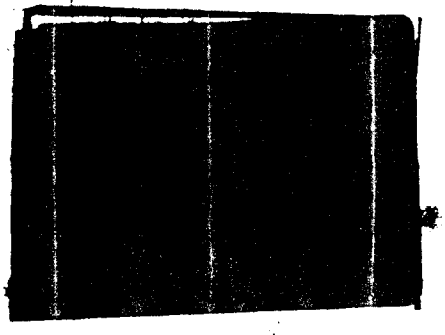
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MEDICAL DEPARTMENT
UNITED STATES ARMY
IN WORLD WAR II

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careful planning for D–day, to be sure that competent planning was being competently implemented.

A large and important part of the administrative background of neurosurgery in World War II concerns personnel. The prewar status of the specialty, even in civilian life, is evident from the fact that it was not until 1941 that the American Board of Neurological Surgery first began to function. The neurosurgeons certified by this Board provided the nucleus of Army neurosurgeons, but it was the special training courses in neurosurgery, set up for young, well-trained general surgeons in civilian medical schools, which provided the majority of neurosurgeons who cared for the neurosurgical casualties of World War II. Intolerable shortages would have occurred in this special field without the outstanding work and judicious placement of these young medical officers.

In World War I, the previous prohibitively high case fatality rate in head injuries was dramatically reduced by the efforts of Colonel Cushing, who served, in effect, as a consultant in neurosurgery in the Surgeon General's Office. He accomplished these results by placement of neurologic surgeons and teams in hospital units near the front, by professional instruction, and by dissemination of detailed instructions for the care of head injuries. The principles of debridement of craniocerebral wounds which Colonel Cushing taught in World War I were the basis of the management of these wounds in World War II. The effects of debridement were enhanced and its risks were reduced by improved technical methods, the use of chemotherapy and antibiotic therapy; modern methods of resuscitation, the bold use of redebridement and delayed debridement when it was necessary, the closure of all dural defects, and the use of such adjunct measures as early ambulation. Debridement, however, remained the keystone.

Of the numerous advances in the management of head injuries in World War II, there should be mentioned cranioplasty with modern agents, especially tantalum; the attention devoted to speech defects in aphasic patients; and the management of posttraumatic epilepsy.

Not the least important and significant chapter in this volume is the one devoted to mortal head injuries. The reluctance of neurosurgeons to admit that any head injury was inevitably lethal is demonstrated by many of the cases described. The corollary of their reluctance was that any wounded soldier who had any chance at all to survive was given the chance, however slim it might be. It was this unconquerable spirit which underlay the brilliant record of neurosurgery in head injuries in World War II.

The editors and authors of this volume have added to their wartime service in the Medical Corps by the time and effort they have devoted to the preparation of this permanent record of their work.

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

Foreword

This volume and a second volume on the same subject now in preparation tell the story of neurosurgery in World War II. The story, as in all other volumes of the history of the Medical Department in this war, includes a frank statement of errors and failures as well as a record of brilliant success achieved in this specialty.

The first neurosurgical volume deals with administrative considerations of neurosurgery and with head injuries. The second volume will cover injuries of the spine, including peripheral nerve injuries and rupture of the intervertebral disk.

As this first volume relates, neurosurgery as a Medical Department specialty was really an outgrowth of World War II. In World War I, in spite of the superb work of a few neurosurgeons, most particularly the late Col. Harvey Cushing, MC, neurosurgery was not a sharply defined specialty as, indeed, it was not in civilian life. After the war, even the slight degree of specialization which had been achieved in the Medical Corps during the war languished completely. Most neurosurgical injuries were handled on orthopedic or general surgery sections of hospitals. Those injuries and conditions which could not thus be treated were referred to civilian neurosurgeons or were cared for in Army hospitals by special arrangements with local neurosurgical consultants. These arrangements, as the text points out, did not constitute a system capable of handling the enormous neurosurgical load which World War II produced. There were no prewar plans for patient loads of such magnitude. The first neurosurgeons, in fact, who entered the service were—very wisely—advised to bring their own instruments and other neurosurgical equipment with them.

In war, as has been repeatedly pointed out, everything must give way to military efficiency. Medical planning is part of the military plan. Without an efficient administrative setup, the most experienced clinician could not apply his best skills to the management of casualties. Not the least important part of this first neurosurgical volume, therefore, is the account of the administrative development of neurosurgery, for it was the administrative background which brought to pass the outstanding clinical care of neurosurgical casualties, the remarkable lowering of the mortality rate as compared with the rate in World War I, and the equally remarkable functional restoration of so many of these wounded men.

Part of the administrative story revolves around the development of neurosurgical centers and their repeated expansion as the casualty load grew heavier. Part of the story concerns the provision of equipment for these centers, the assignment of trained personnel to them, and the evacuation of neurosurgical casualties to them, a policy and practice expedited by the rapid development of air evacuation. Part of the administrative story involves the

Preface

Although the history of neurosurgery in World War II had been conceived many months earlier, the first definite plans for its preparation were not made until March 1942. The comprehensive prospectus which was finally drawn up, in the spring of 1946, differed in many respects from the original outline and reflected the extensive experience with combat-incurred injuries of the intervening months. Work was pursued actively during the first 6 months of 1947, and the completed manuscripts were delivered to the Historical Division, Office of the Surgeon General, in June 1947, with the expectation that they would be published promptly.

This anticipation was not fulfilled, chiefly because of inability on the part of editorial personnel then in the Office of the Surgeon General to agree with us that the story of clinical neurosurgery in World War II is history in the true sense of the word.

When the present determined effort to revive this project was undertaken in 1956, almost 10 years after the original material had been prepared, we were frankly reluctant to resume our activities. It was only when we were assured by the newly appointed director of the Historical Unit, United States Army Medical Service, Office of the Surgeon General, speaking for Maj. Gen. George E. Armstrong, then The Surgeon General, that the manuscripts would be published essentially as they were originally written that we agreed to undertake the task. The material has been re-edited, but the substance is entirely unaltered. In other words, each chapter still represents the original material prepared by the author, at the conclusion of his military service, while all technical matters were fresh in his memory.

We can do no better in this preface than to quote from the preface which was written for the manuscripts prepared for publication in 1947, as follows:

> In the preparation of the history of neurosurgery in World War II, we have endeavored to record faithfully our mistakes as well as our achievements. We have also pointed out the major difficulties under which we labored in that war and the possible solutions for them in future wars. Our objective has been to assemble all pertinent data into two reasonably small, easily readable volumes, which will provide adequate source material for the military neurosurgeons who come after us. We have limited the clinical sections to the three major categories of neurosurgical casualties; that is, head injuries, spinal cord injuries, and peripheral nerve injuries.
>
> The authors for the various chapters were selected on the basis of their interest in the special subjects under discussion and their previous contributions to them. From approximately 200 Army neurosurgeons, an entirely different group might just as well have been selected. Certainly there are missing from the list of contributors the names of many neurosurgeons who might have spoken with complete authority. The opinions expressed by the authors are their own. Editorial revisions were made, but the essential subject matter was not altered in any way by the editors. Unless otherwise indicated, all material in the sections on administration and on the development of clinical policies is derived from the personal

experiences, the official diaries, and the interim and final reports of the authors, supplemented, as necessary, by correspondence and interviews with neurosurgeons who were assigned to the various theaters and areas.

Our gratitude is due to the contributors to these volumes for their cooperation in producing their manuscripts so promptly and so efficiently. Had it not been for their willingness to sacrifice the time which might well have been spent rehabilitating their own private practices after their wartime service, the neurosurgical history of World War II might not have been published for many years, if ever.

In the revived project, there will be two volumes, as originally planned. This volume, the first, deals with (1) administrative details, without which clinical knowledge could not be applied practically; (2) the development of professional policies; and (3) head trauma. The second volume will be devoted exclusively to injuries of the spinal cord and the peripheral nerves.

We are grateful to many persons who have assisted us in the preparation of these volumes, but our first word of appreciation must be to the man who planned the surgical series of the history of the Medical Department of the United States Army in World War II. Had it not been for the late Dr. Fred W. Rankin, formerly Brigadier General, Medical Corps, and Chief Consultant in Surgery, Office of the Surgeon General, there would have been no neurosurgical history. It was under his stimulus that we began to produce it.

Also, there would have been no revival of the neurosurgical history except for the stimulus of Col. John Boyd Coates, Jr., MC, Director, Historical Unit, United States Army Medical Service, and Editor in Chief of the Army Medical Department history. He had to overcome many difficulties, including our initial intransigence, when we declined to have anything to do with the resurrected project, and he deserves our special thanks and appreciation for his determination and his cordial and intelligent cooperation in our work.

Through the years, we have come to know with high regard and personal esteem four Surgeons General of the Army, Maj. Gen. Norman T. Kirk, Maj. Gen. Raymond W. Bliss, Maj. Gen. George E. Armstrong, and Maj. Gen. S. B. Hays, presently The Surgeon General. They have all been helpful to us at various stages in the production of these volumes.

Finally, and perhaps most important of all, we are indebted to Miss Elizabeth M. McFetridge, who did the major editorial work on every chapter in these volumes. Without her efficient help we could not possibly have accomplished our task.

R. GLEN SPURLING, M. D.
BARNES WOODHALL, M. D.

NEUROSURGERY

Volume I

MEDICAL DEPARTMENT, UNITED STATES ARMY

The volumes comprising the official history of the Medical Department of the U. S. Army in World War II are prepared by The Historical Unit, U. S. Army Medical Service, and published under the direction of The Surgeon General, U. S. 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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United States Army Dental Service in World War II

MEDICAL DEPARTMENT, UNITED STATES ARMY

SURGERY IN WORLD WAR II

NEUROSURGERY

Volume I

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Part I

ADMINISTRATIVE CONSIDERATIONS IN NEUROSURGERY

CHAPTER I

The Zone of Interior

Barnes Woodhall, M. D.

HISTORICAL NOTE

As background for an account of the development of administrative and clinical policies in neurosurgery in World War II, it may be useful to outline briefly the comparable policies in effect in World War I,[1] as well as in the interim between the two wars. It should be noted at the outset that in World War I neurosurgery was not a sharply defined specialty as it was in World War II and that neurosurgical cases were for the most part handled under other categories.

Although a Division of Neurology and Psychiatry was created in the Surgeon General's Office in July 1917, medical officers with special training in these fields were seldom utilized in accordance with their training. The service at Letterman General Hospital, San Francisco, Calif., was almost the only exception. When the Surgeon General's Office was reorganized in November 1918, the Division of Neurology and Psychiatry ceased to exist as such and was absorbed into the Division of Internal Medicine. When the armistice was signed on 11 November of that year, a total of 693 officers with some training in neurology or psychiatry or, occasionally, in both fields were in service, 430 in the United States and 263 overseas.

Very few opportunities for training in either neurology or psychiatry had existed before the war. During the war, 6-week courses in these specialties were given at various medical institutions throughout the country. They were attended by between 200 and 300 medical officers, about 20 percent of whom were regarded, at the conclusion of the courses, as qualified specialists.

When a Division of Head Surgery was established in the Surgeon General's Office in July 1917, the plan was that all neurosurgical patients returned to the United States would be cared for in brain sections of special hospitals operating under this division. It was realized that at the close of what was termed the "surgical wound period," patients with injuries involving the nervous system would cease to be surgical and would become neurologic problems, but it would have been impossible, as the Medical Department of the United States

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[1] Bailey, P.: Division of Neurology and Psychiatry. *In* The Medical Department of the United States Army in the World War. Washington: Government Printing Office, 1923, vol. I, pp. 384-394.

Army was then organized, to effect the change from the standpoint of hospital organization.

When neurosurgical patients returned from overseas in greater numbers than had been anticipated, it was found that provisions for their care in the special hospitals established at Cape May and Colonia, N. J., were not adequate in either beds or qualified personnel. Furthermore, the civilian demand that casualties be hospitalized as near their homes as possible had to be taken into account. In response to these needs and demands, more than a dozen general hospitals in various parts of the country were designated to receive neurosurgical casualties, who were assigned to them, as far as possible, on the basis of the nearness of the installations to their homes.

As patients began to be returned from overseas, the Division of Head Surgery in the Surgeon General's Office found it impossible to augment its personnel to meet the expanding demand. Because there was then no special neurologic service in the hospital organization of the Medical Department, neurosurgical patients were sent to divisions of general surgery, to which such neurologists and neurosurgeons as were available were assigned. In the winter of 1918–19, officers with special experience in organic neurology were ordered to general hospitals designated to receive neurosurgical patients, with the recommendation to commanding officers that they be assigned to the surgical services. Although the organizational structure permitted no other arrangement, it was recognized that this plan was not desirable or logical from the clinical standpoint. The surgical necessities of patients who reached the United States, eventually to the number of approximately 5,000, were few. Spinal cord injuries were seldom operable. Secondary surgery was necessary in only occasional head injuries, and not more than 15 percent of peripheral nerve palsies required surgical interference.

The neurosurgical centers of World War I, of which the center at Cape May were representative, were closed promptly after the war, the patient load being transferred to the Veterans' Administration.

In the interim between the wars, injuries of the peripheral nerves and of the spine were cared for on the orthopedic services of general hospitals. Head injuries were assigned to the orthopedic or the general surgery sections. Tumors, tics, brain abscesses, and other neurosurgical conditions were referred to civilian neurosurgeons or were cared for in Army hospitals by local neurosurgical consultants who worked under the contract system.

Without doubt, the system described was efficient for the management of the relatively few neurosurgical injuries which occurred in peacetime in the United States Army and for the similarly few diseases which required specialized neurosurgical care. It was not, however, a system which could envisage in any future war neurosurgical hospital sections of even 150 beds, to say nothing of specialized neurosurgical centers in which the patient census would sometimes reach 2,000 (p.13). There were, moreover, no prewar plans for neurosurgical loads of such magnitudes.

The first neurosurgical service of World War II was set up at Walter Reed General Hospital, Washington, D. C., in May 1942, under Maj. (later Col.) R. Glen Spurling, MC.[2] The peacetime neurosurgical load had not been regarded as sufficiently heavy to warrant the assignment of a full-time neurosurgeon to it. Such neurosurgery as was necessary in the hospital had been done on a contract basis by various neurosurgeons in the immediate vicinity, including the late Dr. Walter E. Dandy of the Johns Hopkins Hospital, Baltimore, Md.

When Major Spurling assumed his duties at the hospital, together with his duties as consultant in neurosurgery to The Surgeon General, the previous status of this specialty was clearly evident. There were no neurosurgical wards; the neurosurgical patients were scattered about all over the hospital. No assistants were assigned to the specialty. None of the nursing staff had had any training or experience in neurosurgery. Electroencephalography was not provided. There was almost no specialized neurosurgical equipment or instruments; Major Spurling, in fact, had been encouraged to bring his own instruments, and he, in turn, made the same suggestion to the neurosurgeons who came into the Army during the next several months. His first official position was assistant chief of the surgical service; later, he served as acting head of the service. As a result, he spent much of his time in administrative and general surgical duties, as did the few other neurosurgeons then in service.

EARLY NEUROSURGICAL PLANNING IN WORLD WAR II

Establishment of Special Centers

Shortly after a neurosurgical service had been set up, as just related, at Walter Reed General Hospital, The Surgeon General authorized the development of similar services in other general hospitals. The details of organization were entrusted to Col. (later Brig. Gen.) Fred W. Rankin, MC, Chief Consultant in Surgery, Office of the Surgeon General, and they occupied much of Major Spurling's time for the next year.

Almost from the first, the planning contemplated the establishment of special neurosurgical centers, in charge of highly trained neurosurgeons, with a staff of younger men to be built up in each center as the work increased. The number of special centers fluctuated as planning proceeded and conditions altered, but the basic policies were never changed; namely, that all neurosurgical casualties received from overseas theaters would be managed in these centers, that all elective neurosurgery would be performed in them, and that clinical conditions would be handled uniformly in all.

The first plan, that five neurosurgical centers be located strategically, to serve designated areas of the country, had to be discarded because it was im-

[2] When he entered the Medical Corps, Major Spurling was Clinical Professor of Surgery (Neurosurgery) at the University of Louisville School of Medicine and Secretary of the American Board of Neurological Surgery. His intimate acquaintance with the diplomates of the board and with the candidates for certification was to prove of great service in the recruitment and assignment of medical officers trained in neurosurgery.—J. B. C., Jr.

practical to have a single center serve more than one corps area. In August 1942, when corps areas were eliminated and service commands set up, a neurosurgical center was established in each of the nine commands, the assignment of patients to be based, as far as bed capacities permitted, on the relation of the centers to the patients' homes.

It was not possible to predict at this time whether hospitals overseas or those in the Zone of Interior would bear the brunt of definitive neurosurgery; it was already clear, however, even at this early date, that the specialized centers would be kept working at full capacity to handle casualties resulting from hostilities overseas as well as from the training program in the United States. Shortly after the 9 original neurosurgical centers had been designated in fact, the concentrations of troops in training in the South and Southwest required the establishment of 3 additional neurosurgical centers in station hospitals in commands in these areas.

By the middle of August 1942, the following centers were ready for operation, under the direction of the neurosurgeons listed:

Walter Reed General Hospital, Washington, D. C., Maj. R. Glen Spurling.
Lawson General Hospital, Atlanta, Ga., Maj. William P. Van Wagenen.
LaGarde General Hospital, New Orleans, La., Capt. Everett G. Grantham.
O'Reilly General Hospital, Springfield, Mo., Maj. Francis Murphey.
Billings General Hospital, Fort Benjamin Harrison, Ind., Maj. Spencer Braden.
Percy Jones General Hospital, Battle Creek, Mich., Capt. Frank H. Mayfield.
Letterman General Hospital, San Francisco, Calif., Maj. Edgar F. Fincher.
Tilton General Hospital, Fort Dix, N. J., Maj. Rupert B. Raney.
Hoff General Hospital, Santa Barbara, Calif., Maj. David L. Reeves.
Lovell General Hospital, Fort Devens, Mass., Capt. Walter G. Haynes.
Fort Sam Houston Station Hospital, Tex., Capt. Robert C. L. Robertson.
Fort Bragg Station Hospital, N. C., Maj. Augustus McCravey.

Conference on Neurosurgery

Although some neurosurgical centers had been established earlier, the beginning of neurosurgery as a specialty in World War II may be said to date from the conference held at Walter Reed General Hospital, 31 August–5 September 1942. It was initiated by Major Spurling, in charge of the neurosurgical center at this installation, in a memorandum to The Surgeon General, 15 August 1942. The substance of this memorandum was as follows:

Although contract service by civilian physicians had in the past provided specialized care as necessary for military personnel, the great expansion in the size of the Army now justified the placing of specialists at hospitals strategically located in the various service commands. In line with this necessity, the Office of the Surgeon General was endeavoring to staff neurosurgical centers in Army general hospitals by securing, from civil practice, neurosurgeons of wide

experience and proved surgical ability, who had been certified by the American Board of Neurological Surgery. The experience at Walter Reed General Hospital indicated how rapidly neurosurgical services were likely to expand elsewhere. Before the establishment of a special neurosurgical service in May 1942, only occasional neurosurgical operations had been performed. Since that date, there had been an average of three major operations in this field each week, including operations for brain tumors, brain abscesses, brain scars with and without skull defects, subdural hematomas, open wounds of the skull and brain, cerebrospinal fluid fistulas, tumors of the spinal cord, progressive exophthalmos, and ruptured intervertebral disk. The latter condition had become a major military problem and would, of itself, amply justify the establishment of a special neurosurgical service, especially since the introduction of Pantopaque (iophendylate) for diagnosis.[3]

At the time this memorandum was written, the patient load in the neurosurgical centers was made up of officers and enlisted men in training camps and battle casualties requiring reconstructive surgery. In some locations, dependents were included in the neurosurgical load. It was expected that the patient load in these centers would increase steadily and that overseas casualties with neurosurgical injuries would eventually be surpassed in number only by orthopedic casualties.

The conference proposed in this memorandum began on 31 August 1942, at Walter Reed General Hospital, and lasted for 6 days. Its purpose was stated to be the establishment of uniform neurosurgical policies, particularly concerning ruptured intervertebral disks. Those present included:

From the Office of the Surgeon General.—Col. Arden Freer, MC, and Colonel Rankin, MC;[4] Lt. Col. William L. Porter, MC, and Lt. Col. (later Col.) Leonard T. Peterson, MC; and Capt. (later Lt. Col.) Albert G. Clark, MC.

From the neurosurgical centers.—Maj. (later Lt. Col.) Spencer Braden, MC; Maj. (later Col.) Joseph M. Cameron, MC; Maj. Edgar F. Fincher, MC; Maj. (later Lt. Col.) John W. Kane, MC; Maj. (later Lt. Col.) Augustus McCravey, MC; Maj. (later Lt. Col.) Francis Murphey, MC; Maj. Rupert B. Raney, MC; Maj. (later Lt. Col.) David L. Reeves, MC; Maj. (later Lt. Col.) Max Schnitker, MC; Maj. (later Col.) R. Glen Spurling, MC; Maj. (later Lt. Col.) William P. Van Wagenen, MC; Capt. (later Lt. Col.) Everett G. Grantham, MC; Capt. (later Lt. Col.) Frank H. Mayfield, MC; Capt. (later Maj.) William Patton, MC; and Capt. (later Lt. Col.) Robert C. L. Robertson, MC.

From the Navy.—Comdr. (later Rear Adm.) Winchell Craig (MC) USNR; Lt. Comdr. (later Capt.) James C. White (MC) USNR; and Lt. (later Lt. Comdr.) Robert H. Pudenz (MC) USNR.

Also in attendance were Lt. Col. (later Col.) Loyal Davis, MC, who had

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[3] Intervertebral disk as a military problem is discussed in detail *in* Medical Department, United States Army. Surgery in World War II. Neurosurgery, vol. II. [In preparation.]

[4] It is fitting, at this point, to mention the services to neurosurgery rendered in World War II by the late Brig. Gen. Fred W. Rankin, MC, Chief Consultant in Surgery, Office of the Surgeon General. General Rankin's particular training and experience were in large-bowel surgery, but he so thoroughly understood the general principles of surgical practice and their universal applicability to all specialties that the success of the neurosurgical program in World War II can be largely attributed to his endeavors.

been designated as Senior Consultant in Neurological Surgery, Office of the Chief Surgeon, ETOUSA (European Theater of Operations, United States Army), and Dr W. Jason Mixter, of Boston, Mass.

Colonel Rankin pointed out, in opening the conference, that its purpose was "to discuss the controversial subjects pertaining to wartime neurosurgery and to make certain recommendations for the guidance of those concerned." Both the discussion and the recommendations were necessary, he observed, because up to this time methods of neurosurgical therapy had been left entirely to the discretion of individual neurosurgeons, subject only to the approval of the chiefs of the surgical services, and uniformity of practice had been completely lacking.

Clinical subjects discussed at the conference included intractable pain (for which the term "causalgia" had not yet come into general use), craniocerebral injuries, low back pain and sciatic pain, posttraumatic head injury syndromes (still not understood or appreciated in their wartime significance), and injuries of the spinal column and spinal cord. The clinical programs of later conferences, the agenda of which often furnished sharp contrasts to the agenda of earlier conferences, reveal how, as the war progressed, the nature of clinical problems changed, as did concepts of their management.

At this first conference, for instance, there was, understandably, an extensive discussion of the ruptured intervertebral disk. This was a relatively new condition, dating back, in its clinical concepts, only to 1934, and Pantopaque myelography was a new diagnostic technique. Rupture of the lumbar disk was, as already mentioned, to prove a serious wartime problem from the standpoint of loss of manpower days, but it was a problem which was to be solved not by the surgical solution proposed at this conference but, in most cases, by separation of the patient from service. The interest expressed at the conference in paraplegia proved well founded; there is no brighter chapter in the history of neurosurgery in World War II than the competent and compassionate long-term management of injuries of the spinal cord. Tantalum was just being introduced in the treatment of skull defects and peripheral nerve injuries, and its use was to attract a great deal of attention later in the war. Its employment was somewhat controversial as far as nerve injuries were concerned, but there was no debate over the value of tantalum in cranioplasty.

An important action of the conference was the adoption for general use of the neurologic form then in use at Walter Reed General Hospital. The chart in use at the same hospital for cases of ruptured intervertebral disk was also adopted for general use. The hope expressed that a uniform system of recording would be instituted in all neurosurgical centers and would make possible an adequate comparison of results was unfortunately never realized.

Early Dissemination of Neurosurgical Information

Newsletter.—Before the conference at Walter Reed General Hospital, neurosurgeons at the various neurosurgical centers had no means, other than

personal letters or other personal contacts, of exchanging information with each other. One of the most useful actions of the conference therefore proved to be the adoption of a plan to distribute a monthly newsletter to all neurosurgeons in service. These letters were to be prepared by the chiefs of neurosurgical sections in the specialized centers and were to be both informal and unofficial. In fact, the mention made of this action in the minutes of the meeting was subsequently deleted.

In spite of their unofficial character, these newsletters were to prove of great importance and value because, until official directives began to be issued, they formed the basis of neurosurgical practice in the Army. The enthusiasm with which they were received, as well as the requests for them from officers to whom they were not distributed, furnishes implicit evidence of the inexperience of neurosurgeons drawn from civilian life in the management of combat-incurred injuries and of their eagerness to compensate for it.[5]

Inter-Hospital Letter No. 2, dated 4 January 1943, makes clear the type of information included in these communications:

* * * * * * *

Major R. Glen Spurling, Walter Reed General Hospital, Washington, D. C. November 25, 1942.—* * * we are opening our new neurosurgical ward tomorrow morning. We have forty-nine beds and it is a beautiful set-up * * *. We are expecting a large group of patients soon. I will give you more information about how they are distributed, particularly the amount of neurosurgery in each hundred casualties, at a later date. This report will be my next contribution to the Monthly Letter, for I think it will indicate to all of you just what you may expect when you get a bunch of casualties.

* * * * * * *

Major Francis Murphey, O'Reilly General Hospital, Springfield, Mo., December 10, 1942.—There is very little to report from here in regard to the service. I have a ward of my own and am naturally responsible for the ward administration, the property as well as writing the histories and preparing the patients for disposition, whether they are returned to duty or C. D. D.

So far I have done about fourteen discs and have already sent two of them back to full duty. I have been held up, of course, because I could not get any Pantopaque. I did finally get six more ampules from Van Wagenen. I have had very little else in the way of surgery except a couple of peripheral nerves and that brain tumor which I had to come back to operate on, which, incidentally, was a glioblastoma * * * and a brain abscess.

I have only one suggestion for the letter and that is in the technique of injecting the Pantopaque. Shortly after I arrived, I had too much difficulty doing the puncture with the patient on his abdomen, so at the present time I do the puncture with the patient on his side in the old-fashioned way and, with the needle still in him, turn him over on his abdomen. I then check to see whether the needle is still in the canal and inject the Pantopaque then. It is just as easy to remove the Pantopaque after the puncture has been done in this manner and saves an incredible amount of time. So far I haven't broken any needles off.

Lt. Robert H. Pudenz, Naval Medical Center, Bethesda, Md., December 17, 1942.—* * * I have just returned from ten days' leave in Montreal, where I picked up a few pearls which may be of interest to the group:

1. Drs. Penfield, Cone, and Elvidge have performed tantalum cranioplasty in eight instances. All of the cases have healed per primam. The longest postoperative period in

[5] The round robin letter prepared and distributed by the American Academy of Neurological Surgery proved of equal value and was received with equal enthusiasm. Its contents were provided by both civilian physicians and medical officers of the Armed Forces.

any one case is about ten months. In one patient a tantalum plate was inserted to fill the skull defect resulting from removal of a neoplasm invading the skull. The neoplasm proved to be malignant and the plate was subsequently removed for radiotherapeutic reasons. Tantalum will allow passage of only 14% of the X-rays.

2. Dr. Arthur Childe informed me that they are using Pantopaque almost exclusively for myelography. When difficulties in procurement present themselves, they have substituted Lipiodol. Their series of cases now exceeds 80. They have not experienced any untoward reaction to the use of this material. No case has been explored unless there is a definite defect in the column on X-ray.

3. There are some very interesting results in the studies of the post-traumatic syndrome, motion sickness, blackout, cerebral edema and the local use of the sulfonamides on the brain. Unfortunately, this work is in the confidential category of the National Research Council and I had better get permission to report their findings. Will try to do so by the time the next circular is ready for publication.

* * * * * * *

Captain Frank H. Mayfield, Percy Jones General Hospital, Battle Creek, Michigan, January 18, 1943.—* * * we received 40 patients on January 15th and had formal flag raising and all.

Today I am doing the Pantopaque myelography. It is the first procedure on the operative schedule. Wednesday I will operate on this man. Glen Spurling will be in town that day and I am holding the case to put on for him * * *.

Data from overseas.—Meantime, Colonel Davis, who had been in the European theater as senior consultant in neurological surgery since early in September 1942, submitted certain observations for transmission to neurologic surgeons in the general hospitals in the United States. His remarks were endorsed by Brig. Gen. (later Maj. Gen.) Paul R. Hawley, Chief Surgeon, ETOUSA, as follows:

The exchange of information with experts in the United States is believed to be highly desirable and is approved in this office. It is hoped, however, that this will be an exchange and not, as has been the case up to this time, information traveling in one direction only.

