitalized with coronary heart disease. Of these 21 outbreaks, there was 1 in which only 17 men were involved, whereas the largest involved 1,637 men. The onset of illnesses following the incriminated meals was reported as varying from 15 minutes to as long as 8 hours. In all instances, the duration of illness varied from 6 to 48 hours and was followed by weakness and diarrhea which lasted as long as 48 hours. From 13 of the 21 outbreaks, laboratory studies were made, and *Staphylococci* were isolated from the incriminated foods.

In the 21 selected outbreaks, the time of onset following a specific meal and the symptoms of illness were typical of those which characterize staphylococcal food poisoning and set it apart from other types of gastrointestinal illnesses. The average period of onset of illness following a meal was slightly less than 3 hours. The symptoms included one or all of the following: Nausea, vomiting, abdominal cramping, and diarrhea. In severe cases, prostration occurred and the patients developed shock. Such cases were treated with fluids parenterally and made rapid recoveries. Fever or subnormal temperatures were rarely reported.

A history of the implicated food is given for 15 of these outbreaks, and in each of these the food stood for several hours at a warm temperature before being served. In two cases, the food item was stored in a warming oven for 7 hours before serving. One outbreak involving ham salad illustrates the folly of attempting to refrigerate large volumes of food. In this outbreak, ground ham was packed in 12-gallon containers and placed in the refrigerator. It is obvious that if the ham was warm when placed in such containers, it would require hours for the ground ham in the center of the container to reach refrigeration temperature. In two outbreaks, frozen ham was cooked and served the same day with no illnesses; the leftover portions were kept out of the refrigerator and served the following morning, causing severe illness. In 1 of these 2 outbreaks, 10 hours elapsed between servings.

Ice cream was involved in two outbreaks. A history of preparation was given in only one. In this incident, a portion of the mix was frozen in the hospital in which it was prepared and served to 30 to 40 people without causing any illnesses. The largest portion of the same mix, however, was kept at room temperature before it was frozen. There were 399 men made ill from the later preparation of ice cream.

One of these 15 outbreaks illustrates the thermostability of the enterotoxin. This outbreak involved chicken pie in which the chicken was cooked and stored at room temperature for 16 hours before being made into the pie which was served immediately after cooking. Apparently, the cooking of the chicken pie did not destroy the enterotoxin which was probably formed during the 16-hour period in which the chicken was out of refrigeration.

Summary of Experiences

Staphylococcal food poisoning frequently occurs in the Army. It is conceivable that its untimely appearance in troops may lose battles, as was pointed out in the case of the Battle of Verdun in World War I. The incidence of staphylococcal food poisoning in World War II is not known, since many outbreaks are not reported and since some of those reported may have been caused by *Staphylococci* but were not so classified because of insufficient data. From the study of 190 tabulated outbreaks involving 22,364 cases of illness, 76 outbreaks were selected where sufficient data were available to classify them as staphylococcal food poisoning. The 76 outbreaks involved 14,214 men. The illnesses usually appeared within from 3 to 4 hours following a meal and were characterized by nausea, vomiting, diarrhea, and, in the acute cases, prostration. The acute symptoms lasted only a few hours, and in general men returned to duty within from 24 to 48 hours. The outbreaks of food poisoning due to Staphylococci were reported for all months of the year but were highest in the warm months, May, June, and July.

Forty-two different food items were involved comprising meat, poultry, desserts, vegetables, dairy products, and miscellaneous items. Of the 76 outbreaks attributed to *Staphylococci*, 21, for which the data were most complete, were selected for study. For 15 of these 21 outbreaks, a history of the handling of the implicated food was available, and in all cases the food had stood for several hours at a warm temperature before being served. The danger inherent in the use of leftovers was demonstrated repeatedly in the series of incidents under study. Examples were cited emphasizing (1) the length of time for enterotoxin production; (2) the failure to obtain rapid chilling in food refrigerated in large containers; and (3) an example of perishable food stored at room temperature and then cooked before serving, thus illustrating the thermostability of enterotoxin.

Although the danger of staphylococcal food poisoning had been emphasized in Army regulations and other written communications in the Army, outbreaks continued to occur. It is important that information such as detailed here be made available to all personnel responsible for handling perishable foods.

STREPTOCOCCAL FOOD POISONING

Outbreaks of gastrointestinal illnesses caused by *Streptococcus faecalis* have been reported.[7] Human volunteers, who were fed large numbers of *Str. faecalis* which had been recently isolated from implicated foods, developed illnesses identical to those described in the outbreaks. Knowledge of this type of food poisoning is incomplete. In food poisoning attributable to *Str. faecalis*, the living organisms are responsible for illness and not preformed toxins as in staphylococcal food poisoning or botulism.

[7] Dack, G. M.: Food Poisoning. Chicago: The University of Chicago Press, 1949.

In outbreaks occurring in the Army, little or no effort was made to look for *Str. faecalis* in the incriminated items of food. Several of the 190 outbreaks tabulated in this series were suggestive of *Str. faecalis* food poisoning. In only 1 of the 6 outbreaks was *Str. faecalis* demonstrated in the implicated food. In the others, there were either no samples of the food available or laboratory tests were not made.

These 6 outbreaks involved 1,015 men. Two were reported for the European theater, one from the Panama Canal Zone, and three from the Zone of Interior. The food items were turkey, lamb, canned meat and vegetable stew, canned creamed corn, veal dressing, and meat-hash sandwiches. The onset varied from 4 hours to 18 hours. The illnesses were usually mild, with little or no vomiting, and were characterized principally by abdominal cramps, nausea, and diarrhea. In general, recovery was complete in from 16 to 24 hours.

In all six outbreaks, perishable foods were kept out of refrigeration for varying periods of time. In the case of the turkey outbreak, 140 turkeys were cooked on an afternoon and evening with facilities that accommodated 8 turkeys at a time. The following morning, the turkeys were sliced and placed in pans and stored in the refrigerator for 24 hours. The meat was served at noon. Alpha-type streptococci were isolated from the turkey in this outbreak, which involved 266 men. The lamb outbreak followed the use of leftover lamb that was kept out of refrigeration for 24 hours. In the meat and vegetable stew outbreak, several of the cans contained gas, and the contents were reported to have had a strong odor, when they were opened. It was suggested that one or two of the spoiled cans may have been used in preparing the meal. The outbreak traceable to creamed corn resulted from use of a product which had been left over from a previous meal. The veal dressing was prepared from chopped frankfurters left over from a meal 5 days previously, stale bread, onions, cheese and macaroni left over from a meal 24 hours before, and scrambled eggs left over from breakfast of the day on which the dressing was prepared. A very pertinent remark for the last outbreak is made by the surgeon reporting it: "* * * so far as health is concerned, food wastage is preferable to food poisoning * * *." The meat-hash sandwiches were prepared a day previous to serving, although they were refrigerated after preparation. There is no statement of the time required for the preparation and the length of time before refrigeration.

Str. faecalis food-poisoning outbreaks occur in the Army, although this type of food poisoning is not generally recognized. Laboratory and epidemiologic studies are necessary to establish the diagnosis.

In 6 selected outbreaks from the group of 190 studied, *Str. faecalis* was considered the probable etiologic agent. In 5 of the 6 outbreaks, there was a definite history of perishable food items having been kept out of refrigeration for several hours.

BOTULISM

There are records of civilian outbreaks of botulism in the United States and Canada dating back to 1899,* and from then until 1918 the number of outbreaks reported yearly varied from none to seven. However, beginning with 1918, when 16 outbreaks were reported, and following through to include 1947, the annual number of outbreaks has varied from 3 to 25, with an average of 13.9 per year. The highest incidence of botulism from canned goods packed in the United States occurred in 1920 (6 outbreaks). Since 1925, with one possible exception, no cases have occurred from commercially canned goods packed in the United States. The sharp rise beginning in 1918 may have been caused by the increase in preservation of foods because of the war. The publicity and better recognition of the disease after 1918 may explain why the number of outbreaks has not decreased since that time.

The control of botulism has been effectively developed by the canning industry in the United States. This disease occurred among only 23 men in the United States Army in World War II, with 9 deaths, giving a case fatality rate of 39 percent. The 23 cases were represented in 4 outbreaks. The first occurred from commercially canned beets packed in Australia in November 1942. In this outbreak, there were 16 cases and 7 deaths, with a case fatality rate of 44 percent. The other three incidents involved home-preserved products. In August 1944, an outbreak occurred in the European theater from home-canned ravioli which was sent to one of the enlisted men by his parents in the United States. There were 3 cases with 1 death. In January 1945, in the United States, a family had sent their son home-canned mushrooms, which caused 7 cases with 1 death among non-Army personnel as well as 1 nonfatal case in a soldier. The fourth outbreak, causing 3 cases with 1 death, occurred in April 1945 in Germany and was due to pickles and preserves found in a cellar of a home.

Since the Army uses commercially canned products and the canning industry in the United States is keenly aware of the botulism hazard, there are no recommendations necessary in this direction. During World War II, the author evaluated a botulism hazard from dehydrated meats, and the industry and using agencies for these products were apprised of the hazard. Instructions were issued for preventing botulism from the use of these products. In recent years, the Quartermaster Corps has been developing canned bread, which is a nonsterile product, and the hazard involved has been investigated under experimental conditions, in the author's laboratory. The specifications for the manufacture of canned bread have been rigidly set to safeguard this product from botulism. If new nonsterile items are introduced, the food-poisoning risk should be considered for each item and control measures instituted before the item is placed in production. Since 3 of the 4 outbreaks were from home-preserved items consumed by soldiers, in theory it might

* See footnote 7, p. 438.

seem desirable to prohibit the sending of food items in which there may be a hazard from botulism, but such selective regulation of items is obviously a practical impossibility. Data for United States Army personnel only indicate that in 3 of the 4 outbreaks antitoxin was administered to 6 cases, 1 of whom died, representing a case fatality rate of 17 percent. Three outbreaks occurred between August 1944 and April 1945. In the other outbreak, which occurred in November 1942 in Australia, there were 16 cases with 7 deaths among United States troops. No mention is made as to whether antitoxin was available or given, and the case fatality rate was 44 percent. The food was of Australian manufacture, and, prior to the arrival of United States troops, both New Zealand and Australia had done very little commercial canning of foods. Under lend-lease agreement, these countries undertook to supply huge quantities of canned food to United States Armed Forces with resulting need for tremendous and rapid expansion of their canning industry. Despite assistance by technical experts from the United States, it was inevitable that time would be required to bring productive methods to a level equal to United States standards. Although the incidence of botulism in the United States Army is small, it is suggested that supplies of botulinus antitoxin be available to take care of emergencies, such as the occasional outbreaks reported here. Furthermore, thought should be given to having available a supply of antitoxin in the event that botulinus toxin is used as an agent in biologic warfare.

Bacterial Infections

CHAPTER XX

Brucellosis

R. A. Kelser, D. V. M., Ph. D.

Over a period of years, brucellosis, or undulant fever, has gradually become a disease of increasing importance in the human family.

Prior to 1918, the malady in man was thought to be limited to infection with the organism responsible for Malta fever in goats and, therefore, more or less restricted to areas where goat raising was well developed and goat's milk more commonly consumed. With the discovery by Alice Evans in 1918 that the causative agent of infectious abortion (Bang's disease) in cattle was a species closely related to the Malta fever organism, the possibility that the bovine bacterium might be capable of producing disease in man became apparent. Subsequent studies and experience have made this early supposition a well-established fact.

There are recognized today three species of *Brucella* organisms, each capable of causing disease in both lower animals and man. The original Malta fever factor (*Micrococcus melitensis*), now commonly termed *Brucella melitensis*, is still looked upon as primarily a caprine strain producing disease in goats, sheep, swine, and also man. *Brucella abortus* is the common cause of infectious abortion in cattle, of brucellosis in man, and occasionally of disease and disease processes in other species of animals. In addition to the caprine and bovine types of *Brucella*, a porcine variety (*Brucella suis*) has also become well known. This type, while not as commonly prevalent as the bovine species, is ordinarily even more virulent for man than the cattle variety.

While more cases of brucellosis have been definitely diagnosed in recent years than previously, it is very likely that there are a great many brucellar infections which are not diagnosed. Since the end of World War II, some 4,000 to 7,000 cases of brucellosis are diagnosed annually in the United States. These figures are, without doubt, far too low.

WARTIME INCIDENCE

While it was anticipated that cases of brucellosis would be encountered in the military forces during World War II, the incidence was relatively very minor. Provisional data based on sample tabulations of primary and secondary diagnoses taken from individual medical records show a total of 1,305 cases of brucellosis in the United States Army during the years 1942 through 1945. Of this total, 956 cases were in the United States and 349 overseas.

The annual incidence rate per 1,000 average strength in the total Army, during 1942–45 combined, was 0.05, the rate in the United States (0.06) being twice the rate experienced overseas (0.03). In one year (1941), the rate reported from overseas areas was considerably higher (0.09) than for subsequent years either in the United States or abroad.

Table 76 indicates the number of cases and incidence rate of brucellosis in the United States Army at home and in foreign areas. Of the 250 cases reported among troops in the United States in 1945 (the only year for which a distribution by service command is presently available), 40 cases were in the Fourth Service Command, 70 in the Ninth Service Command, and 45 in the Eighth Service Command. Thus, approximately 62 percent of the cases was reported from 3 of the 9 service commands during this year. In considering the incidence in these three service commands, it should be remembered that in locating training camps and distributing soldier trainees the Fourth, Eighth and Ninth Service Commands were favored areas because of climatic conditions well suited for year-round training. In overseas areas, the highest incidence of

TABLE 76.—*Incidence of brucellosis in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records of primary and secondary diagnoses]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	956	0.06	151	0.06	253	0.05	302	0.08	250	0.09
Overseas:										
Europe	51	.01	—	0	6	.02	25	.01	20	.01
Mediterranean [1]	154	.10	1	.04	11	.02	52	.08	90	.25
Middle East	5	.03	—	0	4	.08	1	.02	—	0
China-Burma-India	18	.04	1	.11	4	.10	13	.08	—	0
Southwest Pacific	38	.02	3	.04	2	.01	8	.01	25	.02
Central and South Pacific	52	.04	10	.07	14	.05	8	.02	20	.05
North America [2]	12	.02	2	.02	2	.01	3	.02	5	.07
Latin America	16	.04	5	.05	—	0	6	.07	5	.07
Total overseas [3]	349	.03	22	.04	43	.03	119	.03	165	.04
Total Army	1,305	.05	173	.05	296	.04	421	.05	415	.05

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

brucellosis was in the Mediterranean theater. This coincides with the well-known occurrence of the disease in that particular part of the world.

When one considers the brucellosis cases reported by the Army during the period of World War II, it must be realized that while some of the cases, from their history, epidemiology, et cetera, without doubt originated in the military service, not all of them had their origin in the Army. The history and evidence in many cases clearly pointed to the acquisition of the infection in civil life at some earlier period. Of the 370 cases in which brucellosis was the primary cause of admission to medical treatment in 1945, the individual medical records on 70, or about 19 percent of the cases, characterized the disease as having existed prior to the time the individual entered the service. Some were probably inapparent infections, and others with appreciable symptoms were undoubtedly not identified as brucellosis. It is also possible that there were some cases in the Army during the war which were not recognized either because of their minor character or because they were classified as something else.

Brucellosis, as it occurs in man, is commonly classified as an acute, subacute, or chronic disease. Those cases which do not extend over a period greater than 10 to 12 weeks or thereabouts and which do not relapse are generally considered in the acute category. The subacute cases are those in which the acute stage is followed by one or more exacerbations, which may be more or less severe than the primary attack, but in which the patient finally recovers completely within a few weeks. The chronic type of the malady may extend over a period of a number of years with intermittent exacerbations and variable degrees of symptoms.

The period of incubation in brucellosis is quite variable. In some instances, it may be as short as 3 or 4 days, and at the other extreme it may extend over a month or more. In general, the average case will develop within 10 to 16 days.

In some cases of brucellosis, the temperatures in the morning and those in the afternoon may vary only a degree or two in their peaks, while in other cases the height of the afternoon temperature may exceed that of the morning by 4 or 5 degrees. Characteristically, the daily peaks rise gradually, in a wavelike manner, to a peak which may persist for a day or two and then drop to a low point. These wavelike temperature rises may be repeated at intervals varying from several days to several weeks. All patients do not manifest typical fever reactions. In some cases, the fever is low grade in character and may persist over a prolonged period with intermittent intervals of normal temperature; in others, the fever may be well marked in the relatively early stages of the disease and then drop rapidly and remain substantially normal or of little significance.

In addition to the generalized, febrile disease, brucellosis occasionally manifests itself as a local or focal infection in which the lungs, spleen, lymph nodes, eye, brain, bony structures (vertebrae), heart, or skin may be involved.

While the apparent cases of brucellosis commonly are debilitating and disabling, fortunately the case fatality rate is low (about 1 percent in the United States). Fatalities are often due to secondary factors and conditions rather than the specific brucellar infection.

Sources of Infection

Brucellosis in man may be acquired in one of several ways. It is an occupational hazard in some instances, occurring among farmers, dairymen, livestock raisers and handlers, and stockyard and packinghouse employees. Laboratory infections are not uncommon, and at least four cases were thus contracted in the Army during the war. In other individuals, infections more commonly occur as a result of consuming milk or dairy products containing viable Brucella organisms. It is a well-established fact that in dairy cows *Br. abortus*, which has a predilection for embryonic tissues, does not remain in the female genital tract long after parturition or abortion but migrates to the udder where it establishes itself without appreciable damage to the udder tissues. In such locality, where it will often remain for years, the *Brucella* organism multiplies and is shed with the milk. Such milk, if unpasteurized, may well infect man.

Preventive Measures

During World War II, no raw milk was authorized for the use of the United States Army troops. Furthermore, precautions were taken early to protect military personnel from infection through other dairy products such as cheese. With the great demand for cheese for soldiers' rations, lend-lease commitments, and civilian use, the Veterinary Division of the Surgeon General's Office initiated action early in the war to assure that no inadequately ripened cheese was supplied for troop consumption. Specifically, this was accomplished through the promulgation of orders which required that all cheese purchased by the military establishment be held in quartermaster depots or warehouses at least 60 days before shipment to military organizations. The wisdom of this action was attested by the fact that both brucellosis and typhoid fever outbreaks did occur among civilians in several areas as a result of eating "green" or inadequately ripened cheese. Although the Army received several lots of cheese which early tests by the Food and Drug Administration and Army laboratories proved to contain viable *Brucella* organisms shortly after purchase, subsequent extensive tests, following the prescribed 60-day holding period, demonstrated that the cheese was safe for use.

Diagnostic Tests

The clinical diagnosis of brucellosis is not easy. In cases where the disease is suspected, laboratory tests and procedures must be resorted to in order to confirm a tentative clinical diagnosis or to rule out the possibility of brucellosis.

The agglutination test is the simplest and most commonly employed diagnostic aid. Agglutination values, however, in actual cases of the disease vary considerably, and difficulty may be encountered in interpreting agglutination reactions when they occur in relatively low serum dilutions. Agglutination in serum dilutions as low as 1:100 may be diagnostically significant when considered together with clinical manifestations. On the other hand, serum from some cases of chronic brucellosis may give negative agglutination reactions. Some cases of brucellosis will give agglutination reactions in relatively high serum dilutions (one to several thousand). High agglutination titers and those cases in which a rising titer is found in a series of tests are the easiest to evaluate. In some of the chronic cases of brucellosis, the agglutination test gives negative results. It also must be borne in mind, in interpreting agglutination reactions, that cross agglutination reactions with a brucellosis antigen may be encountered in cases of tularemia and also in serum from individuals who have been vaccinated against Asiatic cholera.

The complement fixation test may be used as a diagnostic procedure in brucellosis, but since it is more complicated than the agglutination test it is not utilized as frequently. It, however, is perhaps positive earlier in the disease than the agglutination test, and it may persist for a longer period.

The opsonocytophagic test, in which the ability of the polymorphonuclear leukocytes from suspected cases of brucellosis to phagocytize *Brucella* organisms is compared with the normal, has been utilized to a considerable extent. The results with this test, however, have not been entirely satisfactory. An allergy test, utilizing an agent (Brucellergen) which is comparable to tuberculin and which is administered intradermally, has been of some value in the diagnosis of brucellosis in man. Experience has shown, however, that it is sometimes negative in cases proved culturally to be brucellosis.

Naturally, the most positive evidence of brucellosis is the isolation of the *Brucella* organism from the patient. The bacterium is usually present in the blood in the initial stages of primary attacks of the disease but commonly is there only in relatively small numbers. Repeated blood cultures, therefore, are made at frequent intervals in attempting to isolate the organism.

Where the organism is isolated in pure culture, typing to determine species is not particularly difficult. Bacteriologic procedures involving tests for CO_2 requirements, the production of H_2S, and growth or failure of growth in the presence of certain dyes such as basic fuchsin, thionine, pyronine, and methylene-violet, are commonly utilized in typing *Brucella* organisms. Agglutinin absorption tests are likewise very valuable in determining *Brucella* species. All of this is relatively easy when the specific organism has been isolated from a case of brucellosis. Much greater difficulty is encountered if blood serum from the patient is the only thing available for typing.

The precise incidence of the different specific types of brucellar infections among those cases of brucellosis identified in the Army was not determined. Most of the cases in the Army were diagnosed on the basis of clinical symptoms and agglutination tests. In some instances, in addition, the opsonocytophagic

and skin tests were used. While blood cultures for the isolation of the bacterium were commonly made, recovery of the organism was obtained in only a relatively small percentage of the cases.

Treatment

When the sulfonamides became available and with the development of the various antibiotics, hopes were entertained that one or more of these agents would prove specific in the treatment of brucellosis. In general, the results from the use of all of these agents have been disappointing. In occasional cases, the use of some form of sulfonamides or antibiotics or combinations of them appeared to influence favorably the course of the disease. On the other hand, in similar cases, no appreciable favorable effects were achieved with such therapy. With some patients, rest in bed with little or no treatment has often given results comparable to those seemingly obtained with some of the therapeutic agents which have been employed. The combined use of sulfadiazine and streptomycin gained considerable favor in the treatment of brucellosis after expectations from penicillin failed to materialize. Then, since the advent of aureomycin, it has been considered by some that it is of distinct value. In view of all of the evidence, however, it must be concluded that, while certain beneficial results may be obtained from the use of some of these agents in individual cases, there is as yet no specific therapeutic treatment which can be relied upon in the management of brucellosis.

CHAPTER XXI

Cholera

Kirk T. Mosley, M. D.

HISTORICAL NOTE

The early history of Asiatic cholera in the United States Army is based upon the experience of the military forces with the disease when cholera pandemics originating in the endemic centers of the Far East invaded the North American Continent and gave rise to widespread epidemics within the United States on at least four different occasions in the 19th century.[1] The disease entered the United States through seaports, particularly New York and New Orleans, being brought in by infected immigrants from cholera-stricken areas of western Europe. In the first three epidemics, which swept this country in the fourth, sixth, and seventh decades of the 19th century, troops of the Army often suffered as severely as did the civilian population from outbreaks of the disease. In some instances, troops were responsible for the spread of the disease as infected units were moved from a station to a new post where the disease had not yet appeared. In many instances, the disease was introduced among the troops from civilian communities. One of the first experiences of American soldiers with cholera fully demonstrated its devastating character and showed how disastrous an outbreak can be to a military operation. During the Black Hawk Indian War in 1832, seven companies of infantry troops destined for this campaign embarked on the steamer *Henry Clay* at Buffalo, N. Y., on 1 July. Cholera broke out among the troops on 4 July. By 9 July, only 68 men of the 7 companies that departed from Buffalo were left. Many died of the disease on board ship; many others deserted in panic and died of cholera in the surrounding countryside.

Fortunately, cholera has not been a serious problem to military operations of the Army in any of the major conflicts involving the United States. The disease had not reached the North American Continent at the time of the Revolutionary War and the War of 1812. The Mexican War had just ended when the second American invasion by cholera began in 1849. Cholera was absent from the United States during the Civil War but entered shortly thereafter, in 1866, for the third major outbreak in this country. Cholera apparently played no role in the Spanish-American War, but in 1902 and 1903 the disease broke out among American troops on duty in the Philippine Islands.

[1] Wendt, Edmund C.: A Treatise on Asiatic Cholera. New York: W. Wood & Co., 1885.

The islands at that time were suffering from a severe epidemic of cholera which had spread from the Asiatic mainland.[2] For the next 15 years, sporadic cases of Asiatic cholera appeared in American troops stationed in the Philippine Islands. During World War I, 11 admissions for cholera with 2 deaths occurred among American troops overseas; both deaths and 10 of the admissions were among American troops on duty in the Philippines and China.[3] An additional 6 admissions for cholera, with 5 deaths, occurred among Philippine native troops. In World War II, there were only 13 cases and 2 deaths from cholera among American troops, though large forces were involved in extensive military operations in the highly endemic centers of the disease on the Asiatic mainland. The remarkable achievement of the Army in cholera prevention in World War II is attributable to well-developed and well-executed disease-control measures by our military forces. Principles of sanitation effective in the prevention of infectious diseases transmitted by way of the gastrointestinal tract were particularly emphasized in the preventive measures against cholera.

ADMINISTRATIVE PREVENTIVE MEASURES

With the entry of the United States into the war, necessary revisions were made in Army regulations in order to take full advantage of the latest developments in the field of sanitation and preventive medicine for the protection of troops against infectious diseases, including cholera. Army Regulations No. 40–205, Military Hygiene and Sanitation, was revised and published on 31 December 1942. Army Regulations No. 40–210, Prevention and Control of Communicable Diseases of Man, was published on 15 September 1942 after extensive revision. This revision greatly simplified and condensed a large number of previous regulations and War Department circulars and represented a most valuable accomplishment in the administrative service of the Surgeon General's Office in communicable disease control.

Circular Letter No. 56, published by the Surgeon General's Office on 9 June 1941, provided technical guidance on the prevention of cholera. This letter was revised and published as Circular Letter No. 33 on 2 February 1943. In each of these letters, a section was devoted to cholera. The cholera section in the latter publication was superseded by TB MED 138, published in February 1945. These publications provided medical officers with the latest knowledge concerning the epidemiology and clinical aspects of cholera. The facts that cholera was in the category of an exotic disease and was little more than a name to most medical officers enhanced to a great degree the value of the technical directives on cholera prepared by the Surgeon General's Office.

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[2] Annual Reports of The Surgeon General of the Army, 1902, 1903, 1904. Washington: Government Printing Office.

[3] The Medical Department of the United States Army in the World War. Statistics. Washington: Government Printing Office, 1925, vol. XV, pt. 2, pp. 86–89, 134–137.

Research

Although principles of sanitation for the prevention of cholera were well established and of proved value, there was a constant effort to increase the effectiveness of the known preventive measures against this disease and to develop new ones through research and investigation. This activity of the Surgeon General's Office may be divided into two general fields: Improvement and evaluation of the effectiveness of the cholera vaccine and investigation of drugs and chemicals which might be effective in the prophylactic treatment of individuals exposed to chlorea. Also, knowledge of the pandemic tendencies of cholera and its ability to reach major epidemic proportions under conditions of warfare made it necessary to forestall the menace of cholera even before our country became militarily involved in World War II.

The preliminary precautionary measures were chiefly directed at improving the effectiveness of cholera vaccine. In the spring of 1941, information regarding the use of vaccines for the prevention of cholera and the method of obtaining the best vaccine possible was sought from medical authorities. In the fall of 1941, direct approach was made to the National Research Council for aid and advice in solving the problems connected with the use of immunization procedures against certain infectious diseases, including cholera.[4] Acting quickly on this request, the National Research Council assembled a conference of experts which met on 22 October 1941 and prepared resolutions proposing policies to be followed in the immunization of American troops against certain infectious diseases. These resolutions were submitted to The Surgeon General and were the basis of policies adopted by the Army in its immunization program against cholera.[5] The chapter on immunization in another of the preventive medicine volumes gives details of the cholera immunization policies and practices adopted by the War Department.[6]

The establishment of policies governing cholera vaccine and its use did not lessen the interest of The Surgeon General in investigations to improve the effectiveness of vaccine as a protective measure. Arrangements were made to obtain from East Indian and Egyptian sources new strains of *Vibrio cholerae* for the study of their immunizing properties and for possible use in preparation of vaccines.[7] The Surgeon General made valuable use of the resources of the National Research Council to keep informed about progress in the studies on cholera vaccine, including the chemical aspects, various strains of cholera vibrios, and techniques used in controlling the production and testing the potency of vaccines against cholera. In the summer of 1942, Col. (later Brig. Gen.) James S. Simmons, MC, recognizing the need for organized effort to

[4] Memorandum, Surgeon General's Office (Col. J. S. Simmons), for Dr. L. H. Weed, National Research Council, 8 Sept. 1941, subject: Immunization Against Certain Infectious Diseases, Notably Plague, Cholera, and Typhus.

[5] War Department Circular No. 4, 6 Jan. 1942, sec. 3, Vaccination Against Typhus Fever, Cholera, and Plague.

[6] Long, Arthur P.: The Army Immunization Program. *In* Medical Department, United States Army, Preventive Medicine in World War II. Personal Health Measures and Immunization. Washington: U. S. Government Printing Office, 1955, vol. III, pp. 314–317, 347–348.

[7] Memorandum, Surgeon General's Office (Lt. Col. Rogers), for the Adjutant General's Office, 22 Dec. 1941.

further the Army's knowledge concerning protection against cholera, especially in respect to the value of vaccines, urged that the National Research Council establish a small committee to coordinate cholera research activities. A committee was appointed on 26 August 1942. The committee, in a report made a few weeks later, summarized the existing status of knowledge about cholera vaccines, with particular reference to research being done at the time.[8] It was agreed, on the basis of this report, that the cholera vaccine in current use by the Armed Forces should be continued in its present form, though research was to be encouraged on the antigenic, chemical, and other aspects of the cholera organism, with the hope of improving the vaccine against the disease.

In the latter part of 1942, an informal suggestion was made concerning the advisability of establishing a cholera commission similar to the Typhus Commission. This suggestion, however, did not receive favorable consideration.

In a conference on cholera vaccine held by the National Research Council on 16 June 1943, it was decided that a field experiment in a cholera area was needed to determine the efficacy of the vaccine then being used by the Armed Forces and such other vaccines as might be selected in protecting a population against the disease. By a strange but fortunate coincidence, a very extensive field study on this same problem was actually in progress in India. A preliminary report of this study, conducted in Madras Province, India, under the direction of Dr. R. Adiseshan, Director of Public Health, Madras, with the assistance of Dr. C. G. Pandit and Dr. K. V. Venkatraman of the King Institute of Preventive Medicine, Guindy, Madras, and other scientists, was made available to the National Research Council by the end of the year and indicated that a considerable degree of protection was afforded by immunization.[9] According to the evidence presented in a final report, an immunized population is at least 10 times less susceptible to the disease than an unimmunized population.

Research in the use of drugs in the treatment of cholera, though chiefly the concern of the Medical Consultants Division of the Surgeon General's Office, was of great interest to the Preventive Medicine Division. This interest was an outgrowth of the remarkable success in the use of drugs in small doses for prophylactic or suppressive effects in such diseases as malaria and meningococcal meningitis. Also, pressure for information about drugs effective against cholera developed when it became obvious that an increasingly larger number of troops would be exposed to cholera and that complete protection to exposed individuals is not assured by cholera inoculations. The logical place for studies on the therapeutic effectiveness of drugs is in an endemic area of the disease. Since India is such an area, the National Research Council was requested in February 1945 to consult with the Director General of the Indian Medical Service concerning plans of scientific groups of that country for investigating newer drugs in the therapy of cholera. The council was also re-

[8] Minutes, Committee on Medical Research, National Research Council, 26 Sept. 1942, subject: Conference on Cholera Vaccine.

[9] Minutes, Subcommittee on Tropical Diseases, Committee on Medical Research, National Research Council, 28 Jan. 1944.

quested to ascertain whether a representative of the United States would be welcomed to observe and participate in the investigations. Reports of investigations by Indian scientists conducted under the auspices of the Indian Research Fund Association had been the source of most of the information on the use of newer chemotherapeutic agents in the treatment of cholera. The close liaison of the International Health Division of the Rockefeller Foundation with scientific organizations and agencies of India was of great benefit in making available to The Surgeon General information on the results and progress of research being conducted in that country. While there were indications that Indian authorities and scientists would give favorable consideration to the proposals of the National Research Council, the termination of the war occurred before final arrangements were completed, and the project never materialized. However, the results of an investigation of the treatment of cholera, conducted in Calcutta by a Navy epidemiologic team, led to The Surgeon General's issuance of a letter suggesting certain modifications in the therapy of cholera as described in TB Med 138. These modifications were concerned chiefly with the use of penicillin and plasma in the treatment of acute cases. At the close of the war, no reliable information had been obtained on the use of drugs in the chemoprophylaxis of cholera.

ARMY EXPERIENCE IN THEATERS AND AREAS

India-Burma Theater

The greatest exposure of United States troops to cholera occurred in the China-Burma-India (later India-Burma) theater. Cholera was a constant menace to a large number of troops in this area, especially to those stationed in or near Calcutta or passing through or visiting this metropolis and its environs. In this section of India along the lower Ganges River, the disease is nearly always present with the danger of large epidemics occurring during the cholera season each year.

In spite of the ever present possibility of cholera outbreaks and the occurrence of extensive epidemics among civilians in proximity to military installations, no cases of the disease were reported among United States Army troops stationed in this theater. This excellent record is even more unusual when compared with that of Indian and British troops (table 77) who were stationed in the same area and potentially subjected to the same exposure as the United States troops. While cholera outbreaks were known to have occurred among Chinese troops stationed in India and Burma, reliable information could not be obtained on the number of cases and deaths from this disease among Chinese soldiers assigned to this theater.

Except for immunization directives requiring troops to be immunized against cholera and to receive stimulating doses of vaccine every 6 months, very few administrative actions at theater level were specifically directed against cholera, although a survey on the cholera situation in Assam and

TABLE 77.— *Cholera cases and deaths among enlisted personnel in British and Indian Armies, in India, 1940–46*

Year	British Army		Indian Army	
	Cases	Deaths	Cases	Deaths
1940	1	1	1	Unknown.
1941	25	4	61	Do.
1942	1	1	47	Do.
1943	4	.....	154	Do.
1944	8	2	70	Do.
1945	16	4	67	Do.
1946	7	.....	100	Do.

Source: Annual Reports on the Health of the Army in India.

Bengal Provinces was made by the theater surgeon's office in the latter part of 1943.[10] The absence of theater action specifically dealing with cholera is made apparent in the official compilation of directives affecting the Medical Department which were in force as of 31 December 1944.[11] In that section of the compilation arranged according to subject matter, the subject of cholera did not appear once, since no directives in force at that time pertained specifically to this disease. The explanation for this seeming failure to appreciate the hazard of cholera lies in the fact that, from the very beginning, other gastrointestinal infections, especially the diarrheas and dysenteries, presented the theater with a serious health problem and overshadowed cholera as a cause of concern to the Medical Department.[12] The increasingly high rates for the diarrheas and dysenteries approached a critical level in 1944 and were the basis of two extensive surveys, one by a subcommission of the Army Epidemiological Board [13] and a second by a special commission from the Office of the Surgeon General headed by Brig. Gen. Raymond A. Kelser.[14] Each survey listed a great number of defects in sanitation which might readily account for a high incidence of gastrointestinal diseases, particularly in an environment which is heavily seeded with the infectious agents of these diseases. As a consequence, most of the administrative actions taken in regard to gastrointestinal diseases had as their primary purpose the control and prevention of the diarrheas and dysenteries. In most instances, these measures were by their very nature equally effective against cholera.

[10] (1) Circular No. 50, Rear Echelon, Headquarters, U. S. Army Forces, China-Burma-India Theater, 17 Aug. 1943, subject: Immunization. (2) Circular No. 5, Headquarters, U. S. Army Forces, India-Burma Theater, 20 Jan. 1945, sec. V, Immunization. (3) Essential Technical Medical Data, Headquarters, China-Burma-India Theater, 13 Dec. 1943.

[11] Circular Letter No. 1, Office of the Surgeon, Headquarters, U. S. Army Forces, India-Burma Theater, 1 Jan. 1945, subject: Compilation of Directives Affecting the Medical Department, India-Burma Theater.

[12] Van Auken, H. A.: A History of Preventive Medicine in the U. S. Army Forces of the India-Burma Theater, 1942–45. [Official record.]

[13] (1) Monthly Report for July, Sub-Commission on Dysentery, Army Epidemiological Board, 8 Aug. 1944. (2) Progress and Final Report, Sub-Commission on Dysentery, Army Epidemiological Board, 20 Nov. 1944.

[14] Letter, Headquarters, Services of Supply, U. S. Army Forces, India-Burma Theater, to Commanding General, U. S. Army Forces, India-Burma Theater, 9 Nov. 1944, subject: Report of Medical Department Mission.

There were occasions, however, when the threat of cholera was particularly dangerous and received special attention from the Medical Department in specific commands and areas in the theater. The most serious threat of cholera to American troops in the India-Burma theater occurred in the Calcutta area in the spring of 1945 when a severe epidemic broke out among the civilian population in Calcutta and the surrounding territory, with as many as 100 cases and 35 deaths being reported daily. At the height of the outbreak, the epidemic spread to British troops billeted in the Grand Hotel, with 14 cases reported in this group. The 135 American officers also billeted in the hotel were immediately removed, and the dining quarters of the establishment was put out of bounds for American forces. To Lt. Col. K. R. Flamm, MC, the surgeon of this base section, can be attributed much of the success of the vigorous attack against this threat of cholera. The precautionary measures launched against the disease included: (1) An intensive educational campaign explaining to troops how cholera is spread and how to avoid it; (2) special measures to insure that all troops stationed in the area or passing through were properly immunized against cholera; (3) rigorous inspection of civilian eating establishments in bounds to United States troops, with special attention given to water protection and sanitation, foodhandlers' hygiene, exclusion of serving of fresh raw fruits or vegetables to troops who were patrons, and other sanitation requirements essential for the prevention of cholera; and (4) enforcement of strict sanitary discipline in all the Army messes and other food-dispensing establishments on Army posts, with particular attention to native foodhandlers and their personal hygiene. The use of Indian labor for foodhandling had been discouraged as far as possible, but utilization of cheap labor for this type of service was never completely abandoned by many units. These special measures taken by the commanding general of Base Section 2, which included Calcutta and the surrounding area, represented the only large-scale effort directed primarily at prevention and control of cholera in the India-Burma theater. Because of the strategic importance of Calcutta and the relatively large number of troops (24,500) stationed in the city and its immediate environs, as well as a great flow of Army personnel through this military center, an extensive epidemic of cholera among troops in the area would have gravely impaired the functions of this vital supply base and resulted in serious interference to the theater operations.

Other incidents involving cholera reported in the theater were of much less military significance but were of considerable medical interest. In the spring of 1943, a severe cholera outbreak occurred among the civilian population in several villages, including Pandaveswar, which were in the immediate vicinity of airbases of the 7th Bombardment Group. Special measures were taken by the local commanders to prevent the spread of the disease to personnel in these installations.[15] In the spring of 1945, three suspected cases of cholera in American troops were reported by the 4th Combat Cargo Group stationed

[15] Letter, Surgeon, 7th Bombardment Group, to the Commanding Officer, 7th Bombardment Group, 16 June 1943, subject: Cholera Epidemic Among Natives in Panda.

at Chittagong. These suspected cases and the circumstances surrounding their occurrence were thoroughly investigated by the theater epidemiologist, who was unable to find sufficient evidence to support the tentative diagnosis of cholera. Another incident later in the same year was the occurrence of a case of cholera in a Red Cross worker who was presumably infected in Calcutta but developed her illness in Karachi while she was waiting for passage to the United States. This case, though mild, was confirmed by positive stool cultures.[16]

It is very difficult to draw any satisfactory conclusions concerning the unique record of the India-Burma theater in regard to cholera. While there were no cases among Army personnel, reports of the two separate extensive surveys, which included observations on sanitary conditions in military units, indicate that there were certainly opportunities for exposures to the disease, particularly in those areas where the disease is known to be endemically present at all times. Thus, much of the protection against cholera might be attributed to the effectiveness of the cholera vaccine. While such an assumption seems reasonable, an excellent summary of the American forces' experience with cholera in India contained in a report from the theater, points out certain difficulties in determining the protective value of cholera inoculations as well as in evaluating the relative merits of immunization and sanitation as cholera preventive measures.

China Theater

American troops assigned to the China section of the China-Burma-India theater (later, October 1944, the China theater) faced essentially the same threat from cholera which troops faced in India. The disease was endemic throughout the area of Free China in which American troops were stationed, and epidemic outbreaks occurred periodically. During the war years, 1942–45, widespread outbreaks struck the civilian populations in many of these areas of Free China, in some instances in the immediate vicinity of Army installations. Because of the constant possibility of an explosive outbreak, routine immunization against cholera with stimulating doses at 6-month intervals was administered to all United States Army personnel.[17] Sanitation was emphasized as an important measure for the prevention of cholera and similar enteric diseases. The Fourteenth Air Force published a memorandum which was ordered to be read to all members of that command at monthly intervals.[18]

American troops remained relatively safe from cholera until the summer of 1945, when outbreaks seriously threatened units in various parts of Free China. As a result of these outbreaks, a special cholera commission made up of civilian experts was sent to assist the Chinese National Relief and Rehabilitation Administration in controlling the disease among the civilian population. The first of these threats developed in Chungking, the provisional capital of Free

[16] Essential Technical Medical Data, India-Burma Theater, 1 June 1945.
[17] Annual Report, Office of the Surgeon, Headquarters, China Theater, 26 Oct.–31 Dec. 1944.
[18] Fourteenth Air Force Memorandum 25-8, 10 May 1944, subject: Rules for Health in China.

China and also the headquarters of United States Army forces in China, with the occurrence of a severe epidemic among the civilian population. This outbreak was carefully investigated by the theater medical inspector, and his report is one of the remarkable documents on cholera during World War II. His investigation revealed that sanitary conditions in the American installations were in many instances very unsatisfactory, and of particular concern to him were the serious defects observed in sanitation and in protection of water supplies. Necessary corrective actions were taken in accordance with his recommendations and included the publication of directives on cholera control for troops in the Chungking area. The efforts to prevent cholera from spreading to American forces in the Chungking area were apparently successful, since no cases of the disease appeared in our troops in this area.[19]

However, only a few weeks later, two separate sharp outbreaks of cholera, the only outbreaks to involve American troops in World War II, appeared in units stationed in other sections of China.[20] The first outbreak, which resulted in six cases with one death, occurred during the later part of July 1945 among the enlisted personnel of the 1836th Ordnance Company stationed at the Liangshan Air Base. This outbreak was attributed to the consumption of cakes and cookies which, against instructions, had been purchased from a bakery in the city of Liangshan, where a serious epidemic of cholera was raging. The cakes were served at a snackbar operating on the base. At the same time that these cases of cholera occurred, there was a sharp increase in the number of cases of diarrhea among troops of the 1836th Ordnance Company which operated the snackbar where the questionable food was being served. The six cases of cholera were diagnosed on clinical grounds and treated in the station dispensary. The five who recovered responded well to therapy which included in some instances penicillin and sulfaguanidine in addition to parenteral fluids. According to the immunization records of these six cases, the initial immunizations against cholera were completed in August 1943 with American vaccine. All except one received a stimulating dose of Chinese vaccine in April and May 1944. Five cases received stimulating doses of Chinese vaccine in October 1944; the sixth received American vaccine in September 1944. In April 1945, five cases received American vaccine, and in May 1945 the remaining case received Chinese vaccine. Only two of the cases received another stimulating dose in July 1945.

The other outbreak occurred at Chihchiang during the first week of August 1945 and involved personnel of the 547th Quartermaster Depot Supply Company. In this outbreak, there were seven cases with one death. The vehicle of transmission in this outbreak apparently was contaminated water, since all of the patients were known to have, on one or more occasions, drunk

[19] Essential Technical Medical Data, Headquarters, China Theater, 12 Aug. 45, inclosure 2.