In spite of General Hawley's emphasis on the importance of a free exchange of information and although reports from overseas were prepared frequently and were maintained on a high level of authority, neurosurgeons in the Zone of Interior never found a satisfactory method of implementing his suggestion.

Since Colonel Davis' notes represent one of the first reports on neurosurgery from overseas, his observations are reproduced in full:

After a short initial tour to acquire information concerning neurological surgery in this theatre, the following observations seem to me to be pertinent, although not entirely new in all instances:

1. The length of the period of posttraumatic amnesia following craniocerebral injury is an important factor in determining the prognosis.

2. Patients with craniocerebral injuries should be got out of bed as quickly as possible after they are oriented and are in a stable physical condition. Various types of occupational therapy should be instituted immediately. I have seen enough evidence to change my opinion that such injuries should be kept flat in bed for any given time.

3. It has been possible to return to military duty over 60% of patients with craniocerebral injuries of all types.

4. It is important that all neurological surgeons in the medical corps of the United States Army should keep complete and detailed records of their patients so that all of the material may be pooled after the duration for a significant contribution to military neurological surgery.

5. It is important that careful records of diagnosis, examination, and operative notes accompany neurosurgical patients who are invalided to the United States for further treatment in U. S. General Hospitals so that their therapy may be continued without interruption.

6. Enough clinical evidence should be gathered quickly by neurological surgeons in the United States so that Pantopaque for myelograms can be placed at the disposal of neurological surgeons in this theatre.

7. Craniocerebral injuries which have been immediately debrided superficially, had the head shaven, cleansed carefully, sulfanilamide powder introduced, and a sterile dressing applied, have been operated upon definitively as long as 36 hours later with primary healing of the wound.

8. In one quarter it is believed that delayed secondary peripheral nerve sutures are followed by more perfect nerve healing than primary sutures. I did not see sufficient evidence to justify me in accepting this viewpoint which would mean changing my opinion that a primary end-to-end suture immediately after the receipt of the injury, without or with the use of sulfanilamide in the wound, is the operation of choice.

9. Electrotherapy at frequent intervals coupled with massage and passive movement of the effector mechanisms will beneficially affect muscle atrophy following peripheral nerve injury. The benefit comes through increasing the blood supply to the muscles paralyzed.

10. A series of unilateral and contralateral subdural hematomas have been found following various types of craniocerebral injuries, with symptoms, resembling in their onset, those of middle meningeal hemorrhage.[6]

EXPANSION OF NEUROSURGICAL CENTERS

Early in 1943, it became evident that neurosurgical casualties received in the Zone of Interior might be next in importance to orthopedic casualties in respect to numbers, length of treatment, and proportion of permanent disability. Few neurosurgical patients were evacuated from the Pacific as the aftermath of Pearl Harbor, but, in the first 135 casualties received from North Africa, there were 24 neurosurgical patients (18 percent of the total), who presented 16 peripheral nerve injuries, 4 penetrating wounds of the brain, 4 closed head injuries, and 1 gunshot wound of the spine with paraplegia.[7] The evident need of these patients for specialized neurosurgical treatment, as well as other types of specialized treatment, fully justified the effort already expended in the development of what might be termed "one-man" centers and also emphasized the need for still further expansion of this plan.

War Department Memorandum No. W40–9–43, dated 6 March 1943, definitely established the role of neurosurgery as well as of other specialties in the United States Army Medical Department.[8] It specified that all patients

[6] In a comment on Colonel Davis' notes, Colonel Rankin pointed out that practically all the points mentioned in them had been covered quite well at the neurosurgical conference held at Walter Reed General Hospital. He doubted that it would be possible to return to military duty 60 percent of casualties with combat-incurred head injuries. Colonel Davis' figures referred to closed head injuries, and later events proved Colonel Rankin's doubts entirely justified.

[7] One patient had two injuries.

[8] Some months earlier, neurosurgical conditions had been defined as surgical lesions of the brain, spinal cord, peripheral nerves, and sympathetic nervous system.

received from overseas who required specialized treatment should be classified by the general hospitals receiving them according to types of treatment required and should be promptly reported to The Surgeon General for transfer to an appropriate designated specialized hospital. Neurosurgical centers were designated in this memorandum as follows:

Ashford General Hospital, White Sulphur Springs, W. Va.
Brooke General Hospital, Fort Sam Houston, Tex.
Bushnell General Hospital, Brigham City, Utah
Fitzsimons General Hospital, Denver, Colo.
Hoff General Hospital, Santa Barbara, Calif.
Kennedy General Hospital, Memphis, Tenn.
Lawson General Hospital, Atlanta, Ga.
Lovell General Hospital, Fort Devens, Mass.
McCaw General Hospital, Walla Walla, Wash.
McCloskey General Hospital, Temple, Tex.
Nichols General Hospital, Louisville, Ky.
O'Reilly General Hospital, Springfield, Mo.
Percy Jones General Hospital, Battle Creek, Mich.
Schick General Hospital, Clinton, Iowa
Tilton General Hospital, Fort Dix, N. J.
Walter Reed General Hospital, Washington, D. C.

These centers were strategically located in relation to the distribution of the actual and anticipated neurosurgical load as of March 1943. Centers at the station hospitals in the South and Southwest, as well as at the general hospitals originally designated as centers but not included in this list, were gradually closed, the casualty loads in these particular locations having so altered as not to justify their continuance. By midsummer 1943, changes in the casualty load and its geographic distribution again necessitated changes in the location of the neurosurgical centers. The centers at Lovell General Hospital, Tilton General Hospital, and Hoff General Hospital were transferred to Cushing General Hospital, Framingham, Mass., England General Hospital, Atlantic City, N. J., and Birmingham General Hospital, Van Nuys, Calif., respectively, and additional centers were established at DeWitt General Hospital, Auburn, Calif., and Wakeman General Hospital, Camp Atterbury, Ind. Special neurosurgical sections, with accredited neurosurgeons in charge, were also established at Halloran General Hospital, Staten Island, N. Y., and Letterman General Hospital.

The adequate care of neurosurgical casualties involved not only the establishment of neurosurgical centers and shifts in their locations as regional casualty loads increased or decreased but also the expansion of the centers to several times their original size. The progressive rise in neurosurgical casualties after November 1943, when hostilities in overseas theaters became more active, made it necessary to raise the original 150-bed ceilings in the centers to between 350- and 500-bed ceilings in general hospitals which had average bed capacities

of 1,500 to 2,000 patients. By September 1944, when formal plans were made for neurosurgical ceilings of these levels, continued expansion in most of the centers had already forced a bed census approaching them. At this time, the neurosurgical center at Fitzsimons General Hospital was transferred to Newton D. Baker General Hospital, Martinsburg, W. Va., the center in Schick General Hospital was transferred to Mayo General Hospital, Galesburg, Ill., and the center at Birmingham General Hospital was transferred to Hammond General Hospital, Modesto, Calif. An additional center was established at Northington General Hospital, Tuscaloosa, Ala.

After this reorganization, the 19 neurosurgical centers, which were staffed by 88 medical officers designated as neurosurgeons, had a bed capacity of 9,250 distributed as follows:

General hospital	*Number of beds*
Ashford, Hammond, Brooke, Bushnell, Cushing, England, Newton D. Baker, Halloran, Kennedy, Lawson, McCaw, Nichols, O'Reilly, Percy Jones, Mayo, Wakeman, and Northington	500 each
Walter Reed	400
DeWitt	350

Before the war ended, the neurosurgical load at O'Reilly General Hospital and Halloran General Hospital had sometimes reached 2,000 patients. Even more indicative of the growing importance of neurosurgery was the rapid development of this specialty at Walter Reed General Hospital in 1942, 1943, and 1944 (table 1). In his annual report dated 10 January 1943, Major Spurling recorded that, between the establishment of the service in May 1942 and the end of the year, he had performed 105 major neurosurgical procedures, with only 2 postoperative deaths, both in patients with hopeless malignancies. These figures did not include encephalograms, arteriograms, and myelograms. In August, a department of encephalography had been established, in charge of an experienced organic neurologist. In November, a neurosurgical ward had been set up, with provision for 49 patients and with its own ward officer. The majority of conditions treated in 1942 were still of the kind encountered in civilian practice, but casualties had been received and treated from Pearl Harbor, Australia, and Burma, and casualties from North Africa were beginning to be received.

During 1943, the rising casualty load was reflected in both the number and the kind of neurosurgical admissions. In August 1943, neurosurgical facilities were increased to 56 active beds and 60 convalescent beds; these wards had originally belonged to general surgery, in which the census had decreased as the neurosurgical census increased. In October 1943, a second neurosurgical operating table was provided, and the average weekly neurosurgical operations rose from 6 to between 15 and 20.

Over this same period, the recruitment of two nurses with neurosurgical training and experience had provided for instruction of hospital nurses in this specialty. Unfortunately, the constant shifting of nurses from section to

TABLE 1.—*Workload of the Neurosurgical Center, Walter Reed General Hospital, 1943–44*

Category	Admissions	
	1943	1944
Direct admissions	503	1,138
Admissions from—		
Medical service	71	84
Other surgical services	191	319
Deaths	11	20
Consultations	881	1,114
Dressings	3,352	7,695
Pneumoencephalograms	71	55
Myelograms	280	379
Operations	487	858
Alcohol nerve block	.....	(9)
Aneurysmorrhaphy	.....	(1)
Angiogram	(3)	(2)
Appendectomy	.....	(6)
Biopsy of muscle	.....	(1)
Cranioplasty	.....	(60)
Cranioplasty, with use of tantalum plate	(15)	.....
Craniotomy	(34)	(54)
Debridement	.....	(14)
Debridement, with suture of scalp laceration	(10)	.....
Decompression of nerve root	.....	(2)
Elevation of depressed fracture of skull	(6)	.....
Excision of—		
Amputation neuroma	(10)	.....
Carotid sinus	.....	(1)
Cicatrix	(3)	(46)
Neurofibroma	.....	(2)
Neuroma	(35)	.....
Scalp wound	.....	(1)
Scar	.....	(4)
Sinus	.....	(4)
Subcutaneous tumor	.....	(5)
Exploration of—		
Axilla	.....	(1)
Brachial plexus	.....	(1)
First stage graft	.....	(3)
Nerve	(9)	(24)
Wound	.....	(9)
Evacuation of—		
Epidural abscess of cord	(1)	.....

Category	Admissions	
	1943	1944
Operations—Continued		
Sterile abscess	(1)	.....
Fixation of scapula	.....	(4)
Fusion of cervical spine	.....	(1)
Hemilaminectomy and spinal fusion	.....	(1)
Incision and drainage of abscess	.....	(3)
Interlaminar exploration	.....	(31)
Laminectomy	(23)	(11)
Laminectomy, with removal of foreign body	.....	(1)
Laparotomy	.....	(1)
Ligation of carotid artery	.....	(2)
Myeomectomy	.....	(1)
Naffziger decompression	.....	(1)
Needle exploration of abscess of brain	(2)	.....
Nerve graft	(28)	(26)
Neurectomy	(2)	(4)
Neurolysis	(55)	(131)
Neurolysis, with endoaneurysmorrhaphy	(1)	.....
Neurorrhaphy	(40)	(148)
Osteoplasty	(1)	.....
Periarterial sympathectomy	.....	(1)
Removal of—		
Foreign body	.....	(6)
Ruptured intervertebral disk	(155)	(150)
Tumor of muscle	.....	(1)
Resection of nerve graft	.....	(4)
Resuture of wound	(1)	.....
Revision of wound	.....	(16)
Scalenotomy	(10)	(9)
Secondary closure of wound	.....	(3)
Section of—		
Facial artery	.....	(2)
Nerve	.....	(1)
Sequestrectomy	.....	(1)
Spinofacial anastomosis	.....	(3)
Sympathectomy	.....	(2)
Transplantation of nerve	.....	(4)
Trephine, exploratory	(20)	(12)
Ventriculogram	(20)	(28)
Ventriculostomy	.....	(1)

NOTE.—Figures in parentheses are subtotals.

section made detailed training very difficult. Some enlisted men rendered excellent service in nursing capacities, but the great majority were insufficiently trained for the exacting demands of neurosurgery and never equaled nurses in their ability. During most of the war, it was possible to keep sterile nurses with special training in neurosurgery in their positions in the operating rooms. Before the war ended, however, not a single scrub nurse remained at the instrument tables at this hospital.

In June 1942, the first set of neurosurgical instruments was received at Walter Reed General Hospital. The committee which had selected them had

chosen wisely and well, but, with few exceptions, these tools were poor reproductions of excellent instruments. The drills, the lighted retractors, and the rongeurs were all of good quality, but the silver-clip outfit was classified by Major Spurling as perfectly hopeless. By demonstrating to the supply officers the deficiencies of this equipment as compared to his personal equipment and by pointing out to them how readily a life could be lost from hemorrhage because of these deficiencies, Major Spurling was able to convince them that the original contracts must be cancelled and new specifications for neurosurgical equipment written. It was well into 1943, however, before the problem of neurosurgical equipment and supply was on its way to solution. In February 1943, Major Spurling noted in his official diary that the new silver-clip equipment was not yet ready and that it had taken more than 3 months to secure a supply of Pantopaque.

Organization of 500-Bed Neurosurgical Centers

Personnel.—The authorized basic personnel for a neurosurgical center with a patient load of 500 consisted of a chief of section, 2 assistants, and 1 ward officer for each 100 patients. The staff was fluid, and assistants and ward officers were increased as the patient load increased. At many centers, the authorized staff was never approached. At Percy Jones General Hospital, for instance, when the neurosurgical census was 1,780, the staff consisted of 2 neurosurgeons, 3 general surgeons with neurosurgical training, and 6 ward officers, none with any neurosurgical training at all. In November 1945, when the neurosurgical census was 1,450 patients, the staff consisted of a total of 8 officers. Shortages of ward officers were particularly common and were seriously felt.

The chief of section in all neurosurgical centers was a neurosurgeon certified by the American Board of Neurological Surgery and capable of intervening, with a reasonable hope of success, in any neurosurgical problem, whether of military origin or related to neurosurgical injuries and conditions observed in civilian practice. He carried the administrative responsibility of the section but also gave his personal attention to such difficult neurosurgical conditions as tumors and abscesses and to obscure neurosurgical problems.

Because of the preoccupation of the chief of section with these matters, the assistant neurosurgeons formed the backbone of the neurosurgical effort in the specialized centers. They might not be able to cope with all problems, but in most of the centers these officers were well trained, expert surgeons, who were able to evaluate properly and treat adequately the vast numbers of peripheral nerve injuries and skull defects which comprised the workload. They also operated on the ruptured intervertebral disks which formed a large part of the workload until surgical management of this condition was prohibited.

One point not always taken into consideration by hospital commanders and others in the analysis of neurosurgical workloads was that all neurosurgical procedures are long and that some of them are very long. At the center at

O'Reilly General Hospital, for instance, three neurosurgeons, by spending half of their time in the operating room, managed to average about 90 surgical cases a month. This was a good record, but it did not meet the necessities of the situation. In some instances, operation was delayed so long that it was not as effective as it might have been if it had been performed earlier.

All neurosurgeons carried heavy workloads and worked very long hours. At one general hospital, when the chief of surgery ordered all personnel to work daily until 2100 hours, it was found that the neurosurgical personnel were already working until that time and even later.

The tasks of the ward officer in the neurosurgical center resembled those of an interne in any hospital. It was not feasible, from any point of view, to make him a permanent member of the neurosurgical staff, chiefly because of the fluctuating needs of the services in all hospitals. With the increasing tendency, as the war progressed, to limit the number of special centers in a single hospital, it was possible, under the specific table of organization for the hospital to recruit ward officers as they were needed from various sources, such as other services with lessened responsibilities, limited-duty personnel, and floating personnel undergoing indoctrination.

There was not, of course, general agreement with this point of view about rotation of officers. Some chiefs of sections stated that, if a similar situation ever arose again, fixed ward officer personnel should be authorized, because their rapid turnover militated against the ideal management of neurosurgical casualties. The theory is sound, but the realities make it doubtful that fixed ward officer personnel would ever be justified in a neurosurgical center.

To the more or less permanent staff of the neurosurgical centers were added, at various times, officers on temporary duty, student officers, and officers taking refresher courses. The mainspring and nucleus of a successful neurosurgical center, however, proved to be a permanently assigned and experienced chief of service and his assistants. Their experience, while different from that of neurosurgeons with overseas service, was not gained any more easily, and the point of view was realistic that such a staff should be disturbed as infrequently as was compatible with professional and military necessities. By September 1944, when there was an increasing demand for neurosurgeons in overseas theaters, there was also an increasing workload of rehabilitative neurosurgery in the specialized centers in the Zone of Interior. The Surgeon General therefore gave orders that the personnel of the Zone of Interior centers be frozen, in order to provide a more or less permanent nucleus of neurosurgical personnel for the successful continuation of rehabilitation surgery.[9]

Relationship of specialized centers to general hospital organization.— In most hospitals in the Zone of Interior, the neurosurgical center was a component of the surgical service. The chief of section was responsible to the chief of the surgical service and through him to the commanding officer. On the medical service, the neurologic section was similarly organized. The ad-

[9] Diary, Lt. Col. Durward G. Hall, Director, Military Personnel Division, Personnel Service, Surgeon General's Office, 4 Nov. 1944, section 4: Conference, scarcity of specialists' personnel in the Zone of Interior.

ministrative separation of the neurologic and neurosurgical sections, both of which were vitally concerned with affections of the central and peripheral nervous systems, was not conducive to the utmost efficiency either in the treatment of patients or in the clinical and experimental study of problems of common interest as, for instance, open head injuries (p. 91). It was realized early in the war that a more rational plan of organization would effect a closer relationship between neurosurgeon and neurologist by which their individual identities would be preserved but at the same time their efforts would be fused and the study and treatment of diseases and injuries of common interest would be carried out in the same ward space. The pressure of casualties hastened the evolution of this ideal plan, though it was never completely achieved.

From the standpoint of neurosurgical administration, it would have been highly desirable to divorce neurology and psychiatry and to organize a single section of medical and surgical neurology. Major Spurling pointed this out shortly after the neurosurgical service was organized at Walter Reed General Hospital. At this time, there were 10 neuropsychiatrists, but only 1 organic neurologist, on the neuropsychiatric service. As a result, psychiatrists answered most consultations, and the chances were that a disease that was really organic might sometimes be classified as functional.

Facilities and administration.—The facilities of a neurosurgical center of 500 beds consisted of (1) a diagnostic or preoperative ward; (2) an operative or acute treatment ward, subdivided into a recovery ward, a postoperative ward, and a paraplegic ward; and (3) a convalescent ward (or wards).

The preoperative or diagnostic ward could be operated most efficiently when it was divided into units of 50 patients each, with each unit under a ward officer. Patients in this ward were ambulatory, could go to mess, and were capable of carrying out fatigue duties on the ward under the supervision of an enlisted wardmaster. They presented, in addition to diagnostic problems, the usual types of peripheral nerve injuries, skull defects without disabling neurologic sequelae, and injuries and symptoms referable to the back. After a preliminary survey by the ward officer, the patients in this ward were dispatched to other hospital units for special studies or for consultative review, or even to other hospital installations. At weekly rounds by the chief of section and the staff, decisions were arrived at concerning operation; conservative, nonsurgical therapy; transfer to other therapeutic sections; or some type of military disposition.

The operative or acute treatment ward was also administered in units of 50 patients, each unit being under the immediate care of an assistant neurosurgeon assisted by a ward officer. Patients were admitted to this ward from convoy or from other wards for specialized treatment. When the numbers justified it, special lesions were treated in special subdivisions.

The recovery section of the operative ward was administered as a separate unit, to permit concentration of nursing care for patients who had just been operated on. Patients with acute head injuries or with other acute neurosurgical conditions were also treated in this space.

The main postoperative section of the operative ward contained patients in various stages of recovery from operation, as well as patients undergoing the first stages of rehabilitative treatment for neurosurgical injuries. Patients with septic or chronically infected injuries were isolated in a detached portion of the main postoperative ward or were treated in a separate septic unit. Wound dressings were accomplished from a cart in the main ward, or in adjoining dressing rooms. Casts designed to change the position of the joints in peripheral nerve injuries were changed in the ward under professional supervision; all beds were fitted with overhead frames to facilitate the procedure.

The paraplegic section was part of the main postoperative ward. Patients with paraplegia of traumatic or neoplastic origin were best segregated for medical and nursing care, because of the demanding nature of their care, the nutritional problems involved, and the recurring episodes of bladder and skin infection. Under this plan, the trained personnel who cared for such patients reached their highest peak of efficiency.

Convalescent wards with as many as 200 patients could be supervised under a single ward officer. His tasks, which largely involved channeling treatment and preparing patients for disposition kept him fully occupied. The chief of section made biweekly rounds; the progress of rehabilitation in neurosurgical states, particularly in peripheral nerve injuries, is usually so slow that more frequent observations were not necessary and would not have been useful. As many as 25 patients could be studied at each of these rounds.

Since about 20 percent of all patients with bone and joint injuries had concomitant peripheral nerve injuries, the patient load justified combined neurosurgical-orthopedic wards. This arrangement was found to be most efficient for the management of such casualties, who consisted of both ambulatory and bedridden patients.

Organization of Larger Neurosurgical Centers

In January 1945, in anticipation of the increased casualty load expected by August 1945, the total ceiling for neurosurgical beds in the Zone of Interior was raised from 9,250 to approximately 21,900. Since facilities for further expansion were not available at Brooke General Hospital, the neurosurgical center there was transferred to McGuire General Hospital, Richmond, Va. The center at Walter Reed General Hospital, except for a small neurosurgical section, was transferred, for the same reason, to Halloran General Hospital. The neurosurgical section handled only a few battle casualties, and its activities therefore bore little resemblance to the activities of the other centers.

The ceiling of 21,887 neurosurgical beds allotted in January 1945 represented a practically threefold expansion of the original capacity of the centers. The bed allotment proved none too liberal, and the ceiling was sometimes exceeded in the months before the war ended. Had combat continued, further expansion would undoubtedly have been necessary. It was thought that it

could be accomplished within the centers already established (fig. 1) and that the authorization of additional centers would not be indicated.

The expansion of the bed capacity of the neurosurgical centers was not accompanied by a corresponding increase in professional personnel. At the peak of operations, the 19 centers were staffed by 92 medical officers classified as neurosurgeons, 26 of whom were certified by the American Board of Neurological Surgery. This was a ratio of 1 neurosurgeon to every 237 patients.

The neurosurgical program was also continuously handicapped by a lack of experienced neurologic personnel. Ideally, the diagnostic aspects of all

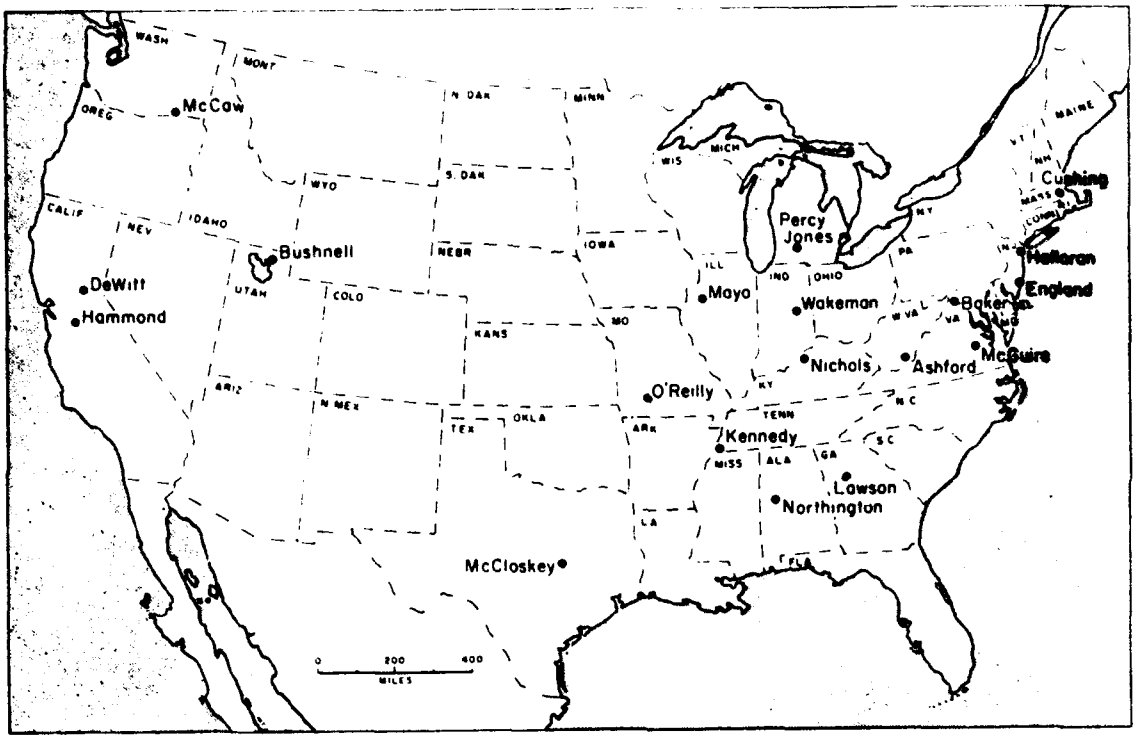


FIGURE 1.— Location of neurosurgical centers in general hospitals in the Zone of Interior in April 1945.

neurosurgical injuries should have been the task of the neurologist, not the neurosurgeon, but it was never possible to secure changes in existing tables of organization which would have provided sufficient neurologic personnel to achieve this objective. This policy, for obvious reasons, would have been particularly desirable in the larger neurosurgical centers.

These were major difficulties, and they prevented the achievement of an ideal organization. Nonetheless, extensive changes in organization were necessarily made when centers with an original capacity of 150 to 500 beds were expanded to centers in which the neurosurgical patient load at times exceeded 2,000. In general, the expanded load required a physical segregation of patients with specific lesions and an administrative separation of the components of diagnosis, treatment, and convalescence or disposition.

In most centers handling a thousand or more neurosurgical casualties, patients were admitted to a common admission ward, from which they were segregated according to whether they needed nonsurgical treatment, neurosurgical treatment, or purely diagnostic surveys. In the latter group were patients with such conditions as closed head injuries, convulsive states, suspected cerebral neoplasms, and sciatica. Patients with cranial injuries, peripheral nerve injuries of the upper and lower extremities, paraplegia, and combined orthopedic-neurologic injuries were further segregated into special wards. This method of assignment promoted efficiency of treatment, led to a better understanding of technical problems, and was conducive to a rapid rate of disposition.

An interesting development of the expanded neurosurgical program was the establishment of so-called nerve laboratories in which unified preoperative and regenerative studies of nerve injuries were carried out. In these laboratories, histories were recorded, photographs were taken, neurologic and electrodiagnostic studies were carried out, and pertinent data were assembled on special forms. The unified methods employed, in addition to facilitating all the studies, permitted a more rapid turnover of patients.

The principles of physical segregation of patients according to their special injuries and the administrative separation of the components of diagnosis, therapy, and disposition were designed (1) to give to the neurologist, when he was available, his proper responsibility for diagnosis; (2) to allow neurosurgeons to spend their time profitably in the operating rooms; and (3) to make ward and administrative officers responsible for the routines of administration and disposition. The use of furlough barracks, where patients returning from furlough were processed in terms of progress, was another useful plan, which prevented the accumulation of ambulatory patients in hospital beds.

Only by a carefully detailed organization such as this could heavy patient loads be efficiently controlled and cared for. Efficient organization was, however, attended with at least one obvious disadvantage, a loss of the doctor-patient relationship. When one group of medical officers worked up patients, another group treated them, and a third group cared for them after operation, there was always the danger that efficiency might degenerate into a production-line type of management as undesirable in military as in civilian practice.

There was also a certain professional loss in the necessity for emphasizing administrative details. As the workload built up, the original plan of staff conferences intended to include not only the neurosurgical staff but the staffs of other sections became increasingly difficult and eventually was abandoned in most of the centers.

Methods of handling the workload varied from center to center. The following (summarized) report from the center at Cushing General Hospital describes a plan of organization which worked admirably when it was tested under heavy pressure.

Organization of the Neurosurgical Center at Cushing General Hospital

The neurologic and neurosurgical sections at Cushing General Hospital always represented a single working unit whether they were combined into a single service, as they were originally, or were separated administratively into medical and surgical services, as they were later. The close cooperation proved as useful from the administrative as from the professional point of view. In times of stress, when there was a heavy influx of patients, the service could be expanded. In quieter periods, it could be contracted. Bed space could be utilized with great efficiency. Personnel and equipment could be shifted from one section to another without delay, according to current requirements. The division of work between the sections made it possible to keep abreast of incoming patients, and, even when large convoys arrived, there was no backlog. The neurosurgeon, who was freed simultaneously from the time-consuming initial examination of patients and from the inevitable paperwork connected with their later observation and final disposition, was enabled to devote his full time to the surgery which was his primary task.

Routine of admission and diagnosis.—Patients who presented neurosurgical emergencies, patients with ruptured intervertebral disks, and paraplegics were admitted directly to the wards on which they were to be treated. All other patients were admitted to the neurologic section, which was set up as an admitting department for both neurologic and neurosurgical patients. Here, within a week of his admission to the hospital, every patient was thoroughly worked up under a routine which was elaborately detailed, yet which operated almost on the principles of an assembly line. One routine was used for head injuries and another for peripheral nerve injuries. Material from both types of injuries was collected in a register for later statistical evaluation.

The objective of the system of examination used at the Cushing General Hospital Neurosurgical Center was twofold: (1) To shorten the time of hospitalization and thus to free beds as promptly as possible for new casualties; and (2) to record neurologic deviations from the normal in numerical values which could be reproduced and reexamined comparatively; that is, in such physical terms as kilograms, millimeters, seconds, and volts. Except for the use of such terms, real evaluation of progress would have been impossible; it was not practical to describe in adequate words, or to bear in mind over long periods of time, the status of the multitude of patients who passed through the centers. These data, however, were essential. The presence or absence of improvement in muscle power, sensitivity to pain and touch, electrical irritability of nerves and muscles, and the presence or absence of sweating, all indicate the course of a peripheral nerve injury, the need for surgical intervention, the success of neurolysis or neurorrhaphy or nerve graft, and the spontaneous recovery of contused or bruised nerves.

Head injuries.—Head injuries were handled on separate wards. The patients were examined according to a special routine, were studied with and

without medication, and were treated according to the indications. They were followed up after discharge on certificate of disability to determine whether convulsions developed subsequently. The clinical, roentgenologic, and neurosurgical findings during debridement or the insertion of a tantalum plate, as well as the results of spinal fluid, electroencephalographic, and psychologic surveys, were correlated with long-term developments in the condition of the patient. The attempt to keep in touch with patients after their discharge, to obtain information concerning their progress, and to advise them concerning medication and other treatment, was carried out with great efficiency by American Red Cross personnel.

Specially trained members of the professional and nursing staff were assigned full time to all aphasic patients, and hemiplegics were thoroughly trained in the use of the intact arm.

Peripheral nerve injuries.—Special methods of testing were employed in instances of peripheral nerve injury as follows:

1. Muscular power was determined by means of the so-called fishhook method, in which an ordinary spring scale was applied to the finger to measure the force in pounds required to overcome voluntary resistance of an affected muscle. The average values for the various muscles and the standard deviations had previously been determined in a large random sample of normal soldiers. Experience soon showed that this method of examination was no more time consuming than the former method of estimating, on an arbitrary basis, the loss of power in terms of percentages of normal. It also showed that the former method, even in the hands of experienced neurologists, frequently provided entirely incorrect information as compared with the data secured by the new method.

2. Muscular atrophy accompanying denervation was recorded, and recovery was checked, by handprints and footprints. A graphic record was provided at the same time of the trophic status of the skin.

3. The presence of, or return of, innervation was checked at the earliest possible time. This information is obviously of the greatest importance in the prognosis of a nerve injury of unknown character. The results of the examination frequently prevented an unnecessary operation, with resultant saving of the time of operating personnel, who were always in short or even critical supply. On the other hand, the results sometimes indicated immediate exploration of the damaged nerve and thus saved the patients weeks and months of disability.

4. No single method of studying the electrical irritability of the nerve-muscle apparatus proved satisfactory, but repeated comparative studies were carried out in which the patient's clinical course was correlated with the electromyogram, strength-duration curve, and cathode-closing tetanus.

5. The permeability or impermeability of a nerve scar or nerve suture was tested 8 to 12 weeks after operation, rather than 8 to 12 months afterward, as was the previous custom. Many months of hospitalization were often saved as a result. Operation under local anesthesia, with stimulation of sensory

fibers distal to the scar or suture, proved by far the most sensitive method employed. This is, however, a qualitative method, which provides no reliable indication of the type or number of nerve fibers passing through the injury. It also supplies no information concerning prognosis. The amplitude and latency of action currents elicited across the injury and recorded during operation were found to supply a better quantitative insight into the type and number of viable nerve fibers in the peripheral stump.

6. Sweat tests were recorded, especially in connection with the determination of the skin temperature by means of thermocouples, before, during, and after injection of sympathetic ganglia before sympathectomy for pain of the causalgic type.

Treatment.—After the patient had been completely worked up, usually within a week after his admission to the hospital, he was presented at a combined conference, held each Saturday morning and participated in by the staffs of the neurologic and neurosurgical sections; the chiefs of the divisions of medicine and surgery; representatives of the orthopedic and physiotherapy sections; and, if necessary, representatives of the plastic surgery section. At this conference, the case was discussed in full detail, and it was decided (1) whether the patient required surgery, (2) what procedure was indicated if he did require it, (3) in what sequence stage surgery should be performed, and (4) whether, if other injuries were associated, the required neurosurgery should be done alone or in combination with orthopedic or plastic surgery. A conference conducted along these lines gave the patient the benefit of the carefully considered opinion of a group of specialists, while, from the administrative side, it reduced the details and delay of consultation to a minimum.

Many patients arrived from overseas with frozen joints, as the result of immobilization in casts applied for fractures or used as a substitute for splints in nerve injuries.[10] These joints had to be mobilized before neurosurgery could be undertaken. The limited facilities of the physiotherapeutic section at Cushing General Hospital made it impossible to treat any single patient for more than 20 minutes daily. A mechanical apparatus devised within the hospital ended this difficulty by providing for the automatic movement of wrist, elbow, and knee joints. The apparatus, which could be operated by the patient himself, released physiotherapists for other duties and also made it possible for each patient to be treated 3 times daily for 30 minutes at a time. An 8-month experience with it proved that mobilization of frozen joints could be achieved by this method in 25 to 30 percent less time than by former methods.

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[10] While some patients with peripheral nerve injuries arrived at this and other centers in poor condition, most of them had been well treated. On a well-organized service such as the one at Cushing General Hospital, errors made repeatedly in the same overseas hospital or by the same surgeon were promptly recognized and reported. Thus, in January 1945, the chief of the neurosurgical center at this hospital wrote to the Senior Consultant in Neurological Surgery, Office of the Chief Surgeon, ETOUSA, that uniformly poor results were being observed in cases treated by a Captain —— of the —— General Hospital. Descriptions of operative findings, the report continued, were too meager to be useful. Infection was common. Joints were more than usually frozen. Conditions in which this officer had to work, the report concluded, might explain the results, but the impression was that he was undertaking too much for his surroundings or his ability. With this sort of specific reporting, the overseas consultant could take action at once to correct the errors which were being made.

Patients with peripheral nerve injuries were transferred from the neurologic to the neurosurgical ward for operation and remained on the latter ward until sutures were removed and extremities were extended. They were then transferred back to the neurologic section. This plan resulted in an effective use of bed space.

The work-furlough system.—The introduction of the work-furlough system also resulted in the release of a large number of needed beds for incoming casualties and had happy results for the patients themselves. Spontaneous recovery from a nerve injury, or nerve suture and regeneration of an injured nerve, always takes months and may require a year or more; combat-incurred injuries did not differ from civilian injuries in this respect. War Department policy required the retention of patients in hospital until they had attained maximum hospital benefits, but rigid observance of this regulation resulted in the distressing spectacle of "barracks happy" patients sitting around wards for week and months, always useless, unhappy because they had nothing to do, and, in addition, occupying bed space which could have been used to better advantage for more urgent needs.

The 90-day work furlough solved this problem. Patients who exercised injured extremities grudgingly 3 or 4 hours daily in a hospital gladly exercised them 8 and 10 hours daily in a factory, while at the same time, they aided the war effort, improved their own financial status, and had the added advantage of living with their own families, which additionally improved their morale. At the end of 3 months or of some other specified period, they returned to the hospital for determination of their progress and for a decision whether they should be returned to duty or discharged on a certificate of disability. At one time, as many as 200 patients with peripheral nerve injuries at Cushing General Hospital had been converted from inpatients to outpatients by the work-furlough system, and the same number of urgently needed beds had been released for incoming casualties.

Clinical research.—The cooperation of the neurologic and neurosurgical sections at Cushing General Hospital permitted the joint investigation of a number of problems. Among them were the use of nerve grafts and their long-term results, studies on the electrophysiologic phenomena already mentioned, and studies of the tensile strength of nerves in connection with their extension after end-to-end suture over gaps up to 13.5 cm. in extent.

TRAINING PROGRAM

In 1941, just before the United States entered World War II, the American Board of Neurological Surgery first began to function. The approximately 200 certified neurosurgeons, even if all of them had been suited by age, health, and academic and other responsibilities to enter the service, would have been totally inadequate to handle the neurosurgical load. The certification listings of the newly organized board, however, served as a reservoir of younger sur-

geons, of certified ability, who could be utilized for service overseas and in the Zone of Interior. The units sent overseas early in the war, many of them affiliated hospitals, were chiefly supplied from this source, as were the neurosurgical centers which were established and rapidly expanded in the Zone of Interior. Evacuation hospitals and neurosurgical teams in auxiliary surgical groups also assimilated an additional number of well-trained neurosurgeons who were physically qualified for service and who had just completed, or were in process of completing, formal training in this speciality.

The situation was extremely serious. The need for neurosurgical personnel was promptly evident, partly in the light of the World War I experience and partly in the light of the high proportion of neurosurgical casualties among the wounded returned from North Africa in the early months of fighting there. To meet the requirements of tables of organization of general and other hospitals would have stripped every civilian hospital and clinic in the country and still left the Army short of needed neurosurgical personnel. Provision for the hospitals which were first organized and for the expanding neurosurgical centers had, as just indicated, taken nearly all the certified neurosurgeons available for service. It had also taken the majority of neurosurgical residents. Additional neurosurgical medical officers were urgently needed, but a period of 6 months was to elapse before another group of medical officers with specialized training in neurosurgery would become available from the training program in this specialty set up by the Office of the Surgeon General.

First Courses of Training

Planning for the training program just referred to was begun in January 1942, by the Office of the Surgeon General, in anticipation of the need for trained neurosurgeons. The Division of Medical Sciences of the National Research Council, at the request of The Surgeon General, recommended that arrangements be made for training medical officers in neurosurgery at the Neuropsychiatric Institute, University of Illinois, Urbana, Ill., and the Neurological Institute, College of Physicians and Surgeons, Columbia University, New York, N. Y. Facilities were available for these courses in May 1942, but, due to a shortage of available candidates, training was not begun until September.

The student officers selected were young general surgeons who, during their hospital experiences, had had some contact with functioning neurosurgical services. The first part of the course included dissection of the central and peripheral nervous systems, cadaveric demonstrations, and lectures on neurophysiology. At the end of a month of intensive instruction, the 24 to 30 officers in each class were divided among the neurosurgical centers for a period of preceptorship that usually lasted 3 months but was sometimes extended for 5 to 6 months. Many of the approximately 125 officers thus trained developed into excellent traumatic neurosurgeons and held responsible posts during the war.

Later Courses

There was, however, rather general dissatisfaction among the students with the kind of training given in these first courses. In the Inter-Hospital Letter dated 22 March 1943, Major Spurling pointed out that their dissatisfaction with the results of the courses of training was shared by the men who were giving them. He then outlined the following plan of training which was to be substituted for them in the future:

* * * the selection of candidates is probably the most important step in the whole procedure. It is contemplated that only men will be selected for training who have had three years of general surgery, preferably in a place where they have seen a large volume of trau- ork. Furthermore, no candidate will be accepted for training unless he indicates a o undertake the course. Each candidate will be interviewed by a representative of geon General's Office. Since all new medical officers entering the Army are sent to Carlisle Barracks, Pennsylvania, for six weeks of basic training, it is contemplated that the selections will be made from this group. However, if any of the Army neurosurgeons are acquainted with any young general surgeons who would like to undertake the course, please write to me immediately.

The first part of the training program will [phase I] consist of four weeks instruction at Columbia University in the anatomy and physiology of the nervous system with particular emphasis placed upon surgical application. Most of the four weeks period will be spent in the dissecting room, where all operations on the head, spine, and peripheral nerves commonly encountered in warfare will be done on the cadaver. A few lectures on the pathology of trauma to the nervous tissue will be given, but otherwise there will be no instruction in this course except anatomy, basic physiology, and cadaver surgery.

At the end of four weeks the class * * * will be distributed to the Army general hospitals having neurosurgical services * * * for a two months' preceptorship [phase II]. * * * it is fully realized that in some of our general hospitals the volume of work is small, and two students might not find enough to do to keep them busy. I would appreciate suggestions from each of the groups regarding the optimal and maximal number of students he can handle.

The method of instruction, once they arrive under your care, will be largely an individual matter. It is anticipated that these men will be placed in evacuation or field hospitals overseas to do the emergency head, spine and peripheral nerve cases. In other words, their problems will be largely those of stopping hemorrhage, preventing infection, and debridement of brain and extremity wounds. Obviously they should be taught to do a simple neurological examination and learn the importance of certain clinical signs with respect to head trauma. They should learn to use the suction, the electrical surgical apparatus, silver clips, muscle implants, etc. After all, these technical details can be taught just as well on one type of operative procedure as another, and the fact that most of us do not have much acute head trauma should not interfere seriously with the teaching of these fundamental details. The students should be assigned to the ward work, writing histories, doing neurological examinations, dressings, assisting at operations; they should be instructed by the roentgenologist in the interpretation of head films; and should study what gross pathology there is available. They should have definite assigned reading to be completed by the end of the course. The two months period * * * might just as well have been three months except that the personnel division anticipates the need for these men for overseas duty at the latter part of the summer.

The course of training described in this letter and designed to replace the earlier courses was put into effect at the Neurological Institute, College of Physicians and Surgeons, Columbia University, (appendix A, p. 401), under the

direction of Dr. Tracy J. Putnam, 24 April 1943. All of the first group of officers chosen for specialized training had had fairly satisfactory previous training in general surgery. An occasional proctologist and gynecologist was included in the first group, but these specialties were not represented in the groups of neurosurgeons subsequently trained.

Forty-eight officers were graduated from the two courses conducted at the Neurological Institute at Columbia University. In January 1944, the course of instruction was transferred to the University of Pennsylvania School of Medicine, Philadelphia, Pa., under the direction of Dr. Francis C. Grant. By the time of the transfer, the source of officer personnel for training had shifted from physicians with general surgical training, who predominated in the courses given at Columbia University, to postgraduate neurosurgical students who had completed available civilian training and wished to continue specialization under Army auspices. The young general surgeon, however, still remained the backbone of the group of 74 officers trained at the University of Pennsylvania.

Theoretical Training

The formal program of instruction originally planned was followed closely in the first course held at the University of Pennsylvania, 17 January to 12 February 1944. Progressive changes in the teaching schedule were partly stimulated by a student critique submitted on 12 October 1944 but for the most part were based on a clearer comprehension of the results desired in the course. They were described as follows by Dr. Grant on 6 December 1944:

> In the first schedule * * * we stuck pretty closely to the Government terms of sixty-four (64) hours for neuroanatomy; sixteen (16) hours for neurophysiology; twenty-four (24) hours for cadaver surgery; fourteen (14) hours for neuropathology; ten (10) hours for neurologic examinations; sixteen (16) hours for clinical neurosurgery; sixteen (16) hours for neurologic and neurosurgical clinics, etc.
>
> At the time of the second course, I rerouted things a little bit and put most of the twenty-eight (28) open hours into neurosurgery and neurology.
>
> In the third and fourth courses we cut out entirely neurophysiology and neuropathology. We abandoned neurophysiology quite frankly because we did not have anyone here who taught it very well. The men for the most part had not had any of it since their Sophomore years in school and neither they nor I could see the practical application of the subject matter. They were definitely allergic to it and I felt that the time could be put to better use in other ways. As far as neuropathology is concerned, again, I could not see that this was necessary.
>
> In the last course, therefore, we kept neuroanatomy at about the required number of hours; put in about ten (10) more hours on neurosurgical and neurologic clinics and devoted the bulk of the remaining time to straight neurology. I also introduced roughly six (6) hours on x-ray film diagnosis and the use of sulfa drugs and penicillin; the care of the bladder in spinal cases, and a couple of hours on neurological ophthalmology.
>
> As far as the textbooks are concerned, I think the books described in the original Government Outline of the courses are excellent, if you can get them.
>
> At the present time, we have twelve copies of Glen Spurling's "Practical Neurologic Diagnosis" hidden away for the next course, but the other textbooks suggested are pretty hard to come by at the present time. Rowbotham and Monrad-Krohn are published in

England and are unavailable. I think the Neurosurgical volume in the Military Surgeons' Manual is pretty useless.

It would be a great help to me if you could send me copies of the various suggestions and orders, etc., that are put out from time to time by the Surgeon General's Office on the treatment of neurosurgical wounds * * * . If they are not too confidential, they would keep me informed of the latest ideas. After all, if I am going to try to teach these men, I don't want to give them information which is currently out of date in Army neurosurgical circles.

Practical training.—In the second or practical phase of neurosurgical training, officers who had completed the first part of the course were assigned by ones and twos to the neurosurgical centers for 60 days each. The ability of the respective centers to follow the specified program of instruction was naturally dependent upon the patient load but was chiefly influenced by the surgical tasks which were uppermost at any given time. Yearly statistics show that when the first two groups of neurosurgeons-in-training reached the centers, most installations were at a low ebb of activity and were preparing for the deluge to come. At the time, they were really serving as hospitals for reconstructive surgery, and instances of acute head trauma were scattered and were quite inadequate for training purposes. Fortunately, the centers expanded rapidly soon after students began to be assigned to them, and those who did not have the opportunity to learn the lessons of acute traumatic neurosurgery in the Zone of Interior rapidly learned them under preceptors overseas.

In actual practice, the second or practical phase of neurosurgical instruction was much less formalized than the outline would indicate (appendix A, p. 401). The students, at least in centers in which the patient load was heavy, became integral, hard-working parts of the neurosurgical staff and by progressive steps participated in all phases of the work. They were placed in charge of preoperative wards, where they could assimilate the many-faceted picture of peripheral nerve injury, study neurologic defects in head injuries, and explore the real mysteries of painful backs. They became acquainted with the often pressing tasks of intersection consultations.[11] They observed the sudden onset of such complicating diseases as malaria and hepatitis. They also learned the administrative details concerned with the issuance of passes and furloughs and the other minutiae of a military hospital.

In the second phase of training, students assisted at operations, and, under guidance, themselves performed neurolyses, neurorrhaphies and concomitant stimulation studies, and maneuvers designed to restore nerve defects. They learned to repair skull defects with tantalum or acrylic resins. They also observed the exposure of the brain for the resection of tumors. Acute battle-incurred head injuries were, of course, not seen in Zone of Interior hospitals, but acute head injuries were seen in practically all station hospitals, as the result of training accidents, traffic accidents, brawls, and similar causes. They were not seen nearly as frequently in the general hospitals of

[11] These consultations were among the important duties of all neurosurgical centers, as a few illustrations will show. At Hammond General Hospital, there were 1,203 consultations in the year ending on 1 September 1945. At McCaw General Hospital, there were 1,595 between April 1943 and September 1945, including 222 on the outpatient service. At O'Reilly General Hospital, there were 4,317 during the total period of operation of the neurosurgical center.

the Zone of Interior except in patients for whom the general hospital was acting as a station hospital.

In the third phase of training, the students learned, in the postoperative wards, the care of patients with battle-incurred injuries and the uses of penicillin and the sulfonamides. They also studied the results of nerve regeneration.

In addition to the clinical work outlined, student officers studied neurosurgical roentgenograms as part of their intensive training, joined the hospital staff at conferences and autopsies, and also continued cadaveric dissection, to complement the study of peripheral nerve visualization at the operating table.

This training was ideal for future work in a neurosurgical center in the Zone of Interior, though it was obviously merely supplementary to the experience with fresh wounds which could be gained only by service in an active theater of operations. In retrospect, however, it can fairly be said that the training program in neurosurgery accomplished the ends for which it was designed. When the students had concluded the course, they could be assigned as assistant neurosurgeons either in overseas units or in neurosurgical centers in the Zone of Interior. No method of formal training can fit a neurosurgeon to care for gunshot wounds of the brain and spinal cord received in battle; he can gain that experience only under field conditions. A background of neurosurgical anatomy and of the principles of diagnosis and therapy is, however, essential, and it was adequately presented in the first phase of the neurosurgical training program, while, in the second phase, training in reconstructive neurosurgery was thorough and adequate. Many of these young neurosurgeons were working in a field entirely alien to their earlier experience, but, through application and devotion to duty, they developed remarkable skill in a new and difficult field, and they were largely responsible for whatever measure of success was achieved by the neurosurgical centers.

Evaluation of training program.—The courses at the University of Pennsylvania were suspended early in 1945, after the supply of qualified general surgeons was exhausted and the neurosurgical needs of the Medical Department had been fulfilled. If they should be reestablished, in some future war, the curriculum (appendix A) should be further scrutinized, with the idea of eliminating unnecessary and pragmatic courses unrelated to the demands of this special field of military surgery.

In the opinion of many observers, the neurosurgical training program was one of the major medical accomplishments of the war. In the prewar years, there was a decided tendency in all of the surgical specialties, including neurosurgery, to underestimate the value of a sound background in general surgery as a preliminary to specialized training. Neurosurgeons, more than other specialists, were inclined to emphasize the theoretical rather than the practical aspects of their specialty. As a result of the wartime experience, it can now be said, without qualification, that a sound background in general surgery is highly desirable in the training of the civilian neurosurgeon and is an essential requirement in time of war.

The success of the training program in neurosurgery, the first such course

to be set up, also taught another important lesson, that the same sort of quick, intensive training is applicable to all specialties. This is a possibility which is of great importance from the military point of view in the prospects it opens of quickly overcoming shortages of specialists in one field or another.

Other Methods of Training

The well integrated and practical training available in the neurosurgical centers in the Zone of Interior was made available to other medical officers in addition to those formally enrolled in courses of instruction:

1. Partly qualified neurosurgeons were in numerous instances admitted directly to the neurosurgical centers for practical training in the military aspects of their specialty, upon recommendation by civilian neurosurgeons of accepted standing. This simplified procedure proved a great saving of time and effort. Indeed, if time had been available for administrative and operative duties and if facilities for neuroanatomic dissection could have been provided, the entire task of neurosurgical training might well have been made a function of the neurosurgical centers under the direction of the chiefs of service.

2. Medical Corps officers with an initial background of neurosurgical experience greater than that of the usual student officer were assigned to the neurosurgical centers on temporary duty, to gain practical experience.

3. Neurosurgeons returned from active theater duty also often found it profitable to orient themselves in terms of rehabilitative neurosurgery through the facilities of the centers.

4. Refresher courses were provided at the centers for small groups of officers, seldom more than eight at a time, who were assigned to the first and second echelons of medical service. The courses lasted 4 days each and were presented under the direct tutelage of the chief of section. During the period of instruction, combat-incurred injuries were rapidly reviewed from the point of view of (1) conditions under which the soldier becomes a casualty, (2) causes of wounds, (3) first aid, (4) emergency medical treatment, (5) treatment in other echelons of medical support, and (6) timelag from battlefield to evacuation hospital. All available types of battle casualties were studied in ward rounds, and rehabilitation was made a more personal matter to these officers by their service as assistants at the operating table. The general relationship between the fresh neurosurgical injuries and their eventual repair was discussed in a final conference with the chief of section at the conclusion of the 4-day period of instruction. Suitable recent reports on neurosurgical subjects were mandatory reading during the course.

All the experience in the training program supported the belief that, in any future war, the procurement of trained neurosurgeons must depend primarily upon a pool of existing talent supplemented by well-grounded general surgeons who are taught intensively the well-defined types of neurosurgical injuries which form a common pattern in warfare. For realistic achievement, surgeons in the second group can best begin their training in a rehabilitation

hospital in the Zone of Interior, after which they can continue it under experienced guidance either in the field or in the United States.

RECORDS AND VISUAL RECORDING

Standard United States Army medical history forms used in World War II had evolved from experience. They were generally adequate but were not satisfactory for recording neurosurgical injuries or illnesses; such records must be very detailed if they are to be useful. Most neurosurgical centers overcame this deficiency by the sort of special notations used in many civilian hospitals. This was an excellent method in trained hands but was very time consuming. Much more satisfactory from every standpoint were the special forms finally employed, such as the forms for peripheral nerve examination, head injury, ruptured lumbar disk, and the dermometer chart.

In cases treated by experienced neurosurgeons, the recording was almost invariably detailed and adequate. In many instances, however, the records were not satisfactory. A record, for instance, which did not indicate the precise condition of a nerve at debridement or the precise details of previous surgery might result in an unnecessary operation because of its inadequacy. When the Peripheral Nerve Registry was set up, the forms prepared for it were sent overseas; when they were properly employed, they furnished all the information necessary for later treatment in the Zone of Interior.

Neurosurgery is a specialty in which graphic recording is always useful and frequently essential. Because all neurosurgical centers did not have adequate provision for such recording, a great many useful and important clinical data were lost. In some centers, equipment was provided but trained personnel to operate it was not, and, again, valuable material was lost. Some of these deficiencies were eventually remedied but too late to salvage many data that it would have been most desirable to preserve.

STATISTICAL DATA

Surveys of representative neurosurgical centers in 1944 and 1945 indicated that about 75 percent of the patient load was made up of casualties from overseas. Peripheral nerve injuries, back injuries, including ruptured disks, and head injuries constituted, in that order, the important technical problems. More than half of all patients admitted underwent some sort of surgery. Disposition, however, was fairly rapid; in 1 center, with a bed allotment of 400, 1,541 patients were admitted in the course of a 12-month period.

The lack of an official recurring report from the neurosurgical centers made it difficult to estimate the patient load in terms of types of injury, character and duration of treatment, and disposition of cases. Some information on patient load, however, can be secured from figures collected in September 1945. Each center was requested to submit its own figures on admissions, consultations, operations, and deaths, so that some idea might be gained concerning

these various items. The figures (tables 2 and 3) are not presented as definitive. The same patients, because of transfers from one installation to another, undoubtedly sometimes appear in the reports of more than one center, and there may also be duplications in the number of consultations for this or other reasons. The number of deaths, however, is entirely accurate. It should be observed that while all fatalities in a neurosurgical center are necessarily classed as neurosurgical in origin, as is the usual clinical practice, a number of the deaths in these installations (tables 2 and 3) were actually due to concurrent disease or to other causes entirely unconnected with the original neurosurgical injury or disease.

TABLE 2.—*Patient load in 26 neurosurgical centers at general hospitals in Zone of Interior, May 1942–September 1945*

Hospital	Admissions	Consultations	Operations	Deaths
Ashford	3, 296	1, 202	2, 410	7
Baker	1, 502	576	874	2
Birmingham [1]	346	101	128	1
Brooke [1]	364	490	183	8
Bushnell	2, 779	2, 304	1, 755	12
Cushing	1, 883	2, 683	1, 289	8
DeWitt	1, 382	493	630	6
England [2]	5, 025	1, 715	1, 838	13
Fitzsimons [1]	305	455	163	12
Halloran [3]	728	798	203	5
Halloran [3]	2, 277	737	908	6
Hammond	2, 505	1, 103	1, 087	17
Hoff [1]	781	943	292	6
Kennedy	3, 880	2, 770	1, 689	23
Lawson	4, 232	985	1, 705	31
Lovell [1]	1, 081	1, 688	742	10
Mayo	1, 935	1, 425	1, 335	13
McCaw	2, 217	1, 595	1, 389	8
McCloskey	4, 500	2, 049	1, 143	23
McGuire	2, 332	610	1, 287	5
Nichols	3, 470	1, 912	1, 315	29
Northington	880	938	691	5
Percy Jones	3, 057	2, 957	1, 948	12
O'Reilly	4, 818	4, 317	2, 110	24
Schick [1]	712	1, 022	432	4
Wakeman	2, 305	837	761	11
Walter Reed [1]	2, 360	2, 247	1, 526	31
Total	60, 952	38, 952	29, 833	332

[1] These centers were transferred or discontinued during the course of the war. Other centers in the list came into existence at various times in 1942 and operated until after V-J Day.

[2] Thomas M. England after 14 November 1944.

[3] The center at Halloran General Hospital was operated as a supplementary installation from 1 July 1943 to 30 June 1944. It was reestablished in January 1945 and thereafter fulfilled all the functions of the usual neurosurgical center. The material for the two periods of operation was not precisely the same and therefore is presented separately.