[20] (1) Essential Technical Medical Data, U. S. Army Forces, Headquarters, China Theater, 2 Oct. 1945, pp. 4-5 and inclosures 2 and 3. (2) Annual Report, Office of the Surgeon, Headquarters, Fourteenth Air Force, 1945 (2d and 3d quarters). (3) Annual Report, Headquarters, Army Air Forces, China Theater, 1945 (3d and 4th quarters), annex III, inclosure 4.

water, unboiled and unchlorinated, from a well at the Catholic Mission in the city of Chihchiang. Samples of water taken from this well on two separate occasions were submitted to the 172d General Hospital for analysis and were found to be positive for colon-group organisms. One nun at the mission developed cholera from which she recovered.

In this outbreak, the patients were hospitalized and treated in the 21st Field Hospital. Stool specimens which were examined in the 172d General Hospital were positive for all seven cases. All patients received adequate amounts of parenteral fluids to combat dehydration and acidosis. Four patients also received penicillin, and, according to one source of information, there was no appreciable difference in response between the patients who received penicillin and those who did not. The patient who died received penicillin therapy. There is lack of information on the immunization histories of these cases, though it is known that one patient had received a stimulating dose of American vaccine on 29 July 1945 and that the other six patients had received stimulating doses of Indian vaccine on 26 April 1945.

The seriousness of the cholera situation in the China theater was called to the attention of all troops in a theater circular dated 2 August 1945 in which the following measures were emphasized: Water sanitation, including purification and handling after treatment; precautions in the preparation and handling of food; avoidance of prepared food and drink from civilian sources; insect-control measures; provision of adequate waste-disposal facilities for American personnel and for Chinese personnel working in American installations; and the maintenance of high immunity to cholera by administration of stimulating doses of vaccine at intervals of not more than 4 months. No further outbreaks occurred among United States forces in China. The absence of additional cases is attributed to the combined effectiveness of sanitation and immunization.

Southwest Pacific Area

The medical history of the United States Army in the Southwest Pacific Area in respect to cholera is an excellent account of wise precautions and careful preparation for eventualities. Although during the initial stages the campaign in this area was far removed from endemic cholera areas, the disease spread far beyond the usual endemic centers, having been seeded by the movement of Japanese troops throughout Southeast Asia including Indo-China, Siam, Singapore, and islands to the south and east of the mainland including the Philippines, Java, Sumatra, Celebes (Makassar), Truk, and possibly New Britain (Rabaul). There were reports that the disease had occurred in units of the Japanese Army in many of these areas, and it was believed that the spread of the disease was partly due to the movement of infected Japanese troops from endemic foci to previously uninfected areas.

The spread of cholera from the mainland of Asia toward areas which

were in the line of the offensive from Southwest Pacific bases, together with the possibility that troops at any time might encounter the disease in infected units of enemy troops, stimulated the development of a careful cholera-control program. Evidence of the careful planning designed to minimize any potential threat of cholera to the success of military operations in this area is contained in a letter from the Chief Surgeon to the Surgeon, Sixth U. S. Army, dated 3 April 1944, recommending (1) vaccination of all troops against cholera; (2) the establishment, in the supplying base of each operation, of a special stock of supplies (such as cresol, gowns, mosquito and fly netting, and salt solution infusion sets), earmarked for epidemic use; (3) equipment of forward laboratory sections for prompt bacteriologic diagnoses of cholera; (4) publication of a technical memorandum for all medical officers on the diagnosis and treatment of cholera and on special precautions to be taken in addition to usual sanitary measures; and (5) a report to the task force surgeon of any evidence discovered of epidemic disease among enemy troops. These measures were all implemented by the necessary official directives and letters.[21] In the planning of the specific preventive measures for cholera, full use was made of the counsel and advice of the Combined Advisory Committee on Tropical Medicine, Hygiene, and Sanitation. The Committee addressed a letter dated 22 June 1944 to the Commander in Chief, Southwest Pacific Area, giving full support to the cholera-control measures outlined in the letter of 3 April 1944 referred to.

Although no cholera cases or incidents occurred among United States troops in the Southwest Pacific, the planning in this area was well designed to meet any cholera situation which might be encountered under conditions of combat.

Cholera in Other Theaters and Areas

Cholera did not present a problem in other theaters and areas. However, each theater had regulations concerning the requirements for cholera immunizations, and, because of pandemic potentialities of cholera, especially under conditions of warfare, a careful watch was maintained in all theaters for any evidence indicating the spread of the disease from its endemic centers. From time to time, false alarms of spread in the Western Hemisphere were received; however, during World War II, the extension of the disease beyond the usual endemic centers occurred only in areas which came under the influence of Japanese military control.

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[21] (1) Technical Memorandum No. 1, Office of the Chief Surgeon, Headquarters, U. S. Army Forces in the Far East, 17 Apr. 1944, subject: Cholera. (2) Letter, General Headquarters, Southwest Pacific Area, to Commander, Allied Land Forces; Commander, Allied Air Forces; Commander, Allied Naval Forces; Commanding General, U. S. Army Forces in the Far East; Commanding General, U. S. Army Services of Supply, 24 Sept. 1944, subject: Cholera. (3) Letter, Headquarters, Advance U. S. Army Forces in the Far East, to Commanding General, Sixth Army; Commanding General, Eighth Army; Commanding General, Far East Air Forces; Commanding General, XIV Corps; Commanding General, U. S. Army Services of Supply; Commanding General, 14th Antiaircraft Command; Commanding General, Replacement Command, 12 Oct. 1944, subject: Cholera.

SUMMARY

The history of cholera in the United States Army is paradoxical in that this disease exacted a heavier toll among troops in times of peace than during periods of major military campaigns, when epidemic diseases such as cholera are usually most prevalent and destructive. Such a record owes much to the fortunate circumstance that peace prevailed on the North American Continent when cholera made its invasions of the Western Hemisphere in the 19th century. Also, there is the factor that military operations by United States troops in highly endemic areas in World War II did not include ground-combat activities to any significant degree. Most of the troops in the India-Burma and China theaters were primarily engaged in opening and maintaining communication and supply lines to Free China by way of India and northern Burma. Air Force personnel engaged in aerial warfare in these theaters were stationed in bases which were in most instances comparatively safe from enemy action, so that it was possible to maintain a high degree of sanitation in these military establishments.

Both sanitation and immunization were emphasized as preventive measures against cholera by the Medical Department of the Army. From the experience in World War II, it is impossible to evaluate the relative merits of each of these measures separately. Used together, they proved very effective under the conditions and circumstances faced by United States troops in the highly endemic areas of the disease.

While a creditable record was made in the prevention of cholera, very little was added to basic knowledge about the epidemiology of the disease. The opportunity for increasing understanding of basic factors influencing its peculiar restricted geographic distribution, epidemic and pandemic properties, and other unsolved problems was not pressed with the same intensity as were the investigations of other diseases of less catastrophic potentialities. Most of the investigative work centered around attempts to improve the cholera vaccine and a somewhat belated effort to discover a chemical prophylactic.

CHAPTER XXII

Typhoid and Paratyphoid Fevers

Dwight M. Kuhns, M. D., and Capt. Donald L. Learnard, MSC

HISTORICAL NOTE [1]

The problem of typhoid and paratyphoid fevers is well recorded in the annals of military history. Until 1818, however, there were no records of disease and epidemiology in the United States Army. With the appointment of Dr. Joseph Lovell as the first Surgeon General of the United States Army and recognition of the Medical Department as a staff department of the Regular Army by Act of Congress,[2] the first system of reports was initiated.

Between 1818 and 1865, little of importance in regard to typhoid fever was recorded. Some data on the subject are contained in the Medical and Surgical History of the War of the Rebellion.[3] Following the work of Pettenkoffer in the middle of the 19th century, in which he demonstrated the importance of water supply in the epidemiology of typhoid fever, water sanitation was adopted by many cities and communities throughout the world. Following 1857, the mortality rate of continental Europe dropped markedly.[4] In the Army, field sanitation became a matter of immediate concern, and problems of water and food supply were investigated. As a result, strict sanitary discipline was invoked, and efforts were made to educate personnel involved in food and water procurement.

With the onset of the Spanish-American War in 1898 and the campaigns in Cuba, the Philippine Islands, and Puerto Rico, typhoid fever became widespread throughout the Army. As a result of the alarming pace at which the death rate increased month by month, the Army set up a Typhoid Fever Board to study the epidemiology of typhoid fever in United States military camps, and Maj. Walter Reed, MC, Maj. Victor C. Vaughan, MC, and Maj. Edward O. Shakespeare, MC, were appointed as members. The board brought to light much of the present knowledge of the disease and its epidemiology.[5]

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[1] Acknowledgment is hereby made to Martin Roth, M. A., for his faithful assistance in the research for historical material for this section and in the final preparation of the manuscript.

[2] Military Medical Manual. 5th ed. Harrisburg: The Military Service Publishing Co., 1942.

[3] Medical and Surgical History of the War of the Rebellion. Medical History. Washington: Government Printing Office, 1888, vol. I, pt. III.

[4] Bulloch, W.: History of Bacteriology. *In* A System of Bacteriology. [Great Britain] Medical Research Council. London: His Majesty's Stationery Office, 1930, vol. 1, pp. 15–103.

[5] Reed, Walter; Vaughan, Victor C.; and Shakespeare, Edward O.: Report on the Origin and Spread of Typhoid Fever in United States Military Camps During the Spanish War of 1898. Washington: Government Printing Office, 1904, vol I.

In the year 1898, with a mean strength of 147,795 men, the Army reported 20,926 cases of typhoid fever resulting in 2,192 deaths. These figures are perhaps not too accurate since at that time typhoid fever was a term used to describe a wide variety of enteric conditions and since the disease was often diagnosed as malaria. Records of the Office of the Surgeon General indicate an admission rate of 14,158.80 per 100,000 in 1898 with a death rate of 1,483.14 per 100,000.

The one important concept not appreciated by the Typhoid Fever Board was that of the carrier. It remained for Robert Koch (1902) to describe the various carrier states and the role of the carrier in typhoid epidemiology.[6]

In 1908, the Army sent Maj. (later Brig. Gen.) F. F. Russell, MC, to study the epidemiology of typhoid in foreign armies. His report was the basis for the appointment of a board which reviewed the history of vaccination against typhoid. The recommendations of the board led to the adoption of antityphoid inoculation in the United States Army, which was the first organization to establish the use of typhoid vaccination on a large scale and to determine definitely its value in preventive medicine. In March 1909, the first vaccine used as a protective measure against typhoid fever was introduced in the United States Army on a voluntary basis. In March 1911, the first large-scale immunization program was carried out in Texas on 15,000 troops used in quelling hostilities along the Mexican border, and by the end of that year 85 percent of the Army had been vaccinated. Siler, Dunham, and Longfellow[7] reported that, as a result of this vaccination program, the admission rate per 100,000 dropped from 30.52 in 1912 to 4.41 in 1913. In 1910, one of the most important steps ever taken in the control of typhoid fever was made by Maj. (later Brig. Gen.) C. R. Darnall, MC, who introduced the liquid chlorine method of water purification, a method that has since been adopted the world over.[8]

The next significant date in the historical review of typhoid and paratyphoid fevers is 16 September 1916, the date the Army Medical School supplied the Army with its first paratyphoid A vaccine. The standard routine then became the administration of 6 doses of vaccine— 3 of antityphoid and 3 of antiparatyphoid A.

By July 1917, the Army Medical School had perfected a new vaccine which included antityphoid, antiparatyphoid A, and antiparatyphoid B fractions. This new product was known as TAB vaccine (triple typhoid saline vaccine) and was administered to all newly inducted personnel as well as to individuals already in the Army. For the first time in history, typhoid fever became a disease of secondary importance in military operations, no longer ranking among the 30 diseases of most frequent occurrence.

Siler and his associates reported that the annual typhoid fever admission

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[6] Gay, Frederick P.: Agents of Disease and Host Resistance. Springfield: Charles C Thomas, 1935, p. 1557.
[7] Siler, J. F., Dunham, G. C., and Longfellow, D.: Typhoid Fever. Baltimore: The Johns Hopkins Press, 1941.
[8] See footnote 2, p. 463.

rate for the World War I period was 37 per 100,000 strength. Typhoid fever contributed only 0.04 percent of the total admissions to hospitals for all diseases and, of all deaths from disease during World War I, only 0.39 percent were attributed to typhoid fever. There were 1,529 admissions to hospital for typhoid fever among approximately 4,000,000 troops to give an annual rate of 0.37 per 1,000 strength throughout the Army. As had been anticipated, a higher incidence rate was experienced in Europe than in the United States. The greater prevalence overseas was, in all probability, due to breakdown of induced immunity in certain individuals by numerous and repeated exposures to massive doses of the causative organism in specific sections where food, milk, and water were contaminated, sewage disposal inadequate, and a general low civilian health standard prevailed. Paratyphoid fevers were likewise not a major problem during the World War I period. Admission rates for the entire United States Army for this period were 0.05 per 1,000 strength annually, considerably lower than the admission rate for typhoid fever. Six deaths from paratyphoid fever were reported in 1918 and five in 1919.

The contrast between the typhoid-paratyphoid problem in World War I as compared with that of the Spanish-American War can be attributed to the continuous application of control measures. In those days, it was thought that: "The control of typhoid fever is based first, on preventing the transmission of massive or continuous infection by water, food and contact, that is, by water purification, food control and waste disposal, and second, on immunization by prophylactic vaccination."[9]

For the 10-year period 1920–29, the average number of admissions for typhoid and paratyphoid was 7.5 and 0.8, respectively, per annum, and during this time nine deaths were reported for typhoid with none from paratyphoid. The average annual admission rates per 1,000 troops during this period were 0.05 for typhoid and 0.01 for paratyphoid.

Since July 1917, all Army personnel had been inoculated with the triple typhoid vaccine containing *Salmonella typhosa*, *Salmonella paratyphi*, and *Salmonella schottmülleri* suspensions. In May 1928, the paratyphoid B constituent was eliminated from the vaccine in view of the very low incidence of the disease, no cases having been encountered since 1922, and because of the belief that it increased the toxicity of the product.

At the beginning of the 1930's, it was noticed that typhoid fever was more in evidence in the Army than it had been for a long time. Some attributed this to a loss of immunogenicity due to genetic aberrations in the organism. In 1933, Seckinger[10] published a report discussing the growing ineffectiveness of the vaccines produced from the old Rawlings strain which Russell had brought

[9] Dunham, George C.: Military Preventive Medicine. 3d ed. Harrisburg: Military Service Publishing Co., 1940, p. 163.

[10] Seckinger, D. L.: Epidemiological Studies Upon Typhoid Fever in Georgia. Problems Associated With Its Control. South. M. J. 26: 933-941, November 1933.

back from England in 1908.[11] On 4 October 1936, the Panama strain of *Sal. typhosa*, known as "Boxill" and "Chronic Carrier" and designated as "Strain 58" by the Army Medical School, was adopted for vaccine production, and it has been the one used throughout the Army from that time.[12]

Throughout this decade, the typhoid rate remained below the level of 0.10 per 1,000 average strength with the exception of 1931, when 18 cases occurred at Fort Des Moines, Iowa, in a command in which proper hygiene was not enforced. In this instance, all personnel had received the required vaccination some months previously, but heavily polluted well water was drunk with no attempt made at purification. Paratyphoid fevers were rarely encountered during this decade. According to Siler, 15 cases in all were reported; 3 of paratyphoid A, 11 of paratyphoid B, and 1 unclassified. On 1 August 1934, it was decided to remove the paratyphoid A fraction from vaccines for much the same reasons the paratyphoid B portion had been eliminated in 1928.

During the period 1933–39, some of the most important advances in the development of the understanding of typhoid epidemiology were made as a result of experience with the Civilian Conservation Corps. The organization had enrolled during its existence approximately two and one-half million youths who served for periods of 6 months or more in various camps throughout continental United States. Immunization to typhoid fever was routinely carried out by the United States Army Medical Department immediately subsequent to enrollment. Sanitary measures were applied to food, water, and the removal of waste.

Outbreak of typhoid fever in a partially immunized and nonimmunized group.—Despite the precautionary measures usually taken, outbreaks did occasionally occur. One such outbreak occurred at Ely, Minn., in 1934. One hundred and forty men were vaccinated at Fort Leavenworth, Kans., with the standard Army initial dose, followed a week later by the second inoculation. The unit was shipped to Ely, and, 10 days following arrival at camp, a high percentage came down with typhoid fever. In the cases that were clinically observed, the diagnosis of typhoid fever was confirmed by the laboratory of the Minnesota State Health Department. At this camp, there were a number of workmen of approximately the same age group as the Civilian Conservation Corps members, and many of them also contracted the disease. Eleven deaths occurred among the workmen, the course of the disease running for a longer time and with a greater severity than in the case of the immunized Civilian Conservation Corps personnel, all of whom recovered. Investigation

[11] (1) Grinnell, F. B.: A Study of the Comparative Value of Rough and Smooth Strains of *B. typhosus* in the Preparation of Typhoid Vaccines. J. Immunol. 19: 457–463, November 1930. (2) Perry, H. M., Findlay, H. T., and Bensted, H. J.: Antityphoid Inoculation. The Role of *Bacterium typhosum*, Strain Rawlings. J. Roy. Army M. Corps 60: 241–254, April 1933. (3) Perry, H. M., Findlay, H. T., and Bensted, H. J.: Antityphoid Inoculation: Observations on the Immunizing Properties and on the Manufacture of Typhoid Vaccine. J. Roy. Army M. Corps 62: 161–177, March 1934. (4) Perry, H. M., Findlay, H. T., and Bensted, H. J.: Antityphoid Inoculation: Observations Relative to the Immunological Value of Different Cultures of "*Bacterium typhosum*." J. Roy. Army M. Corps 63: 1–12, July 1934.

[12] See footnote 7, p. 464.

revealed that the camp's water supply was contaminated with coliform bacteria, indicating a fecal contamination. It was discovered that blasting operations conducted in preparation of the camp had opened fissures in the rock strata, allowing seepage directly from the latrines to the water source.

This outbreak serves to add to the evidence that immunization is not entirely protective when large numbers of the causative organisms of typhoid fever are ingested, but such immunization does lower the mortality of the disease and check the seriousness and duration of the illness.

Generally speaking, between World War I and 1939, the Army had learned through its own experience, civilian health authorities, and the Civilian Conservation Corps, that sanitary precautions were as necessary in times of peace as in war. It had found that only through observance of strict water purification, inspection of foodstuff, examination of foodhandlers, control of human carriers, and continued immunization of individuals could typhoid and paratyphoid fevers become a disease of minor importance.

Table 78 furnishes an over-all view of typhoid and paratyphoid fevers in the Army from 1900 to 1941. It shows the number of admissions or cases per annum per 1,000 average strength for each year during that period. It

TABLE 78.—*Occurrence of typhoid and paratyphoid fevers in the U. S. Army, 1900–41*

[Rate expressed as number of cases per annum per 1,000 average strength]

Year	Incidence [1]	Rate	Year	Incidence [1]	Rate
1900	978	9. 74	1921	11	0. 05
1901	594	6. 42	1922	2	. 01
1902	565	6. 99	1923	7	. 05
1903	348	5. 14	1924	4	. 03
1904	293	4. 35	1925	4	. 03
1905	206	3. 14	1926	9	. 07
1906	373	5. 72	1927	12	. 09
1907	237	3. 79	1928	12	. 09
1908	239	3. 20	1929	8	. 06
1909	288	3. 43	1930	10	. 07
1910	201	2. 47	1931	24	. 18
1911	72	. 87	1932	8	. 06
1912	31	. 35	1933	9	. 07
1913	10	. 11	1934	8	. 06
1914	18	. 19	1935	4	. 03
1915	17	. 16	1936	4	. 02
1916	506	2. 87	1937	15	. 09
1917	317	. 47	1938	13	. 07
1918	875	. 35	1939	7	. 04
1919	553	. 55	1940	33	. 10
1920	14	. 07	1941	27	. 02

[1] From 1900 through 1923, data presented represent admissions only. Incidence data are not available for these years.

Source: Annual reports of The Surgeon General.

can be seen that the annual rate for typhoid and paratyphoid combined was reduced to a remarkably low level by 1920 and maintained there until 1931 when there was a slight increase.

WORLD WAR II EXPERIENCE WITH TYPHOID AND PARATYPHOID FEVERS

Control Measures

By 1940, standard regulations had become well established from actual experience gained in previous military operations in war and peace. These rules were described in Army regulations.[13] Generally, they provided for the responsibility of commanders in regard to housing; water supplies; waste and garbage disposal; food and drink preparation; cleansing of dishes, messkits, and utensils; and examination of permanent foodhandlers. The Medical Department was charged with investigation of sanitary conditions, checking of water supplies, recommending types of water purification, and making periodic surveys of the potability of water.

Methods of water purification were the standard settling, filtration, and chemical (chlorine) processes, all water being considered impure until declared potable by the proper medical officer. In the field, the water sterilizing bag was to be used with calcium hypochlorite treatment, and, in the absence of sterilizing bags, the use of galvanized iron cans was authorized. In addition, units for individual use such as halazone tablets were to be available with proper instruction in their use. The men were advised to boil the water for 1 minute whenever chemical agents were not available.

All individuals known to be or suspected of being ill with any of the intestinal diseases were to be hospitalized unless recommended otherwise by the surgeon. Once in the hospital, the men were isolated and treated with due respects to body wastes, bedding, and linen. All cases of these diseases were investigated to ascertain, if possible, the source and route of infection.

Carriers.—No discussion of typhoid or paratyphoid fever would be complete without a discussion of the problem of carriers. According to Nichols,[14] carriers may be classified in three categories, the names of which are self-explanatory: Incubationary, convalescent, and contact. During the World War I period, it was the general policy in the United States to send all Army typhoid carriers to Walter Reed General Hospital, Washington, D. C. There it was felt that surgery was indicated in certain cases. Occasionally such procedure effected a cure, though in general it was not considered to be indicated and many chronic carriers were never cured.