TABLE 3.—*Distribution by cause of 332 deaths in 60,952 neurosurgical admissions in 26 neurosurgical centers in Zone of Interior, May 1942–September 1945*

Cause of death	Number
Cerebral causes:	
Traumas:	
Acute injuries	49
Gunshot wound, acute	2
Gunshot wound, old, postoperative hemorrhage	1
Gunshot wound, old, residual infection	7
Subdural hematoma, bilateral	1
Neoplasms:	
Glioma, type not specified	30
Glioblastoma multiforme	25
Meningioma	6
Pinealoma	5
Metastatic tumors	5
Medulloblastoma, cerebellar	4
Neurinoma, acoustic	4
Ependymoma	4
Craniopharyngioma	4
Astrocytoma	3
Adenocarcinoma, pituitary	2
Adenoma, pituitary	1
Teratoma	1
Sarcoma, dura, recurrent	1
Infections:	
Abscess, cerebral	26
Fungus, cerebral, cerebritis	1
Arachnoiditis	1
Meningoencephalitis	3
Encephalitis	1
Interventricular block, cerebral abscess, postoperative	1
Tuberculoma	1
Vascular causes:	
Subarachnoid hemorrhage	2
Cerebellar hemorrhage, cause unknown	1
Thrombosis, cerebral	1
Aneurysm, cerebral	3
Miscellaneous causes:	
Tic douloureux, postoperative hemorrhage	1
Atresia aqueduct of Sylvius	2
Hydrocephalus, cause undetermined	1
Status epilepticus	2
Total	202
Spinal cord:	
Paraplegia:	
Urinary sepsis	14
Urinary sepsis, with cerebellar abscess	1
Uremia, cerebral edema	1
Meningitis, abscess about foreign body	1
Removal of foreign body, postoperative	1
Cachexia	4
Infectious hepatitis	3
Bronchopneumonia	2
Bronchopulmonary fistula	1
Pulmonary abscess, multiple	1
Respiratory failure, acute	1
Rheumatic endocarditis, acute cardiac dilatation	1
Cardiac embolus	1
Aneurysm of aorta, dissecting	1
Septicemia, *Staphylococcus aureus*	1
Retroperitoneal abscess, decubitus ulcers	2
Amyloidosis, decubitus ulcers	1
Cause not reported	9
Infections:	
Meningitis, tuberculous	3
Meningitis, pneumococcic	1
Meningitis, pneumococcic, after myelogram	1
Meningitis, coccidioidal	1
Meningitis, with chronic arachnoiditis, postoperative	1
Meningitis, type undetermined	5
Neoplasms:	
Lymphoma	1
Syringomyelia	1
Hodgkin's disease	1
Type not specified	5
Traumas:	
Fracture, cervical cord	7
Gunshot wound, cervical cord	2
Ruptured lumbar disk:	
Aneurysm common iliac artery, postoperative	1
Spinal analgesia	1
Hemorrhage iliac artery, postoperative	1
Total	78
Miscellaneous causes:	
Hepatitis, acute	2
Pancreatitis, acute hemorrhagic	1
Carcinoma, cervical region	1
Stab wound, chest	1
Overdosage drug	1
Melanosarcoma, chest wall	1
Total	7
Cause not stated [1]	45
Grand total	332

[1] Including 2 deaths while on furlough.

VISITS TO HOSPITALS

One of the duties of the consultant in neurosurgery in the Office of the Surgeon General was to visit the neurosurgical centers, to evaluate personnel, observe the organization and management, disseminate information, demonstrate techniques, correct errors, and, in general, survey the center and report on its performance and results to the Chief Consultant in Surgery, Office of the Surgeon General.

The following (summarized) reports of two of these visits, the first in early December 1944 and the second in the middle of April 1945, are presented as typical.

December 1944

When the —— general hospital was visited, there were 1,820 patients, of whom 587 were on the neurosurgical section. Of the 38 medical officers, 6 were on the neurosurgical service. The center was in full action, heavy operating-room schedules being kept, with teams working both morning and afternoon. In November, 132 neurosurgical operations had been done. There was a backlog of 22 cases, estimated as a week's work. The operating rooms were well equipped, except that there was no stimulator, and were well supervised.

The following report was made:

Staff.—Major —— is an adept, hard-working neurosurgeon, with Board qualifications. He is particularly interested in head injuries and their sequelae and is a student of electroencephalography. He is an able administrator and has a closely knit, efficient organization. He may lack some initiative and drive, possibly because he came from a very inactive professional service elsewhere. Another explanation may be that the chief of surgery is an extremely dynamic individual, with interests in another field, and Major —— may possibly be submerged by the association.

Lieutenant —— is a scholarly, technically adept military surgeon of considerable promise. He is held in high regard by the chief of the neurosurgical service.

Lieutenant —— is a very industrious and conscientious surgeon, though he lacks experience and training in nerve surgery.

The student officer will never be a military neurosurgeon and should be reclassified as a ward officer.

General organization.—Patients are admitted directly to the neurosurgical section, and there is no close association with the recently established neurological section as far as military injuries are concerned. The usual preoperative studies are carefully made, but no special studies are undertaken, and few patients have undergone electrical diagnostic studies. These, when requested, are done very well, but they are unduly prolonged, and the timelag may be 2 or 3 weeks. It is obvious that a simplified method of electrical diagnosis is needed, probably to be carried out under the supervision of the neurological section.

There is some confusion at present about the type of cases to be sent to the convalescent hospital for the center, particularly in regard to the facilities available there for physiotherapy and followup care. Patients are therefore held at the center until definite signs of regeneration are available.

A combined orthopedic-neurosurgical ward, where combined injuries are studied by members of both staffs, is serving a valuable purpose.

Specific professional problems.—These problems are as follows:

1. *Nerve injuries.*—Nerve explorations are done under general anesthesia, and no stimulator is available. The staff seems somewhat doubtful about the rather obvious advantages of local anesthesia, even without a stimulator.

The observed incisions were far too limited in scope. The staff seemed unfamiliar with standard methods of far-reaching nerve mobilization and, indeed, doubted the wisdom of this procedure. As a result, far too many bulb sutures had been performed with scant indication for this distinctly second-grade type of nerve repair. The impulse for secondary operation was lacking.

There was also a lack of appreciation of the value of early definitive treatment of nerve injuries. The significance of detrimental changes in the distal stump was not well appreciated. Many patients were undergoing posture changes in various types of splints, to further joint movement, or were undergoing nerve stretching, to facilitate, theoretically, subsequent operative procedures. This is a theory that stems directly from lack of knowledge of other mobilization methods.

There was no neuropathologic check of distal or proximal stumps, although some valuable work in this field had only recently been carried out in this center.

Finally, both postoperative splinting and other hand splinting seemed poorly understood. With short incisions and lack of nerve mobilization, the hand was frequently—and quite unnecessarily—dressed in acute flexion. Periods of postoperative immobilization were far too short, averaging about 3 weeks for sciatic nerve, and immobilization was limited to a knee case. The radial nerve splint was misused, the hands being immobilized, to all intent, in the Percy Jones splint, which was not correctly applied, so that no patient could flex fingers or hands. Patients in casts were also not using mobilizing splints.

These various points were discussed with the staff. It was recommended that two of the officers be sent to another center, on temporary duty, for further training in peripheral nerve surgery. It was also recommended that the hospital pathologist be urged to review all peripheral nerve material.

2. *Head injuries.*—These were very well handled by Major ——, and a valuable and intensive study of the late results of combat-incurred wounds is under way. An electroencephalograph is available.

3. *Paraplegia.*—There are 13 such patients, with 8 personnel in attendance. The patients are on the urological ward, and intermittent urethral bladder irrigations are carried out with solution M. There are no overhead frames or reconditioning equipment, but they have been ordered. One patient was

relieved of pain by cordotomy. There are very few decubitus ulcers, because of careful nursing, and all arrangements are very satisfactory.

4. *Ruptured disks.*—The new regulations concerning treatment were explained. Too much effort had been made previously to return to duty obviously unfit men who had no special qualifications for service.

Summary.—This is one of the best neurosurgical centers, though the personnel need further training in more refined peripheral nerve surgery. It is unfortunate that for a number of reasons the former interest in combined vessel nerve injuries has diminished. The explanation seems to be that the emphasis is now entirely on the vascular lesion, which represents a threat to life, and not on the neural dysfunction, which is a threat to rehabilitation. The opportunity for a very fruitful study of these combined disabling injuries is being neglected. The solution might be the direct assignment of a neurosurgeon to the vascular service. The orthopedic-neurosurgical ward is of value in expediting the treatment of the peripheral nerve injury.

April 1945

The neurosurgical bed allotment in the —— general hospital, formerly 1,440, had just been reduced to 1,000. At the time of the visit, there were 320 neurosurgical patients in the hospital and 300 on furlough. The surgical backlog of 50 patients consisted in large part of recent acquisitions. Eight officers were assigned to the neurosurgical service.

The following report was made:

Staff.—Major —— is a well-trained, mature, hard-headed surgeon, a fine organizer, and a good leader.

Major —— is probably a good neurosurgeon but is a disappointing section chief with a poor viewpoint toward military service.

Captain ——, who is on limited service, is an enthusiastic, very energetic, fine military surgeon, though still in the developmental stage.

Captain —— is an older man, a recent student, but adept in routine affairs. He is on limited service.

Lieutenant —— is another promising military neurosurgeon, who should be promoted. He is a former student and is on limited service.

There are three ward officers.

Only three of the officers assigned to neurosurgery are first-rate military neurosurgeons. Major —— will shortly report from overseas, and Major ——, who is being held at —— General Hospital for no good reason, should be returned to this hospital.

Organization.—Lack of neurologists has previously prevented a common admitting ward, and the reduced size of the neurosurgery section at present makes this plan unnecessary. Exchange of patients with the one neurologist is prompt, and no cases are held on the section unnecessarily. At this time, the section is divided into head, peripheral nerve, paraplegic, combined ortho-

pedic-neurosurgical, and officers' wards, and these divisions fit well into the permanent and semipermanent structures of the hospital.

Head injuries.—All head injuries, skull defects, tumors, and the like are segregated under Captain ——, who, with Major ——, is directly interested in several important problems in this field. The most impressive work, now under way for 2 years, has been a study of speech defects, in the recognition and treatment of which Captain —— is a recognized authority. Ninety-nine patients have been followed to date, and treatment has been initiated in all severe aphasics. These studies will be reported in detail in the *Bulletin of the U. S. Army Medical Department* and in a speech journal. This work is very worthwhile.

A second fruitful study is concerned with a description of occipital lobe injuries with visual field defects and their prognosis. This work will also be reported.

A third study concerns the use of tantalum foil between the dura and cerebral scar after removal of dural-cortical scar adhesions. It is related to the method of cranioplasty used in this section. Plates are not swaged but are formed by hammering. Operations are under general anesthesia. The dura is opened and the meningeal-cortical scar is divided; its extent and depth are determined by brain puncture. Tantalum foil (0.00025) is then placed as noted, and cranioplasty is carried out by the tantalum wedge inlay method.

This study is completely uncontrolled as an effort to control either post-traumatic symptoms or convulsions, particularly the latter, since no electro-encephalographic studies are available. In past reports, two adverse reactions to tantalum foil have been noted. The advantage of using local anesthesia in cranioplasty was discussed with the staff.

Thirty-seven other head cases were reviewed. The technical results were good. There were no complicated cases. Six early cranioplasties were reviewed. Various simple techniques were used, and all results were good, without complications. No patients were observed with infection or retained skull fragments.

Peripheral nerve injuries.—Peripheral nerve cases were also segregated. Operations on several patients were observed. The technique was delicate, but there was a lack of use of major transposition procedures. The advantages of local anesthesia were not well appreciated. Tantalum wire was used, but foil was used infrequently. Postoperative positioning was adequately maintained for sufficient periods of time. Splinting was poor, and the mobilizing radial nerve splint was introduced. Few complications were seen. The Peripheral Nerve Registry was explained in detail. It is believed that the use of the registry forms will stimulate corrections of any gross defects in the care of these patients.

Combined orthopedic-neurosurgical section.—This (new) section embraces three large, well-designed wards, on which 70 patients were examined, most of them recently admitted. As in other centers, combined injuries of the radial nerve and humerus and of the sciatic nerve and femur were most common.

One patient was a striking illustration of the advantages of early surgical attack on sciatic lesions. None of the patients with injuries of the upper extremity had been received in any kind of mobilizing splints; at that, their hands looked considerably better than some observed after splinting. Only two frozen hands were observed in which cast fixation could be implicated, and, in both cases, vascular ligation had been necessary. Major —— and Lieutenant ——, who are detailed to this section, have an intelligent point of view about the extremity as a whole. Mobilizing radial splints were instituted in all appropriate cases, and the necessity for early sciatic nerve exploration was stressed.

Paraplegia.—The paraplegia ward, situated on the second floor of a permanent building, is large, airy, sunny, and rather impressive. Only the very ill were segregated. The few officers were placed in large 4-bed wards. All beds have Balkan frames, and reconditioning is established. The mattresses are somewhat soft, but bedboards have been ordered.

The control of urinary sepsis is in the hands of Captain ——, an enthusiastic, impeccably trained urologist. Details of his self-controlled bladder irrigation sets and reports of his various studies have been published.

Forty-five patients were examined, 19 with complete and 26 with incomplete injuries. Sixteen of the patients with complete injuries had decubiti, though the nursing care was thorough and good. Surgical treatment of the ulcers had been a little tardy but was now under way. Only a few nutrition studies have been made. The incomplete paraplegics were mostly ambulatory, but wheelchairs were few, and this deficiency has retarded more active ambulation for the complete paraplegics. Metal wheelchairs should be available.

Morale is high, and the whole ward, though new, already represents a high degree of aggressive professional care.

Ruptured disks.—There were few patients and no evidence of overemphasis upon this problem.

General comments.—Physiotherapy is at present quite inadequate for the patient load. This is to be remedied by a new building.

Six fairly large operating rooms are available, but equipment has been slow in arriving.

Neuropathology is nonexistent.

The electroencephalograph is at the hospital but still in crates because permission to build the necessary auxilliary equipment and installation has not yet been received.

There appears to be scant central control of the neurological load.

Electrodiagnostic methods are almost nonexistent because of the heavy physiotherapy load and the lack of apparatus.

Photography, for some reason, is under Public Relations, which seems a poor arrangement for securing clinical illustrations.

Summary.—In all, 145 patients were examined. This is a good neurosurgical center, and there is nothing beyond a few technical and supply details to worry about in it.

CHAPTER II

The Mediterranean (Formerly North African) Theater of Operations

Eldridge H. Campbell, Jr., M. D.[1]

EVACUATION AND HOSPITALIZATION

Neurosurgical policies in the Mediterranean (formerly North African) Theater of Operations, United States Army, were so largely dictated by local conditions of fighting and terrain that some mention of these considerations is essential for backgound. In the Tunisian campaign, evacuation of casualties, like supply, was extremely difficult because of the wide dispersion of fighting, the frightfully bad roads, the inadequate means of transportation, and the distances from the battlefield to the nearest hospitals. During the fighting in southern Tunisia, it was sometimes necessary to move patients 60 to 110 miles by ambulance to the nearest evacuation hospital, from which another trip of 400 miles by train (later by air) was necessary to reach the nearest general hospital at Oran. As a result of these unfavorable conditions, some of the more severely wounded patients died, chiefly of shock, before an evacuation hospital could be reached.

The fluid status of the fighting frequently made it impractical or entirely impossible to bring evacuation hospitals farther forward. The plan was therefore evolved of placing field hospital platoons within litter-carrying distance of the divisional clearing stations. These hospitals, the mission of which was limited to the care of patients with any type of injury which would make them nontransportable, were small, flexible, and highly mobile. After a little practice, it was possible for their personnel to move and set them up within a few hours. Professional work in them was chiefly done by specialized teams from the 2d and 3d Auxiliary Surgical Groups. Living conditions in field hospitals were often difficult, and the workload was likely to be heavy, but the outstanding service rendered in them resulted in the saving of many lives and limbs.

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[1] A consultant in neurosurgery was never formally appointed in the Mediterranean theater. When the need arose, Dr. Campbell, then a lieutenant colonel in the Medical Corps, represented the consultant in surgery to the Theater Surgeon. Gaps in this chapter are due to this fact; they could not be overcome because of Dr. Campbell's death while the history was in preparation.

NEUROSURGERY IN FORWARD HOSPITALS

Except in unusual circumstances, however, neurosurgery did not prove practical in field hospitals, for several reasons, as follows:

1. There was seldom enough work in a single platoon to keep members of a neurosurgical team occupied with their specialty.

2. The attachment of neurosurgeons to field hospitals therefore involved a waste of specialized personnel, which was always in short supply and which could be put to better use in larger installations, in which there was more neurosurgery to be done.

3. The policy of placing neurosurgeons in field hospitals and assigning them to other surgery when there was no neurosurgery to be done did not solve the problem of neurosurgery in forward hospitals. It involved the waste of neurosurgical personnel already mentioned, and it did not promote efficient surgery, since neurosurgeons had no training in the type of surgery (abdominal, thoracic, and vascular) which chiefly occupied the personnel of field hospitals.

The problem was therefore solved in two ways. The young general surgeons with broad training who were placed in the field hospitals carried out such immediate treatment as was necessary in neurosurgical patients, who were then evacuated to the rear as promptly as possible. If serious neurosurgical injuries occurred in nontransportable patients, a neurosurgeon came forward from the nearest evacuation hospital and performed the necessary surgery.

The management of any type of neurosurgical injury at the level of the field hospital presupposed, of course, the provision of special equipment, such as suction apparatus, electrocautery, and silver clips, but this contingency was readily cared for by slight changes in the table of equipment for field hospital units.

Evacuation Hospitals

Evacuation hospitals proved the most practical installations for definitive neurosurgery in forward areas. Under favorable circumstances, they were located between 10 and 50 miles behind the frontline, on the direct line of evacuation or as near to it as possible. They could practically always be reached within a maximum of 4 hours, and sometimes within a minimum of 30 minutes, after casualties had left the clearing station. They possessed numerous advantages over the field hospital. They were usually behind the zone of enemy shelling, though at the Anzio beachhead this was not true, nor was it always true elsewhere. Reasonably adequate operating facilities were usually available. A special ward, with trained personnel, could be set aside for neurosurgical patients. Allied specialties, such as ophthalmology and maxillofacial surgery, were usually represented, which simplified the consultations frequently necessary in neurosurgical injuries. Finally, patients could be held at an evacuation hospital after surgery until they were ready for the much longer trip to the general hospital in the base section.

Neurosurgical Teams

The advantages listed for evacuation hospitals often proved purely theoretical in the North African theater, as has already been indicated, and sometimes in the later Italian fighting also. The trip from the frontlines to the hospital was frequently long and rough. Furthermore, from the standpoint of the neurosurgeon even more than of other surgical specialists, there was likely to be too much or too little to do. Because of long distances between medical installations in which definitive care could be given, it was the natural tendency of evacuation officers to send the majority of patients with head injuries to the nearest hospitals, with the result that, within a few days, the neurosurgical teams working in them would be overwhelmed with work while teams in hospitals farther to the rear would be almost idle.

The uneven distribution of casualties was compensated for by supplementing neurosurgical teams in forward hospitals with teams from hospitals farther to the rear. Although this plan had the effect of concentrating neurosurgical casualties at the most forward establishments, it was found practical to handle such concentrations without difficulty. Later in Italy, in the Fifth U. S. Army area, where the plan was fully effective, it became evident that two neurosurgical teams working 12-hour shifts could accomplish better work, and proportionately far more, than a single team working for 24 hours.

The flexible use of neurosurgical teams in North Africa was found impractical only in the few instances in which lines of evacuation did not converge for long distances. The wisdom of the judicious placement of specialized medical personnel was clearly established. Had the principle of pools of surgical teams, rather than inflexible tables of organization, been in effect for all hospitals, including station and general hospitals, in the communications zone, and had all army and theater consultants had complete authority to move personnel quickly, a more efficient use could have been made of the limited supply of neurosurgeons.

NEUROSURGICAL CENTERS

It was originally the plan of the surgical consultant to the Surgeon, Mediterranean theater, to organize at the general hospital level two or more centers in which patients with neurosurgical injuries and neurosurgeons with specialized talent could be brought together. Each neurosurgical service was to be in charge of an experienced neurosurgeon, under whose direction less experienced neurosurgeons from other base hospitals would work. As part of the program, neurosurgeons in forward hospitals would be exchanged from time to time with those at the special centers.

The advantages of this plan were obvious. Neurosurgeons working at the base would thus meet at first hand the problems of the management of head injuries, for instance, while they were still acute, while surgeons working in forward hospitals would become familiar with early reparative neurosurgery and with the diagnosis and treatment of complications of neurosurgical injuries.

The medical center at Bizerte was selected as admirably suited for the institution of this plan, since its five general hospitals and several station hospitals were situated within an hour's ambulance haul of each other. The 33d General Hospital was selected as the neurosurgical center for several reasons. It was centrally located in respect to the other hospitals at Bizerte. It was readily accessible otherwise, being near the Sidi Ahmed airport and the Karouba docks. All patients coming into the base underwent triage at this hospital. It already had two neurosurgeons on its staff, and it had operating-room and ward nurses already experienced in neurosurgery. Still later, a medical artist was assigned to the hospital.

In spite of these advantages, however, the program devised worked only reasonably well when the 33d General Hospital was in operation as a neurosurgical center. The influx of neurosurgeons, it is true, engendered new enthusiasm on the part of all concerned. Better work was accomplished. Information on neurosurgical policies and practices was more easily spread. On the other hand, the system of exchange of personnel between army and base areas was not put into operation with any real degree of efficiency for more than a year. Even more important for the success of the plan, some hospital commanders recalled their organically assigned nuerosurgeons who had been attached to the other installations after they had served in them only a short time. The resulting loss of talent, already in critically short supply, was serious because of the heavy concentration of neurosurgical casualties at the 33d General Hospital. The result was that specialized talent was wasted in the hospitals not serving as specialized centers because they did not receive neurosurgical casualties, who were still being sent to the neurosurgical center. The neurosurgeons recalled to them frequently had so little to do in their own field that they were put to work in general and orthopedic surgery.

UTILIZATION OF NEUROSURGICAL PERSONNEL

In spite of the limited success of the plan of specialized centers in North Africa, the opinion persisted that only by this or some similar method could critically short specialized personnel be used to the best advantage. Existing shortages, furthermore, were aggravated by an uneconomical use of neurosurgeons for administrative and other nonspecialized duties. On one occasion, for instance, at the time of extremely heavy fighting at Anzio and Cassino, several forward hospitals each had 150 or more wounded men awaiting operation, and all hospitals in the base were also extremely busy. The 33d General Hospital was then staging in Naples, and its commanding officer was requested, through the Surgeon, Peninsular Base Section, to furnish 11 general surgeons and neurosurgeons to work in forward medical installations. These officers were assembled on a volunteer basis, but, instead of being taken to the forward hospitals for which they had volunteered, they were taken to Naples, where they were assigned such nonmedical duties as checking prostitutes.

Transfer of personnel was also frequently very slow. At the beginning of the North Apennines campaign, the Consultant in Surgery, Office of the Surgeon, Fifth U. S. Army, Maj. (later Col.) Howard E. Snyder, MC, urgently requested, through channels, that several surgical and neurosurgical teams be loaned from the base. Since the 33d General Hospital was then inactive, several general surgical and neurosurgical teams were provided, all on a volunteer basis. Although there was complete agreement on the part of all concerned that these teams were needed and should be supplied, it was 10 days before the necessary orders for their movement were received.

These were exceptional instances, it is true, but they were not isolated, and they help to explain why, until late in the war, neurosurgical personnel in the Mediterranean theater was always in short supply and was not used as efficiently as it might have been.

CHAPTER III

The European Theater of Operations

R. Glen Spurling, M. D.

The Professional Services Division created in the Office of the Chief Surgeon, Headquarters, ETOUSA (European Theater of Operations, United States Army), in the summer of 1942 was composed of senior consultants in all major specialties. After their appointment, these consultants originated all policies pertaining to specialized care and settled such problems concerned with it as were referred to them. From an administrative standpoint, the activities of the senior consultants were limited to hospitals in the rear echelons of the theater; that is, in the communications zone and in the advanced section zone. Actually, however, their services were fully utilized by the surgeons of the armies, and at no time during the active campaign, from a neurosurgical standpoint, were difficulties encountered in coordinating the professional services of hospitals in the forward and rear echelons.

This account of the activities of the neurosurgical consultants in the European theater therefore properly begins with the statement that, at all times, the senior consultant in neurosurgery had a completely free hand in all professional matters, as a result of the full cooperation given him by the chief of the Professional Services Division and the Theater Chief Surgeon. Numerous difficulties were encountered, but they were never on this level. Not a single recommendation of professional policy was disapproved by either of these superior officers, and their advice, which was always available for the asking, was invaluable in expediting and correlating the suggestions made regarding professional practice.

The first senior consultant in neurological surgery, in the European theater, Lt. Col. (later Col.) Loyal Davis, MC, was appointed in August 1942 and served in the United Kingdom from 6 September 1942 until 13 September 1943, when he was relieved because of illness. Maj. (later Lt. Col.) John Scarff, MC, acted as consultant in the specialty until 15 March 1944, when Col. R. Glen Spurling, MC, succeeded him. Colonel Spurling served until 12 July 1945, when the neurosurgical consultant system in the European theater was terminated.

FUNCTIONS OF THE SENIOR CONSULTANT IN NEUROSURGERY

In the structural organization of the Medical Corps in the European theater, the senior consultant in neurosurgery was directly responsible to the

chief of the Professional Services Division and, through him, to the Theater Chief Surgeon. The over-all function of this consultant was to formulate professional policies relating to his specialty, to plan the structural organization of neurosurgical care in terms of day-to-day as well as long-term requirements, and to recommend alterations in plans and policies to meet special situations as they arose.

Evaluation and Assignment of Personnel

The most important function of the senior consultant in neurosurgery concerned the supervision of neurosurgical personnel in the theater. Initial assignments had frequently, and of necessity, been made without special reference to professional ability and training, either on the basis of sheer expediency or to satisfy table of organization requirements. With the objective of placing each neurosurgeon in a position in which his abilities and capacities could best be utilized, the evaluation of all personnel in the European theater was a continuous process both before and after D-day. Both senior consultants in neurosurgery already knew personally many of the neurosurgeons assigned to the theater. It was their business to study the training, experience, ability, and temperament of those they did not know, and to evaluate the performance of all neurosurgeons.

Changes in assignment were frequently recommended as a result of this analysis. Some recommendations had to do with more efficient utilization of personnel, who were always in critically short supply and wastage of whom was particularly to be deplored. Other recommendations concerned the transfer of less experienced neurosurgeons to positions in which their work could be supervised by more experienced officers. A few assignments were deliberately changed in order to utilize officers who, under certain conditions, might easily have become emotionally upset and who, because of the changed assignment, proved to be useful and productive surgeons.

The intimate knowledge concerning each neurosurgeon, necessary for his appropriate assignment, was acquired by the senior consultants in neurosurgery by regular visits to hospital installations at all levels, as well as special visits when special needs or difficulties arose. These opportunities were utilized in various ways. Meetings were held with neurosurgeons in evacuation and general hospitals, in an endeavor to unify policies and practices, clarify new directives, and generally insure not only that the intentions of the neurosurgical section of the Professional Services Division, Office of the Chief Surgeon, Headquarters, ETOUSA, were carried out but also that their principles were understood, so that they could be applied intelligently. The senior consultant acted as a professional consultant on difficult, obscure, or important cases and frequently was in the operating theater for purposes of demonstration and instruction.

Before D-day, and in the early days of the European campaign, when all general hospitals were located in the United Kingdom, it was possible for the

consultants to exercise personal supervision over all professional personnel in them. As general hospitals were transferred to the Continent in increasing numbers, and as the battlefield extended over a greater area, more of Colonel Spurling's time was spent on the Continent and less could be spent in the United Kingdom Base. The difficulties which might have arisen from his prolonged absences were avoided by the appointment of a regional consultant in neurosurgery for each hospital center in the base.

The chief responsibility of the regional consultants concerned the prompt and accurate triage of neurosurgical patients, but their services were also available to the surgical coordinator of each hospital center for the implementation of neurosurgical policies and for advice concerning the assignment and reassignment of neurosurgical personnel. They were responsible to the senior consultant for any departures from official policy.

At the request of the consulting surgeons of the armies, neurosurgical consultants had been named for each army before it went into action on the Continent. These consultants were assigned to evacuation hospitals on temporary duty while functioning in a consultant capacity under the supervision of the surgical consultant for each army. It was their duty to advise the surgical consultants concerning neurosurgical policies in the army; to visit the evacuation hospitals where, as a result of the critical shortage of neurosurgeons, less experienced men were necessarily in charge of services; and, by instruction and demonstration in the hospital wards and operating theaters, to attempt to improve the quality of neurosurgical care of battle casualties. Whenever facilities prevented the prompt evacuation of casualties to general hospitals, patients with complicated neurosurgical injuries were transferred to hospitals to which experienced neurosurgeons were assigned.

Informal conferences regarding the care and evacuation of neurosurgical cases in the field were held almost weekly with Col. Joseph Augustus Crisler, MC, Consultant in Surgery, Office of the Surgeon, Headquarters, First U. S. Army, both before and after D-day, and relations with the surgical consultants of the other armies in the theater were equally close and cordial. The facilities of the Office of the Senior Consultant in Neurosurgery, Office of the Chief Surgeon, Headquarters, ETOUSA, were always available to army neurosurgical consultants, but, on the whole, the press of circumstances made it necessary for them to make their own decisions. The competent performance of their duties by these consultants played an important part in elevating the standards of neurosurgical practice in evacuation hospitals.

Equipment

Perfecting the distribution of physical equipment and supplies was another important phase of the work of the senior consultant in neurosurgery in the European theater (p. 74). As he moved from one hospital to another, he was able to observe deficits, to note what was in short supply and what was in excess, and to recommend the correction of shortages and excesses alike. He

was also able to report on the usefulness and value of new equipment and to see to it that it was properly used in the installations in which it had been placed.

Liaison

Liaison with appropriate medical officers in the Allied forces was another duty assigned to the senior consultant in neurosurgery. The liaison was productive of good will as well as the exchange of medical information, which often was of great importance. It enhanced the medical prestige of all Allied medical services and was a matter of necessity in forces destined to operate as a unit under a single supreme commander.

During his period of service, Colonel Davis accompanied the chief surgical consultant in the theater on the joint Anglo-American Medical Mission which visited the Soviet Union early in the summer of 1943.

ASSIGNMENTS OF NEUROSURGICAL PERSONNEL

The consultant system in neurosurgery was set up in the European theater in September 1942. The first activities of the neurosurgical section were conducted under considerable difficulty because of the personnel situation. Although neurosurgery is a highly specialized field, neurosurgeons had been removed from the tables of organization for general hospitals early in 1942. When Colonel Davis reached the theater, in September 1942, he found that the only two neurosurgeons then in the United Kingdom were functioning in their respective organizations under the guise of "septic" surgeons. A third neurosurgeon, who arrived a little later, functioned in the same capacity. When the 3d Auxiliary Surgical Group arrived in England, the two neurosurgeons assigned to it were sent on with it to North Africa.

In the fall of 1942, when neurosurgical work was limited to civilian-type injuries, the two general hospitals in the theater, to which qualified neurosurgeons were assigned, were entirely capable of handling all the craniocerebral, spinal cord, and peripheral nerve injuries that were encountered. All such casualties had to be transferred to one or the other of these organizations because the three other general hospitals then in England did not have on their staffs officers with the proper professional qualifications to care for them. This situation obviously would not meet the requirements when battle casualties would be received in large numbers.

In spite of Colonel Davis' endeavors to secure additional personnel, experienced neurosurgeons continued to reach the European theater very slowly, and, as of 1 April 1944, there were only seven fully qualified specialists in the United Kingdom. Twenty-one other medical officers had had, according to their records, some neurosurgical experience. All of them had been interviewed and their qualification cards had been completed by Major Scarff, who had acted as neurosurgical consultant during the interim between Colonel Davis' return to the Zone of Interior and Colonel Spurling's arrival in England in March 1944, but their actual abilities were unknown. During April 1944,

general hospitals arrived in the theater almost daily; however, few had neurosurgeons assigned, for the revised tables of organization for 1,000-bed general hospitals, which provided for a neurosurgeon (C-3131),[1] had not yet become effective. As a result, by 1 May 1944, the number of qualified neurosurgeons in the United Kingdom with full training and sufficient experience to head services in large general hospitals had increased to only 11. Concurrently, the number of medical officers with some neurosurgical training, whose qualifications were, however, either unknown or unproved, had reached only 30.

Visits to Hospitals

Summarized accounts of visits to hospitals in the United Kingdom Base before D-day will indicate how these inspections were utilized for the evaluation and proposed reassignment of neurosurgical personnel.

15 December 1943.—The chief of the surgical service at the ——th General Hospital is a forceful, forthright officer, who takes the direct approach to all problems and pursues them aggressively until results are achieved. He has the strong loyalty of his officers, and the morale of the unit is high. He is a general surgeon of considerable experience and competence.

There is no neurosurgeon in this unit and nobody in it who professes to know very much about the care of neurosurgical injuries. This hospital, fortunately, is less than an hour away from the ——th General Hospital, where there is a good neurosurgeon. It was therefore agreed that whenever patients with serious neurosurgical injuries were received, a consultation would be held with this neurosurgeon and with the regional consultant in neurosurgery for the area and that the patient would immediately be transferred to this hospital if these neurosurgeons so advised.

16 December 1943.—The ——th Station Hospital is a very well run organization, one indication being the excellent mess. There is, however, no neurosurgeon here, or anybody who knows much about neurosurgery. This hospital is about an hour and a half from the ——th General Hospital, and the same arrangements for the transfer of neurosurgical patients were made as had been made at the ——th General Hospital.

19 January 1944.—The neurosurgeon at the ——st General Hospital is an attractive young man with a nice personality but with only meager neurosurgical training. During the course of a 3-year rotating residency in surgery at ——, he spent 6 months as assistant to a neurosurgeon, Dr. ——. He knows how to deal with acute trauma of the cranial vault and the surface of the brain and could probably deal satisfactorily with penetrating wounds of the brain, but he knows little or no neurology and is not competent to do elective cranial surgery. He would be a good man for some hospitals, but he is not competent to meet the demands likely to be placed upon the neurosurgeon in this hospital. Plans should be made to reinforce this hospital with a stronger and more

[1] The classification symbol for neurosurgeons was MOS (Military Occupational Specialty) 3131, with the letters "A," "B," "C," and "D" indicating the degree of professional proficiency.

experienced neurosurgeon, though the present incumbent should also be given a chance to prove his worth.

26–27 March 1944.—The neurosurgical section in the ——th General Hospital is functioning in a station hospital setup. The neurosurgeon has had 4 years of general surgical training and 2 years of neurosurgery with —— at ——. He is bright, alert, and well rounded in the subject. Although he has had very little experience in electroneurosurgery, he has seen and helped with a great deal of excellent work. A lumbosacral disk then on the ward was correctly diagnosed and localized on clinical findings, and he was preparing to transfer the patient to one of the general hospitals because he did not feel that he had had enough experience with this type of case to handle it himself. His patients with head trauma, some of them with difficult, complicated injuries, had all been treated with fine judgment. The state of the wounds indicated that this man is a careful, technical surgeon. An expertly trained operating-room nurse is available to assist him.

Neurosurgical equipment was incomplete, but orders had been put through channels for the necessary instruments. A portable electrosurgical unit was particularly needed. Captain —— had invented several very ingenious devices, one of them a bulb syringe with a two-way valve which could be used to provide continuous irrigation of a wound. It would be useful to any neurosurgeon.

30 March 1944.—The ——th Station Hospital is performing a vital function in caring for Army Air Force personnel, and the neurosurgeon on the staff is thoroughly qualified to handle all types of neurosurgery. He is doing a good job but should have an assistant at the earliest possible moment.

12 April 1944.—The neurosurgeon at the ——th General Hospital in the ——th Medical Center is thoroughly qualified. He has had adequate training and a number of years of experience in private practice. All his cases were exceedingly well done. Several patients with disks had had complete symptomatic relief. He understands neurosurgical problems thoroughly, and he is the logical man to head the neurosurgical setup in this hospital group.

At the ——th General Hospital, the surgeon in charge of neurosurgery has had a very limited experience and apparently would not be a good team man. He would be better in an auxiliary surgical group in an evacuation hospital, where he would be working under supervision.

At the ——th General Hospital in the center, there is no recognized neurosurgeon, although two surgeons on the staff have had considerable experience. They could handle emergency neurosurgery but could not take on the exclusive responsibility of a neurosurgical service.

At the ——d General Hospital, the neurosurgeon is intensely interested in the subject but has had only sketchy training. He is careful and studious and could go far if given the proper opportunities. He would be well qualified to do peripheral neurosurgery but has had little experience in brain surgery. Certainly he has had insufficient experience to be in a setup in which he would be required to handle acute head trauma without support. He would do better as an assistant.

As a result of the inspection of the hospitals in this medical center, it was recommended to Col. James C. Kimbrough, MC, Chief, Professional Services Division, Office of the Chief Surgeon, Headquarters, ETOUSA, that the proposed neurosurgical center be established in the hospital nearest the airport at which casualties from the Continent would be received. Since this location was not known at the time, it was recommended that neurosurgical casualties be concentrated at the ——th General Hospital in the center, where Major ——was already well established, with trained assistants and nurses. This setup, however, should be regarded as only temporary and should not be permitted to overshadow the final decision, which should depend upon the location of the airport. Various recommendations for reassignment of personnel were made in the light of the comments on the neurosurgeons observed and evaluated during the visit to this center.

Neurosurgical Centers

As of 1 May 1944, when the total number of neurosurgeons of all degrees of training and experience in the United Kingdom numbered only 41, as just stated, there were 59 general hospitals in the theater. Heavy neurosurgical casualties were anticipated, and it was obvious that the available personnel, who were of all degrees of training and experience, could not possibly be spread out over all these hospitals. Evacuation hospitals staging for the invasion of the Continent also had to be provided with neurosurgeons. The conclusion was inescapable that competent care could be given to neurosurgical casualties only in specialized neurosurgical centers, so located as to facilitate triage and evacuation to them.

In a memorandum to Brig. Gen. (later Maj. Gen.) Paul R. Hawley, Chief Surgeon, ETOUSA, dated 6 May 1944, dealing with plans for the care of neurosurgical casualties, Colonel Spurling recommended the establishment of 10 specialized centers for neurosurgery at strategic points throughout the United Kingdom. The proposal was approved, with minor changes, on 17 May 1944. On 10 June 1944, Circular Letter No. 81 was issued listing nine hospitals for specialized neurosurgical treatment (fig. 2).

Approval of the proposal to establish these specialized centers permitted an economical use of the neurosurgical personnel available in the theater. The chief of service in each center was a fully qualified neurosurgeon of considerable experience. The assistants on the service had considerably less experience, though some of them were graduates of the short courses in neurosurgery given in the Zone of Interior (p. 24).

An effort was also made to assign all nursing personnel with neurosurgical experience to the neurosurgical centers, to work in the operating rooms and on the wards, and a similar effort was made to assign to them enlisted personnel with neurosurgical experience. Finally, personnel with experience in physical therapy and physical education were assigned to the neurosurgical centers.

Since few other types of wounds require for their proper treatment the combined efforts of as many different specialists as do neurosurgical casualties,



Key:

1. 67th General Hospital, Musgrove Park, Taunton, Somersetshire.
2. 100th General Hospital (later 117th General Hospital), Frenchay Park, Bristol, Gloucestershire.
3. 91st General Hospital, Churchill, Headington, Oxfordshire.
4. 49th Station Hospital, Diddington, near Saint Neots, Huntingdonshire.
5. 231st Station Hospital, Morley Hall, near Wymondham, Norfolk.
6. 65th General Hospital, Redgrave Park, near Botesdale, East Suffolk.
7. 1st General Hospital (later 7th General Hospital), North Mimms Park, North Mimms, Hertfordshire.
8. 98th General Hospital, Hermitage, near Newbury, Berkshire.
9. 158th General Hospital, Odstock, near Salisbury, Wiltshire.

FIGURE 2.—Hospitals with facilities for specialized neurosurgical treatment in the United Kingdom Base, ETOUSA.

special pains were taken, through the cooperation of the consultants group, to assign to each of the neurosurgical centers competent specialists in radiology, medical neurology, orthopedic surgery, plastic surgery, urology, otolaryngology, and ophthalmology.

Many difficulties were naturally encountered in effecting the large transfer of personnel required by this program, and a number of compromises had to be made, but, by 1 June 1944, the task was completed, and the neurosurgical services in the designated centers were ready to receive casualties.

In the fall and winter of 1944, acute special treatment hospitals were established on the Continent, at Liége, Bar-le Duc, and Nancy, and in the Paris area. This plan had to be adopted, because, with the coming of winter, bad weather often made air evacuation to the United Kingdom uncertain or impossible. These hospitals, however, were not intended to receive neurosurgical patients for definitive care unless air evacuation was impossible or unless the patients were not transportable, for other reasons, after they could be transferred from evacuation hospitals.

With the expansion of general hospital facilities on the Continent, which began after the liberation of Paris in August 1944, it was expected that there would be a demand that more and more definitive neurosurgery be done in these installations. The policy was occasionally discussed, but it was consistently discouraged by Colonel Spurling. Practically all neurosurgical casualties had to be evacuated eventually to the Zone of Interior, and it was his opinion that as long as neurosurgical beds were available in the United Kingdom and evacuation facilities were available from it, the better plan was to continue to provide definitive neurosurgical care in the hospitals in this base rather than on the Continent. With the exceptions just noted, this general policy continued in effect to the end of the war in Europe.

Evacuation Hospitals

A problem of pressing importance in the planning for D-day was the staffing of the evacuation hospitals of the First U. S. Army, which was to spearhead the invasion of the Continent. Colonel Davis, in January 1943, had stated an excellent set of principles for the employment of neurosurgical personnel in wartime, and they were generally adhered to. They were as follows:

1. Qualified, experienced neurosurgeons, of recognized ability, are not necessary in the combat zone. They will be of greatest usefulness in general hospitals in the communications zone, in which definitive, complete neurosurgical care should be given.

2. Medical officers trained in general surgery and instructed in the most effective methods for the immediate surgical treatment of neurosurgical wounds should be assigned to the combat zone.

3. Casualties with neurosurgical injuries, after initial wound surgery, should be evacuated as rapidly as possible to general hospitals in the communications zone for definitive treatment by experienced neurosurgeons.

4. Mobile neurosurgical teams, with appropriate equipment, should not be sent forward into the combat zone. It would be wiser to attach to these mobile teams young general surgeons, who, preferably, have had some neurosurgical experience during their residencies and whose basic surgical training and experience have been of such a quality that they would do neither too much nor too little in the initial wound surgery of neurosurgical casualties.

By a rather complicated shifting of personnel, it was eventually possible to assign to each evacuation hospital a so-called working neurosurgeon. A surgeon of this class had usually had 3 or 4 years of general surgical training and, in addition, had completed the short course in neurosurgery given in the Zone of Interior (p. 24). He had not usually had a great deal of independent surgical experience, but, since his general surgical background was good, it was believed that he would function well at the evacuation hospital level, and certainly better at that level than at the level of the general hospital, in which it was planned that definitive neurosurgery would be done.

The situation in the Third U. S. Army, which had been engaged in intensive training in northern England for several months before D-day, differed from the situation in the First U. S. Army in that each of the evacuation hospitals attached to it had arrived with its own neurosurgeon. At a meeting arranged with these neurosurgeons and with the chiefs of the surgical services of these hospitals, it became clear that a number of changes in personnel could profitably be made.

If a designated neurosurgeon seemed to possess insufficient qualifications for his assignment in the evacuation hospital, he was relieved of duty in it and transferred to a general hospital as a neurosurgical ward officer. His replacement was furnished from the general hospital list. The shifting of personnel, while based on correct principles, unfortunately had to be done on a rather haphazard basis in many instances because the capabilities of many of the officers for doing independent neurosurgery were completely unknown. It was believed, however, that the experiences in handling neurosurgical casualties which would be reported by the First U. S. Army could be used as a guide for the wiser allocation of neurosurgical personnel in the Third U. S. Army when it eventually went into action.

Transit Hospitals

Medical planning for the invasion of France called for casualties wounded in the period after D-day to receive their first definitive neurosurgery in England, at the level of transit hospitals on or near the coast. These were fixed hospital installations which served in the United Kingdom Base (and later, occasionally, on the Continent). They operated as evacuation hospitals and held casualties only long enough for the performance of sufficient definitive surgery to make them safely transportable to general hospitals located farther inland. These hospitals were placed with reference to their accessibility by rail and ambulance.

Individual neurosurgeons were not assigned to transit hospitals, which were staffed, instead, by neurosurgical teams. These teams, six of which were alerted for D-day, were composed of personnel supplied by general hospitals farther north in England. Each team consisted of a surgeon, an assistant surgeon, an anesthetist, a scrub nurse, and two trained aidmen, and each team had its own allocation of specialized neurosurgical equipment. The training of these teams had included practice in knocking down their equipment, transporting it, and setting it up quickly in another location, ready for instant use. Special training in operating-room technique was also provided.

The allocation of the neurosurgical teams to transit hospitals was left to the decision of the senior consultant in neurosurgery and the surgical consultant to the Southern Base Section surgeon, under whose jurisdiction most of the early casualties from the Continent naturally fell.

In March 1944, Colonel Spurling recommended to Col. (later Brig. Gen.) Elliott C. Cutler, MC, Consultant in Surgery, Office of the Chief Surgeon, Headquarters, ETOUSA, that it would be advisable to supplement the teams to be assigned to the transit hospitals by three additional standby neurosurgical teams, which would be called upon if neurosurgical casualties exceeded the estimates and the provisions for their care proved inadequate. In such an eventuality, these teams could be mobilized quickly and assigned as they were needed. It was recommended that personnel for them be obtained from fixed hospitals then assigned to locations in what would probably be a negative zone immediately after D-day. Finally, it was recommended that each team, like the teams from the auxiliary surgical groups, consist of a neurosurgeon of considerable experience, an assistant surgeon, an anesthetist, an operating-room nurse, and two enlisted surgical technicians.

This recommendation was accepted and the teams described were provided, but they did not have to be called upon, since the pre-D-day provisions for the care of neurosurgical casualties proved adequate.

Auxiliary Surgical Groups

Auxiliary surgical groups proved to be among the most effective medical units functioning in the European theater. Their efficiency was chiefly due to two factors. The first was that they were carefully staffed and contained some of the best surgical talent in the theater. The second was that, from the standpoint of administration, these teams could be utilized more simply than could the personnel of other medical organizations. The use of the teams, which was dictated by the tactical situation, was controlled, for the most part, by the surgical consultant to the army surgeon. Their movement from one location to another, as the need for their services arose, could therefore be achieved without a complicated series of administrative orders.

The auxiliary surgical groups, as originally constituted, provided for four neurosurgical teams each. The 3d Auxiliary Surgical Group, however, which had been assigned to the First U. S. Army, had only two neurosurgical teams

on its table of organization. After consultation with Colonel Crisler, surgical consultant in the First U. S. Army, it was decided to create two additional neurosurgical teams in this group. This was accomplished by transferring so-called working neurosurgeons from the general hospital pool. The 1st Auxiliary Surgical Group had been assigned to work temporarily with the general hospitals in the United Kingdom. Its ranks had been depleted of one neurosurgical team, and it was decided that no replacement would be attempted immediately.

Changes in Assignment of Personnel

As circumstances of evacuation changed from time to time after D-day, neurosurgical personnel were shifted to meet the changing circumstances. In northeastern England, for example, where casualties were derived for the most part from the Army Air Forces, the neurosurgical load gradually decreased as fighter protection for bombers became more effective. As a consequence, early in the summer of 1944, it was possible to close a neurosurgical center in East Anglia and to shift its personnel to a hospital in southern England, where the need for trained neurosurgeons was then acute. At about the same time, a general hospital near London was found inconvenient for evacuation, and its neurosurgical personnel were transferred to a hospital in a location where neurosurgical casualties were being received in large numbers.

Neurosurgical services in hospitals designed for specialized treatment increased steadily in size, and by December 1944 the patient load at these centers varied from 150 to 650 each. Neurosurgical staffs in the larger centers had from time to time been augmented by neurosurgeons newly arrived in the theater; all officers with neurosurgical qualifications had been utilized for this purpose. Current needs, however, continued to be so pressing that in most of the neurosurgical centers it was necessary to withdraw surgeons from general surgical services and rapidly train them in neurosurgical principles and techniques. In one of the largest centers, at the 160th General Hospital, Stowell Park, Gloucestershire, England, 16 neurosurgical officers were once assigned at the same time; morning and afternoon operative schedules were in effect, and as many as 150 major neurosurgical procedures were carried out in a single month. The average patient-officer load in most centers was consistently greater on the neurosurgical than on any other service.

In order to care for the heavy load of neurosurgical casualties, it was found expedient to increase the personnel of existing installations rather than to establish additional special treatment hospitals. The supply of fully qualified neurosurgeons was still extremely limited, and a service headed by a well-qualified neurosurgeon, assisted by less well qualified medical officers, functioned more efficiently than did new services headed by indifferently qualified neurosurgeons. The arrangement described permitted experienced neurosurgeons to apply their skills in complicated cases and also to function as teachers and demonstrators for younger, less well trained officers.

Shifting personnel to meet the requirements of changing circumstances proved a never-ending difficulty. Commanding officers, however much they might desire to improve professional services in their installations, were obliged to adhere rigidly to tables of organization. This difficulty in the assignment of neurosurgical personnel was circumvented by assigning neurosurgical officers to hospitals in the vicinity in which table of organization vacancies existed and carrying them on a temporary duty status in the hospitals in which the need for their services existed. Although the arrangement was not desirable, if only because it frequently deprived officers who had rendered outstanding services of their chances for advancement in rank, as matters stood it seemed the only way in which the more important need of rendering specialized professional care where it was most needed could be met.

Free movement of highly trained professional personnel between hospital installations in a theater of operations is obviously necessary for proper professional care of casualties. It should be a relatively simple procedure. The senior consultant in neurosurgery, however, frequently recorded in his official diary that he had found it the most complicated task he had to perform in the European theater and one which neither the passage of time nor his increasing experience in administrative matters in any way simplified.

Rotation of personnel.—After the initial phases of the European campaign had been concluded, a plan was worked out in the Chief Surgeon's Office for the rotation of personnel for 60-day periods between forward hospital units and units in the rear echelons. As far as neurosurgeons were concerned, it was promptly evident that personnel working in general hospitals had very little conception of the problems facing surgeons in evacuation hospitals and that surgeons in forward units knew little of surgical procedures undertaken in general hospitals. Also, surgeons in forward units were anxious to follow up patients they had treated and to enjoy what they believed to be the quiet activity of installations in the year.

A rotation system was finally put into effect in December 1944, and approximately half of the neurosurgical personnel in the European theater had the benefit of the program before the end of hostilities. The free exchange of ideas, the fuller appreciation of each other's problems, and the heightening of morale which comes with changes of assignment made rotation of personnel a valuable innovation.[2]

An important byproduct of the rotation program was that army neurosurgeons became fully aware, some of them for the first time, of the importance of thorough debridement at the initial operation for craniocerebral injury. Observations in a general hospital made clear the devastating effects which could result from incomplete debridement. Neurosurgeons from general hospitals, for their part, learned in forward installations how difficult it is to do superior

[2] *Rotation was an excellent plan, in theory, for all personnel. It was entirely practical for neurosurgical personnel,* whose numbers were limited. Their exchange could always, therefore, be handled on a personal and individual basis, and very few medical units were involved. Rotation, however, was entirely impractical for most other medical personnel, especially battalion and regimental surgeons, the very nature of whose missions would have made it particularly desirable.—J. B. C., Jr.

neurosurgery under field conditions. Even experienced officers were often chagrined, on their return to the general hospital level, to find that debridements had been incomplete and inadequate and that, as a result, wounds were infected, sometimes in cases which they had handled themselves in forward hospitals.

Some neurosurgeons ended their periods of service with a remarkably wide experience. One of them, for instance, was chief of the neurosurgical service in a named general hospital in the Zone of Interior for 18 months before he went overseas. During 30 months overseas, he served in the field with an auxiliary surgical group in North Africa, Sicily, France, Belgium, Holland, and Germany. During a portion of this time, he acted as consultant in neurosurgery to an army surgeon. He then worked in a neurosurgical center in England, where the later results of primary procedures in forward areas could be evaluated. Finally, he was in charge of a neurosurgical center in the Zone of Interior. His experience included hundreds of ordinary skull fractures and simple lacerations of the scalp, more than a hundred penetrating wounds of the brain, 62 brain abscesses, 31 cases of cerebral fungi, over a hundred laminectomies for injuries of the spine, and more than 300 peripheral nerve lesions. His total experience comprised more than 1,100 neurosurgical procedures, the great majority done in evacuation or field hospitals near the frontlines. It ranged from emergency procedures in clearing stations to peripheral nerve surgery in a general hospital.

It was Colonel Spurling's opinion, as a result of this brief experience with rotation of personnel of forward and rear echelons, that such a policy should be arranged for, in advance, in all future medicomilitary plans, on the ground that it contributes to better professional care and improves morale among medical personnel at all levels in the chain of evacuation.

CRITIQUE OF ASSIGNMENT OF NEUROSURGICAL PERSONNEL

At the end of the campaign in Europe, in a memorandum to General Hawley dated 25 May 1945, Colonel Spurling, on the basis of the neurosurgical experience during the active fighting, made further comments regarding the use of auxiliary surgical groups in the management of neurosurgical casualties.[3] The original plan, he pointed out, had been to provide each evacuation hospital with a working neurosurgical team. In order to do this, it had been necessary to cut corners on qualifications, and, as a result, some of the work done by less experienced men in the early days of the European invasion was not up to standard.[4]

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[3] Semiannual Report, Senior Consultant in Neurosurgery, Office of the Chief Surgeon, Headquarters, ETOUSA, 1 Jan. 1945 to 30 June 1945.

[4] In previous analyses of the neurosurgical experience, Colonel Spurling had warned against the pitfalls which had to be avoided in the assignment and reassignment of personnel. In particular, he stated, great care had to be exercised, in disturbing good units to build up the staff of poor units, that all units, by the time the job was completed, would not be uniformly second rate.

An analysis of the neurosurgical experience seemed to indicate that the plan adopted had been responsible for considerable wastage of trained personnel. These neurosurgical teams had not worked actively more than a third of the time that they were attached to evacuation hospitals. As the tactical situation constantly shifted, these hospitals were often moving, were shut down, or were awaiting movement. At these times, although their neurosurgeons were idle, they could be reassigned or detached from them only with difficulty, because of the natural reluctance of commanding officers to part with their professional personnel for fear that they would never get them back.

On the basis of this experience, Colonel Spurling suggested that neurosurgeons in an active theater of operations should not be authorized in the table of organization of an evacuation hospital but should be organized into teams assigned to auxiliary surgical groups, to be dispatched to functioning evacuation hospitals as needed, at the discretion of the consultant in surgery to the army surgeon. One neurosurgical team could thus care for the casualties in two evacuation hospitals, and the services of the neurosurgeons who composed it would be utilized almost continuously. He further suggested that each team should be composed of a chief neurosurgeon; an assistant neurosurgeon capable of handling an operating table under the supervision of the chief neurosurgeon; a qualified anesthetist; an assistant anesthetist, preferably an aidman; two aidmen trained as scrub nurses; and four aidmen trained as operating-room assistants.

Colonel Spurling called attention to the fact that nurses did not appear in these proposed teams, because it had been the rather general experience of neurosurgical officers that trained aidmen met conditions better in army areas. He also pointed out that aidmen had been substituted for assistant anesthetists. The reason was that more than half of all neurosurgical procedures are done under procaine infiltration anesthesia and that trained aidmen, under the supervision of an anesthetist, could take blood pressures, administer fluids, and be responsible for the general care of the patient on the operating table.

The opinions just expressed by Colonel Spurling concerning the utilization of neurosurgical personnel in the European theater in World War II were quite generally held. The average number of surgical cases handled during the active fighting, from D-day to V–E Day, by each neurosurgeon was 150. Each of these officers, had he been moved freely from installation to installation as he was needed, could easily have done a hundred or more cases each month. War is wasteful, but this wastage was not necessary.

There are at least two reasons why it would probably be better not to authorize a neurosurgeon (or any other critical specialist) on the tables of organization of any field or evacuation hospital. The first reason is that these units are necessarily deactivated for longer or shorter periods for staging and other purposes; during these periods, specialists do not function, and their services are therefore totally lost. The second reason is that when once a medical officer is authorized on a table of organization of any unit by specialty

MOS, it is a major operation to attach him to another organization for any reason.[5]

It would therefore seem more efficient to make neurosurgeons, along with other critical specialists, members of teams which would be freely mobile and entirely self-contained, so that they could be moved in a few hours' notice from one location to another as the need for their services arose. Movement could be by small plane or command car. Standard operating supplies are available at all installations, and the teams would need to carry nothing beyond their own specialized equipment. A mobile hospital truck, such as was used by British and Canadian neurosurgeons in World War II, is somewhat more elaborate than is necessary and retards mobility in bad weather. Moreover, United States Army forward hospitals are better equipped than either British or Canadian hospitals, and specialized equipment for these teams could be correspondingly reduced.

During World War II, neurosurgical teams from auxiliary surgical groups which were attached to evacuation hospitals were, in effect, supplemental personnel. They served in addition to the neurosurgical personnel provided in the table of organization, and neither they nor the organic neurosurgical personnel were fully utilized. By the plan just proposed, there would be no organic neurosurgical personnel in evacuation hospitals; instead, neurosurgical teams would be attached temporarily to these hospitals as the need for their services arose. The administrative difficulties of such a plan should not be insuperable. Had it been in effect in the European theater in World War II, the same number of casualties could have been handled, and handled far more effectively, with half the neurosurgical personnel used.

CARE OF NEUROSURGICAL CASUALTIES

Care of Civilian-Type Injuries

When the European theater was first established, neurosurgical injuries encountered in United States Army hospitals, with the exception of a few combat injuries sustained by Army Air Force personnel, were the result of accidents comparable to those encountered in civilian practice. Originally, most United States Army soldiers with this type of injury were treated in the British EMS (Emergency Medical Service) hospitals, where they remained for various periods of time before discharge, without supervision by United States Army medical officers.

Eventually, on Colonel Davis' request, a census of these patients was furnished General Hawley's Professional Services Division, with the name,

[5] While this plan undoubtedly has the advantages stated, there are certain disadvantages connected with it which must be borne in mind. Against the gain in efficient use of personnel in short supply in this specialized field must be set the loss in morale and in esprit de corps invariably sustained by personnel who are not permanently assigned to some unit. This loss, although intangible, is almost always ultimately reflected in performance. Furthermore, the matter of promotion must be considered; this always presents difficulties for personnel who are never in one place for any length of time. Finally, if this plan were adopted for neurosurgical personnel, it might, with equal reason, be applied to other specialist personnel, which would inevitably produce a reductio ad absurdum.—J. B. C., Jr.

organization, date of admission, and diagnosis of each man. At a meeting with the chief consultant in neurosurgery of the EMS, Mr. Geoffrey Jefferson, and his regional associates, a plan was evolved whereby the senior consultant in neurosurgery would be informed of the admission to all EMS hospitals of all United States Army soldiers with neurologic injuries. The United States Army consultant would then see the patient personally, or would arrange to have him examined by a United States Army neurosurgeon, or would delegate the man's care to the regional EMS neurosurgeon until one or the other of these plans could be put into effect. When this policy was fully implemented, it proved most effective. The use of the facilities of EMS hospitals was reduced to a minimum, and, if they had to be employed, the nature of the injury and the course of the patient were known to the senior neurosurgical consultant or his delegated representative.