During World War II, known carriers of the causative organisms of these diseases were excluded from food handling, and regulations were established for keeping close contact with their assignments and movements. Com-

[13] Army Regulations No. 40-205, 31 Dec. 1942, and No. 40-210, 15 Sept. 1942.

[14] Nichols, Henry J.: Carriers in Infectious Diseases. Baltimore: Williams and Wilkins Co., 1922.

manders were notified whenever carriers were to be transferred to their commands. Reports were made to The Surgeon General of any individual who retained his carrier state for more than 6 months. Through close cooperation with certain State boards of health, known typhoid and paratyphoid carriers were detected and reported to The Surgeon General by State health authorities. No provisions were made, however, to exclude or discharge carriers from the Army, although this problem had been discussed at length by members of the Army and the National Research Council.[15] Known carriers were placed on duty not involving handling of food or other materials that might be contaminated.

World War II did not bring medical science closer to a solution of the typhoid carrier problem than did World War I. During the second world conflict, the possibilities of antibiotic therapy in such cases had not been envisioned. Following the war, antibiotics such as Chloromycetin (chloramphenicol) were used but with little or no effect.

Preliminary data on admissions for typhoid and paratyphoid carriers, based on sample tabulations of individual medical records, are available for the years 1942–45 as shown in table 79.

Immunization.—The immunization policy of the Army directed that all military personnel on active duty receive routine immunizations. After

TABLE 79.—*Admissions for typhoid and paratyphoid carriers in the U. S. Army, by area and year, 1942–45*

[Rate expressed as number of admissions per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate
	Typhoid carriers									
United States	26	0.00	6	0.00	15	0.00	5	0.00	.....	0
Overseas	12	.00	..	0	5	.00	7	.00	.....	0
Total Army	38	.00	6	.00	20	.00	12	.00	.....	0
	Paratyphoid carriers									
United States	47	0.00	3	0.00	15	0.00	9	0.00	20	0.01
Overseas	125	.01	2	.00	11	.01	82	.02	30	.01
Total Army	172	.01	5	.00	26	.00	91	.01	50	.01

[15] Memorandum for file, Capt. P. E. Sartwell, 21 Aug. 1944, subject: Conference With Professional Service on Typhoid Carriers.

1 September 1940, the paratyphoid fractions were reinstated, and the prescribed course included three 1 cc. subcutaneous injections of triple typhoid vaccine given approximately 3 years after the initial immunization. Only two complete series were required for any individual except, where deemed advisable by the medical officer concerned, in such cases as (1) outbreaks of the disease; (2) when personnel were assigned to a theater of operations; and (3) upon boarding an Army transport or airplane for travel beyond the limits of the United States, unless vaccination had been completed within 12 months prior to departure. Individuals over 45 years of age were not required to complete revaccination unless they were leaving the United States.

Special measures.—Special measures were executed during epidemics of any intestinal disease. Extra efforts were made to detect and hospitalize early cases and to detect and restrict carriers. Suspected common sources of infection (such as milk, water, and food) as indicated by epidemiologic study were excluded from use by the command until the proved cause or causes of the epidemic had been discovered or until adequate corrective measures had been applied to the suspected source or sources. Special attention was given to the examination of foodhandlers, sanitation of kitchens, mess, garbage-disposal methods, latrines, and the control of flies. Close cooperation with civilian health authorities in determining sources of infection was established wherever indicated.

Other required sanitation measures included compliance with local regulations regarding sewage disposal, collecting and processing of liquid wastes from kitchens and bathrooms, avoidance of water and stream pollution, analysis of sewage, and control of flies.

In the summer of 1942, The Surgeon General, recognizing the need for preventive measures in the control of typhoid and paratyphoid as well as other diarrheal diseases, consulted with the National Research Council. The recommendations of the National Research Council were later incorporated in Circular Letter No. 33, Office of the Surgeon General, dated 2 February 1943, Treatment and Control of Certain Tropical Diseases, a copy of which was furnished to each officer of the Medical Department. The circular advocated various preventive measures including the supervision of milk and water supplies and the protection of foods from flies and carriers. Fly breeding was to be combated at all times. All foods in which bacteria could multiply were to be refrigerated at 40° F. or less. Oysters were not to be served raw unless known to be from a safe source. Also prescribed were sanitary disposal of excreta, the isolation of known cases of the disease with epidemiologic investigation of sources of infection, and continuation of the standard policy of vaccinating all personnel with triple typhoid vaccine.

The preventive medicine officer in each of the major theaters of operations established policies and procedures for prevention of intestinal infections, and port authorities—both sea and air—made every effort to apprehend and place under medical supervision each individual suspected of harboring a contagious disease. This was perhaps more effective in the European theater than in

other areas because medical facilities were fairly well established before large numbers of troops had arrived. Special epidemiologic reports were devised for typhoid which were forwarded to The Surgeon General when completed. In many cases these were not conveniently accomplished, the patient and the medical officer both being far removed from the source of infection.

Special methods for control of typhoid, paratyphoid, and other intestinal diseases included culturing of stools for the causative organism in all cases of diarrhea. This was feasible, even in theaters of operations, because laboratory service was available in most instances at hospitals, Army laboratories, and when necessary in regular public health laboratories. A period of 4 days between the time of specimen collection and the time of culturing was permitted for transit. Dish culturing was instituted throughout the various service commands of the Army for the purpose of periodically evaluating the thoroughness achieved in mess sanitation. Laboratories and laboratory technicians were evaluated by proficiency studies whereby known specimens were forwarded by the various service command laboratories thereby promoting skill in the laboratory detection of pathogenic organisms.

Incidence in the Army

In the initial phase of mobilization, the incidence of typhoid fever was no higher in the Army in the United States than in the 2 previous years of 1938 and 1939. There were 3 reported cases of typhoid fever in home troops in 1940 and 12 cases in 1941. The annual rates per 1,000 average strength for typhoid fever in the United States (among enlisted men only) were 0.02 for 1938, 0.02 for 1939, and 0.01 for 1940. In 1941, the rate was 0.01 indicating that control measures were effective during a period of inducting large numbers of troops from every section of the country.[16]

No significant difference in typhoid incidence among seasoned troops as opposed to fresh recruits was noted nor did any epidemics or sporadic occurrences take place among new inductees at induction and basic training centers. Paratyphoid fever was similarly of no great importance, though one minor epidemic was reported to have occurred at Camp Claiborne, La., in October 1941, when seven cases were reported to have been discovered. There are no records to indicate that this ever received official diagnostic confirmation. As the etiologic agent responsible had not been previously encountered, it was named *Salmonella claibornei*.

The history of typhoid and paratyphoid is different in the various theaters depending upon the level of sanitation of the areas concerned. Thus, in 1944, there were 25 cases of typhoid in the European theater (12 of which were brought in from North Africa) with a case rate of less than 0.1 per 1,000 troops against 38 cases reported in the China-Burma-India theater where a case rate of 0.23 per 1,000 troops per annum existed. The cases can be traced to breaches

[16] Holt, R. L.: Current Status of Immunization Procedures. Typhoid Fever. Am. J. Pub. Health 38: 481-484, April 1948.

of water discipline, food from unauthorized sources, or human carriers. In the China-Burma-India theater, local conditions were unhealthy, there was little or no enforcement of sanitary laws, and deplorable facilities existed for the disposal of garbage and human excreta.

Strange as it may seem, it was frequently found that, regardless of the theater, troops actually engaging in combat were less disposed to acquire typhoid and paratyphoid infections than supporting, reserve, or resting troops. While military control was not wanting at such places as rest camps, the opportunity for contact with native food and water facilities existed, and it was the belief of some of the United States troops that consumption of such commodities could lead to no ill effects. Thus, overconfidence in the protection conferred by vaccination may have led some Americans to believe they were completely immune to typhoid and paratyphoid fevers. One may note at this point that in many cases, especially in those that were of a sporadic nature, the source of infection was never determined.

The incidence (total cases) of typhoid fever in the United States and in overseas theaters and areas during World War II is shown in table 80. Table 81 shows the incidence of paratyphoid fever. A comparison of the two tables shows that the Army was somewhat less successful in controlling paratyphoid

TABLE 80.—*Incidence of typhoid fever in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on individual medical record sample tabulations of primary and secondary diagnoses]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	91	0.01	20	0.01	25	0.00	16	0.00	30	0.01
Overseas:										
Europe	64	.01	1	.01	3	.01	25	.01	35	.01
Mediterranean [1]	135	.09	1	.04	83	.18	46	.07	5	.01
Middle East	23	.16	2	.33	12	.23	9	.19		0
China–Burma–India	78	.18	5	.57	25	.63	38	.23	10	.05
Southwest Pacific	73	.04	1	.01	18	.09	4	.01	50	.05
Central and South Pacific	28	.02	1	.01	3	.01	9	.02	15	.04
North America [2]	3	.01	1	.01	1	.01	1	.01		0
Latin America	10	.03	4	.04	5	.04	1	.01		0
Total overseas	414	.04	16	.03	150	.09	133	.03	115	.02
Total Army	505	.02	36	.01	175	.03	149	.02	145	.02

[1] Includes North Africa.
[2] Includes Alaska and Iceland.

TABLE 81.—*Incidence of paratyphoid fever in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on individual medical record sample tabulations of primary and secondary diagnoses]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	125	0.01	19	0.01	35	0.01	41	0.01	30	0.01
Overseas:										
Europe	84	.02	2	.02	2	.01	20	.01	60	.03
Mediterranean [1]	212	.14	1	.04	141	.31	45	.07	25	.07
Middle East	34	.23	2	.33	13	.25	9	.19	10	.24
China-Burma-India	96	.22	1	.11	8	.20	37	.22	50	.23
Southwest Pacific	183	.10	1	.01	18	.09	24	.04	140	.14
Central and South Pacific	33	.03	3	.02	4	.01	6	.01	20	.05
North America [2]	1	0	1	.01	-----	0	-----	0	-----	0
Latin America	70	.18	7	.07	49	.41	9	.10	5	.07
Total overseas [3]	714	.07	18	.03	236	.14	150	.04	310	.07
Total Army	839	.03	37	.01	271	.04	191	.02	340	.04

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admission on transport in 1943.

fever than in controlling typhoid fever, although its program was, on the whole, efficient and admirable.

Experience during 1942.—By 1942, a large number of troops had been shipped to various theaters of operations, all immunized in accordance with existing regulations and instructed in sanitation procedures for field service. During the year, 36 cases of typhoid fever were reported for the total Army, 16 from overseas areas. In Latin America (Panama and the Antilles) and the China-Burma-India theater, admissions totaled 4 and 5, respectively, or 0.04 and 0.57 per annum per 1,000 troops. Although these commands reported the highest number of admissions, the case rate in the Middle East theater was relatively high, 0.33 per annum per 1,000. Only one case was recorded in the European theater, from Northern Ireland, but data concerning the source and mode of transmission in this case are obscure.[17]

The incidence of paratyphoid fever in 1942 was about the same as that of typhoid fever, totaling 37 admissions, 18 of which were from overseas. The case rate for the total Army was 0.01 per 1,000 troops. Two cases were reported from the Middle East Command, one in September and one in October. The

[17] Gordon, J. E.: A History of Preventive Medicine in the European Theater of Operations, U. S. Army, 1941–45. [Official record.]

only other locality to report more than 5 cases was Latin America with 7. It is readily discernible from this account that soldiers stationed in areas of good local sanitation, such as the United Kingdom, were no more likely to contract the disease than troops in the United States, the case rate per 1,000 men being approximately the same. In the Middle East and the Antilles Department, clinical and subclinical paratyphoid and other enteric fevers were rampant in the native population. Necessarily, Americans were exposed to local conditions and, unknowingly or carelessly, consumed highly contaminated foods and water. Considering at the same time the excellent preventive measures practiced in the Latin American theater, where the case rate for paratyphoid fever was 0.07 per 1,000 troops per annum, it is apparent that adequate military control can be exercised to prevent enteric fevers even in communities where natives are suffering from the disease.

Experience during 1943.—In the year 1943, United States troops were widely distributed all over the world. Large numbers of soldiers were engaged in active combat operations in North Africa, Sicily, Italy, and throughout the Pacific Ocean area on isolated islands. They were stationed in northern regions such as Alaska, the Aleutian Islands, Iceland, Greenland, and Newfoundland, as well as in built-up areas in the United Kingdom, the Persian Gulf, Burma, China, Australia, New Zealand, India, and Hawaii. Air installations were in remote and farflung places such as the Azores, the Caribbean Islands, South America, and the Pacific. There were reported 175 cases of typhoid fever throughout the Army, 150 of which were from overseas, for a total Army case rate of 0.03 per 1,000 troops. In the Mediterranean theater, there were 83 cases, partly associated with an outbreak in a prisoner-of-war camp in North Africa during October, November, and December, the case rate being 0.18 per 1,000 troops. The Middle East theater reported 12 cases and a rate of 0.23 per 1,000 troops.

Eighteen cases were known in the Southwest Pacific, records not being available to state the exact locations. Twenty-five cases were reported from the China-Burma-India theater for a case rate of 0.63 per 1,000 average strength, the highest incidence throughout the Army during the year.

Paratyphoid fever was more prevalent than typhoid, with 271 cases reported of which 236 occurred in overseas commands. Of the various overseas areas, the Mediterranean area had 141 cases, though the area where the annual rate was highest was Latin America. Only 13 cases were reported from the Middle East, but the case rate of 0.25 per 1,000 troops was third highest throughout the Army. The China-Burma-India theater annual rate was significantly high at 0.20 per 1,000 troops, although there were only 8 admissions. The Southwest Pacific Command reported 18 cases, with a rate of 0.09 per 1,000, again omitting names of countries or islands involved. For the total Army, including the United States, paratyphoid fever was evidenced in only 0.04 per 1,000 average strength.

Experience during 1944. In 1944, typhoid fever cases ranked second in the World War II years with a total of 149 and an annual rate per 1,000 average

strength of 0.02. The majority of these cases were overseas where the case rate reached 0.03 per 1,000 strength. The highest number of cases was in the Mediterranean theater where 46 were reported, but the rate was exceeded in the China-Burma-India theater with 0.23 for 38 cases. In the Southwest Pacific, there were only four cases with a rate of 0.01

An interesting outbreak of typhoid fever occurred during the year (1944) in Company G, 349th Infantry in Northern Italy. In November and December, 19 members were admitted to hospital and diagnosed as having typhoid fever. One man, who had become aware of symptoms 30 days prior to reporting sick, died in a toxic state 10 days after being admitted. Although an extensive investigation was carried out to determine the source of the infection, no definite conclusions could be reached. Possible reasons for the outbreak were given as (1) indiscriminate drinking of water from wells, shellholes, and other places without using halazone or other purification methods (some patients explaining that they had been issued halazone tablets only once and after they were used, no reissue was made); (2) consumption by some soldiers of civilian food, including potatoes and salami, though there were no samples of the food available for examination; and (3) presence in the unit of individuals with subclinical typhoid fever. The outbreak followed a carrier pattern, 2 individuals from other units acquiring the disease after eating one or two meals with Company G, but the possibility of a carrier was not confirmed.[18]

A decrease in paratyphoid fever was noticed in 1944, with 191 cases for the total Army of which 150 were from overseas commands. *No command* suffered a serious outbreak, and the rate of 0.02 per 1,000 troops per annum was half the 1943 rate. The Mediterranean theater had the highest number of admissions, while China-Burma-India experienced the highest rate of the various theaters (0.22 per annum per 1,000 average strength).

Experience during 1945.—By 1945, although a great number of troops were in the field, the total admissions were slightly lower than the previous year with 30 cases in the continental United States and 115 overseas. The case rate, however, remained constant at 0.02 per 1,000 per annum for the total Army, with a slight increase to 0.01 in the United States. The overseas rate dropped to 0.02 per 1,000 troops as compared with 0.05 in 1944. The largest number of admissions was reported from the Southwest Pacific, a total of 50 being recorded. There were no cases of typhoid in the Middle East, and the European theater showed 35 cases with a rate of 0.01 per 1,000. The China-Burma-India theater continued to show a comparatively high rate incidence. All other theaters had few admissions and no specific outbreak is recorded. It is to be noted, however, that the Central and South Pacific areas had a rate of 0.04 per 1,000 average strength.

Paratyphoid fever was twofold higher in incidence than the previous year with 340 cases, case incidence throughout the Army rising to 0.04 per 1,000 average strength. Overseas commands contributed 310 cases with a

[18] Bruner, D. W.: Salmonella Infections of World War II. [Official record.]

rate of 0.07. The Southwest Pacific reported 140 cases with a rate of 0.14. The Pacific Ocean area, including the Central and South Pacific, reported 20 cases, but in view of the large number of troops involved the rate amounted to 0.05.

Comparative incidence of typhoid and paratyphoid fevers and diarrheal diseases. Since the epidemiology of the diarrheal diseases is similar to that of typhoid and paratyphoid fevers, it is of interest to show comparative incidence of the diseases (table 82). It is generally considered that neither typhoid fever nor paratyphoid fever was a particularly serious problem to the Armed Forces in either World War I or World War II. For the two world conflicts, these diseases were even less of a problem in the second than they were in the first war. While this may not be altogether apparent upon cursory inspection of these statistics, it is readily acknowledged when it is considered that many more men were under arms in the Second World War than in the First. It becomes clear on examination of the rates, especially when dealing with preliminary figures, that they are a more reliable index of incidence than are the absolute numbers of cases reported. The diarrhea-dysentery problem in World War II was at least as serious as in World War I. This lends support to the belief that typhoid-paratyphoid vaccine played a tremendously important role in the control of typhoid and paratyphoid fevers. In the occurrence of diarrheal diseases and dysentery, no such reduction took place since no effective vaccine for these diseases has ever been perfected.

TABLE 82. *Admissions for typhoid and paratyphoid fevers and diarrhea and dysentery, by area, World War I and World War II*

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	Typhoid fever		Paratyphoid fever		Diarrhea and dysentery	
	Admissions	Rate	Admissions	Rate	Admissions	Rate
World War I						
United States	546	0.24	43	0.02	39,854	17.83
Overseas	983	.52	169	.09	52,658	27.82
Total	1,529	.37	212	.05	92,512	22.41
World War II						
United States	86	0.01	120	0.01	296,176	20.43
Overseas	379	.04	649	.06	481,072	44.81
Total	465	.02	769	.03	777,248	30.50

Includes gastroenteritis.

Source: World War I: Data taken from The Medical Department of the United States Army in the World War, vol. XV, pt. II. World War II: Data are tabulations of individual medical records.

Incidence in Civilian Populations

The fact that United States troops were relatively free from typhoid fever should not be interpreted to indicate that there was little opportunity for exposure. The disease, when it did occur in military personnel, often did so against a background of it in civilian populations, the latter having been far less immune to typhoid fever than were the Armed Forces of the various nations engaged in combat. The disease was prevalent throughout war-ridden Europe, especially in France, Belgium, Holland, and Germany. Figures for typhoid in Russia are not available but were probably substantial. By way of illustration, the city of Saint-Étienne, France, had 570 cases with 27 deaths in the autumn of 1944. At Greater Liége, in Belgium, there were 104 cases from 1 August to 18 November 1944. Thus, civilian populations were always a potential source of infection.

Incidence Among Prisoners of War

During the early years of the war, prisoners of war had an excellent record with respect to typhoid and paratyphoid fevers. A minimum of sanitation seemed to suffice in maintaining the low rates. It was soon learned that the German Army had been perhaps as effectively immunized as the United States Army. However, under certain conditions such as extreme deficiencies in sanitation, immunity proved *inadequate. It is for this reason* that most epidemiologists believe that the control of typhoid fever is basically more a matter of sanitation than of vaccination. Another set of conditions that brought about an increased typhoid rate in prisoners of war was the large-scale breakup and disruption of the German Army. In May and June 1945, there were 482 cases of typhoid fever in German prisoners of war. Chart 48 shows the incidence of typhoid fever among a sample of 415 prisoners of war having typhoid fever during May and June 1945 in the European theater.[19]

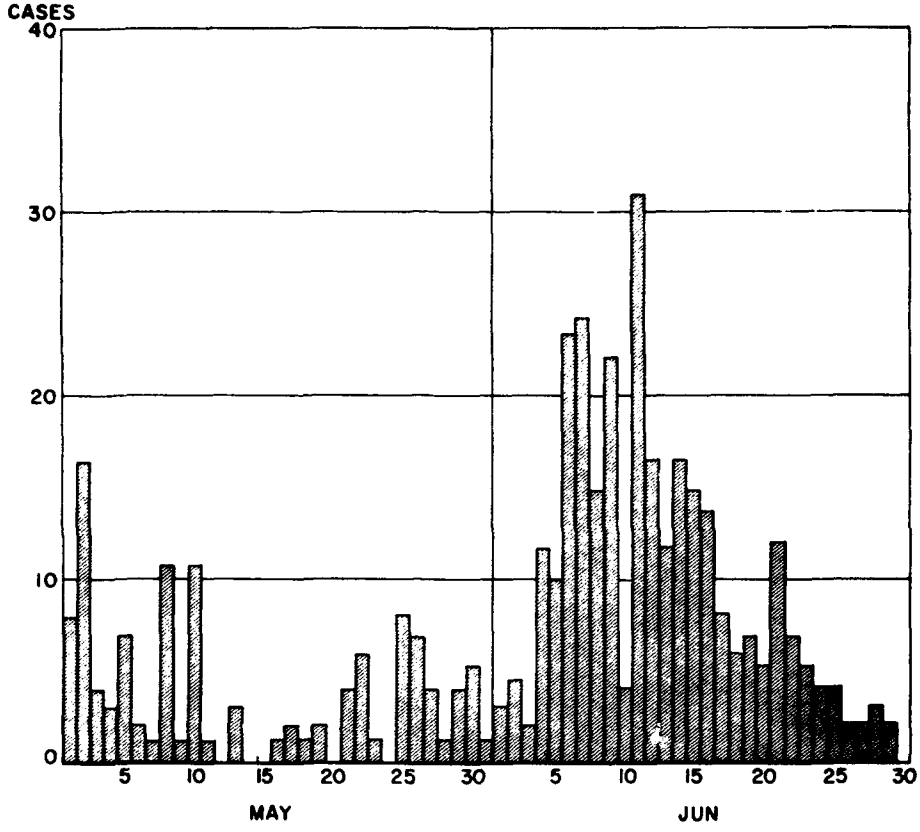
A marked difference can be seen in chart 48 between May and June and even between consecutive days. Not all of the infections were in newly captured prisoners. Many of them occurred in prisoners who had been held for an appreciable period of time. In the Advance Section, Communications Zone, among 403,142 prisoners of war, there were 30 deaths from typhoid in the 6-week period from 1 May to 15 June 1945. It is to be noted that it was not possible to give the prisoners of war the best medical care, and it is not unreasonable to hypothesize that typhoid cases among prisoners of war may not always have been correctly diagnosed.

In certain instances, it was quite possible to trace the cases in prisoners of war. Prisoners as a rule were held for a few days together with other prisoners not too far from the frontlines. They were then moved to the rear to a collecting point and again moved further back to larger camps. Some of the collecting points were strongly suspected of being the point of origin of the

[19] See footnote 17, p. 473.

CHART 48.—*Typhoid fever among 415 prisoners of war in European theater, May and June 1945*

[Distribution by day of admission to hospital]



infection. The following report from a prisoner-of-war camp illustrates some of the symptoms of the disease as it occurred among prisoners of war:

Epidemiologic case report.—Prisoner-of-War Enclosure No. 11 was a collecting point in Normandy Base Section for prisoners of war captured in Germany. Three prisoners received on 6 June 1945 were ill on arrival with a disease characterized by severe headache, prostration, and high fever. The next day, 9 more men reported ill, and thereafter daily additions were made to the roster of patients with a similar disease, so that by 25 June 1945 the number was 101. About a third of the patients had a rash of the abdomen, not affecting the extremities, face, or back, and the fever was typhoidal in type. The infectious process was confirmed by laboratory examinations as typhoid fever in a significant proportion of patients and by pathologic examination after death in a number of instances. Most of the prisoners had been captured in Germany about the first of May. They had been held in various enclosures in Germany and then forwarded to the Normandy concentration point. Those with typhoid fever were in large part from PWTE, A7, near Welgesheim, but a number of different enclosures were represented. In general, the prisoners had left Germany in the early days of June and had arrived in Normandy between 4 and 8 June 1945. There had been no previous typhoid fever in the Normandy camp, and the period of incubation would indicate that in most instances infection had been acquired after capture and in prisoner-of-war enclosures near the frontlines.

As for the Pacific Ocean Area and the China-Burma-India theater, there were no reports of typhoid or paratyphoid among American prisoners of war.

Undoubtedly, there must have been some cases, but they were either undiagnosed or unreported. It is known that there was a high rate of diarrhea in the Philippines following the fall of Bataan and in the American prisoner-of-war camp in the Asiatic area. When the Japanese conquered the Philippine Islands, the Japanese Army Medical Department took over a Philippine vaccine-production laboratory and produced a vaccine that contained typhoid, cholera, and dysentery bacteria antigens. The typhoid and cholera components may have been effective. It is doubtful whether the dysentery component was effective, although the death rate in infants with dysentery was reported to be lowered.

On Bataan, in the reports of the patients hospitalized, there is no record or mention made of true cases of typhoid fever. In 1945, the senior author of this chapter observed 3,000 sick Japanese prisoners of war at the new Bilibid Prison in Manila. Although there were many cases of bacillary dysentery and also amebic dysentery, there were few if any cases of typhoid fever among the prisoners.

ADVANCES IN CONTROL DURING WORLD WAR II

During and following World War I, the safeguarding of water and food supplies had approached and attained so high a standard that there developed a tendency to relegate its importance to secondary consideration. In World War II, in order to correct this tendency, Army engineers were given greater responsibility in such aspects as the location and appraisal of water supplies and the proper treatment of water, including chlorination, while the role of the Medical Department became more advisory in nature.

Laboratory Techniques

Constant supervision of laboratory technicians was achieved by evaluation studies that had been initiated at the beginning of the war in the Fourth Service Command and adopted as standard procedure. Through studies of results obtained by the laboratories under evaluation, supervisory laboratories were able to determine and direct necessary training. Thus, the early diagnosis of diseases dependent on laboratory results was greatly aided by increasing the proficiency of technicians.

Dish culturing.—A constant evaluation of mess sanitation was maintained by the periodic culturing of dishes, utensils, and other culinary apparatus, as well as by directing better sanitary practice wherever and whenever indicated.

Detection of typhoid and paratyphoid fevers.—World War II brought with it several improvements and refinements in the techniques of the detection and confirmation of typhoid and paratyphoid fevers through laboratory methods. The main advance in this connection has been the introduction of new media for culturing typhoid and paratyphoid organisms, a field in which Hardy and

Watt [20] were among the first important workers. It has been said that the reason for the supposedly low incidence of paratyphoid in the continental United States during the decade 1930–40 was because effective techniques for the isolation of the organisms had not as yet been developed. The use of S.-S. (*Shigella-Salmonella* thiosulfate-citrate-bile) agar, modified brilliant-green agar, sodium selenite, and tetrathionate broths are examples of the progress made in the development of differential media for the growth of the various *Salmonella* species.

There was no marked change in the routine serologic diagnostic methods, although a great step forward was made when the Kauffmann-White Schema was introduced with the resulting simplification. The ultimate diagnosis of the infections from *Salmonella* organisms (including *Salmonella typhosa*) during World War II depended on the classical standby method of culturing blood, urine, and stools of the patient.

Controlled examination of water.—In addition to local examination of water supplies, it was found that more constant results could be obtained by submission of water samples to central Army laboratories where highly trained technicians could perform the necessary examinations with a higher degree of accuracy than could be done by smaller laboratories not completely equipped for such procedures. Acting from a central location where complete results of examinations were known, personnel charged with water sanitation could more readily direct such measures of purification as were necessary. The basic reason for this centralization was the need for greater administrative rather than technical improvement.

Routine culturing of stools.—The practice of culturing stool specimens in all cases exhibiting diarrhea proved to be a valuable one, and many cases of salmonellal infections were detected in this manner which might otherwise have remained unnoticed. A point which may be mentioned at this time is the erroneous impression of some bacteriologists and even physicians that only formed stools are of value in culturing organisms of enteric and gastrointestinal diseases. One of the methods of improving the routine culturing of stools was found to be that of inoculating known specimens at regular intervals and letting the bacteriologists and technicians study the number of organisms present to produce any positive culture and diagnosis.

Carrier control and detection.—As has been mentioned earlier, little was accomplished in the therapy of typhoid and paratyphoid carriers. However,

[20] Hardy, A. V., Watt, J., and DeCapito, T. M.: Studies of the Acute Diarrheal Diseases. Pub. Health Rep. 57: 521-524, 10 April 1942.

vigilance was not relaxed, and great zeal was displayed in the finding and apprehension of carriers. The sulfonamide drugs were found to be ineffective, and more recently the antibiotics have proved ineffective in completely eliminating the organisms from carriers.

Mobile laboratories.—Mobile laboratories were tested and found to be of considerable value in field operations where fixed-laboratory service was nonexistent or simply not feasible. Immediate laboratory diagnosis of typhoid and paratyphoid fevers may prevent large outbreaks when sporadic infections occur.

Prophylaxis.—For the purpose of record, it is to be noted that there are two schools of thought on the merits of the Army's prophylactic vaccination. Some consider it most effective, while others attribute less value to it. The authors of this chapter accept the moderate viewpoint that while vaccination is effective on the whole, under extremely heavy infections the conferred immunity may break down. However, even in such cases, experience showed that the disease generally pursued a milder course in "immunized" persons. Thus, as is the case in a number of other diseases, vaccination does not provide absolute immunity to typhoid and paratyphoid fevers for the artificially induced immunity is overcome when organisms in sufficiently high numbers are introduced into an individual. Indeed, most of the recorded cases of typhoid and paratyphoid fevers occurred in immunized personnel. Rarely was it contracted prior to induction. Nevertheless, the mortality rates do indicate that the virulence of the disease was indeed modified.

The occurrence of typhoid fever in "immunized" individuals in the Army is shown in table 83 formulated from data obtained by Lt. Col. (later Col.) Arthur P. Long, MC, who collected data during the war on immunized personnel who had developed the disease.[21]

Effective immunization against typhoid fever has been a problem to all armies and for that reason many nations have developed vaccines, often with considerable success, as for example, TAB endotoxoid which was used so successfully in South America. Considering the low rate of 0.02 per 1,000 troops during World War II, it is perhaps not unreasonable to suggest that despite the many modifications and variations in typhoid vaccines thus far devised, the United States Army vaccine probably is as effective as has been developed anywhere.[22]

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[21] The Medical Department of the United States Army. *Preventive Medicine in World War II, Personal Health Measures and Immunization.* Washington: U. S. Government Printing Office, 1955, vol. III, ch. VIII.

[22] Batson, H. C.: Typhoid Fever Prophylaxis by Active Immunization. Pub. Health Rep. Supp. 212, August 1949.

TABLE 83.—*Immunization status of 42 cases of typhoid fever among Army personnel in the United States, 1942–45* [1]

Immunization record	1942	1943	1944	1945	Total
Typhoid vaccine received:					
Basic series completed, stimulating dose(s) given	----	1	5	6	12
Basic series completed, no stimulating doses	4	9	3	2	18
Basic series not completed	6	1	1	1	9
No vaccination	1	----	1	1	3
Total	11	11	10	10	42
Number of stimulating doses given:					
1 dose	----	1	5	2	8
2 doses	----	----	----	3	3
3 doses	----	----	----	----	----
4 doses	----	----	----	1	1
Total	----	1	5	6	12
Interval from last dose to onset of disease: [2]					
Less than 7 days	----	----	1	1	2
1 week to 1 month	1	----	1	1	3
1 to 6 months	2	4	2	1	9
6 months to 1 year	1	5	1	2	9
More than 1 year [3]	----	----	3	3	6
Not stated	----	1	----	----	1
Total	4	10	8	8	30

[1] Cases (proved by positive cultures) on whom histories were received by Epidemiology Division, Office of the Surgeon General.

[2] For cases who had completed basic series.

[3] Of the 6 cases in this group, the interval was 12 to 15 months in 3 cases.

Parasitic Infections

CHAPTER XXIII

Amebiasis

Henry E. Meleney, M. D.

Before World War II, amebiasis had not been a serious problem in the United States Armed Forces, except during the Philippine Insurrection following the Spanish-American War and in one epidemic on the Mexican border in 1916. Attention has been called to these two episodes by Craig.[1] During World War I among American Armed Forces,[2] only 38 out of 934 laboratory tested cases of dysentery were proved to be of amebic origin. The only indication of acquisition of infection with *Endamoeba histolytica* overseas was furnished from a fecal survey at Debarkation Hospital No. 3 by Kofoid, Kornhauser, and Plate[3] in which they found 12.8 percent of 2,300 overseas troops infected, as compared with 4.3 percent of 576 troops who had not been overseas.

The British had considerable experience with amebiasis during World War I especially in the Gallipoli campaign and in the Middle East. This led to intensive studies of intestinal amebiasis of man and to the discovery of two previously unrecognized species, *Endolimax nana*[4] and *Dientamoeba fragilis*.[5] Within the next 20 years, surveys of the prevalence of amebiasis were made in many parts of the world including several in the United States, and Craig estimated that the over-all prevalence in this country was about 10 percent of the population with wide variations in different regions.

Many other important contributions to the knowledge of amebiasis were made between the First and Second World Wars. In 1925, Boeck and Drbohlav[6] devised a practical culture medium for *E. histolytica*, and this was improved upon by Dobell and Laidlaw.[7] In 1929, Craig[8] described a comple-

[1] Craig, Charles F.: The Etiology, Diagnosis, and Treatment of Amebiasis. Baltimore: Williams and Wilkins Co., 1944. (2) Craig, C. F.: The Occurrence of Endamebic Dysentery in the Troops Serving in the El Paso District From July 1916 to December 1916. Mil. Surgeon 40: 286-302; 423-434, March and April 1917.

[2] The Medical Department of the United States Army in the World War. Washington: Government Printing Office, 1926, vol. VI, p. 1101.

[3] Kofoid, C. A., and Kornhauser, S. I., and Plate, J. T.: Intestinal Parasites in Overseas and Home Service Troops of the U. S. Army; With Especial Reference to Carriers of Amoebiasis. J. A. M. A. 72: 1721-1724, June 1919.

[4] Wenyon, Charles M., and O'Connor, Francis W.: Human Intestinal Protozoa in the Near East. London: John Bake, Sons and Danielsson, 1917.

[5] Jepps, M. W., and Dobell, C.: *Dientamoeba fragilis*, a New Intestinal Amoeba From Man. Parasitology 10: 352-367, 1917-18.

[6] Boeck, W. C., and Drbohlav, J.: The Cultivation of *Endamoeba histolytica*. Am. J. Hyg. 5: 371-407, July 1925.

[7] Dobell, C., and Laidlaw, P. P.: On the Cultivation of *Entamoeba histolytica* and Some Other Entozoic Amoebae. Parasitology 18: 283-318, September 1926.

[8] Craig, C. F.: The Technique and Results of a Complement Fixation Test for the Diagnosis of Infections with *Endamoeba histolytica*. Am. J. Trop. Med. 9: 277-296, September 1929.

ment fixation test for amebiasis. Dobell [9] worked out the life cycle of *E. histolytica* in culture. Studies of pathogenicity were made in kittens [10] and dogs,[11] and the influence of associated bacteria and diet received attention. The Chicago epidemic of 1933 [12] emphasized the importance of contaminated water as a source of infection. Studies of water purification indicated that cysts of *E. histolytica* are more resistant to chlorination than are fecal bacteria.[13] Chemotherapy was improved by the introduction of carbarsone and the iodohydroxyquinoline compounds, chiniofon, Vioform, and Diodoquin. The zinc sulfate flotation technique for concentration of cysts in fecal diagnosis was introduced.[14]

INCIDENCE

General Considerations

The incidence of amebiasis in the Army included mild cases of amebic dysentery and amebic colitis as well as those with the classical picture of dysentery. Available statistics for the Army have been obtained from sample tabulations of primary and secondary diagnoses on individual medical record cards and include admissions for amebic dysentery as well as cases admitted for different diagnosis but in which amebic dysentery existed concurrently or developed subsequent to admission. Although dysentery is the most prominent clinical manifestation of amebiasis and is a good measure of its importance as a cause of noneffectiveness, it represents only a small proportion of the infections with *E. histolytica*. This is important because these infections are usually of long duration, probably many years, and are the source of spread to contacts under suitable circumstances. Furthermore, symptomless infections may give rise to clinical manifestations only after a long period of time.

Preliminary statistical data on the incidence (total cases) of amebic dysentery in the United States Army for the years 1942–45 by area, as calculated from samples of individual medical records, are presented in tables 84, 85, 86, and 87. These estimates, though subject to considerable sampling error, furnish comparative information of value concerning the relative importance

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[9] Dobell, C.: Researches on the Intestinal Protozoa of Monkeys and Man. II. Description of the Whole-Life-History of *Entamoeba histolytica* in Cultures. Parasitology 20: 365-412, December 1928.

[10] Meleney, H. E., and Frye, W. W.: The Pathogenicity of *Endamoeba histolytica*. Tr. Roy. Soc. Trop. Med. & Hyg. 29: 369-379, January 1936.

[11] Faust, E. C., and Kagy, E. S.: Studies on the Effect of Feeding Ventriculin, Liver Extract and Raw Liver to Dogs Experimentally Infected With *Endamoeba histolytica*. Am. J. Trop. Med. 14: 235-255, May 1934.

[12] Epidemic Amebic Dysentery: The Chicago Outbreak of 1933. Nat. Inst. Health Bull. No. 166, pp. 1-187, March 1936.

[13] Chang, S. L., and Fair, G. M.: Viability and Destruction of the Cysts of *Entamoeba histolytica*. J. Am. Water Works Assoc. 33: 1705-1715, October 1941.

[14] Faust, E. C., D'Antoni, J. S., Odom, V., Miller, M. J., Peres, C., Sawitz, W., Thomen, L. F., Tobie, J., and Walker, J. H.: A Critical Study of Clinical Laboratory Techniques for the Diagnosis of Protozoan Cysts and Helminth Eggs in Feces. Am. J. Trop. Med. 18: 169-183, March 1938.

TABLE 84.—*Incidence of amebic dysentery in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records of primary and secondary diagnoses]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1942-45		1942		1943		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	3,455	0.24	289	0.11	515	0.10	621	0.16	2,030	0.71
Overseas:										
Europe	1,637	.37	.....	0	37	.14	610	.36	990	.42
Mediterranean [1]	1,849	1.25	11	.48	340	.74	1,008	1.55	490	1.38
Middle East	1,171	8.01	29	4.80	601	11.33	391	8.46	150	3.67
China-Burma-India	10,351	23.61	66	7.55	626	15.80	4,809	28.50	4,850	21.91
Southwest Pacific	12,048	6.56	25	.35	83	.44	645	1.20	11,295	10.90
Central and South Pacific	3,097	2.46	17	.11	207	.71	1,118	2.55	1,755	4.67
North America [2]	24	.05	2	.02	5	.03	7	.05	10	.15
Latin America	269	.71	78	.77	32	.26	84	.98	75	1.03
Total overseas [3]	30,546	2.85	230	.39	1,934	1.15	8,677	2.27	19,705	4.24
Total Army	34,001	1.34	519	.16	2,449	.36	9,298	1.19	21,735	2.87

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

of amebic infection as a cause of noneffectiveness in the Army. The following observations on these figures are of interest:

1. The over-all rate of 1.34 per 1,000 average strength is low as compared with that of many other infections requiring medical attention.

2. The rate each year more than doubled the rate of the previous year.

3. The rate for troops in the United States was uniformly very low, increasing only in 1945 when many overseas troops had returned to this country.

4. Among the overseas theaters, the China-Burma-India theater had by far the highest rate, with the Middle East theater second, and the combined Pacific theaters third. The rates in the other overseas theaters never reached important proportions.

TABLE 85.—*Admissions for amebic dysentery in the U. S. Army, by area and year, 1944–45*

[Preliminary data based on sample tabulations of individual medical records]

[Rate expressed as number of admissions per annum per 1,000 average strength]

Area	1944–45		1944		1945	
	Number	Rate	Number	Rate	Number	Rate
Continental United States	1,981	0.29	481	0.12	1,500	0.51
Overseas:						
Europe	1,040	.26	395	.24	645	.27
Mediterranean [1]	998	.99	683	1.05	315	.89
Middle East	496	5.69	366	7.92	130	3.18
China-Burma-India	8,734	22.39	4,324	25.63	4,410	19.92
Southwest Pacific	8,865	5.63	420	.78	8,445	8.15
Central and South Pacific	2,393	2.94	978	2.23	1,415	3.77
North America [2]	12	.06	2	.02	10	.15
Latin America	129	.81	59	.69	70	.96
Total overseas [3]	22,757	2.69	7,232	1.89	15,525	3.34
Total Army	24,738	1.61	7,713	.99	17,025	2.25

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

TABLE 86. *Admissions for amebic dysentery carrier in the U. S. Army, by area and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records]

[Rate expressed as number of admissions per annum per 1,000 average strength]

Area	1942–45		1942		1943		1944		1945	
	Number	Rate	Number	Rate	Number	Rate	Number	Rate	Number	Rate
Continental United States	408	0.03	10	0.00	103	0.02	60	0.02	235	0.08
Overseas:										
Europe	125	.03		0	5	.02	85	.05	35	.01
Mediterranean [1]	103	.07		0	2	0	36	.06	65	.18
Middle East	104	.71		0	29	.55	55	1.19	20	.49
China-Burma-India	750	1.71	1	.11	5	.13	179	1.06	565	2.55
Southwest Pacific	706	.38		0	1	.01	20	.04	685	.66
Central and South Pacific	311	.25	1	.01	1	0	59	.13	250	.67
North America [2]	5	.01		0		0		0	5	.07
Latin America	83	.22		0		0	3	.03	80	1.10
Total overseas [3]	2,193	.20	2	0	43	.03	438	.11	1,710	.37
Total	2,601	.10	12	0	146	.02	498	.06	1,945	.26

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

TABLE 87. – *Incidence of amebic dysentery carrier state in the U. S. Army, by area and year, 1944–45*

[Preliminary data based on sample tabulations of individual medical records of primary and secondary diagnoses]

[Rate expressed as number of cases per annum per 1,000 average strength]

Area	1944–45		1944		1945	
	Number of cases	Rate	Number of cases	Rate	Number of cases	Rate
Continental United States	770	0. 11	175	0. 04	595	0. 20
Overseas:						
Europe	225	. 06	125	. 07	100	. 04
Mediterranean [1]	161	. 16	71	. 11	90	. 25
Middle East	95	1. 09	70	1. 51	25	. 61
China-Burma-India	949	2. 43	239	1. 42	710	3. 21
Southwest Pacific	1, 270	. 81	70	. 13	1, 200	1. 16
Central and South Pacific	529	. 65	94	. 21	435	1. 16
North America [2]	5	. 03	--	0	5	. 07
Latin America	83	. 52	3	. 03	80	1. 10
Total overseas [3]	3, 323	. 39	673	. 18	2, 650	. 57
Total Army	4, 093	. 27	848	. 11	3, 245	. 43

[1] Includes North Africa.
[2] Includes Alaska and Iceland.
[3] Includes admissions on transports.

AMEBIASIS IN OVERSEAS THEATERS AND AREAS

India-Burma Theater

Amebic dysentery was recognized and reported in Army units soon after they began to arrive in the theater in the summer of 1942.[15] It increased moderately in incidence during 1943 and more in 1944 reaching a peak of 50 per annum per 1,000 average strength in August and September 1944. This represented 675 and 884 cases, respectively, in these 2 months. In 1945, the incidence decreased considerably when intensive measures to prevent diarrheal diseases became effective. It is interesting that the incidence of bacillary dysentery was almost consistently somewhat lower than that of amebic dysentery throughout the entire period of operations in this theater. Both of these infections, however, were greatly overshadowed by acute diarrhea of unrecognized etiology, which reached a peak incidence of 215 per annum per 1,000 strength in July 1944.