The clinical principles by which neurosurgical injuries were managed at this time in hospitals in the United Kingdom Base are described under appropriate headings in this volume and in volume II [6] of the neurosurgical history. Circular Letter No. 75, which covered the management of craniocerebral, spinal cord, and peripheral nerve injuries, as well as the management of ruptured disks, was issued from the Office of the Chief Surgeon on 4 December 1942.

At the same time that these principles of clinical management were set forth, the policy was established that patients with neurosurgical injuries should be treated only in general hospitals in which qualified neurosurgeons were on the staff. Until a general hospital was established in East Anglia, it was necessary to use two station hospitals in this area, the 49th and the 231st, for the definitive surgical treatment of these patients. Otherwise, the policy just stated was strictly adhered to.

Care of Combat-Incurred Injuries

In planning for the care of neurosurgical casualties before D-day, it promptly became evident that a forthright definition of what constitutes such a neurosurgical casualty must be formulated and must be authorized by the Chief Surgeon. The definition which was finally approved, and which remained in effect throughout the European campaign, was that all surgical lesions of the central, peripheral, and sympathetic nervous systems were to be considered neurosurgical problems. This definition was in contrast to that currently in force in the British Army, by which neurosurgical injuries were limited to surgical lesions of the central nervous system.

Operation of Specialized Centers

After the plan for the establishment of specialized centers for the treatment of neurosurgical casualties in the United Kingdom had been approved (p. 51)

[6] Medical Department, United States Army. Surgery in World War II. Neurosurgery. Volume II. [In preparation.]

on 17 May 1944, the following proposed circular letter was drafted by Colonel Spurling:

Subject: Care of Neurosurgical Casualties in the U. K.

1. The following hospitals are designated as special treatment centers for neurosurgical patients: 91st General Hospital, 67th General Hospital, 158th General Hospital, 98th General Hospital, 100th General Hospital, 160th General Hospital, 55th General Hospital, 83d General Hospital, 65th General Hospital, 49th Station Hospital, 231st Station Hospital, and 1st General Hospital.

2. All neurosurgical casualties in the U. K. will be evacuated to the nearest designated center for treatment and disposition as soon as the condition of the patient permits safe transportation.

3. The senior neurosurgeon in each center will act as regional consultant.

This letter, it will be noted, made it mandatory to transfer to these specialized centers all neurosurgical and other casualties requiring specialized care as soon as their condition permitted transportation. For some reason, never fully clarified, the publication of the circular letter dealing with the neurosurgical centers was delayed in administrative channels until 4 days after D-day. Then the following letter [7] was issued:

1. Certain diseases and injuries require treatment in which a high degree of specialization is needed. To provide this specialized treatment, certain hospitals in the U. K. have been provided with the necessary equipment and staffed with qualified personnel.

2. There are three (3) hospital centers in operation in the U. K.; the 12th Hospital Center, Malvern Wells, Wores., the 15th Hospital Center, Cirencester, Glos., the 6810 Hospital Center, Whitchurch, Flintshire. In each of the above listed hospital centers, there are one or more hospitals provided with equipment and personnel for highly specialized treatment in:

a. Neurosurgery
b. Thoracic surgery
c. Plastic and maxillofacial surgery
d. Surgical treatment of extensive burns
e. Urological surgery

3. In addition to hospital centers, there are located throughout the U. K. a number of hospitals with personnel and equipment necessary for specialized treatment:

Neurosurgery

Location	*Plant No.*
Churchill, Headington, Oxon	4144
Musgrove Park, Taunton, Somerset	4108
Odstock, Nr. Salisbury, Wilts	4124
Hermitage, Nr. Newbury, Berks	4140
Frenchay Park, Bristol, Glos	4166
Redgrave Park, Nr. Botesdale, East Suffolk	4209
Diddington, Nr. St. Neots, Hunts	4204
Morley Hall, Nr. Wymondham, Norfolk	4210
North Mimms Park, North Mimms, Herts	4211

4. The Commanding Officer of any hospital lacking facilities and sufficiently qualified personnel for the specialized treatment of any patient will transfer the patient to the most available hospital center or hospital designated in paragraphs 2 and 3 above. The Com-

[7] Circular Letter No. 81, Office of the Chief Surgeon, Headquarters, ETOUSA, 10 June 1944, subject: Hospitals With Facilities for Specialized Treatment.

manding Officers of the centers will designate the hospital within the center to which the patient will be admitted.

Because of the wording "lacking facilities and sufficiently qualified personnel," this letter was interpreted by some commanding officers of general hospitals as permissive and not mandatory. As a consequence, a period of considerable confusion ensued, in the course of which operations on neurosurgical casualties were sometimes done in general hospitals by personnel with little or no neurosurgical experience. Eventually, although the circular letter was never rescinded, the situation was clarified, and thereafter casualties who required specialized neurosurgical care were transferred, without unnecessary delay, to the special treatment hospitals which had been set up for the care of such casualties.

Evacuation

After the initial rush of casualties following D-day and dissemination of information concerning the location and objectives of the specialized neurosurgical centers, triage of neurosurgical patients proceeded efficiently. In particular, delay in evacuation from the far shore steadily lessened, chiefly as the result of improvement in the management of port facilities.

In the initial planning for D-day, hards had been established for the unloading of craft at Weymouth, Plymouth, and Southampton, on the southern shore of England. All three points were conveniently located in reference to the invasion beaches on the Normandy coast. The hard at Plymouth was seldom used for neurosurgical casualties, and the hard at Southampton never functioned to full capacity.

About D-plus-15-day, small numbers of casualties began to be evacuated by air, and by the end of June approximately half of the wounded were thus reaching England. As air evacuation steadily increased in efficiency, more and more freshly wounded neurosurgical patients began to arrive in England, and hospital needs changed correspondingly. Two new transit neurosurgical centers were therefore established in close proximity to airfields, the corresponding decrease in the number of sea-evacuated casualties making it possible to shift the personnel from one of the original transit hospitals in southern England to meet the new conditions.

Once the system of air evacuation was fully established, most of the seriously injuried neurosurgical casualties were flown to the United Kingdom Base hospitals during the summer of 1944. Only the more lightly wounded were brought over by sea. Weather conditions were fortunately good, and air transport was seldom hindered, so that it was not unusual to receive neurosurgical casualties in transit hospitals in England 12 to 14 hours after they had been wounded on the Continent. In one recorded instance, a soldier with a gunshot wound of the brain was being prepared for surgery in a general hospital in England only 4 hours after he had been injured in France. In another instance, the timelag was only 5 hours. During the winter, air

evacuation was erratic because it was at the mercy of the weather, and most casualties were transported by sea.

A system of air evacuation of neurosurgical casualties to the Zone of Interior was set up in the summer of 1944, and for a time it was thought that this was the ideal method of transportation for them. With the coming of bad weather in the fall and winter, air transportation became less and less satisfactory, and, at the same time, reports from neurosurgical centers in the Zone of Interior indicated that casualties with injuries of the spinal cord, who furnished the most serious problems of evacuation, were arriving in better condition by ship than by air. Air evacuation was resumed in the spring and was more satisfactory because of the lessons which had been learned about how to care for spinal cord injuries in transit.

Inspection of Neurosurgical Facilities in the United Kingdom Base

On D-plus-4-day, by which time casualties were being received in a steady flow from the Normandy beaches, Colonel Spurling made complete rounds in all transit hospitals, evacuation hospitals, and field hospitals in southern England, to determine the effectiveness of the plans which had been set up for the triage and professional care of neurosurgical casualties. His report of 29 June 1944 to Colonel Kimbrough made the following points:[8]

1. Not all commanding officers of hospital installations had had advance information concerning the location of specialized centers for neurosurgical treatment. As a result, some neurosurgical casualties had not been well handled, especially in hospitals to which neurosurgical teams were not assigned.

2. Treatment had been delayed too long in some instances. Some casualties, seen immediately on their arrival, had been wounded 5 days previously in France and had been without definitive neurosurgical care during this time. On the whole, however, first aid neurosurgical care had been excellent.

3. Delay in treatment of neurosurgical casualties in transit hospitals could readily be corrected. Reasons for the delay included overeagerness on the part of administrative officers to evacuate seriously injured patients farther to the north; use of neurosurgical casualties who should have been considered nontransportable to fill up hospital trains because evacuation directives had been interpreted to mean that trains must be completely filled before they moved; failure to use ambulances to move neurosurgical casualties to special treatment hospitals; and a basic delay, which was steadily being reduced, inherent in evacuation of casualties from the far shore.

In this report, Colonel Spurling expressed himself as being, in general, greatly impressed with the excellent care rendered by neurosurgical personnel and with their ingenuity in improvising equipment and methods to meet military conditions. In this connection, he wrote to Col. (later Brig. Gen.) Fred

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[8] Informal Routing Slip, Lt. Col. R. G. Spurling, to Col. J. C. Kimbrough, Chief, Professional Services Division, Office of the Chief Surgeon, Headquarters, ETOUSA, 29 June 1944, subject: Tour of Special Treatment Hospitals and Transient Hospitals for Neurosurgery in the SBS.

W. Rankin, MC, Chief Consultant in Surgery, Office of the Surgeon General, as follows: "I was greatly impressed with the manner in which the young neurosurgeons took hold of a difficult situation and delivered a thoroughly competent job. Many of them worked without rest for 3, 4, and 5 days. I failed to see one instance of negligence, and I saw very few cases in which mature judgment had not been exercised."

Inspection of Neurosurgical Facilities on the Normandy Beachhead

On the invitation of Col. (later Brig. Gen.) John A. Rogers, MC, Surgeon, First U. S. Army, Colonel Spurling visited the Normandy beachhead on 12 July 1944 to study the management of neurosurgical casualties in army evacuation hospitals and other installations and to make such recommendations as seemed indicated for the future functioning of these units. After a 4-day inspection trip in all forward units, including collecting stations, division clearing stations, field hospitals, and evacuation hospitals, and after consultation with the consultants assigned to the First U. S. Army, the following recommendations were made to the surgeon of that army:

1. The tendency in evacuation hospitals to do too much definitive surgery should be restrained, and all transportable casualties should be evacuated to the United Kingdom for this type of care.

2. All patients with head injuries should be evacuated without surgical treatment except patients with insignificant injuries, such as scalp wounds, who may be expected to return to duty within 10 days, and patients with brain wounds which do not permit of transportation. Injuries in the latter category were defined as compound brain wounds with presenting cerebral tissue (fungus), brain wounds associated with progressive deterioration in the patient's condition, and brain wounds associated with other injuries which in themselves contraindicated prompt evacuation. Unconscious patients were always to be held and treated until the exact status of the intracranial injury was determined, after which they would be evacuated or held according to the indications.

3. Patients with injuries of the spine accompanied by paralysis should be promptly evacuated by air when this was possible. They should be left on stretchers and an attempt made to improve their condition according to instructions in the Manual of Therapy. If air evacuation was not possible, and if there was reasonable doubt about complete severance of the spinal cord, exploratory operation should be carried out, under local analgesia, without opening the dura unless definite indications existed for this procedure. It was emphasized that the decision concerning the need for operation should be made by the nearest available neurosurgeon.

4. Two neurosurgical teams should be outfitted in a mobile surgical unit and made available for roving assignments in the Omaha and Utah sectors of the Normandy beachhead. The duties of these teams were defined as supervision of work in evacuation hospitals in their respective zones, and operation

of their mobile units in whatever evacuation hospitals the Consultant in Surgery, First U. S. Army, should designate as most in need of their services. It was also suggested that the personnel of the teams should go forward to field hospitals and, if necessary, to division clearing stations to care for nontransportable neurosurgical casualties.

5. The basic suggestions made in this report were incorporated in a letter issued from the Office of the Surgeon, Headquarters, First U. S. Army, 20 July 1944.[9] Later, these same policies were adopted by the Third, Ninth, Seventh, and Fifteenth U. S. Armies as they successively became operational. Uniform management of neurosurgical casualties was therefore in effect, for all practical purposes, throughout the period of active hostilities in Europe. They were followed consistently except when the local tactical situation, adverse flying weather, or difficulties of evacuation for other reasons made it necessary to modify them.

CRITIQUE OF CARE OF NEUROSURGICAL CASUALTIES

On the basis of his observations of the care of neurosurgical casualties during the active fighting in Europe, the senior consultant in neurosurgery on 25 May 1945, submitted to Colonel Kimbrough a memorandum entitled "Some essential facts we have learned in this theater about the care of neurosurgical casualties." In it, the following points were made:

1. A competent neurosurgical team can handle the neurosurgical work of at least 5,000 active hospital beds.

2. The present tables of organization for general hospitals, which provide a neurosurgeon (C–3131) for each 1,000-bed hospital, are wasteful of specialized personnel. The neurosurgical workload in an installation of this size is not heavy enough to keep him occupied.

3. By utilizing the 1,000-bed hospital as part of a hospital center or hospital group and by designating one of the hospitals in the center as a specialized treatment center for neurosurgery, as was done in the United Kingdom Base, wastage of neurosurgical personnel is overcome. In some such centers in the United Kingdom, a chief of service supervised 4 or 5 operating teams, and a high degree of efficiency was attained. Loads of 600 patients were carried month after month, and between 100 and 150 major neurosurgical operations were performed each month. Difficulties about the transfer of personnel which arose from table of organization requirements were readily overcome in hospital centers. Finally, early triage of casualties, which is essential if the best results in neurosurgery are to be secured, was extremely efficient in hospital centers.

4. If hospital centers are not considered practical, hospitals of 1,500- to 3,000-bed capacity might solve the neurosurgical problem. Hospitals of 2,000-bed capacity were brought to France in support of the Seventh U. S.

[9] Medical News Letter No. 14, Office of the Surgeon, Headquarters, First U. S. Army, 20 July 1944, subject: Management of Neuro-Surgical Cases.

Army and fully justified the assignment of first-rate neurosurgical talent to them.

5. If general hospitals in the Pacific were to be organized on a 1,000-bed basis, the only practical solution of the neurosurgical problem in them would be to group them into hospital centers and to designate one hospital in each center as a specialized treatment center for neurosurgery, with the assignment of neurosurgical personnel adjusted according to the workload. The chief of section should be a well-qualified man (A, B, or C–3131), but his assistants could be young general surgeons who had had some neurosurgical experience (C or D–3131).

Colonel Spurling concluded the memorandum by pointing out that if, in a future war, the establishment of hospital centers should not prove practical, neurosurgical personnel would best be conserved by the organization of the neurosurgeons available at the general hospital level into neurosurgical teams, to be attached either to an auxiliary surgical group or a headquarters pool, whence they could be dispatched to whatever points might be most in need of neurosurgical services. This plan would eliminate table of organization difficulties which had complicated the shifting of neurosurgical personnel in World War II in the European theater. A similar plan had, in fact, worked very satisfactorily in army evacuation hospitals during that period.

REDEPLOYMENT OF NEUROSURGICAL PERSONNEL

After V–E Day, neurosurgical casualties in evacuation hospitals on the Continent were quickly cleared to general hospitals in the Communications Zone, and most of them (about 90 percent) were eventually transferred to the United Kingdom for further treatment and disposition. As a result, most neurosurgeons in forward hospitals soon found themselves with nothing productive to do. A reasonable period of inactivity was naturally welcome, but, after it had extended for several weeks, activity became desirable. Whenever possible, therefore, these officers were sent on temporary duty, for periods of 30 to 60 days, to neurosurgical centers in the United Kingdom, and, while they were there, every effort was made to permit them to visit British neurosurgical installations also.

This program could not be completed because of the urgency of redeployment of units to the Pacific, which made it necessary to recall the officers to their units. Staging, unfortunately, again resulted in long periods of inactivity, without contact with professional work, which had a very bad effect upon morale, in addition to the obvious waste of highly skilled personnel.

Table of organization requirements for hospitals to be redeployed directly from the European theater to the Pacific called for 59 neurosurgeons, with the fulfillment of the requirement mandatory as long as a qualified neurosurgeon was available, regardless of the length of his overseas service or any other consideration. As soon as the requirements for the Pacific became known and a survey of the situation could be made, the following memorandum, repro-

duced almost in full because of the gravity of the shortage of personnel it indicates, was addressed to General Hawley on 11 June 1945, under the title "Review of neurosurgery in the ETO with respect to redeployment":

1. An analysis has been made of all neurosurgical personnel in this theater, based upon all available information in the Personnel Division [of the Theater Chief Surgeon's Office]. Critical scores are not yet available, but the records of most of these men I know personally.

2. *Available personnel:* There are 92 men listed and classified as 3131 and they are rated as follows:

a. *B*–3131—12 (fully qualified neurosurgeons).

b. *C*–3131—31. For the most part this group consists of young general surgeons who have been trained in traumatic surgery through short courses in the States.

c. *D*–3131—49. Of this group 25 are known to be capable of acting as chiefs of services in evacuation hospitals or auxiliary surgical groups. The others have been acting as assistants or ward officers on established neurosurgical services. *Eight* are nine-month internes who have worked as ward officers on neurosurgical services for a period of months. The reason many of these men were given the classification of 3131 was to keep them in neurosurgery as assistants or ward officers. No lower classification to accomplish this purpose was available. Therefore, there is a wide spread in the capabilities within the group.

3. I have listed, with some misgiving, the personnel as follows:

a. Qualified for chiefs of neurosurgical services in evacuation hospitals—32.

b. Qualified for chiefs of neurosurgical services in general hospitals—40.

c. Qualified for chiefs of neurosurgical teams, auxiliary surgical groups—8.

d. Qualified as assistants (ward officers) in general hospitals, evacuation hospitals, or auxiliary surgical groups—12.

4. *Requirements:* According to the present T/O, approximately 170 neurosurgeons are required. Fulfillment of this quota is obviously impossible. By eliminating most of the neurosurgeons from the indirectly deployed general hospitals, the urgent need is as follows:

a. For direct redeployment:

(1) 34 neurosurgeons for general hospitals.

(2) 17 neurosurgeons for evacuation hospitals.

(3) 8 neurosurgeons for auxiliary surgical teams.

b. For the Army of Occupation: 2 neurosurgeons for general hospitals.

c. For the U. K. Base: 2 neurosurgeons for general hospitals.

d. Total number of neurosurgeons urgently required—63. All of these men should be capable of heading a service.

5. *Balancing of requirements with available personnel:*

a. There are 38 places required for general hospitals, and there are 40 qualified, or questionably qualified, men available to fill these posts.

b. There are 17 evacuation hospitals that must be staffed immediately, and there are 32 men thought to be qualified for these assignments. This allows a surplus which may be carried over to the indirectly redeployed evacuation hospitals.

c. There are 8 auxiliary surgical teams requiring neurosurgeons. There are 8 men believed to be qualified as neurosurgeons who are available for these posts.

6. *Discussion of the neurosurgical situation.*—Neurosurgeons are properly classified as critical personnel in all theaters of operation, as well as in the ZI. Months or years are going to be required to discharge these responsibilities, and, therefore, no neurosurgeon who is physically qualified will be released from service for many months, or even years. As a consequence, the neurosurgeons cannot anticipate a reasonably early separation from the service. Approximately 35 of the neurosurgeons in this theater have had long periods of overseas service, many of them having been through two or more campaigns, and many of them with high point scores. If the present requirements are met, it will mean that practically every one of them must go to the Pacific Theater direct. This is a particularly grave hardship to the men who have had long periods of overseas service and will result,

in many instances, in a breaking of spirit which will greatly hamper their future usefulness to the service. These men have carried as large a patient load, or larger, during these campaigns as any other group of surgeons or physicians. Many of them are tired, and justifiably so.

7. *Solution:*

a. There are 19 neurosurgical centers in the ZI, with staffs ranging from 5 to 16 officers. Very few of these officers have had any overseas service—in many instances through no fault of their own—and many of them would welcome an opportunity for overseas duty. Some of the men in this theater could take over the ZI work without a hitch and do it better, for that matter, because of their experiences in active campaigns.

b. There should be an immediate transfer of personnel between the ZI and the ETO. This, I believe, could only be done by an arrangement with The Surgeon General's Office by which officers of like grade and ability could be exchanged as casual officers. Such a move would, I believe, be to the best interest of the service, and it would provide better neurosurgical care for the wounded soldier, both at home and abroad.

The personnel injustices which it was thought would be necessary at the beginning of redeployment were somewhat relieved by a request from the Office of the Surgeon General for the immediate return to the Zone of Interior of about 25 fully qualified neurosurgeons. It was expected that these officers, most of whom had had long overseas service, would be assigned to neurosurgical centers in the Zone of Interior, thus releasing personnel without overseas service for assignment to Pacific. After this request had been fulfilled, enough personnel were still available in the European theater with sufficient training and with relatively short periods of overseas service to meet the requirements of units being deployed directly to the Pacific and also to meet the neurosurgical needs of the army of occupation.

All redeployment difficulties were terminated by the ending of hostilities in the Pacific in August 1945.

TRAINING, EDUCATIONAL ENDEAVORS, DISSEMINATION OF INFORMATION

Training Before D-Day

Methods of training.—The special treatment hospitals established in the United Kingdom Base in March 1944 provided facilities for the training of officers, nurses, and enlisted personnel of newly arrived units. As soon as each neurosurgical center was established, a training program was set up in it. Each chief of service prepared his own standing orders of procedure and saw to their distribution among the professional specialized and general staff, nurses, and enlisted men. Such local regulations were extremely important in the smooth conduct of the affairs of a ward; they concerned both neurosurgical professional policies and the paperwork connected with records, disposition boards, and similar matters.

As neurosurgeons came into the European theater, whether as casuals or assigned to general or evacuation hospitals or auxiliary surgical groups, they were placed on temporary duty with hospitals with long experience in the

theater or, more often, with one of the neurosurgical centers for evaluation and further training. Officers assigned to evacuation hospitals and auxiliary surgical teams often had long waiting periods before their units were called into action, and many of them could be utilized in the centers for periods of 3 or 4 months. The plan not only provided valuable additional training for officers thus assigned but also helped materially in carrying the load of neurosurgical casualties in the early days of the European campaign.

Another great advantage of this plan was the opportunity it afforded the senior consultant in neurosurgery for a proper evaluation of newly arrived personnel and for their reassignment if necessary. Most of the officers arriving in the theater in 1944 and 1945 were general surgeons whose only neurosurgical training was the short courses given in the Zone of Interior. They had been assigned to their units on the basis of their qualification cards, without firsthand knowledge of their experience, temperament, or other qualities, and many of them proved to be misassigned. It was possible, by reassignment, to remedy most of these errors before D-day.

Chiefs of neurosurgical sections were encouraged to hold weekly conferences dealing with all important cases and with other matters of clinical and administrative interest. These conferences, to which staffs of other services were invited, proved of great value in the general training program.

Since the end results of combat wounds largely depend upon the type and quality of the initial wound surgery by which they are managed, special attention was paid to the instruction of medical officers in divisions staging in the theater just before they went into combat. This instruction increased in practical value as experience in combat increased. It supplemented the training in fixed hospitals just described and was an essential part of the training of young medical officers with no combat experience and limited neurosurgical training.

One of the difficulties of the training program was the frequent practice of rotating nurses through various hospital services.[10] The principle may be sound, but the practice had to be discontinued in neurosurgical centers. There are far too many technical details in the specialty of neurosurgery to make ward or operating-room nurses of great value before training, which could not, of course, be undertaken when casualty loads were heavy.

Medical field service training school.—A medical field service training school was established at Shrivenham, Berkshire, England, by the Chief Surgeon, ETOUSA, in 1943. It was particularly designed for general duty medical officers assigned to combat units. Officers of all grades were, however, given the opportunity to attend it as circumstances permitted. Instructors were drawn, for the most part, from the Professional Services Division, Office of the Chief Surgeon, Headquarters, ETOUSA, and each class was instructed in the fundamentals of first aid care of various types of casualties. Neurosurgical instruction consisted of lectures, lasting from an hour to an hour and a half each,

[10] Although rotation of nurses was the practice in some hospitals in the European theater, it was not theater policy.—J. B. C., Jr.

supplemented by demonstrations on the emergency care of injuries of the head, spine, and peripheral nerves.

Utilization of British neurosurgical clinics and organizations.—During the long period from the arrival of the first neurosurgical personnel in the United Kingdom in 1942 to D-day, British and Canadian neurosurgical clinics provided the only facilities available for active training of United States Army neurosurgical personnel. The cooperation of these Allied neurosurgeons with their American colleagues during this interim was superb. The facilities placed at the disposal of American neurosurgeons included the clinics of Brigadier Hugh Cairns, RAMC, and Air Vice Marshall C. P. Simon, RAMC, at St. Hugh's Hospital for Head Injuries, Oxford; the clinics of Mr. John O'Connell at Hill End Hospital, London, of Mr. Norman Dott, Edinburgh, and of Mr. Geoffrey Jefferson, Manchester; the Canadian Neurological Hospital No. 1, at Basingstoke, under Lt. Col. Harry Botterell, RCAMC, and his colleagues; and Mr. Herbert Sedden's unique hospital for peripheral nerve injuries at Oxford. So thoroughly were these fine clinics utilized that scarcely a single American neurosurgeon failed to visit or study in one or more of them during his tour of duty in the European theater. The opportunities thus afforded not only provided valuable educational opportunities but also were of great assistance in keeping up the morale of the neurosurgeons during the long period of waiting.

The consultants in neurology, neurosurgery, and allied specialties, in both the British Army and the British Emergency Medical Service, together with a large number of other neurosurgeons in military and civilian services, extended every courtesy to the senior consultants in neurosurgery. After September 1942, the United States Army consultants were guests at every meeting of neurologic sections of the British Army consultants in this specialty. They were also guests of the subcommittees on craniocerebral injuries, spinal injuries, and peripheral nerve injuries of the Medical Research Council. In turn, many British and Canadian neurosurgeons visited United States Army hospital installations in the United Kingdom Base and on the Continent and were also guests at neurosurgical conferences in both areas.

The meetings of the British Society of Neurological Surgeons, which were held regularly throughout the war, were always open to United States Army neurosurgeons. The stimulus afforded by these meetings played an important part in the educational program of the American group, and the close comradeship which resulted from the association had important implications for the war period and for the future.

Similarly, the contacts with British neurologists and neurosurgeons through the Section of Neurology of the Royal Society of Medicine contributed greatly to the success of the American neurosurgical program. The meetings were always open to United States neurosurgeons, who were present whenever conditions permitted their attendance and some of whom became Fellows of the Royal Society of Medicine.

Similar liaison on the Continent was developed as rapidly as circumstances permitted. Neurosurgery on French casualties was done, for the most part,

in United States hospitals. Immediately after the liberation of France, contacts were established with French neurosurgeons in civilian hospitals, and neurosurgeons in the French Army visited neurosurgical services in United States Army hospitals in the Paris area and elsewhere. Visits by the senior consultant in neurosurgery to the clinic of Prof. Clovis Vincent, at the Hôpital de la Pitié in Paris, were particularly productive and helpful. With General Hawley's authorization, Professor Vincent's first assistant at this hospital and two other neurosurgeons who were with French outfits on D-day entered Paris with the Chief Surgeon's Office after that city fell.

Critique of methods of training.—The results of the various expedients adopted in the European theater to train professional personnel without previous neurosurgical experience and to supplement the training of those with limited experience were, on the whole, exceptionally good. Should a similar situation ever arise again, most of these techniques might well be employed again.

The greatest defect of the training program was that it did not make the fullest possible use of the experience accumulated by the consultants in neurosurgery. If these consultants had been permitted to return to the Zone of Interior at intervals, to relay their experiences to division medical officers about to be sent overseas, the tasks of these latter officers would have been greatly simplified. This plan might not have been feasible after the invasion of the Continent, but it could profitably have been employed before D-day, when the theater was inactive.

This plan, too, might well have been the first step in a program of close liaison between professional services in the European theater and the Office of the Surgeon General. Undoubtedly, it would have prevented further misdirection of effort in the operational training directed to neurosurgery. Consultants in neurosurgery to the Royal Army Medical Corps were firm in their opinion that the plan described was the best possible way of providing instruction in military neurosurgery for division medical officers, and it is unfortunate that it was never implemented in the United States Army Medical Service.

Conferences

Preliminary plans for the European theater had envisaged regular conferences to be attended by all available neurosurgical officers. Because of the stress of work, however, the first formal conference was not held until 20 December 1944. An informal meeting of all neurosurgeons in the United Kingdom Base had previously been held in conjunction with the fall meeting of the British Neurosurgical Society at Basingstoke.