Insanitary conditions conducive to the transmission of amebiasis existed almost everywhere in the theater. The medical and sanitary departments

[15] Van Auken, H. A.: A History of Preventive Medicine in the U. S. Army Forces of the India-Burma Theater, 1942–45. [Official record.]

were not prepared to deal with this complicated problem. Most medical officers had had no practical experience in dealing with the type of environmental conditions which existed and could not foresee or detect the hidden sources of infection. Directives for prevention and control were general in nature and not specifically adapted to the local situation. Equipment for mess sanitation, fly control, and water purification was inadequate or not efficiently used. Facilities for accurate diagnosis were provided only at the 9th Medical Laboratory and at some of the general hospitals.

As experience was gained by investigation of conditions associated with high incidence or epidemics of diarrheal diseases in individual units, unsupervised native foodhandlers appeared to be the greatest source of infection, although surveys for *E. histolytica* regularly showed a much lower incidence of infection in natives than in American personnel. In two instances, however, contaminated water supplies appeared to be the source of outbreaks of amebic dysentery. Other possible sources of infection were difficult to evaluate.

In June 1944, the Preventive Medicine Service of the Surgeon General's Office sent a Sub-Commission on Dysentery of the Army Epidemiological Board to Calcutta to make an intensive study in that area and in the Ledo area of Assam. The Sub-Commission[16] reported that at the 112th Station Hospital in Calcutta during July 1944 about one-fourth of the admissions for diarrhea or dysentery were amebic. In Advance Section 3 (Assam) for the year ending 30 September 1944, there had been 444 hospital admissions for amebic dysentery. In one combat unit, one-third of the first 150 admissions were for amebic dysentery. The Sub-Commission concluded that, from the standpoint of days lost and disability, amebic dysentery was the most important of the diarrheal diseases. Trained clinical and laboratory personnel and equipment for accurate diagnosis were inadequate, and sources of infection were poorly controlled. The chief source appeared to be native foodhandlers, followed by water, flies, and food in order of importance. Instruction of personnel was started, and recommendations were made for more rapid and accurate diagnosis, additional trained personnel, thorough treatment, and the control of possible sources of infection.

A preventive medicine section was set up in the Office of the Chief Surgeon and certain control activities were initiated through the theater commander. Among these were the assignment of a special sanitary officer to check mess sanitation through the theater, the issuance of letters of inquiry to organizations with high diarrheal rates, the inauguration of a better method for the distribution of sanitation supplies, and the preparation of a circular on water supplies.

The complete program of control instituted in the fall of 1944 included the issuance of directives, periodic inspections, provisions of necessary supplies, education of commanders and lectures to personnel, investigations of undue increase of cases of diarrhea, and the placing of responsibility for control on unit commanders. A theater laboratory consultant was appointed to raise

[16] Progress and Final Report, Sub-Commission on Dysentery, Army Epidemiological Board, 20 Nov. 1944.

the standards of diagnostic performance throughout the theater, and the 9th General Laboratory gave apprentice training and longer courses of instruction to laboratory officers and enlisted men.

The problem of symptomless carriers of *E. histolytica* among American personnel and natives received considerable attention. Beginning during the summer of 1944, numerous surveys were conducted by the 9th Medical Laboratory and by local hospitals and dispensaries. In some instances, the entire personnel of an organization was examined. Infection rates varied from 3 to 33 percent. Rates tended to be higher in units having many cases of diarrhea. The lowest recorded rates may have been due partly to lack of training of laboratory personnel. The highest rate was at the 24th Station Hospital where an epidemic of amebic dysentery among the hospital personnel was traced to a contaminated water supply.

Most units had their foodhandlers examined periodically when laboratory facilities were provided and found that the elimination of infected personnel from the kitchens was followed in a number of instances by a definite drop in the incidence of diarrhea.

The intensive control measures instituted in the fall of 1944 are reflected more in the lower reported incidence of common diarrhea in the summer of 1945, which was only about one-third of that in 1944, than in the incidence of amebic dysentery which reached a rate of 37 per 1,000 per annum in July 1945 as compared with a rate of 50 per 1,000 per annum in August and September 1944. This lesser decline is to be expected because of the insidious and chronic nature of amebic infection.

In the India-China Division of the Air Transport Command, water supplies in the China stations were unsatisfactory, and coolie labor increased the hazard of contamination. A report by Maj. Clifton W. Bovee, SnC, in July 1945, to the division surgeon, described the deficiencies that still existed and made recommendations for each of the bases. Basic water supply systems or diatomaceous silica filters provided safe drinking water, but water supplied washrooms, showers, and some messes remained highly inacceptable. Additional mechanical equipment and simple distribution systems were required.

Southwest Pacific Area

With the invasion of individual islands, insanitary conditions were encountered and contact with native populations occurred which were conducive to the development of a high infection rate with *E. histolytica*.

An illustration of the experience of one division is contained in a report by Maj. Harry J. Bennett, SnC, on a survey made of intestinal parasites in the 37th Infantry Division.[17] The division arrived in the Fiji Islands from the United States and New Zealand in June and August 1942. It was transferred to Guadalcanal in April 1943, participated in the New Georgia campaign from

[17] Report, Maj. Harry J. Bennett, SnC, to Chief Malariologist, Headquarters, U. S. Army Forces, Far East, 18 Nov. 1944, subject: Intestinal Parasite Survey for the 37th Infantry Division.

July to September 1943, then returned to Guadalcanal for 1 month, and arrived in Bougainville in November 1943. The first six cases of amebic dysentery were diagnosed on Guadalcanal in May 1943. There was no great increase in cases until after the division reached Bougainville. Beginning in January 1944, when 17 cases occurred, there was an increase to 67 cases in July, a rate of 55 per 1,000 per annum. Between 1 November 1943 and 1 November 1944, 460 cases of amebic dysentery were admitted to hospitals. A stool survey of 1,072 individuals, mostly kitchen personnel from the various units of the division, was conducted from July to September 1944, and 27.3 percent were found to harbor *E. histolytica*. Since only one specimen was examined from most individuals, it was estimated that the actual prevalence was at least 50 percent. Analysis of sanitary conditions under which the division had operated suggested that no single source of infection could be incriminated but that flies, inadequate sanitary facilities, and polluted water all probably played a part in building up a high incidence of infection.

Another illustration is the experience of the 81st Infantry Division which invaded the island of Peleliu, one of the Palau Islands. A high incidence of amebic dysentery led to a preliminary stool survey by Capt. E. C. Nelson, SnC,[18] while the division was still on the island. On a single stool examination of 2,210 troops of a regiment, 30 percent were found to harbor *E. histolytica;* the prevalence in different companies varied from 17 to 44 percent. When the division returned to New Caledonia, the survey was continued with 14,534 (88 percent) of the personnel receiving 1 stool examination. An over-all prevalence of 18.7 percent was found. This study was reported by Murray, Winter, and Sears.[19] Captain Nelson investigated the conditions possibly responsible for the high incidence of cases shortly after arrival on the island and concluded that flies were probably the principal source of infection since the water supply was found to be protected from surface contamination and individual food rations were used. The incidence of infection was, in general, highest where the fly population was greatest. One course of specific treatment of all infected personnel reduced the incidence in these individuals to 5.1 percent.

Following the invasion of the Philippine Islands, beginning with Leyte in October 1944, there was an increase in the number of clinical cases of amebiasis through the quarter ending June 1945, after which the number of reported cases declined. This is indicated by the following data from the quarterly reports of the 116th Station Hospital, which was located on Leyte and served the XXIV Corps, the 81st Division, and later the IX Corps and other troops assigned to Base K:

1945	*Cases of amebiasis*
January–March	330
April–June	761
July–September	215
October–November	72

[18] Essential Technical Medical Data, Headquarters, Pacific Ocean Area, March 1945, inclosure 7.
[19] Essential Technical Medical Data, Headquarters, South Pacific Base Command, March 1945.

Amebiasis was considered the most serious disease problem in the Philippine area except for infectious hepatitis. The increase in the number of new cases of amebiasis and the rates per thousand per annum for Base K during the first 5 months of 1945 are shown in the following tabulation:[20]

	Cases	*Rates*
January	275	4. 57
February	432	7. 20
March	822	11. 37
April	911	15. 32
May	999	16. 52

In the Philippines, contact of military personnel with the civilian population was greater than in most of the Pacific islands. In Manila, the destruction of buildings and the general breakdown of sanitation added to the conditions favorable for the transmission of amebiasis. The following measures were taken to control amebic dysentery on Base K:

1. All water was treated before consumption by boiling or filtration and then by chlorination. Wells were cased and properly located.
2. Fly control was rigidly enforced by screening and spraying with DDT.
3. Civilians were prohibited from any food handling.
4. The preparation and use of ice was controlled.
5. Messgear was dipped in boiling water before use.
6. Cases and carriers of amebic infection were excluded from food handling.
7. Foodhandlers received stool examinations monthly and were instructed in personal sanitation.
8. Temporary kitchen police were instructed in personal sanitation.
9. Military personnel were forbidden to live with civilians.
10. Civilians were not permitted to live within unit areas.
11. Animal pets were discouraged.
12. Consumption of food from civilian sources was forbidden, and green vegetables grown locally were not eaten uncooked.

In the Eighth U. S. Army, which ultimately went from the Philippines to Japan, amebiasis was a relatively unimportant cause for hospitalization at the beginning of 1945 but by June increased to an admission rate of 40 per annum per 1,000 average strength.[21] It was believed that a large proportion of the cases resulted from the practice among Army personnel of eating food and drinking untreated water in Filipino homes and restaurants. By means of the issuance of directives and personal visits by medical officers from Army headquarters, an intensive sanitary control program was put into effect in all operational areas. This was followed by a drop in admissions in July to a rate of 9.7 per annum per 1,000, with no later significant increase, and a continued decline after arrival in Japan.

In Hawaii and the mid-Pacific islands, amebiasis was apparently not an important infection.

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[20] Essential Technical Medical Data, Headquarters, U. S. Army Forces, Pacific, July 1945.

[21] Annual Report, Surgeon, Headquarters, Eighth U. S. Army, Southwest Pacific Area, 1945.

Middle East Theater

The data in tables 84, 85, 86, and 87 indicate that amebiasis was not so prevalent in this theater as in the China-Burma-India theater and that it reached a peak of 11.33 per 1,000 per annum in 1943, after which it declined. A report on amebiasis from the 38th General Hospital for the period November 1942 to November 1944 stated that 464 cases of amebiasis had been admitted to the hospital during that period. The highest rates of admission were in April and May 1943. About one-fourth of these were classed as carriers; the remainder had chronic or acute symptoms. Of the hospital personnel, 816 complained of abdominal symptoms during this period, and 144 (17.8 percent) were found to be suffering from symptomatic amebiasis. Purged stools of foodhandlers were examined monthly. None was found infected until the hospital had been in that locality for about a year. Of 77 individuals, 28 (36.4 percent) were found infected on one of the examinations. Twenty-two of these infected persons were entirely symptom free.[22]

A report of the Middle East Service Command, 1 June 1944, gave results of a survey of 147 foodhandlers at Camp Russell B. Huckstep. A prevalence of 18.3 percent of infection with *E. histolytica* was found.

Mediterranean Theater

Amebiasis was a minor problem in the Mediterranean theater. A report on dysentery and common diarrheas by Maj. H. M. Hurevitz, MC, in June 1945, indicated that the admissions to hospitals and quarters in the entire theater for protozoal dysentery totaled 156 cases in 1943 and 851 in 1944. Some of the cases in 1944 were asymptomatic, having been admitted only because *E. histolytica* had been found in the feces. Surveys of military personnel had revealed a prevalence in different units of only 2 to 11 percent. Many patients with diarrhea or dysentery in whom *E. histolytica* was found were considered to be suffering from other infections.

The potential menace of diarrhea and dysentery in the forces in North Africa was predicted by Col. Perrin H. Long, consultant in medicine to the surgeon of the Mediterranean theater, in January 1943.[23] He made recommendations to the Deputy Surgeon, Allied Force, for sanitary-control procedures, but these were not acted upon at that time. In May, when flies became abundant, bacillary dysentery broke out in American troops in all areas of North Africa. Investigation indicated that the outbreaks were due to bad sanitation in certain units. This led to the publication of a theater circular which dealt with the measures to be employed in the control of dysentery and stressed command responsibility for sanitation.[24] Supplies for flyproofing

[22] Tallant, E. J., and Maisel, A. L.: Amebiasis Among the American Armed Forces in the Middle East. Arch. Int. Med. 77: 597-613, June 1946.

[23] Long, Perrin H.: A Historical Survey of the Activities of the Section of Preventive Medicine, Office of the Surgeon, Mediterranean Theater of Operations, United States Army, 3 January 1943 to 15 August 1943. [Official record.]

[24] Circular No. 106, Headquarters, North African Theater of Operations, United States Army, 9 June 1943.

were released from engineer stocks; sanitary inspectors, armed with punitive powers, visited the base sections to check up on sanitation; and great efforts were made to bring fly breeding under control. As a result, the admission rates for diarrhea and dysentery which had reached 445 per 1,000 per annum in June were cut to 213 per 1,000 per annum in July despite the invasion of Sicily early in that month. Measures specifically directed against the transmission of amebiasis emphasized prohibition of the use of raw fruits and vegetables without prior sterilization.

In 1944, with the shifting of the campaign to Italy, control measures against diarrheal diseases were successfully continued.[25]

European Theater

Amebiasis was not a serious problem in the European theater.[26] The total number of admissions for the years 1942 to 1945 was 1,637, an annual rate of 0.37 per 1,000 average strength. This is broken down further into an annual rate for 1942 of 0, for 1943 of 0.14, for 1944 of 0.36, and for 1945 of 0.42 per 1,000 average strength (table 84). Very few cases occurred in troops in England; the number increased with the invasion of France. This increase appears to have been partly due to infections acquired in the Mediterranean theater by troops who invaded Southern France. Edson, Ingegno, and D'Albora [27] reported 39 cases of amebiasis from a United States Army general hospital in North Ireland during a period of 10 months. All patients had symptoms and physical signs. Twenty-six were members of one division (presumably the 5th) which was composed largely of troops from the Southern United States who had been through maneuvers in Tennessee and Louisiana. The authors suggested that the original infections had occurred in the United States and that some had been acquired subsequently from contact with carriers. A survey of 162 foodhandlers with the 5th Infantry Division revealed 18.5 percent positive for *E. histolytica* on one stool examination. A second survey of a random selection of troops by another laboratory revealed a prevalence of 19 percent.

A survey of troops from a division in England showed a rate of 16.1 percent for *E. histolytica*, indicating that the infection was more common than is estimated in the civilian population of the United States. Cases of diarrhea in which *E. histolytica* was identified were classed as amebic dysentery. It was considered probable that some of these cases were actually cases of bacillary dysentery or common diarrhea and that the actual incidence of active amebic dysentery was less than that reported.

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[25] Annual Report, Surgeon, Mediterranean Theater of Operations, United States Army, 1944, vol. 2.

[26] Gordon, J. E.: A History of Preventive Medicine in the European Theater of Operations, U. S. Army, 1941–45, pt. III, sec. 2. [Official record.]

[27] Edson, J. N., Ingegno, A. P., and D'Albora, J. B.: Amebiasis: A Report of Thirty-nine Cases Observed in an Army General Hospital Stationed in Northern Ireland. Ann. Int. Med. 23: 960–968, December 1945.

PREVENTION AND CONTROL

At the request of the Chief, Preventive Medicine Service, Office of the Surgeon General, the Subcommittee on Tropical Diseases of the National Research Council early in 1941 prepared a directive entitled "Notes on the Treatment and Control of Certain Tropical Diseases," which was issued by the Office of the Surgeon General on 9 June 1941 as Circular Letter No. 56. A revision of this directive issued on 2 February 1943 as Circular Letter No. 33 contained data on the prevention of amebiasis including the necessity for superchlorination or boiling of water to kill cysts, avoidance of raw fruits and vegetables wherever exposed to human excreta, exclusion of infected persons from food handling, and control of flies and cockroaches. Reference was also made to AR 40–205 and AR 40–210. Circular Letter No. 33 was superseded by TB MED 159, Amebiasis, issued May 1945, which contained a section on prevention, including considerable detail concerning mess sanitation, foodhandlers, water purification, excreta disposal, and fly control by the use of DDT.

In general, the measures taken to prevent amebiasis in individual areas of military operation were part of those taken to prevent all forms of gastrointestinal infection. The only additional requirement for the prevention of amebiasis was based upon the fact that a higher concentration of residual chlorine is required to kill the cysts of *E. histolytica* than to kill bacteria in water. Superchlorination and subsequent dechlorination of water in Lyster bags and canteens was introduced to some extent in 1944 and 1945, but its effect on the incidence of amebiasis cannot be determined. Several directives were issued by theater surgeons [28] describing the disease and its modes of transmission, and methods of diagnosis, treatment, and prevention.

The effective measures of control of amebiasis seem to have been (1) the provision of safe water supplies, (2) the exclusion of native foodhandlers, (3) the examination of military foodhandlers and the exclusion of those found to be infected, (4) the sanitary disposal of human excreta, (5) the prohibition or sterilization of raw fruits and vegetables, (6) the control of flies by screening and DDT spraying, (7) the prohibition of eating in native homes or restaurants, and (8) the enforcement of individual sanitary discipline.

A survey of single, normally passed stool specimens from 4,000 men at the time of separation from military service was made at Fort McPherson, from January to May 1946.[29] The specimens were examined by the direct smear and zinc sulfate flotation techniques and doubtful specimens were checked by iron-hematoxylin stain. *E. histolytica* was identified in 14.3 percent of the specimens. The prevalence by area of service is shown in table 88.

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[28] (1) Circular No. 9, Office of the Surgeon, Headquarters, U. S. Army Forces, India-Burma Theater, 2 Apr. 1945, subject: Amebiasis. (2) Technical Memorandum No. 20, Office of the Chief Surgeon, Headquarters, U. S. Army Forces in the Far East, 24 Nov. 1944, subject: Amebiasis: Amoebic Dysentery.

[29] Survey of Intestinal Parasites in Soldiers Being Separated from Service. Bull. U. S. Army M. Dept. 6: 259–262, September 1946.

TABLE 88.—*Prevalence of infection with* E. histolytica [1] *among 4,000 troops from various areas, at time of separation from service, Fort McPherson, Ga., January to May 1946*

Area of service	Number examined	Percent positive
Continental United States and Western Hemisphere (nontropical)	1,317	10.2
Europe, Africa, Middle East, and Italy	1,347	18.5
Pacific	952	13.2
China-Burma-India	160	21.9
Western Hemisphere (tropical)	27	7.4
Europe and Pacific areas	197	13.7

[1] Based on results of examination of fecal specimens.

The results of this survey coincide in general with the clinical and laboratory experience in overseas theaters and indicate that many infections acquired overseas were introduced into this country.[30]

RESEARCH ON WATER PURIFICATION

The importance of water as a vehicle for the transmission of *E. histolytica* was emphasized by the epidemic of amebiasis in Chicago in 1933. Although some work was done before World War II on the effectiveness of chlorine in killing the cysts of *E. histolytica*, Chang and Fair [31] in 1941 were the first to demonstrate the degree to which cysts were resistant to chlorination under various conditions of water quality. Their work was confirmed by Brady, Jones, and Newton [32] in 1943. Later studies by Fair [33] demonstrated that, if calcium hypochlorite solution were used in Lyster bags, a residual chlorine of 7.5 parts per million would be required at a temperature of 10° C. with a 30-minute contact to assure the killing of cysts. Under these conditions, other chlorine demands of the water would probably raise the initial chlorine demand to 25 to 30 parts per million; acidification would also be necessary to ensure a desirable pH value of the water, and, in order to produce a palatable water, dechlorination by sodium sulfite in the receiving vessel would be necessary. The use of halazone (p-dichlorosulfamidobenzoic acid) tablets presented an even greater problem because of their slow dissolving time and the more objectionable taste which they convey to water. These difficulties reduced the practicability of employing chlorine compounds to disinfect water in canteen quantities, although

[30] An estimate of the prevalence of amebiasis among veterans of World War II may be obtained from records of veterans hospitals showing the number of cases discharged from the hospitals since the war. It was reported at a meeting of Regional Consultants on Tropical Medicine, Veterans' Administration, held at Savannah, Ga., on 5 November 1950, that during the years 1945–48, a total of 3,673 patients treated for amebiasis were discharged. There was a rapid increase in the number of cases diagnosed after the return of troops from overseas, and the increase was continuous through 1949.

[31] See footnote 13, p. 486.

[32] Brady, F. J., Jones, M. F., and Newton, W. L.: Effect of Chlorination of Water on Viability of Cysts of *Endamoeba histolytica*. War Med. 3: 409–419, April 1943.

[33] Fair, G. M.: Interim Report No. 4; Disinfection of Water; Contract OEMcmr-251. NRC Bull. Sanit. Eng. Div. 12 July 1945, pp. 195–219.

provision for hyperchlorination and dechlorination was made in some areas of military operation.

For these reasons, Fair and his associates investigated other halogens and found that iodine was a more useful cysticidal agent. The first iodine tablet developed contained Bursoline, a mechanical mixture in the proportion of 2 moles of glycine hydriodide to 1 mole of elemental iodine. On solution in water, tablets containing this mixture liberated free iodine. This element has the advantage over chlorine of not being so easily hydrolyzed and not reacting with ammonia or organic nitrogenous substances to form iodoamines.[34] For field disinfection it was found that, with a contact time of 10 minutes, 4 parts per million of free iodine were required to kill 60 cysts per ml. at 23° Centigrade. A dose of 7.5 to 8 parts per million was considered sufficient to take care of the great majority of waters and leave sufficient residual for disinfecting action in 10 minutes, unless the water was very cold. Tablets containing Bursoline, disodium dihydrogen pryo-phosphate as an acid-buffering agent, and filler were prepared for use in canteens, and field tests showed that they were satisfactory because of their stability, rapid solution, and the lack of disagreeable taste of the treated water. There was also no evidence that iodine, in the dosage employed, would be harmful to consumers. Bursoline tablets were, therefore, produced on a small commercial scale and were used to some extent in the India-Burma and Pacific theaters late in the war.