After the December 1944 meeting, conferences for the base neurosurgical personnel were held at bimonthly intervals. Mimeographed abstracts of the discussions were sent to every neurosurgeon in both base and army areas, and by this means, as well as by the informal discussions possible at the conferences, unification of therapeutic practices in general hospitals was largely achieved.

The meetings provided the opportunity for reports on new clinical methods, sometimes with arrangements for subsequent clinical trials on a large scale. They also made it possible for army neurosurgeons to secure details concerning the condition in which their patients were arriving in the rear echelon.

Two conferences each were held with the neurosurgeons of the First and Third U. S. Armies and one conference each with the neurosurgeons of the Seventh and Ninth U. S. Armies. Although these conferences were held during the active campaign, it was possible for almost every army neurosurgeon, as well as the chiefs of services in evacuation hospitals, to attend them. The exchange of information between forward and rear echelons was important, and the meetings, like the base conferences, provided opportunities for unifying treatment throughout the theater and also played an important part in keeping professional practices at a high level.

It is unfortunate that these conferences were not begun earlier. They were of great value. One of the final recommendations of the senior consultant in neurosurgery was that, in future planning, regular monthly meetings of the neurosurgeons assigned to each army and to general hospitals in the communications zone should be provided for from the start.

Publications

Neurosurgical experiences in the European theater were reported at the conferences just described, in the *Bulletin of the U. S. Army Medical Department* and in the current medical literature. About 25 formal papers were published from the theater. The substance of these reports, with additional data, is incorporated in appropriate sections of the clinical sections of this volume and volume II [11] of the neurosurgical series.

Manual of Therapy

A manual of therapy setting forth principles of treatment to be followed in the European theater was issued 5 May 1944. This manual was in preparation when Colonel Spurling arrived in the theater in March 1944, and one of his first duties was the revision of the sections dealing with neurosurgical casualties (appendix B).

The introduction to this manual, by General Hawley, well sums up the objectives followed in the preparation of the neurosurgical sections:

* * * * * * *

This manual sets forth principles of treatment which have been tested in active operations by both our own forces and those of our Allies. In it are incorporated many of the professional policies of the medical service of ETO. These policies will be followed habitually. Any one of them may, and should, be disregarded in an individual case where there is sound reason for departing from policy. Personal preference for other methods of treatment as a routine is not a "sound reason." Departures from policies will be made because of special circumstances associated with individual cases.

[11] See footnote 6, p. 61.

The objectives sought in the preparation of the neurosurgical sections of this manual were to lay down principles to be followed by medical officers who, without specialized neurosurgical training, would be required to handle neurosurgical casualties on their own responsibility. The booklet was not intended for experienced neurosurgeons; unification of definitive treatment was provided for by other means, including frequent contact between them and the senior consultant in neurosurgery.

At the close of the European campaign, the Professional Services Division undertook the revision of the Manual of Therapy in the light of the experiences gained during the period of hostilities. In neurosurgery, the only really important changes had to do with the transportation and care of paraplegics.

SUPPLY AND EQUIPMENT

In the early days of hostilities in Europe, one of the major difficulties of the senior consultant in neurosurgery had to do with supplies and equipment. The few United States Army hospitals in the United Kingdom in 1942 suffered from an almost complete lack of the specialized instruments and equipment essential in the performance of neurosurgical operations. This was because such supplies as were arriving were being used to outfit hospitals for the North African invasion. Attempts to make up the neurosurgical deficit from local instrument houses were not successful.

As of March 1944, when Colonel Spurling assumed his duties as senior consultant in neurosurgery in the theater, the neurosurgical supply situation was still chaotic. The supplementary list of neurosurgical instruments, which was presumably standard for each general hospital, was not complete in any installation in the theater. The supplementary neurosurgical kits, originally intended for use in evacuation hospitals and by auxiliary surgical teams, were also, without exception, found incomplete. Individual neurosurgical items were scattered through various medical supply depots all over the United Kingdom.

Major Scarff, who had been acting as consultant in neurosurgery before Colonel Spurling's arrival in the theater, had made a beginning in the correction of the supply situation by having collected at a single depot (G30 in London) enough individual neurosurgical items from other depots to complete 50 neurosurgical kits for auxiliary surgical teams. Whenever an individual standard item was lacking, a similar item was purchased from British sources.

It had been recognized that the assembly of 50 neurosurgical kits was little more than a stopgap procedure, and in March 1944 general measures were taken to correct the whole supply situation. The first step, a complete inventory of all neurosurgical equipment available anywhere in the European theater, revealed that almost enough standard items were available in scattered locations to complete 100 neurosurgical kits. The Supply Division of General Hawley's office agreed to assemble all these items in a single depot (G30) and further agreed that, in the future, all supplies for neurosurgical purposes should

be concentrated in the same depot. This agreement meant that future requisitions for neurosurgical supplies could be automatically forwarded to G30 to be filled.

As soon as the supplies scattered over the United Kingdom had been concentrated in a single depot, it was possible to complete 100 neurosurgical kits, all of which had previously lacked one or more standard items. Incomplete kits which had been issued to general and evacuation hospitals and to the neurosurgical teams of auxiliary surgical groups were recalled and exchanged for complete kits. After all these installations had been provided for, there was still a sufficient reserve of completed kits to meet immediate future demands. Items in excess supply were held for replacement purposes. Although the standard neurosurgical kits (fig. 3) were not ideally suited, in a number of respects, to the needs of military neurosurgery, their distribution as described provided the various medical organizations with enough equipment and instruments to permit the performance of almost any neurosurgical operation likely to be necessary.

Evaluation of Equipment and Supplies

Col. (later Maj. Gen.) S. B. Hays, MC, after his assignment as Chief, Supply Division, Office of the Chief Surgeon, Headquarters, ETOUSA, did an excellent job of procurement and distribution of neurosurgical supplies and equipment. After the initial difficulties just described had been overcome, the supply situation was gradually clarified, and equipment and instruments began to be received in sufficient amounts to be adequate for all purposes.

It was sometimes found that deficiencies in special hospitals were the result of poor organization in the hospital rather than of any fundamental deficiency in the central supply system. In one general hospital, for instance, surveyed in the spring of 1944, there was no table upon which the cerebral headrest would fit, and no Pantopaque (iophendylate) for myelography. The neurosurgical instrument list was also very incomplete. No effort, as far as could be determined, had been made to overcome any of these deficiencies.

In general, both equipment and instruments were of excellent quality and in ample supply. The following general criticisms, however, were almost universal among neurosurgeons and therefore cannot be attributed to individual practices and desires:

1. Fine-pointed mosquito forceps, which are of great importance in traumatic neurosurgery, as civilian experience had long since proved, were missing from the kits. Had this type of hemostat been supplied, in amounts up to 6 dozen for each kit, neurosurgical work would have been greatly facilitated, particularly in general hospitals in which large amounts of peripheral nerve surgery were done, and the quality of surgical technique would have been greatly improved.

2. Scalp clips for controlling bleeding from scalp wounds and scalp incisions, which had also been proved to be of great importance in civilian

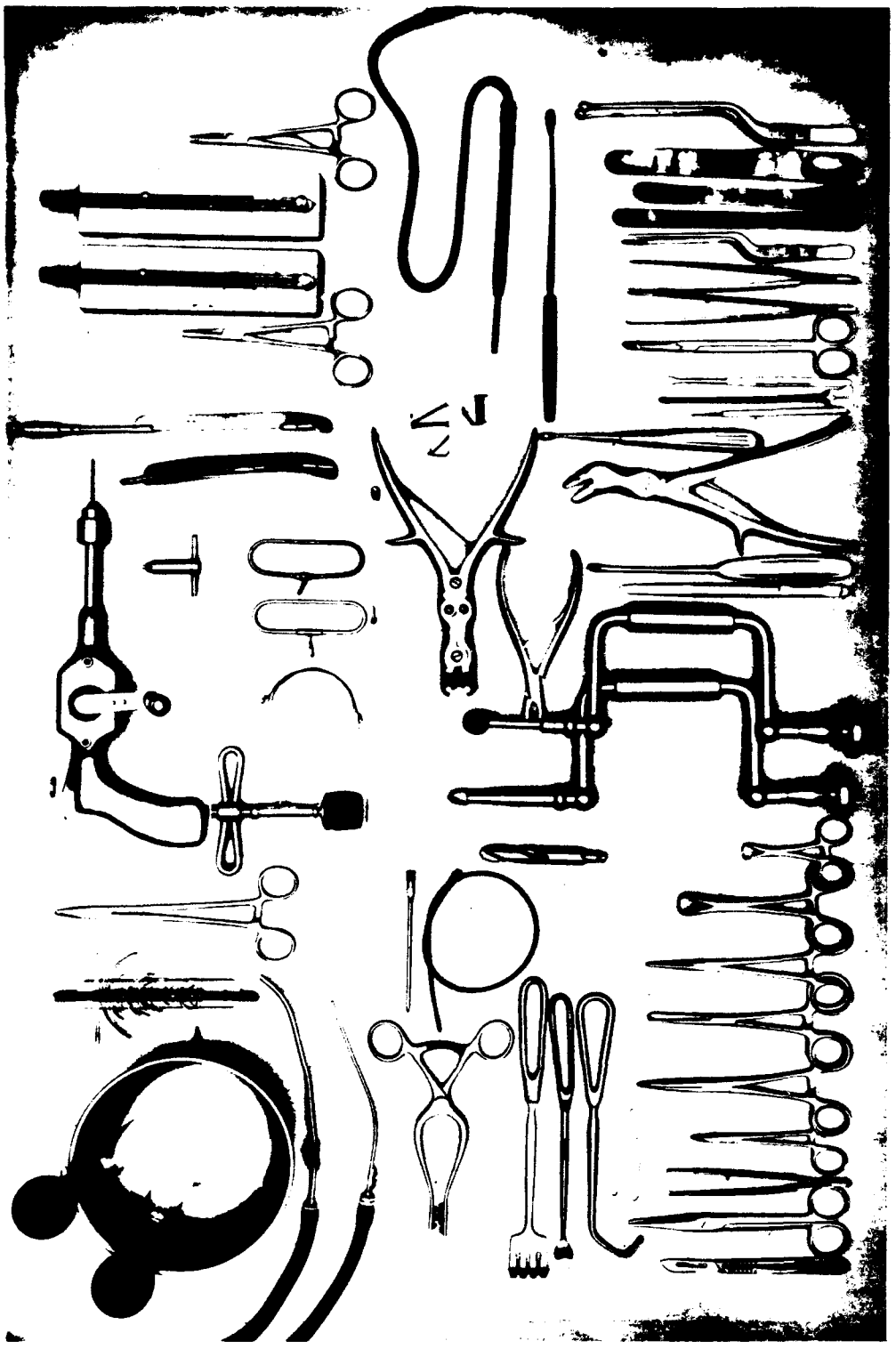


FIGURE 3.—Typically arranged sterile supply table used in neurosurgical operations. All instruments specially designed for neurosurgery were assembled in an auxiliary neurosurgical kit and made available to all Army hospitals where neurosurgical procedures were performed.

neurosurgery, were missing from the neurosurgical kits. A clip of this type (preferably the Raney clip) should have been supplied in each kit in amounts up to 6 dozen, with appropriate clip-carrying racks and with forceps for their application. The control of hemorrhage from the scalp without the aid of these clips is a difficult and time-consuming process.

3. Too much emphasis had been placed upon instruments for turning bone flaps. Experience in civilian practice had shown that the use of bone flaps in traumatic neurosurgery is most infrequent, and it could reasonably have been presumed that, as actually happened, it would be similarly infrequent in military neurosurgery. As a consequence of the inclusion of these instruments, the neurosurgical kit was unnecessarily cumbersome, and the critically short material used to manufacture the instruments could profitably have been diverted to more useful purposes.

4. Before D-day, tantalum plate for the repair of skull defects and tantalum wire and tantalum foil for use in peripheral nerve surgery were almost impossible to secure in the European theater. British neurosurgeons had tantalum plate available for their needs long before United States Army neurosurgeons could secure it. Experiences accumulated in the Zone of Interior had proved that tantalum in various forms was essential neurosurgical equipment, but initial attempts to procure supplies of it in the European theater were unsuccessful and accompanied by many frustrations. Although ample supplies were available in the theater after November 1944, tantalum was still being carried as a nonstandard item at the end of the war in Europe, a policy which was not justified in view of its established value in neurosurgery.

5. Fibrin foam and thrombin also never became standard items. Both had proved of the greatest value in the control of hemorrhage and were to prove of particular importance in frontline neurosurgery, in which it was necessary to deal with the complex and difficult problem of hemorrhage from large venous sinuses. There is, in fact, almost no neurosurgical procedure in which these agents are not of value. The first supplies were secured as the result of a personal appeal to Dr. E. J. Cohn and Dr. Franc D. Ingraham, of Harvard University Medical School. They always had to be secured on special requisitions, though they were both available in limited quantities by this means after November 1944.

Difficulties of supply of particular items were partly due to difficulties of production. Both fibrin foam and thrombin had to be secured from animal or human sources. Gelatin foam, which can be used with either human or bovine thrombin, is now an acceptable and readily procurable substitute for fibrin foam, and oxidized cellulose cotton has excellent hemostatic properties when saturated with bovine thrombin. Neither substitute, however, was available during hostilities to relieve the situation in Europe in World War II.

6. Electrosurgical units of the portable Bovie type were standard equipment in all general, evacuation, and station hospitals but were not part of the equipment of auxiliary surgical teams. This was unfortunate. When auxiliary surgical teams functioned in general or evacuation hospitals, two or more

neurosurgical tables were often in use in the operating room at the same time, and the efficiency of the teams was greatly reduced because of their dependence on the single electrosurgical unit with which the hospital was equipped. From the standpoint of portability, the unit, which is no bulkier than the accessory neurosurgical kit, could readily have been included in the equipment of auxiliary surgical teams.

The Bovie electrosurgical unit proved the best of its kind in any Allied army. It did not break down even under field conditions, and it should be made standard equipment in any installation in which neurosurgery is done. Colonel Spurling pointed out soon after his arrival in the theater in March 1944, at a conference in the Chief Surgeon's Office, that the fact that a neurosurgeon was not assigned to an installation did not mean that it should not be provided with this unit. The reason was that head injuries might have to be handled on an emergency basis by the staff of the hospital, or by an adjacent neurosurgeon who would come to the hospital to operate on the patient, and electrosurgical equipment is the most essential item in surgery for penetrating wounds of the head.

7. The small portable electric cortical stimulator, delivering faradic and sinusoidal current and provided with a variety of electrodes, was made available to neurosurgical centers in the Zone of Interior in 1942 but was never made available in the European theater. The substitute (inductorium) purchased from British sources was of inferior quality and much less satisfactory. A cortical stimulator is essential equipment in any hospital in which peripheral nerve surgery and elective brain surgery are performed, and it should be standard future equipment in all neurosurgical centers.

8. The portable suction pump, which was standard, proved satisfactory in all respects except that it was provided in insufficient quantities in the tables of equipment. With the number of neurosurgical casualties treated, each general hospital could readily have used at least six.

9. In neurosurgical centers, there was need for a suction pump of greater capacity than the portable suction pump provided in tables of equipment. Twenty-five stationary pumps, from each of which as many as six leads could be taken, were purchased from British sources and greatly facilitated the handling of the large volume of neurosurgical work.

10. The foot suction pump developed by Lt. Col. Ralph M. Tovell, MC, Senior Consultant in Anesthesia, Office of the Chief Surgeon, Headquarters, ETOUSA, proved a most valuable piece of accessory equipment, particularly when the electric current failed, as it not infrequently did, or when equipment was out of order for one reason or another.

11. The standard portable operating table supplied to evacuation and field hospitals proved unsuitable for neurosurgery. A particular objection was that it could not be adapted to the prone position in which operations on the back of the head must be done. The standard operating table supplied to general hospitals proved too heavy and cumbersome for neurosurgery. The

table used by the Royal Army Medical Corps was excellent for neurosurgical purposes, and early in the war it was possible to purchase it in sufficient quantity from British sources to equip the 400-bed evacuation hospitals then in the theater. Enough tables could not be purchased, however, to equip forward hospitals in the later days of the fighting in Europe.

12. Air mattresses of simple design, without ridges, constructed to fit hospital cots or beds, were not available in the European theater. They are essential in the management of paraplegic patients, whose care is always a serious neurosurgical problem. The nursing and transportation of paraplegic battle casualties involved difficulties far greater than those encountered in similar patients in civilian practice, and air mattresses of the type described would have simplified these difficulties and immeasurably reduced the incidence of pressure sores.

In the absence of an ideal article, the standard operating-table pad was substituted. Its inner-spring construction made it useful for this purpose, and, because the pad exactly fitted the litter, its narrow width facilitated transportation. The excess supply of operating pads in the European theater made it possible for most hospitals handling paraplegic casualties to secure a pad for each patient during the long evacuation from frontline hospitals to general hospitals in the United Kingdom Base. It was unfortunate, however, that air mattresses were not made available in sufficient quantity for all hospitals handling neurosurgical casualties to use them for all paraplegic patients and to have at least a dozen in reserve.

13. Although the use of casts, splints, and braces was kept to a minimum in neurosurgical injuries in the European theater, because the modern therapeutic trend is toward early motion and minimum fixation, they sometimes had to be used. Splints were particularly necessary in peripheral nerve injuries. It would therefore have been advantageous if a small braceshop had been attached to each neurosurgical center. A properly fitted spring-type brace, which is necessary for the correction of footdrop, was never available in the European theater.

Physical Therapy Equipment

Physical therapy is an integral part of peripheral nerve repair, and the program in the European theater suffered because tables of equipment proved totally inadequate for the diagnostic and therapeutic load in the neurosurgical centers. At least three times the allotted amount of floor space could profitably have been used. The standard machine which delivers faradic, galvanic, and sinusoidal current proved quite satisfactory for both diagnostic and therapeutic work, but the need was frequently urgent for half a dozen to be used at the same time instead of the one or two which each general hospital possessed. Deficiencies in physical therapy equipment were particularly unfortunate in centers in which both neurosurgical and orthopedic patients were concentrated.

RECORDS AND PHOTOGRAPHS

Records

The maintenance of uniform, adequate clinical records in the European theater was always seriously handicapped by lack of clerical help. The result was that medical officers had to spend an excessive amount of time writing records in longhand and that operative notes, examination reports, and followup records were generally inadequate. In the general hospitals, particularly those in the United Kingdom, the elaborate forms required for disposition boards inevitably slowed up evacuation and produced an unnecessary backlog of patients on the neurosurgical services. Some of these difficulties were corrected during the latter part of the European campaign by elimination of the clinical abstracts originally required.

The importance of complete operative reports, particularly from forward hospital units, was fully realized but never fully implemented, and the inadequacy of the notes sometimes led to unnecessary neurosurgical explorations in the Zone of Interior (p. 31). Once the policy of rotation of neurosurgical personnel between frontline hospitals and general hospitals in the rear was put into effect, however, frontline records showed an almost overnight improvement. When neurosurgeons from evacuation hospitals saw for themselves in general hospitals exactly how important it was to record operative findings accurately, as well as the difficulties and complications which might arise when the records were not exact, they bent their efforts toward correcting the situation, with strikingly good results.

The movement of requests for followup data from one echelon to another was always slow and inadequate. This was unfortunate. Better organized relations between evacuation and general hospitals overseas, and between overseas hospitals and hospitals in the Zone of Interior, would undoubtedly have improved the quality of the work done in each echelon. Neurosurgeons in forward hospitals who wanted to know the outcome in cases they had handled frequently attached followup cards to the patients' emergency medical tags. The cards were often lost en route to the neurosurgical centers. Even more often, overworked neurosurgeons at the centers failed to fill them out and return them.

When planning was in progress for the invasion of the Continent, it was realized that, because of the expected pressure of work, records were likely to suffer. It was appreciated that when the patient load became heavy, records would necessarily receive less attention. Routine Army reports did not contain the details of neurosurgical data necessary for satisfactory analysis of neurosurgical material, and while it was agreed that Army medical officers should not be burdened with additional, superfluous paperwork, it was also thought that as many of these details should be preserved as the burden of work would permit.

In an endeavor to suit the method to the circumstances, a simplified

questionnaire was devised, which each neurosurgical chief of section was to prepare monthly and to return to the senior consultant in neurosurgery. The essential data requested concerned penetrating wounds of the brain, spinal cord injuries, and peripheral nerve injuries. Data were kept as a day-to-day record and could readily be assembled at the end of the month. The reports were tabulated bimonthly by the senior consultant in neurosurgery and were distributed in mimeographed form to all neurosurgeons in the theater.

At the close of the European campaign, a final statistical analysis of neurosurgical data from all United Kingdom Base neurosurgical centers was thus available (p. 82). The summary of the work done in these installations from D-day to V–E Day may not have been entirely accurate, but it would not have been possible at all if the plan outlined had not been carried through.

There were numerous reasons why statistical analysis of neurosurgical casualties similar to the one just described was not accomplished in hospitals in army areas. The chief reason was that monthly reports limited to neurosurgical data were not deemed practical by the army surgeons and their surgical consultants, and their point of view is quite understandable, in view of the very heavy load carried by neurosurgeons during the European campaign. On the other hand, such details, were they available, would be of immeasurable value in planning for future neurosurgical casualties, and it is unfortunate that they were not secured, as they could have been without undue hardship, had closer liaison been possible between the senior consultant in neurosurgery and individual neurosurgeons. Most of the neurosurgeons in the army areas kept personal records, and, had they been provided with a uniform worksheet, uniform data would have been available at the close of the campaign with little more effort on their part—perhaps even less effort—than was expended in keeping their individual forms. Because such a worksheet was not provided, a final, accurate compilation of neurosurgical data proved impossible. The records which had been kept individually were so diversified and were collected from such varying points of view that those which were examined proved almost useless for this purpose.

Pictorial Records

From the standpoint of the neurosurgeon, far too little emphasis was placed upon photography as a means of recording important professional work. Large numbers of pictures were collected in the neurosurgical centers, but they were collected by the initiative of individual medical officers. The Army did not furnish cameras, photographic supplies, or personnel. The only team of photographers and artists from the Army Pictorial Service which functioned in the European theater was attached to the 1st Medical Laboratory, and its services, because of a series of errors, the responsibility for which must be shared by all concerned, were never fully utilized.

No matter how efficiently this team might have functioned under happier circumstances, it could not possibly have supplied adequate coverage for all

the neurosurgical centers in the United Kingdom Base Section. Furthermore, its unwieldy organization was not conducive to efficient work in more than two general hospitals at one time.

From the neurosurgical standpoint, a better solution of the photographic problem in the future would be to provide equipment and supplies, and perhaps a technician, for each general hospital of 1,000 beds or more in which specialized neurosurgical work is done, and to attach a photographic consultant to the staff of the Chief Surgeon to supervise the work of the individual photographic laboratories. For lack of this or some similar system, an enormous amount of important neurosurgical material was completely lost during the European campaign in World War II.

RESEARCH

No formal research programs in neurosurgery were undertaken in the European theater, chiefly because the burden of clinical work was so heavy during the period of active combat that neurosurgeons had little [illegible] anything but the treatment of casualties. Many worthwhile [illegible]udies were, however, undertaken and reported. Attention is called [illegible]em at appropriate points in this volume and in volume II [12] of the neurosurgical history.

STATISTICAL DATA

In the preliminary planning for the invasion of the Continent, it had been estimated by Colonel Spurling that about 10 percent of the seriously wounded casualties reaching evacuation hospitals would be neurosurgical. The estimate was based on the United States experience in North Africa, the British experience at Dieppe, and his own experience on the neurosurgical service at Walter Reed (p. 5). Head injuries were expected to account for 2 to 3 percent; spinal injuries including fractured vertebrae, penetrating wounds of the spine, and acute disk lesions, would account for 0.5 to 1 percent; and peripheral nerve injuries, including extremity wounds in which one or more major nerve trunks would be involved, would account for 7 to 8 percent.

These predictions proved quite accurate. In a survey a month after D–day, Colonel Spurling found that about 10 percent of all casualties reaching general hospitals were neurosurgical. Head injuries represented about 4 percent, injuries of the spinal cord about 1.5 percent, and peripheral nerve injuries 5 to 6 percent.

Statistical data on peripheral nerve injuries and penetrating wounds of the brain collected from neurosurgical centers in the United Kingdom, as well as the limited data collected in the army areas, appear in this volume and volume II of the neurosurgical series under appropriate headings. In order to illustrate the amount of material and the types of cases handled by neurosurgical teams in various echelons, three individual (greatly condensed)

[12] See footnote 6, p. 61.

reports to the senior consultant in neurosurgery are appended, each covering the period from the inception of the service through V–E Day or later.

Report No. I.—Neurosurgical Team No. 2, 3d Auxiliary Surgical Group, worked in First U. S. Army installations between 13 July 1944 and 13 May 1945, in 9 evacuation hospitals (95th, 34th, 45th, 67th, 96th, 97th, 102d, 103d, and 128th) and in 4 field hospitals (13th, 45th, 47th, and 51st). During this period, 283 major neurosurgical procedures were performed (260 craniotomies, 23 laminectomies), and approximately 200 additional operative procedures of a minor nature, such as suture of lacerations of the scalp and debridement of wounds, were also carried out. No statistical record was kept of the other cases treated, which included large numbers of closed and compound head injuries, closed and compound spinal injuries, and peripheral nerve injuries. These injuries were seen in consultation in various hospitals in the First U. S. Army area but did not require surgery.

The case fatality rate in the 283 major neurosurgical operations was 9.5 percent (27 cases). Of the 27 deaths, 26 occurred in penetrating wounds of the brain; the remaining fatality followed laminectomy.

There were 24 deaths in the 161 penetrating wounds of the brain treated in evacuation hospitals and 2 in the 31 similar wounds treated in field hospitals. There were no deaths in the 58 compound depressed skull fractures treated in evacuation hospitals or in the 10 treated in field hospitals.

The single death which followed laminectomy occurred in 1 of the 6 cases treated in field hospitals.

The surgical case fatality rate in the 147 United States soldiers with penetrating wounds of the brain was 8.8 percent, compared with a rate of 30.0 percent in 45 German prisoners of war with the same injuries.

Compound head injuries.—The following observations were made in 192 penetrating wounds of the brain treated by surgery:

Metallic foreign bodies were retained in 59 wounds and bone fragments in 102 wounds; the latter were of the gutter type. There were 31 through-and-through injuries.

In 46 intracranial injuries, one frontal sinus, or both, were involved 26 times, the maxillary sinus 12 times, and one or both ethmoids 17 times. The mastoid process was injured 9 times. One eye was destroyed in 20 cases and both eyes in 5.

In 25 injuries complicated by involvement of the dural sinuses, the longitudinal sinus was involved 16 times, the transverse sinus 11 times, and the straight sinus once.

In 60 cerebral injuries, associated injuries (exclusive of facial wounds) included 7 penetrating wounds of the chest, 7 penetrating wounds of the abdomen, 2 thoracoabdominal wounds, and 24 wounds of the extremities without fractures.

The following additional data were collected from the 192 penetrating wounds of the brain treated surgically: There were 11 extensive subdural and 3 extensive extradural hematomas. There were 39 transventricular wounds, 14 of which were fatal. The dura was left open in 6 cases and closed in 67. In the other 119 cases, free pericranial grafts were used in 102, temporal fascia grafts in 14, fascia lata grafts in 2, and a free galeal graft in 1.

Analysis of the 26 fatalities following craniotomy revealed the following data: Twenty-one patients were in deep coma on admission and did not recover from it any time before death. Eight patients exhibited Cheyne-Stokes breathing when operation was undertaken. Five had normal, regular respirations at this time, but 6 had audible evidence of pulmonary edema and 7 had irregular, stertorous breathing. Seven had bilateral fixed pupils at operation. It was the experience of Neurosurgical Team No. 2 that no patient who had bilateral fixed pupils when he was first seen ever recovered, no matter what other signs he might or might not present.

In 15 cases, the brain lesions involved both hemispheres, and in 11 they were unilateral. Fourteen lesions were transventricular.

Three patients presented extensive thrombosis of the longitudinal sinus. Two patients were found at necropsy to have postoperative hematomas of the surgical wounds, following

frontal and occipital lobectomy, respectively. It is possible that these clots contributed to the fatal outcome.

One patient died on the operating table. He was moribund when first seen and presented uncontrolled bleeding from multiple lacerations of the dural sinuses and cortical vessels. Of the remaining patients, 10 died within 24 hours after operation, 8 between 24 and 72 hours afterward, and 7 between 4 and 8 days afterward.

The causes of death were as follows: One patient (as already noted) died of hemorrhage on the operating table. Seventeen patients died with the syndrome of progressive respiratory irregularity and failure, hyperthermia, and terminal pulmonary edema. Two patients, both with extensive thoracoabdominal wounds, died of massive pulmonary congestion 6 days after operation. Two patients, both German prisoners of war, one 50 and one 60 years old, died on the second and the fourth postoperative day, respectively, of circulatory failure, pulmonary edema, and anuria. One patient died on the fifth postoperative day of overwhelming meningitis, 12 hours after the first clinical evidence of the disease, and in spite of daily intrathecal and intramuscular injections of penicillin. The local wound was clean at necropsy. The etiology and type of the meningeal infection were not determined. One patient died on the eighth postoperative day, 36 hours after a transfusion followed by circulatory and respiratory collapse, hyperpyrexia, and anuria. One German prisoner of war, 60 years old, died 8 hours after operation. Only 75 cc. of whole blood had been given on the operating table when the transfusion had to be stopped because of a severe chill and circulatory collapse. One German prisoner of war died on the third postoperative day, immediately after the intravenous administration of anti-gas-gangrene serum for gas gangrene of both legs.