Further investigation by Fair and his associates indicated that Globaline (crystalline tetraglycine hydroperiodide) could replace the Bursoline mixture and thereby reduce the amount of inactive iodine added to the water. Buffered tablets of this compound, which would liberate 8 mg. of iodine per tablet, were found to be stable under normal conditions of storage and use. They dissolved in less than a minute and disinfected most waters in 10 minutes with the use of one tablet per canteen. Two tablets were required for highly colored waters and 20 minutes for disinfection of very cold water. High turbidity, alkalinity, ammonia, urea, and salt had no appreciable effect on the disinfecting efficiency. Field tests in the armed services showed great superiority of Globaline over chlorine compounds because of palatability, rapidity of disinfection, and convenience.

Synthetic detergents were tested for cysticidal activity under contract with the Office of Scientific Research and Development by Fair, Chang, Taylor, and Wineman [35] and by Kessel and Moore.[36] A number of cationic compounds were found to be effective. Fair reported that Ceepryn (1-n-hexadecyl pyridinium chloride) and Fixanol (cetyl pyridinium bromide) were cysticidal in water at concentrations of 25 to 50 parts per million, but that it was not practical to

[34] Fair, G. M.: Water Disinfection and Allied Subjects. *In* United States Office of Scientific Research and Development: Advances in Military Medicine. Boston: Little and Brown Co., 1948, vol. II, pp. 520–531.

[35] Fair, G. M., Chang, S. L., Taylor, M. P., and Wineman, M. A.: Destruction of Water-Borne Cysts of *Endamoeba histolytica* by Synthetic Detergents. Am. J. Pub. Health 35: 228–232, March 1945.

[36] Kessel, J. F., and Moore, F. J.: Emergency Sterilization of Drinking Water With Heteropolar Cationic Antiseptics. Am. J. Trop. Med. 26: 345–350, March 1946.

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use them in the Armed Forces because at that concentration they foam badly and have a bitter taste.

The effectiveness of ozone as a cysticidal agent was investigated during the war by Kessel and his coworkers,[37] and more recently by Newton and Jones.[38] In laboratory tests they found ozone more active than chlorine and not influenced by hydrogen ion concentration or organic matter, but difficulty of production and rapid dissipation in water, leaving no residual, made it unsuitable for practical application.

Ultraviolet light as a cysticidal agent was also tested on a small scale during the war by Stoll, Ward, and Mathieson at the Naval Medical Research Institute.[39] Although it was found to be effective under experimental conditions, it did not appear to be a practical agent for the purification of military water supplies.

The efficiency of the portable sand filters of the Army for the removal of cysts of *E. histolytica* from water was investigated by the Corps of Engineers during the war in cooperation with the National Institutes of Health.[40] It was found that when these filters were operated at the rate for which they were designed they allowed some cysts to pass through. This danger was increased by the tendency to operate the filters at a higher rate when a larger quantity of water was required. Diatomaceous earth was found to be efficient in removing all cysts, irrespective of the nature of the water, at flow rates as high as 7 gallons per square foot per minute. Portable filters of this type were manufactured and were used to a small extent during the latter part of the war in the China section of the Air Transport Command.

RESEARCH ON SEWAGE TREATMENT

Cram [41] studied the survival of cysts of *E. histolytica* under experimental conditions simulating sewage treatment processes in common use. The cysts were not removed by primary settling, but passed out in the effluent, and also passed through trickling filters and survived activated sludge treatment. They were removed by alum floc precipitation during secondary settling, and also by intermittent sand filtration. They did not survive sludge digestion. These results indicated that effluents from sewage disposal plants would be likely to contain cysts of *E. histolytica* which might be transported for long distances in streams.

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[37] Kessel, J. F., Allison, D. K., Kaime, M., Quiros, M., and Gloeckner, A.: The Cysticidal Effects of Chlorine and Ozone on Cysts of *Endamoeba histolytica*, Together With a Comparative Study of Several Encystment Media. Am. J. Trop. Med. 24: 177-183, May 1944.

[38] Newton, W. L., and Jones, M. F.: The Effect of Ozone in Water on Cysts of *Endamoeba histolytica*. Am. J. Trop. Med. 29: 669-681, September 1949.

[39] Naval Medical Research Institute, Research Project X-110, Report 5, 2 Feb. 1945, subject: The Effect of Ultraviolet Radiation on Cysts of *Endamoeba histolytica*.

[40] (1) Black, H. H.: Army Field Water Supply Developments. Am. J. Pub. Health 34: 697-710, July 1944. (2) Engineer Board, Corps of Engineers, Report 834, 3 July 1944, subject: Efficiency of Standard Army Water Purification Equipment and of Diatomite Filters in Removing Cysts of *Endamoeba histolytica* from Water. (3) Newton, W. L.: Water Treatment Measures in Control of Amebiasis. Am. J. Trop. Med. 30: 135-138, March 1950.

[41] Cram, E. B.: The Effect of Various Treatment Processes on the Survival of Helminth Ova and Protozoan Cysts in Sewage. Sewage Works J. 15: 1119-1138, November 1943.

RECOMMENDATIONS

If the experience gained in World War II is to be utilized in the prevention of amebiasis and other intestinal infections in future military operations, it is necessary to make preparations in advance and to educate each new generation of physicians in the part it must play in a preventive program. The following recommendations are suggested:

1. Sanitary discipline of camps, messes, and combat units is a responsibility of the commanding officer and should be given equal importance with other phases of military training. Instruction and practical experience should be given to all personnel from highest to lowest rank. This must include specific directives, and the participation of medical and sanitary officers well trained in the details of preventive measures.

2. Continuous adequate training should be provided for medical, sanitary and laboratory personnel in the accurate diagnosis, treatment, and prevention of amebiasis, considering it as a specific infection of equal importance to bacillary dysentery and other intestinal infections.

3. Efficient portable and emergency water supply equipment and supplies should be devised and prepared which can be furnished to overseas forces and combat units as soon as they are activated.

4. Efficient equipment for mass sanitation, excreta disposal, and fly control should be provided and given high priority in supplies accompanying overseas forces.

5. Special instruction in personal sanitation and continuous supervision of all foodhandlers in military units should be required.

6. On the occurrence of any epidemic of diarrhea in troop units, stools should be examined particularly for *E. histolytica* as one of the causative agents. If it is found, examination of stools from all members of the unit and treatment of all infected individuals should be carried out. Search should be made for the source of infection and appropriate preventive measures should be instituted.

7. Special stool examinations for *E. histolytica* on all troops returning from overseas or on discharge from military service should be performed whenever practicable and those found to be infected, should be treated.

CHAPTER XXIV

Nematode and Cestode Infections

Clyde Swartzwelder, Ph. D.

With the exception of filarial and hookworm infections, parasitism of nited States Army troops by nematodes and cestodes during World War II id not constitute a very important military problem. The total United tates Army admissions during the years 1942 through 1945 for infections ith selected parasites are shown in table 89. The distribution of hospital nd quarters admissions, in 1944, of troops infected with these parasites is resented by area and theater in table 90. Except for trichinosis and tapeworm nfections, a large majority of the nematode and cestode infections apparently ere acquired overseas. The number of hospital and quarters admissions is ndicative of the problem of acute disease. These admissions do not indicate, xcept indirectly perhaps, the number of troops infected.

The report of Stoll, entitled "This Wormy World," [1] represents a classic n the field of the geography of parasitism. The number of human helminthic nfections in millions, calculated by Stoll, is shown in table 91. This table ives ample evidence of the presence of reservoirs of parasitic worms throughout he world which might provide a source of infection and a potential hazard to roops unless adequate protective sanitary measures were employed. Military rganizations made numerous surveys of native groups living in the vicinity of roop concentrations; these surveys provide additional evidence of the presence f a source of infection for troops.[2]

NEMATODE INFECTIONS

Strongyloidiasis

There was a total of 1,242 admissions for infection with *Strongyloides ercoralis* during the years 1942 through 1945. In 1944, there were 336 ecorded admissions for this infection. Of these, 237 represented admissions n overseas theaters. Ninety-nine cases were admitted in the United States uring that year. The areas with the largest numbers of reported cases in 944 were the Central and South Pacific, Southwest Pacific, Latin America,

[1] Stoll, N. R.: This Wormy World. J. Parasitol. 33: 1-18, February 1947.

[2] (1) Hansen, M. F., and Bern, H. A.: A Roundworm Problem in the Philippines. Air Surgeon's Bull. 2: 377, ovember 1945. (2) Fink, H.: A Helminth Survey From an Autopsy Series on Natives of Okinawa, With Comments on mplications of Ascariasis. Am. J. Trop. Med. 28: 585-588, July 1948. (3) History of Preventive Medicine, Headarters, U. S. Army Forces, Middle Pacific, December 1941-September 1945, pp. 201-206. [Official record.]

TABLE 89.—*Admissions for selected nematode and cestode infections in the U. S. Army, by diagnosis, area, and year, 1942–45*

[Preliminary data based on sample tabulations of individual medical records]

Diagnosis	1942–45	1942	1943	1944	1945
	United States				
Strongyloides infection	453	49	125	99	180
Ascaris infection	1,401	62	110	59	1,170
Enterobius infection	383	87	105	81	110
Trichinosis	208	32	80	61	35
Nematode infection, other [1]	697	98	200	104	295
Echinococcus infection	15	2	5	3	5
Cysticercosis	[2]	[2]	[2]		
Cestode infection, other	[3] 1,334	[4] 293	[4] 455	321	265
	Overseas				
Strongyloides infection	789	25	82	237	445
Ascaris infection	3,630	30	150	430	3,020
Enterobius infection	889	107	173	299	310
Trichinosis	77	4	6	22	45
Nematode infection, other [1]	1,578	32	241	310	995
Echinococcus infection	7			7	
Cysticercosis	[5] 1	[3]	[3]	1	
Cestode infection, other	[4] 679	[5] 53	[5] 131	220	275
	Total Army				
Strongyloides infection	1,242	74	207	336	625
Ascaris infection	5,031	92	260	489	4,190
Enterobius infection	1,272	194	278	380	420
Trichinosis	285	36	86	83	80
Nematode infection, other [1]	2,275	130	441	414	1,290
Echinococcus infection	22	2	5	10	5
Cysticercosis	[5] 1	[2]	[2]	1	
Cestode infection, other	[3] 2,013	[4] 346	[4] 586	541	540

[1] Except filariasis and ankylostomiasis.
[2] Cases, if any, included with "Cestode infection, other."
[3] Includes cysticercosis cases, if any, in 1942 and 1943.
[4] Includes cysticercosis, if any.
[5] 1944 and 1945 only.

China-Burma-India, and the Mediterranean (table 90). A single stool examination will fail to diagnose all cases of strongyloidiasis. Many infections require duodenal aspiration for demonstration of diagnostic forms. On a number of occasions, the writer observed cases of strongyloidiasis erroneously

TABLE 90. *Admissions for selected nematode and cestode infections in the U. S. Army, by diagnosis and area, 1944*

[Preliminary data based on sample tabulations of individual medical records]

Area	Strongyloides infection	Ascaris infection	Enterobius infection	Trichinosis	Nematode infection, other [1]	Echinococcus infection	Cysticercosis	Cestode infection, other
Continental United States	99	59	81	61	104	3	.....	321
Overseas:								
Europe	5	100	30	5	20	5	.....	60
Mediterranean [2]	16	151	107	6	23	.....	1	54
Middle East	6	1	3	.....	.....	.....	.....	6
China-Burma-India	26	83	21	1	15	.....	.....	18
Southwest Pacific	45	30	99	3	56	.....	.....	21
Central and South Pacific	99	29	30	4	48	.....	.....	42
North America [3]	1	.....	3	.....	3	2	.....	9
Latin America	39	36	6	3	144	.....	.....	10
Total overseas [4]	237	430	299	22	310	7	1	220
Total Army	336	489	380	83	414	10	1	541

[1] Except filariasis and ankylostomiasis.
[2] Includes North Africa.
[3] Includes Alaska and Iceland.
[4] Includes an admission aboard transport.

diagnosed as hookworm infection in overseas areas. Thus the number of infections acquired no doubt greatly exceeded those correctly diagnosed and recorded as strongyloidiasis.

Maj. A. A. Liebow, MC, and 1st Lt. C. A. Hannum, SnC,[3] reported that infection with *S. stercoralis* stood next in frequency to hookworm among admissions of troops from the Solomon Islands to the 39th General Hospital for helminthiases. Routine stool examinations which were performed at the hospital between February and December 1943 showed that 7.4 percent of 633 specimens were positive for rhabditiform larvae of *S. stercoralis*. Denhoff [4] pointed out the striking similarity between the symptoms of patients with strongyloidiasis and those of other troops with anxiety neurosis. Delay in diagnosis of *Strongyloides* infections for long periods, without clinical relief, and the lack of a satisfactory treatment contributed to the difficulty in separating complaints from psychoneurosis and those from strongyloidiasis in troops. Denhoff called attention to the fact that duodenal intubation as a diagnostic method for *Strongyloides* infection had not been stressed sufficiently. The 8th General Hospital, stationed at New Caledonia in 1944, reported that

[3] Liebow, A. A., and Hannum, C. A.: Eosinophilia, Ancylostomiasis and Strongyloidiasis in the South Pacific Area. Yale J. Biol. and Med. 18: 381-403, May 1946.

[4] Denhoff, E.: The Significance of Eosinophilia in Abdominal Complaints of American Soldiers. New England J. Med. 236: 201-206, February 1947.

TABLE 91.—*The calculated number of human helminthic infections, in millions* [1]

Infections	Northern America [2]	Middle and South America [3]	Africa	Europe (without U.S.S.R.)	U.S.S.R. in Europe [4]	U.S.S.R. in Asia	Asia (without U.S.S.R.) [5]	Oceania [6]	Total
Trichinella spiralis [7]	21. 1	1. 3	0. 2	3. 9	1. 3	.....	(8)	(8)	27. 8
Taenia saginata	. 1	. 7	12. 0	. 8	12. 0	7. 2	6. 0	0. 1	38. 9
Taenia solium	(8)	(8)	. 5	(8)	. 5	. 3	1 [illegible]	(8)	2. 5
Hydatid [7]	(8)	(8)	(8)	(8)	(8)	(8)	(8)	(8)	(8)
Hymenolepis nana	. 1	. 7	. 6	1. 6	2. 3	. 9	14. 0	(8)	20. 2
Diphyllobothrium latum	(8)	.....	(8)	2. 8	3. 3	3. 1	1. 2	.....	10. 4
Clonorchis sinensis	.....	.....	.....	.....	.....	.....	19. 0	.....	19. 0
Opisthorchis felineus	.....	.....	.....	. 1	. 4	. 6	(8)	.....	1. 1
Fasciolopsis buski	.....	.....	.....	.....	.....	.....	10. 0	.....	10. 0
Paragonimus westermani	.....	.....	(8)	.....	.....	.....	3. 2	(8)	3. 2
Schistosoma japonicum	.....	.....	.....	.....	.....	.....	46. 0	.....	46. 0
Schistosoma haematobium	.....	.....	39. 0	(8)	.....	.....	. 2	.....	39. 2
Schistosoma mansoni	.....	6. 2	23. 0	.....	.....	.....	.....	.....	29. 2
Dracunculus medinensis [7]	.....	(8)	15. 0	.....	.....	3. 3	30. 0	.....	48. 3
Onchocerca volvulus [7]	.....	. 8	19. 0	.....	.....	.....	.....	.....	19. 8
Mansonella ozzardi [7]	.....	7. 0	.....	.....	.....	.....	.....	.....	7. 0
Acanthocheilonema perstans [7]	.....	8. 0	19. 0	.....	.....	.....	.....	(8)	27. 0
Loa loa [7]	.....	.....	13. 0	.....	.....	.....	.....	.....	13. 0
Wuchereria bancrofti and *W. malayi* [7]	.....	9. 0	22. 0	(8)	(8)	.....	157. 0	1. 0	189. 0
Enterobius vermicularis	18. 0	16. 0	8. 9	62. 0	25. 0	7. 5	71. 0	. 4	208. 8
Hookworm	1. 8	42. 0	49. 0	1. 4	2. 8	(8)	359. 0	. 8	456. 8
Ascaris lumbricoides	3. 0	42. 0	59. 0	32. 0	13. 0	6. 9	488. 0	. 5	644. 4
Trichuris trichiura	. 4	38. 0	28. 0	34. 0	23. 0	4. 2	227. 0	. 5	355. 1
Strongyloides stercoralis	. 4	8. 6	3. 3	. 6	. 7	. 2	21. 0	. 1	34. 9
Trichostrongylus species	(8)	.....	(8)	.....	. 5	. 5	4. 5	.....	5. 5
Total	44. 9	180. 3	311. 5	139. 2	84. 8	34. 7	1, 458. 3	3. 4	2, 257. 1

	Number in millions	Number in millions	Number in millions	Number in millions	Number in millions	Number in millions	Number in millions	Number in millions	Number in millions
Population	143. 5	130. 8	148. 0	387. 5	125. 0	45. 4	1, 176. 5	10. 1	2, 166. 8
	Percent	*Percent*	*Percent*	*Percent*	*Percent*	*Percent*	*Percent*	*Percent*	*Percent*
Infections of population	31	138	210	36	68	76	124	34	104. 2

[1] Reproduced by permission of Norman R. Stoll, Sc. D.

[2] Northern America includes the United States of America and Canada.

[3] Middle and South America includes the West Indies, Mexico, and south.

[4] The U.S.S.R. in Europe includes Transcaucasia (with Dagestan) and excludes Bashkiria and the Ural Area.

[5] Asia includes Japan and the islands off southeast Asia to the eastern line of Netherlands New Guinea.

[6] Oceania includes Australia and New Zealand.

[7] Broadly speaking, these infect man due to his ineffective insulation from transfer hosts, the other helminthiases are due, in final analysis, to man's ineffective insulation from his own excretory products (and, to a minor, unknown extent to man's ineffective insulation from the excretory products of reservoir hosts for the few species which are considered to employ other mammalian hosts optionally).

[8] Represents less than 100,000 infections.

hookworm and *S. stercoralis* were the commonest intestinal parasites encountered. The infections with *S. stercoralis* were more resistant to treatment and appeared to be more clinically significant than the hookworm infections.[5] A prevalence of strongyloidiasis of 12 percent was reported in 150 Puerto Rican enlisted men at Fort Brooke, P. R.[6] One infection with *Strongyloides fülleborni*, normally a parasite of monkeys, was recorded.[7] The patient had kept a pet monkey while stationed on the island of Leyte in the Philippines.

The following information supplied by Dr. C. A. Jones, chief of the medical service of the Veterans' Administration Hospital, New Orleans, La., provides excellent information on the problem of strongyloidiasis in veterans of World War I and World War II. Between 4 May 1946 and 11 October 1950, 21,784 patients were admitted to the hospital. The diagnosis of strongyloidiasis was established in 123 of these patients—approximately 0.6 percent of the total admissions. Forty-two of these patients were veterans of World War I. Of this group, 14 served in areas outside the United States. Thirteen had service within the continental limits of the United States. In 15 cases, data on foreign service were not recorded. The available records indicate that all except one of these patients had no military duty outside the continental limits of the United States for approximately 30 years. The World War I group of patients probably acquired their infections in areas around their homes. Most of these World War I veterans were farmers or had other occupations which brought them in close contact with the ground. The homes of 35 of these World War I veterans were in rural Louisiana, Mississippi, or Arizona. Seven lived in New Orleans. Each of this older group of patients had received an average of 66 days of hospital care. This prolonged period of hospitalization resulted from repeated admissions to the hospital and complicating or accompanying chronic degenerative diseases. The relative proportion of veterans of World War I and of World War II in the series of 123 cases was approximately the same as the ratio of their admission to the hospital.[8]

There were 81 patients whose service occurred during World War II. One of these had service in both the First and Second World Wars. In contrast to the patients of World War I, at least 46 had duty outside the continental limits of the United States. Twenty-five of this group served in England, France, Italy, North Africa, or in the Middle East. The remainder had duty in the Pacific. Twenty-three had service exclusively within the continental limits of the United States. In 12 cases, the precise location of military duty other than the United States was not stated. The World War II veteran group included farmers, laborers, truck drivers, and skilled mechanics. The majority of these veterans lived in rural areas of Louisiana, Mississippi, and Texas. Thirteen came from larger communities such as New Orleans,

[5] Annual Report of the Activities of the 8th General Hospital, New Caledonia, for the year ending 31 December 1944.

[6] History of Medical Department Activities, Antilles Department, Preventive Medicine, p. 67. [Official record.]

[7] Wallace, F. G., Mooney, R. D., and Sanders, A.: Strongyloides Fülleborni Infection in Man. Am. J. Trop. Med. 28: 299-302, March 1948.

[8] (1) Personal communication to author. (2) Jones, C. A.: Clinical Studies in Human Strongyloidiasis; Semeiology. Gastroenterology 16: 743-756, December 1950.

Birmingham, Ala., and Houston, Tex. The epidemiologic data indicate that these patients, with few exceptions, live in areas where strongyloidiasis is endemic. In the patients who served in World War II, exposure to polluted soil in endemic areas in the United States is probable. However, over half of these veterans served outside the limits of the United States in areas where strongyloidiasis is also endemic and prevalent. The hazard of infection was probably greater under overseas combat conditions than in these men's civilian occupations or around their homes.

Of the 123 patients in whom a diagnosis of strongyloidiasis was established, approximately 90 percent had gastrointestinal symptoms of mild or severe nature. These symptoms were mainly those of abdominal pain, diarrhea, and allergic manifestations such as urticaria and asthma. Many have had repeated recrudescences of symptoms requiring repeated hospitalization; this has already resulted in an average period of hospitalization of approximately 45 days for each of these World War II veterans. Many of this latter group had no other disease to account for their illness, in contrast to veterans of World War I, many of whom had other chronic diseases to prolong their hospital stay. Data on the first 100 patients with the diagnosis of strongyloidiasis have been reported by Dr. Jones in *Gastroenterology*.

It is interesting to note that Hall [9] reported in 1918 that Dr. C. W. Stiles recommended during World War I that soldiers infected with *S. stercoralis* should be discharged from the service. The reason for this recommendation was that no satisfactory treatment for this condition was known.

It is the opinion of the writer that strongyloidiasis constitutes an important postwar problem among veterans. In many cases, the disease is difficult to diagnose, with the result that infection may be overlooked for long periods. The duration of some infections apparently may be 10 or more years. The natural reduction or loss of infection without treatment appears to be less marked in strongyloidiasis than in many other intestinal helminthiases. Treatment of strongyloidiasis is not satisfactory, although clinical relief may be afforded by repeated courses of gentian violet. The search for an effective therapeutic drug merits investigation and support in order to remove infections acquired during military service. Most of the measures designed to prevent hookworm infection in troops would also serve to prevent strongyloidiasis. The initial mode of infection, that is, penetration of the skin by larvae, is similar in both infections.

Ascariasis and Trichuriasis

Maj. C. A. Kofoid, SnC, 2d Lt. S. I. Kornhauser, SnC, and 2d Lt. J. T. Plate, SnC, recorded a prevalence of 1 percent of ascariasis in overseas troops and a 0.3 percent prevalence in home-service troops in World War I. They also reported that there was a definite indication of acquisition of *Trichuris*

[9] Hall, M. C.: Parasites in War Time. Scient. Monthly 6: 106-115, February 1918.

trichiura, the whipworm, in troops with overseas service during the First World War. The study involved 1,200 overseas and 300 home-service troops of the United States Army. The prevalence of whipworm infection was 6 percent in the overseas troops and 2 percent in the home-service group. This threefold increase was all the more significant if allowance was made for the fact that 5 of the 6 infections in home-service troops were in recent immigrants from Italy and Russia. If these infections were deducted, the prevalence in the remaining home-service troops was 0.3 percent.[10]

In World War II, there were 5,031 admissions of United States Army troops for ascariasis between 1942 and 1945. The admissions for infection with *Ascaris lumbricoides* in overseas theaters totaled 3,630. The remainder, 1,401, represents admissions in the continental United States. In 1944, the largest numbers of admissions for ascariasis occurred in the Mediterranean and European Theaters of Operations. No figures are available for hospital admissions for trichuriasis. Usually only heavy infections with *T. trichiura* produce clinical manifestations. Light infections, which ordinarily are subclinical, are not treated for lack of a completely satisfactory therapeutic agent. A few reports of experiences with these two intestinal nematodes in troops follow.

Capt. D. R. Lincicome, SnC, and 1st Lt. John R. Shaver, SnC, reported a 34.4 percent prevalence of ascariasis in the 13th Engineer Battalion, 7th Infantry Division, which had been in combat on Leyte.[11] An infantry battalion stationed near Manila, P. I., were examined by personnel of the 26th Medical Laboratory, who found 18 percent of the troops infected with *A. lumbricoides* and 10.7 percent infected with *T. trichiura*.[12] Incidentally, a prevalence of 83 percent infected with *A. lumbricoides* was recorded in a group of Philippine civilians who lived adjacent to troops.[13] May [14] surveyed 400 American soldiers interned by the Japanese and found that 35 percent had ascariasis and 40 percent had trichuriasis. *A. lumbricoides* was second in prevalence among parasitic helminths in continental soldiers stationed in the Canal Zone. The infection rate per 1,000 per annum from 1940 to 1945 was 3.96.[15] Intestinal parasitism was found in about 80 percent of 150 Puerto Rican soldiers examined at Fort Brooke. The prevalence of *T. trichiura*, the whipworm, was 55 percent.[16] Other surveys of Puerto Rican troops stationed in the United States revealed prevalence of 53 and 72 percent infected with *T. trichiura* despite postinduc-

[10] (1) Kofoid, C. A., Kornhauser, S. I., and Plate, J. T.: Intestinal Parasites in Overseas and Home Service Troops of the U. S. Army. J. A. M. A. 72:1721, June 1919. (2) The Medical Department of the United States Army in the World War. Communicable and Other Diseases. Washington: U. S. Government Printing Office, 1928, vol. IX, pp. 529-549.

[11] Medical Bulletin No. 19, Headquarters, Army Service Command 1, Office of the Surgeon, Okinawa, 17 Sept. 1945.

[12] Essential Technical Medical Data, Headquarters, U. S. Army Forces, Pacific, October 1945, p. 35.

[13] Semimonthly Report of Activities of the 19th Medical Service Detachment (General Laboratory), U. S. Army Services of Supply, for the First Half of March 1945, to the Chief Surgeon, Headquarters, U. S. Army Services of Supply, Southwest Pacific, 17 Mar. 1945.

[14] May, E. L.: Parasitologic Study of Four Hundred Soldiers Interned by the Japanese. Am. J. Trop. Med. 27: 129-130, March 1947.

[15] Professional History of Internal Medicine in World War II, 1 January 1940 to 1 October 1945, the Panama Canal Department, vol. II, p. 252. [Official record.]

[16] See footnote 6, p. 508.

tion treatment.[17] A routine stool survey of 1,456 insular troops who were admitted to hospitals in the Canal Zone revealed that 76 percent were positive for ova of *T. trichiura*. The whipworm rate in insular troops in the Panama Canal Department per 1,000 per annum as determined by stool examination during hospitalization (1943–45) was 21.31.[18] The 105th General Hospital, which was located in the Southwest Pacific, received a large number of patients who were infected with *T. trichiura*. The infections were asymptomatic, and no treatment was administered.[19]

In general, infections with *A. lumbricoides*, the large intestinal roundworm, and with *T. trichiura*, the whipworm, were of minor medical significance in United States Army troops during World War II. No deaths in troops were caused by either of these parasites. About 5,000 admissions for ascariasis were recorded. Effective anthelmintics were available for therapy of cases of ascariasis. The treatment is of short duration and does not require prolonged hospitalization. Trichuriasis was apparently prevalent in many overseas theaters. High prevalences were recorded in numerous surveys. Most cases of whipworm infection have light worm burdens which represent subclinical infections. Some of the 2,275 admissions listed under "Nematode infection, other" (table 89) possibly include clinical cases of trichuriasis. Since an effective anthelmintic for the treatment of whipworm infections is lacking, search for a satisfactory drug for use in clinical infections should be initiated and supported.

In countries where human feces are commonly used as fertilizer, troops generally were prohibited from serving and eating vegetables raw. The regulation probably prevented numerous cases of ascariasis. Embryonated eggs in feces-polluted soil frequently provide the source of infection. Troops often are, of necessity, in contact with soil from which their hands may easily be contaminated. Ingestion of these eggs will produce infection. Personal hygiene, therefore, is very important in the prevention of ascariasis. Proper disposal of excreta is necessary to prevent spread of the infection.

Enterobiasis

The total number of admissions for *Enterobius vermicularis*, the pinworm, is recorded as 1,272 (table 89). Of these, 889 occurred in overseas troops. The symptoms of many pinworm infections probably were not severe enough to induce men to seek medical treatment. Anal-swab techniques were not universally employed in military establishments for the diagnosis of *Enterobius* infections. Since stool examination is notoriously inefficient for the detection

[17] (1) Report of Disinfestation Program in 762d and 891st Antiaircraft Gun Battalion, by Lt. Col. G. H. Houck, MC, Capt. P. L. Burlingame, SnC, Capt. M. S. Watts, MC, and 1st Lt. G. T. Marconis, MC, Fourth Antiaircraft Command, Post Office Box 3552, San Francisco 19, Calif., 5 Aug. 1945. (2) Letter, Lt. Col. G. H. Houck, MC, Army Air Forces Regional and Convalescent Hospital, Office of Chief of Medical Service, Santa Ana Army Air Base, Calif., to the Surgeon, Headquarters, Fourth Air Force, San Francisco, Calif., 22 June 1945.

[18] See footnote 15, p. 510.

[19] Annual Report of 105th General Hospital, Southwest Pacific, 29 Jan. 1944.

of pinworm infections, the statistics probably do not reflect the degree of parasitism even in clinical cases of enterobiasis in troops. The small number of admissions recorded suggests that *Enterobius* infection was not a serious problem. Pinworm infection reaches high incidence under institutional conditions. Troops living in barracks or otherwise housed in large groups provide similar conditions which might predispose to mass infection with *E. vermicularis*. Viable pinworm ova may occur in household dust, bedclothing, toilet fixtures, and innumerable other objects indoors. Rigid personal hygienic measures and cleanliness of troop quarters are necessary to minimize and suppress this infection.

Trichinosis

There were only 285 hospital and quarters admissions for trichinosis in the United States Army recorded from 1942 through 1945 (table 89). About three-fourths (208) of these admissions occurred in the United States. One death from trichinosis was recorded in 1944. Additional cases were recorded in May 1941 by Maj. Alexander A. Marble, MC, Capt. Allen P. Skoog, MC, and 1st Lt. Donald J. Bucholz, MC.[20] Thirteen soldiers in L Company, 104th Infantry Regiment, 26th Infantry Division, Camp Edwards, Mass., were admitted to the station hospital with clinical trichinosis. Based upon the presence of eosinophilia, it was estimated that from one-third to one-half of Company L (142 troops) probably had asymptomatic trichinosis.

The low incidence of trichinosis in United States Army troops suggests that pork and pork products generally were cooked thoroughly before being served in military installations.

Trichinosis was more of a problem in German prisoners of war than in United States Army troops. Several outbreaks occurred among interned prisoners. Many German prisoners of war preferred and were accustomed to eating meat raw. The outbreaks of trichinosis among these prisoners were due primarily to this habit and to the failure of prisoner-of-war cooks to comply with clear and precise instructions to cook all pork and pork products thoroughly before serving to their fellow prisoners. To obtain better compliance with orders for the proper preparation of meat to prevent trichinosis, closer supervision of mess personnel and oral as well as written instruction of all prisoners of war would be necessary. Section VII, Army Services Forces Circular No. 160, 4 May 1945, specifically prohibited permitting German prisoners of war to consume raw pork.

An outbreak of trichinosis occurred at Camp Atterbury, Ind., in December 1945. One hundred and three prisoners of war with trichinosis were admitted to Wakeman General Hospital. The clinical diagnosis was adequately substantiated by laboratory studies. Because of lack of hospital space, 77 others with symptoms similar to those exhibited by patients sent to the hospital

[20] Marble, A., Skoog, A. P., and Bucholz, D. J.: Trichinosis: Report of an Outbreak at Camp Edwards, Massachusetts. Mil. Surgeon 90: 636–643, June 1942.

were admitted to isolation barracks. In addition there were 409 patients with similar but milder symptoms who were kept in their quarters. There were no cases of trichinosis reported in American personnel at the camp. Epidemiologic evidence indicated that canned bacon and pork which were consumed without cooking caused the infections. Eleven prisoners of war from the prisoner-of-war camp at Austin, Ind., who had trichinosis were also sent to Wakeman General Hospital in December 1945.[21]

Eighty-three German prisoners of war with clinical trichinosis were admitted to an Army Air Force regional station hospital in the United States in December 1945.[22] Uncooked sausage presumably provided the source of infection. An outbreak of trichinosis also occurred at Fort Custer, Mich.[23] It resulted in the hospitalization of 256 German prisoners of war. Raw pork sausage which had been made into sandwich spread and inadequately cooked meat loaf, prepared contrary to published orders, presumably were the sources of infection. A board of officers recommended that:

1. All meals should be inspected by American personnel at the time the meal is being served.

2. All mess personnel, American and German, should read, in the presence of the commanding officer of the respective company, all instructions relative to preparation and serving of food to prisoners of war.

3. All members of the respective mess details should sign the above instructions after reading them.

4. Copies of these instructions should be posted in a conspicuous place in each messhall.

CESTODE INFECTIONS

There were 2,036 admissions for cestode infections recorded during the period 1942–45. Of this total, 1,349 admissions were in the United States. Twenty-two admissions for *Echinococcus* infection occurred during the period. Cysticercosis was reported in one patient during 1944 and 1945. There were 2,013 other cases of cestode infection. Most of these were probably due to *Taenia saginata*, the beef tapeworm. Had any cases of cysticercosis occurred in 1942 and 1943, which seems relatively unlikely, they would have been included among 932 of the above 2,013 cases of cestode infection (footnote 5 of table 89).

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[21] (1) Letter, Col. W. O. H. Prosser, MC, Post Surgeon, Camp Atterbury, Ind., to the Commanding General, Fifth Service Command, Army Service Forces, Fort Hayes, Ohio, 29 Dec. 1945, subject: Investigation of Outbreak of Trichinosis Among German Prisoners of War. (2) Davis, W. A., and Cleland, R. R.: Trichinosis in Prisoners of War. Bull. U. S. Army M. Dept. 7: 973-976, November 1947.

[22] Hathaway, F. H., and Blaney, L.: Trichinosis: Report of an Epidemic. Ann. Int. Med. 26: 250-262, February 1947.

[23] (1) Annual Report, Surgeon, Sixth Service Command, Calendar year 1945, to the Commanding General, Army Service Forces, 27 Feb. 1946. (2) Report of Proceedings of Board of Officers, Army Service Forces, Sixth Service Command, 1611th Service Command Unit, Prisoner of War Camp, Fort Custer, Mich., 17 Mar. 1945. (3) Oppenheim, J. M., Whims, C. B., and Frisch, A. W.: Clinical and Laboratory Observations on 256 Cases of Trichinosis. Bull. U. S. Army M. Dept. 6: 581-593, November 1946.

Echinococcus Infection (Hydatid Disease)

Military personnel were no doubt exposed to *Echinococcus* infection in endemic areas such as New Zealand, Australia, the Mediterranean littoral, and Iceland. Although 15 of the 22 cases of echinococciasis in United States Army troops were admitted to hospitals in the United States, their origin may well have been in overseas theaters either prior to or during World War II. The disease is endemic in the United States, but it is also rare in occurrence.[24] The incubation period in some cases of hydatid disease may be many years in duration. One death from *Echinococcus* infection occurred in 1944. In 1947, Mathieson [25] reported a case of hydatid disease of the lung in a United States Army veteran of World War II. According to Magath,[26] the patient had been in Casablanca, French Morocco, 4 months, another 8 months elsewhere in North Africa, and 16 months in and about Naples and elsewhere in southern Italy. The only contact which the patient had with dogs was in Italy, where the soldier's infection was most likely acquired.

Occasionally troops kept dogs as pets and unit mascots in overseas areas where hydatid disease was endemic. Trained dogs were also used by military services. Dogs constitute the chief source of human infection with *Echinococcus granulosus*. Since the parasite may, in many cases, require years of development in man before clinical manifestations result, it is possible that this disease may occasionally be detected in the future in veterans. If troops were prohibited from keeping dogs as pets, the hazard of infection might be reduced. However, complete enforcement of such a regulation is difficult to attain.

Cysticercosis

The results of a recent statistical investigation of individual records of cysticercosis indicated that there was actually only one case of this disease. The admission occurred in the Mediterranean theater in 1945. Cysticercosis is a relatively rare infection with the larval stage of *Taenia solium*.

Man is the only host of the pork tapeworm, *T. solium*. Since infection with *Cysticercus cellulosae* ordinarily originates from ingestion of *T. solium* eggs (except in cases of internal autoinfection), contamination of food or drink with human excrement must occur for cysticercosis to be transmitted. Proper disposal of excreta and rigid personal hygiene are necessary to avoid infection. In areas where *T. solium* is heavily endemic, care should be taken to avoid or minimize transmission of the infection from the native population to troops.

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[24] Swartzwelder, J. C.: Echinococcus Infection (Hydatid Disease) in Louisiana. New Orleans M. & S. J. 99:617-619, June 1947.

[25] Mathieson, D. R.: The Present Status of Tropical and Exotic Diseases Among Servicemen. Journal Lancet 67: 37-40, January 1947.

[26] Personal communication to author from T. B. Magath, Mayo Clinic, Rochester, Minn., 18 Aug. 1950.

Other Cestode Infections

The number of other cestode infections was approximately 2,000. *T. saginata* presumably was the commonest tapeworm in these cases. Two-thirds of these cases were admitted to hospitals in the United States. In 1944, the European and Mediterranean theaters and the Central and South Pacific contributed most of the overseas admissions for cestode infections. *Taenia* infections are acquired from eating inadequately cooked beef or pork, depending upon the species of tapeworm involved. The necessity of cooking these meats and their products adequately is obvious. Many of the *T. saginata* infections in troops in the United States may have been acquired by eating in civilian restaurants or in homes as well as in Army installations.

INTRODUCTION OF INFECTIONS INTO THE UNITED STATES

A survey for intestinal helminths in 4,000 soldiers processed for separation at Fort McPherson, Ga., revealed that the prevalence of *A. lumbricoides*, *T. trichiura*, *S. stercoralis*, *E. vermicularis*, and *Hymenolepis nana* infection was less than 1 percent for each. About two-thirds of the group had overseas service, and the remainder had service only in the continental United States. The prevalence indicated a low acquisition rate of these intestinal helminths.[27] Mackie and Sonnenberg[28] found a low prevalence of infection with most helminths in 484 veterans studied. The number of infections for each parasite was *A. lumbricoides*, 7; *S. stercoralis*, 10; *T. trichiura*, 22; and *Taenia* species, 1.

Wright and McCoy predicted that the return of infected troops would make little difference in the public health status of these infections in the United States.[29] The relatively small number of hospital admissions of United States Army troops from 1942 through 1945 (table 89) and the apparently low prevalence of infection found in separatees with overseas service and in veterans, if representative, support the views of Wright and McCoy that these helminth cases in returned servicemen offer no basis for exceptional concern from a public health point of view. The helminths considered in this section are already endemic in this country. The slight increment of infection contributed by returned troops probably is extremely small in comparison with the number of infections already present in the civilian population. This does not necessarily apply to *Ancylostoma duodenale*, the Old-World hookworm.

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[27] Special Article: Survey of Intestinal Parasites in Soldiers Being Separated From Service. Bull. U. S. Army M. Dept. 6: 259-262, September 1946.

[28] Mackie, T. T., and Sonnenberg, B.: Tropical Disease Problems Among Veterans of World War II. Am. J. Trop. Med. 29: 443-451, July 1949.

[29] (1) Wright, W. H.: Present and Post-War Health Problems in Connection With Parasitic Diseases. Science 99: 207-213, 17 Mar. 1944. (2) McCoy, O. R.: Public Health Implications of Tropical and Imported Diseases; Imported Malaria. Am. J. Pub. Health 34: 15-19, January 1944. (3) McCoy, O. R.: Precautions by the Army to Prevent the Introduction of Tropical Diseases. Am. J. Trop. Med. 26: 351-355, May 1946.

SUMMARY

Parasitism of United States Army troops by nematodes and cestodes, with the exception of filarial and hookworm infections, did not constitute a serious military problem during World War II. There is abundant evidence of a high incidence of helminthiases in native inhabitants of certain overseas areas. The practice of basic sanitary preventive measures must have contributed to the over-all low incidence of infection in troops stationed in these areas.

There were 1,243 recorded admissions for strongyloidiasis from 1942 through 1945. About two-thirds of these occurred in overseas theaters. Most of the diagnoses were based upon stool examination which is of limited value as a diagnostic measure for this infection. The similarity between the symptoms of patients with strongyloidiasis and those of troops with anxiety neurosis created a diagnostic problem. Of the common nematode and cestode infections, strongyloidiasis possibly represents the most potential postwar problem among veterans. Measures designed to prevent hookworm infections no doubt were to a great extent operative against strongyloidiasis.

Admissions for ascariasis during this 4-year period numbered 5,031. Of this total, 3,630 were in overseas theaters. In view of the high incidence of ascariasis in native inhabitants of many overseas areas, appropriate preventive measures must be taken to minimize transmission of this infection to troops. Whipworm infection, caused by *T. trichiura*, apparently was frequent in troops in some overseas areas. The incidence of *T. trichiura* was high in native troops from Puerto Rico and the Canal Zone. Whipworm infections usually were subclinical.

The small number of admissions for enterobiasis, 1,272, suggests that this infection did not constitute an important problem in troops during the war. It is known that pinworm infection attains high incidence under institutional conditions. Troops are often housed in large groups. This enhances the opportunity for dissemination of this parasite. Therefore, rigid personal hygienic measures and cleanliness of troop quarters are mandatory if the spread of this infection is to be prevented.

About three-fourths (208) of all hospital admissions for trichinosis (285) in United States Army troops were in the United States. One death from trichinosis occurred in 1944. There were a few outbreaks of trichinosis in German prisoner-of-war camps. These resulted from the habit of some prisoners of eating pork raw and from the failure of prisoner-of-war cooks to comply with instructions to cook all pork and pork products thoroughly. Closer supervision of prisoner-of-war mess personnel and instruction of all prisoners of war would have secured better compliance with orders for the proper preparation of pork to prevent trichinosis. Trichinosis was more of a problem in these prisoners of war than in United States Army troops.

There were over 2,000 admissions for cestode infections in United States Army troops. The majority occurred in the United States. *Echinococcus* or

hydatid infection was recorded 22 times. One death was caused by echinococciasis. The long incubation period of hydatid disease may result in the clinical appearance of this infection in veterans in future years. The danger of acquiring this infection from dogs should be emphasized to troops.

At least one case of cysticercosis occurred in troops. In areas where *T. solium*, the pork tapeworm, is heavily endemic, care should be taken to avoid transmission of infection from natives to troops through fecal contamination of food and drink.

Troops should be instructed to eat only adequately cooked beef at Army installations or at civilian establishments to prevent *T. saginata* infection. The need for such instruction is indicated by the occurrence of approximately 2,000 admissions for cestode infections.

The number of infections with these helminths in troops returned from overseas is small in comparison with the number already present in the civilian population of the United States where the infections are endemic. The return of the troops with these helminthiases probably will not make any significant difference in the public health status of the infections in this country.

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