Compound injuries of the spine.—The indications for the 23 laminectomies performed by Neurosurgical Team No. 2 were as follows: Partial, (clinically) favorable lesions in 5 cases; gross spinal fluid fistulas in 5 cases; associated injuries which made transportation impossible in 17 cases; and nontransportability due to the patient's poor general condition and unsatisfactory respirations in 1 case.

The neurologic picture in the first 48 hours after operation showed no improvement in 9 of these 23 patients, slight improvement in 8, and marked improvement in 4. In 2 instances, there was no record of the neurologic status.

The single death in the series occurred in a patient with a through-and-through wound of the abdomen involving the transverse colon, the liver, and the cauda equina. Laminectomy was performed 3 days after laparotomy; the dura could not be closed. Death occurred on the 10th postoperative day from meningitis of *Bacillus coli* origin.—Capt. DONALD D. MATSON, MC.

Report No. II.—Neurosurgical Team No. 4, 1st Auxiliary Surgical Group, functioned in five United States Army hospitals between September 1943 and V–E Day. During this time, this team worked in 2 general hospitals, 2 evacuation hospitals, and 1 station hospital. It performed a total of 269 neurosurgical procedures with 9 deaths, 3.35 percent. Periods of inactivity were caused by staging for the channel crossing (6 weeks), lack of supplies of tantalum (14 days), and movements with an evacuation hospital (several days).

The policy of the team was to make no selection of cases but to operate in the order of seriousness on all patients who, it was believed, would not succumb to the trauma and stress of surgery. In some instances, delays of as long as 48 hours after admission to the hospital were deliberately practiced, to permit the use of supportive and resuscitative measures. The shortest elapsed time between injury and wound closure was 6 hours and the longest 7 days. The majority of the patients were observed for periods of 10 to 14 days after operation except when evacuation at the end of 5 to 7 days was necessary because of the tactical situation. The small number of observed infections (7) was attributed to the good results achieved by the concentration of all neurosurgical procedures in a separate operating room, removed from other cases.

A breakdown of the 269 operations performed by this team is presented in table 4.

TABLE 4.—*Analysis of 269 neurosurgical procedures performed in general, evacuation, and station hospitals by Neurosurgical Team No. 4, 1st Auxiliary Surgical Group*

Injury or operation	Cases	Deaths	Case fatility ratio
Head wounds:	*Number*	*Number*	*Percent*
Penetrating wounds	67	4	5. 9
Perforating wounds	5		
Depressed fractures with intact dura	22		
Scalp lacerations:			
Severe lacerations	26	1	3. 8
Minor lacerations [1]	30		
Skull defects [2]	9		
Brain abscess	1		
Laminectomies	25	2	8. 0
Subdural explorations	13	2	15. 4
Herniated nucleus pulposus	1		
Peripheral nerve injuries:			
Neurorrhaphy	41		
Neurolysis	29		

[1] Secondary closure.
[2] Tantalum plating.

Head injuries.—One of the 4 deaths in the 67 penetrating wounds of the head occurred after a relatively mild left frontal wound, which had penetrated the brain for only 2.5 cm. The patient also had a fracture of the left radius and three compound fractures of the pelvis. Although he was received in profound shock, he responded well to therapy except that voiding could not be induced. He was operated on for the head injury 24 hours after admission to the hospital, under local anesthesia. Death occurred on the fifth postoperative day. During this interim, he passed no urine. The nonprotein nitrogen of the blood was greatly increased, but ureteral catheterization failed to disclose any pathologic changes. Post mortem examination revealed a clean head wound, in which healing was progressing satisfactorily. The kidneys were rather pale, but no blood or crystals were found in the tubules.

The second patient in this group had two penetrating wounds of the brain, in the right frontal and right parietal regions, respectively. In each area, multiple foreign bodies had traversed both lateral ventricles and lay beneath the skull on the left side. The patient was deeply unconscious, and his condition was so poor that the wounds were attacked separately. He died 48 hours after the second operation. Post mortem examination revealed great destruction of both frontal and parietal lobes and almost complete necrosis of the corpus callosum.

The third patient had an extensive left parieto-occipital wound. He was operated on 20 hours after wounding and died 4 days later. His depth of unconsciousness was always profound. At autopsy, a fracture was found across the base of the middle fossa. The left petrous pyramid was fractured anteriorly and posteriorly, so that, except for its dural, vascular, and neural attachments, it lay free.

The fourth patient had an enormous left frontoparietotemporal wound, with destruction of most of the left frontal lobe and part of the temporal lobe. At operation, the brain was extremely dirty and foul smelling, and organisms were easily smeared from necrotic tissue. After debridement, the wound was left open, and the brain was treated as a fungus cerebri. The patient died on the 12th postoperative day, but details are not known, since he was never transportable and had to be left in a holding unit when the hospital was moved.

The single fatality in the 56 scalp lacerations occurred on the operating table. The patient had been wild and belligerent since admission. The roentgenograms, which for this reason were not entirely satisfactory, showed a possible small defect beneath the laceration. The patient did not take the anesthetic (ether, by the endotracheal route) satisfactorily, but the wound was quickly explored and closed. No fracture was found. As he was recovering from anesthesia, he had a generalized convulsion and died almost at once. Post mortem examination showed lacerations of the tips of the frontal and temporal lobes on both sides.

Both patients who died after closed head injuries were received after prolonged periods of unconsciousness. Neither responded to exploratory burr holes, oxygen, nasal feedings, and other routine measures.

Spinal injuries.—The two patients who died after laminectomy both had injuries of the cervical spine. One of them was received a week after wounding. He had had no previous surgery. Meningitis was present, with profuse leakage of cerebrospinal fluid. The wound was debrided and the dura closed after a great deal of dirt had been removed from the spinal canal. His course was progressively downhill after operation, and he died in 36 hours. The second patient withstood laminectomy and had a satisfactory convalescence until the fifth day, when a massive atelectasis developed. Response to bronchoscopy was good. On the eighth day, when the patient was ready to leave the hospital, he died suddenly and without warning. Autopsy revealed a massive right-sided pulmonary infarction.—Capt. R. M. PEARDON DONAGHY, MC.

Report No. III.—The report of the neurosurgical section of the 15th Hospital Center in the European theater covers the period from 1 June 1944 to 1 July 1945 for all cases except the combined nerve-orthopedic injuries, which were observed between 1 March and 1 July 1945.

Penetrating wounds of the head.—There were 777 such injuries, of which 25 terminated fatally. Surgical procedures, which were performed on an average of 5.2 days after injury, included 29 primary debridements; 198 secondary operations (debridements, cranioplasties, operations for osteomyelitis, wound closures); 41 craniotomies for repair of dural fistulas, removal of foreign bodies, and excision of brain abscesses; and 67 miscellaneous procedures, including burr holes, drainage of abscesses, and plastic procedures on the scalp.

In 270 instances, dural repair was carried out at the primary operation. After complete debridement, 122 patients still had retained bone fragments.

Infections included 65 superficial scalp infections, 63 subgaleal infections, 72 brain abscesses, and 43 instances of meningitis.

Spinal cord injuries.—There were 334 injuries of the spinal cord, 47 at the cervical, 164 at the thoracic, and 123 at the lumbar level. One hundred and sixty-five patients presented complete physiologic loss and 169 incomplete physiologic loss. There were 12 fatalities.

Of 141 patients operated on, 23 showed prompt improvement after surgery. There were 19 wound infections and 9 instances of meningitis.

Peripheral nerve injuries.—Exclusive of 154 combined nerve and orthopedic injuries, 1,251 peripheral nerve injuries were observed at the 15th Hospital Center, of which 256 were treated by neurolysis and 165 by nerve suture, on an average of 61.5 days after injury. There were no fatalities. Primary wound healing occurred in 414 cases. Tantalum wire was used in 164 cases and foil cuffs in 397. Silk was used in only one case.

In 123 wounds of the extremities with peripheral nerve paralyses, secondary wound suture was performed on an average of 24 days after injury. There were no fatalities. Primary wound healing occurred in 92 cases, and 4 wounds broke down completely; superficial infections occurred in the remaining 27 cases. Preparation for surgery included chemotherapy in 92 cases and local treatment of the wound in 96.

Miscellaneous cases.—There were 467 cases not covered by any of the categories described. In 251 cases in this group, encephalography, repair of ruptured intervertebral disks, and various minor operations were carried out.—Lt. Col. STUART N. ROWE, MC.

Part II

THE MANAGEMENT OF HEAD INJURIES

CHAPTER IV

Historical Note

Barnes Woodhall, M. D.

By the end of World War I, as a result of the experiences of surgeons in the United States Army and in Allied armies, a considerable body of knowledge had been accumulated in the field of military neurosurgery. The comment is frequently made that the written record in this area of experience, and in other areas as well, was buried in the official medical history of the war, which did not attain wide circulation. The criticism is not fair. The official medical history was available to all surgeons who wished to study it, as were a number of texts on neurosurgery and numerous contributions on military neurosurgery in the periodical literature.

Between the two World Wars, some neurosurgeons in teaching institutions demonstrated their interest in military neurosurgery by means of clinics and occasional clinical notes and thus transmitted their knowledge, experience, and definitive conclusions to the medical students whom they taught. Years of peace, however, are not conducive to the maintenance of interest in gunshot wounds of the brain, the peripheral neuraxis, and similar subjects, and it is an indisputable fact that the new generation of military neurosurgeons entered the neurosurgical centers which were established in the Zone of Interior in 1942 poorly prepared for the management of battle-incurred neurosurgical injuries. Much of the old had to be relearned, and new developments occurred with almost startling rapidity.

During World War I, as the record shows, there were no striking advances in many of the previously accepted methods of neurosurgical treatment. This was not true of injuries of the head. Between June 1918, when the late Col. Harvey Cushing, MC, was appointed neurosurgical consultant in the Surgeon General's Office, and the signing of the armistice that ended the war in November of the same year, the modern neurosurgical management of battle-incurred injuries of the brain came into existence.

Early in World War I, the results of these injuries were poor. The neurosurgical section of the official history of the Medical Department contains the statement: "The results of the early operations for penetrating wounds of the skull, so far as figures rendered them available, had been lamentable, the estimated operative mortality from reports in literature varying from 50 to 65 percent * * *." [1] Later in the war, although the Deuxnouds Hospital, which was supporting the First U. S. Army, did not receive casualties until an average

[1] The Medical Department of the United States Army in the World War. Washington: Government Printing Office, 1927, vol. XI, pt. I, p. 749.

of 36 hours after injury, the surgical case fatality rate in 106 craniotomies was only 33 percent and the total case fatality rate in 175 craniocerebral injuries was only 20 percent.[2]

This improvement in results was brought about through (1) the placement of neurologic surgeons and neurologic teams in hospital units near the front, (2) professional instruction, and (3) the dissemination of detailed directions for the care of craniocerebral wounds. More important, however, than any of these considerations were the personal professional activities of Colonel Cushing himself. The principles of debridement of craniocerebral wounds which he established in World War I proved equally valid in World War II. They were enhanced, but they were not altered, by the addition of modern chemotherapy and antibiotic therapy. The experience of World War I was of inestimable value in the management of battle-incurred injuries of the brain in World War II.

With the exception of cranioplasty, the late treatment of head wounds in World War I offered a limited field for neurosurgical procedures or neurosurgical interest. It was generally agreed that autogenous bone, most often secured from adjacent intact skull but sometimes from the tibia, ribs, or ilium, was the cranioplastic material of first choice. In an occasional selected case, a celluloid plate was inserted.

Because of the higher incidence of infection in World War I as compared with World War II, considerable interest was necessarily shown in brain abscess and cerebral fungus. The important part played by infection is illustrated by the extensive experimental studies made at the Army Neurosurgical Laboratory in Baltimore, which were chiefly concerned with these aspects of head injuries.[3] The reconstructive surgery of cerebral scars, the medical prophylaxis of posttraumatic epileptic seizures, and the rehabilitation of hemiplegic and aphasic patients were all problems for the future.

In the late management of head wounds, neurosurgery in World War II advanced considerably beyond the achievements of this specialty in World War I. In the early management of these wounds, neurosurgeons in World War II merely had to build upon the firm foundation laid in World War I by the activities and practices of Colonel Cushing.

[2] Ibid., p. 774–775.
[3] Ibid., p. 848.

CHAPTER V

Head Injuries in the Zone of Interior

Barnes Woodhall, M. D.

When the United States entered World War II, Zone of Interior hospitals, which were to bear the brunt of the sequelae of head injuries, had the benefit of three recent advances to assist them in the achievement of this mission. The first was electroencephalography, which had become of established value as an adjunct measure in the localization and interpretation of convulsive disorders. The second was the new drugs available for the medical therapy of convulsive states. The third was tantalum and the acrylic resins which, although they were new and comparatively untested, seemed to offer great possibilities as cranioplastic materials.

During the first months of United States Army participation in World War II, the neurosurgical emphasis, rather naturally, was upon penetrating head wounds as compared with closed head injuries. It soon became evident, however, that in a mechanized army, closed injuries and the factor of blast (p. 215) could not be casually dismissed as potential causes of cerebral trauma. At this time, when neurologists and psychiatrists were fully occupied with large numbers of psychiatric casualties, all patients with cerebral trauma were, of necessity, treated on neurosurgical sections. Sequelae of head injuries in the fields of both neurology and neurosurgery were widely various, ranging from speech defects, headache, and vertigo through mental disturbances and convulsive disorders to retained foreign bodies including bone chips, skull defects, and cerebral abscesses.

ADMINISTRATIVE CONSIDERATIONS

Correlation of the functions of the neurologist and the neurosurgeon in the management of these patients, who were their joint responsibility, was accomplished at various centers in various ways. In some, it was achieved by constructive personal arrangements between the respective chiefs of sections. In others, the cooperation was less intimate. In still others, a comparable division of labor and interest was never accomplished. These results might have been anticipated; by hospital regulations, electroencephalography, speech control, and the estimation of the neurologic defect were under the general management of the medical service, while the same patient's difficulties as they were related to the causative skull defect were the responsibility of the surgical service. Administrative regulations, therefore, for all practical purposes, made it necessary to consider the patient with a damaged nervous system as the

result of a head injury not as a single person with a multitude of complaints and with objective signs of injury but rather as two persons. Till the end of the war, the medical officers responsible for the solution of these problems always found it difficult and frequently found it impossible to transcend the regulations which kept this fallacious point of view operative.

RECONSTRUCTIVE MANAGEMENT

The majority of patients with head injuries received from overseas had been excellently treated. In comparison to the late evacuation, undue prolongation of immobilization, frozen joints, and other errors which characterized the early experience with peripheral nerve injuries, patients with head injuries were, almost without exception, received in good condition.

Only about a third of all patients with penetrating injuries were, however, entirely free of disabling defects or symptoms, while in others who were presently asymptomatic there was electroencephalographic evidence of epileptogenic foci. No unequivocal statements, therefore, could be made that their future would be secure from sequelae of their wounds. This consideration, plus the fact that a large proportion of men with head injuries who were returned to duty had to be separated from service within periods of months, led to the development of the policy that all casualties with penetrating injuries should be treated adequately and then promptly separated from service. When this policy was fully implemented, patients with cranial injuries were being disposed of so rapidly that many of them were out of service within a month of their return from overseas.

As a result of excellent treatment overseas and the policy of separation from service after adequate treatment in the Zone of Interior, the mission of hospitals in the United States was clear. It chiefly concerned penetrating wounds. Patients with such wounds always had to be surveyed, and many of them had to be treated, from one or more of the following points of view: (1) Residual infection; (2) retained metallic or other foreign bodies, particularly bone fragments, and their influence upon infection and other manifestations of injury; (3) neurologic defects, with emphasis upon pneumoencephalography; (4) skull defects and their repair; and (5) convulsive states, with particular attention to electroencephalography and the resection of cerebral scars.

A well-rounded clinical description of the syndrome of the acutely injured brain was soon available through the contributions of neurosurgeons experienced in, and adept at, the surgery of these injuries. Diagnostic refinements were introduced. A technical bulletin on the technique of electroencephalography and the interpretation of electroencephalographic findings was issued.[1] The indications for, and the technique of, cranioplasty were also described in detail.

Evaluation of all features of the cerebral injury, which was a laborious task, received only limited attention while the war was in progress. This was partly because attention was devoted to other, more immediately urgent, mat-

[1] War Department Technical Bulletin (TB MED) 74, 27 July 1944.

ters but chiefly because this phase of reconstruction is essentially a long-term problem.

Infection

The first responsibility of the neurosurgeon in Zone of Interior hospitals was to complete the task, begun overseas, of eliminating infection. This was usually a minor problem. In the relatively few cases of late residual infection observed in neurosurgical centers in the United States, the influence of inadequate debridement and inadequate postoperative care was clearly manifest. In most of these instances, retained bone fragments, which were always an evidence of incomplete debridement, were the cause of persistent infection, and the practice of radical resection was soon developed for the management of such patients. In an occasional case, the location of the fragments was such that only drainage was employed, for fear that resection might destroy whatever residual vision the patient had.

In occasional instances, a solitary abscess had developed about a retained metallic fragment, just as it might have developed in any other area of the body. In the absence of clear indications for surgery, conservatism was the rule in this type of case, though occasionally a well-encapsulated abscess was removed in toto. Penicillin proved of great value in the conservative management of brain abscesses, particularly in the early stage, in which cerebritis is a prominent feature.

Cranial Defects

A second responsibility of the neurosurgeon in the Zone of Interior was the restoration of the integrity of the skull after wounding. Most patients with penetrating wounds of the brain were left with unsightly scars and other defects, for which some sort of reconstructive surgery was necessary. Although this was often a major task, because the defects were numerous and were frequently technically difficult to repair, a technique was rapidly evolved which proved safe and effective for general use.

Cranioplasty was performed overseas in a limited number of cases, but the results are inconclusive because of the lack of official reports. When the dura and brain were not lacerated, it was a safe procedure when performed early, and it undoubtedly expedited returns to duty, at least theoretically, when manpower was in short supply. Schorstein,[2] who commanded a British mobile neurosurgical unit, reported that 28 of 33 patients who underwent primary skull closure with acrylic plates had been back to duty, mostly in a lower category, for 3 months or more at the time of writing. It was later learned that two patients in this group had sustained delayed infections. The United States Army experience seems to have been less satisfactory. Neurosurgeons in Zone of Interior hospitals observed a number of postoperative infections and did not regard the cosmetic effect as good in all cases.

[2] Schorstein, J.: Primary Skull Closure With Acrylic Plates. Brit. J. Surg., Supplement No. 1, Wounds of the Head, pp. 256-257, 1947.

In their opinion, this procedure was better done in a general hospital some months after primary healing.

Neurosurgeons in the Zone of Interior also found the use of tantalum foil over the injured brain not only useless but dangerous. The foil was invariably found in a crumpled mass, adherent to the tissues. Almost every patient thus treated had either an associated cerebritis or a distinct increase in the local scar, and at least two patients were known to have developed status epilepticus shortly after the foil was employed.

Indications.—The indications for repair of cranial defects remained those which had been standard in prewar neurosurgery; that is, localized pain, headache, vertigo, vertigo upon change of posture, deforming defects, and fear of further injury. The mere presence of a skull defect 3 cm. or more in diameter was usually regarded as a sufficient indication for cranioplasty, especially in view of the military directives which precluded the return to duty of a patient with a skull defect of larger area or with a defect which would prevent the ready use of military headgear.

Tantalum.—In World War I, as already mentioned (p. 90), cranioplasty seems to have been carried out almost entirely with autogenous bone, usually derived from adjacent calvarium or from the tibia and less often from other bony structures such as the ilium, the scapula, or the rib. In complicated and larger defects, the use of celluloid plates was advised.

By the onset of World War II, a number of alloplastic substances had become available for clinical trial, such as Vitallium, zirconium, tantalum, and certain synthetic resins such as methyl methacrylate. The most promising of these, tantalum, was introduced into the neurosurgical centers in the fall of 1942. Certain statements concerning possible adverse reactions after its use appeared from time to time, but objective evidence to substantiate them was never forthcoming in the whole cranioplastic experience with this agent. The thin, translucent connective tissue capsule which appears about a tantalum plate after it is in place is apparently nonprogressive in nature.

Even before the war, experimental and clinical studies had demonstrated that tantalum possesses at least two properties which are essential in any material used in the repair of the skull. The first is that it is so relatively inert in tissues that, for all practical purposes, it can be considered absolutely inert. Experimental studies indicate that it does not react to heat, cold, or electrical fields.

The second desirable feature of tantalum is its malleability. When it was originally introduced, it was applied by crude molding, cutting, and contouring. These original crude methods had obvious disadvantages. One was loss of time at the operating table. Another was the fact that although plates shaped in this manner were adequate for the convexity of the skull surface, they could not be used for multicontoured defects, such as those likely to occur in the supraorbital region. Refined techniques of molding the malleable plate before cranioplasty were rapidly developed. The preparation of plates of complicated shapes gradually evolved by the use of a die and counter die or

by the use of a hard rubber piston, by means of which the plate could be contoured and swaged over the original impression.

Materials required for the formation of tantalum plates, as well as the technicians needed to make them, were available in all dental laboratories, and the skill and experience of Army Dental Corps officers had much to do with the successful treatment of patients in neurosurgical centers, particularly those with large skull defects.

Techniques.—All techniques devised for the insertion and fixation of tantalum plates proved to have features of distinct value. In no field of reconstructive surgery, indeed, was the ingenuity of United States Army neurosurgeons so clearly demonstrated as in the many technical methods devised for this purpose. A sound knowledge of the blood supply of the scalp and of the principles of shifting flaps of scalp, both derived from experience in acute head injuries, proved essential in the exposure of skull defects preparatory to cranioplasty with tantalum.

In the actual insertion of the plate, it was soon found that because of the predominance of linear scars, approach to the defect was usually possible by simple excision of the original scar, with dissection and retraction of a flap of scalp. Cranioplastic incisions about horizontal scars were promptly abandoned, it being observed that they were almost always followed by necrosis of the incisional line, or, occasionally, by actual gangrene of the original scar. Fixation was obtained with wire sutures, with screws, or with tantalum points applied by various techniques.

The experience at Kennedy General Hospital, Memphis, Tenn., may be cited as typical. Up to September 1945, 316 tantalum plates had been inserted in the skull as part of cranioplastic procedures required by combat-incurred defects. Various types of fixation were tested and discarded before fixation with glazier's pegs was adopted. Still later, slots were cut near the edge of the plate for the insertion of the pegs, or, when slots were not practical, notches were cut into the edge. The best cosmetic results were achieved when the plate was placed directly on the outer plate of the skull, without any countersinking.

At Hammond General Hospital, Modesto, Calif., 152 skull defects were repaired in the year ending on 21 August 1945. In only a few instances was it necessary to open the dura for exploratory purposes before adjusting and fixing the plate in position. At first, the plates were held in position by the use of tantalum wires strung through perforations in the outer table of the skull and the tantalum plate; the perforations were made by means of an electric motor and dental drill. This method was discarded when the use of tantalum wedges was found to afford a firmer fit and also to save time at operation. Perforations in the plates, however, continued to be used, partly to permit a free drainage of fluids after operation and partly because the growth of connective tissue through the openings helped to hold the plates firmly in position.

At this hospital, the policy at first was to inlay plates only in the frontal region, the time taken to inlay them elsewhere being thought to be an unneces-

sary requirement. Later, the inlay method was used in most operations because the fit was better with this technique and there was less likelihood of the development of ridges under the scalp.

Postoperative removal of tantalum.—Although there was definite evidence that perforations in the tantalum plate somewhat weakened its strength, there was also considerable evidence that they possessed certain advantages. One of the most important, as just noted, was that they provided for drainage of the fluid which invariably collected beneath the plate. They also permitted complete expansion of the cranial content and the dura to the under surface of the plate. The latter fact became increasingly apparent during the later phases of the neurosurgical experience, when a small number of tantalum plates had to be removed as part of the attack upon epileptic foci. With a tantalum plate in situ, it is not possible to secure complete pneumoencephalographic studies of the cerebral scar because the plate acts as a barrier to roentgen rays. Considerable evidence accumulated, however, that the electroencephalographic recording of such a focus is not obstructed by an intervening tantalum plate, although the exact location of the focus is perhaps made more diffuse by the presence of the intervening element.

Complications.—Some tantalum plates had to be removed for postoperative infections, which could usually be traced to the original acute infection. Scattered instances of pneumatocele were observed, all in cases in which plates had been placed over skull defects affecting the air-containing sinuses. With these exceptions, no complications of any consequence followed the use of tantalum in the correction of skull defects, and not many plates had to be removed.

Results.—On the whole, the results of cranioplasty were good both cosmetically and clinically. Headaches and dizziness, particularly on changes of position, were generally less marked. Neurologists who followed these patients reported improvement in both neurologic and aphasic disturbances, but the period of observation during the war was not long enough to warrant generalizations, and whether there was an actual improvement in cerebral function or merely the psychic improvement associated with correction of the skull defect could not then be determined.

Acrylic resins.—It is undeniable that the emphasis upon, and the good results of, cranioplasty with tantalum influenced adversely the development of other materials for the same purpose, such as the acrylic resins. The situation was recognized early in the war, and studies with these resins were instituted at the neurosurgical centers at Fitzsimons General Hospital, Denver, Colo., and Newton D. Baker General Hospital, Martinsburg, W. Va., where approximately 70 cranioplastic procedures were performed with them. The immediate technical results were good in all cases, and no undue tissue reactions occurred. In retrospect, however, cranioplasty with tantalum met all the objectives of the repair of large numbers of skull defects, and, subject to the few qualifications mentioned, it seems likely that this material will maintain its status as a cranioplastic agent.

Convulsive States

The treatment of the somatic functional sequelae of head injuries, such as aphasic and convulsive states, differs from the treatment of infection and the restoration of skull defects in that the passage of time is the deciding factor. The very nature of the lesions makes the two tasks incompatible. They demand a different armamentarium and different, though equally highly specialized, professional care.

Pneumoencephalography was performed in only a relatively small percentage of penetrating head injuries, chiefly in patients with convulsions or with striking neurologic defects. Electroencephalography was carried out, whenever possible, in all head injuries and was of considerable value in the identification of preclinical convulsive states, the localization of convulsive foci and the differential study of cerebral injury following blast.

The experience with encephalography was particularly extensive at Kennedy General Hospital. Late in 1943, a civilian physician experienced in this field was employed, and a special laboratory was set up. At the peak, 6 technicians and 2 secretaries were necessary to handle the load. Of the 2,617 studies made, 411 were for skull defects or depressed fractures in which surgery, usually plating, was required, and 425 were made on patients who had suffered blast concussions, with no evident trauma of the skull or body.

Early in the history of the neurosurgical centers, it became apparent that some policy must be established regarding the surgical treatment of cerebral scars. Previous experience had shown that, when the brain is penetrated by a missile, an incidence of subsequent convulsions in the neighborhood of 40 or 45 percent might be expected. About half of the attacks could be expected to develop during the first year after injury and the remainder within the next 4 years. The problem was particularly serious in neurosurgical centers during the war. The neurosurgeons assigned to them were working under a great deal of pressure because of the heavy patient load, and not all of them were talented in this special field. Even if they had been, there was little time to dedicate to a specialized group of casualties.

The fundamental issues were twofold: (1) Whether prophylactic excision of cerebral scars should be done, and (2) whether, if the policy of excision was established, it should be carried out in all neurosurgical centers or whether this highly specialized group of patients should be concentrated in one or two centers.

Expressions of opinion on the subject were invited from well-recognized authorities in the field of posttraumatic convulsive states, including Dr. Wilder Penfield of the Neurological Institute in Montreal. As a result of the survey, a policy of conservatism was decided upon. Prophylactic excision of cerebral scars was not advised, and patients who developed convulsive states were treated under a long-term regimen of medical care, including the administration of the new anticonvulsive drugs. The results were not impressive in the few cases in which prophylactic excision of scars was carried out, and in some

instances the neurologic status of the patients was worse after operation than before.

In November 1945, a posttraumatic epilepsy center was established at Cushing General Hospital, Framingham, Mass., under the direction of Maj. A. Earl Walker, MC, for the specific purpose of pursuing studies on this problem under ideal conditions. Approximately 300 patients formed the nucleus of the study. At the present time, only tentative opinions can still be expressed concerning the incidence, diagnosis, and therapy of convulsions following gunshot wounds of the brain. Many more years must pass before definitive statements can be made upon these points. The data presently available concern such matters as infection, the influence of retained bone chips, and results of electroencephalographic studies (p. 279).

CHAPTER VI

The Mediterranean Theater of Operations

Eldridge H. Campbell, Jr., M. D.

A review of 14,000 battle wounds treated in Fifth U. S. Army hospitals in the Mediterranean Theater of Operations, United States Army, in 1944 disclosed that, exclusive of maxillofacial wounds, 6.17 percent involved the head. One-third of the head injuries were classified as intracranial. The remainder involved only the scalp. The case fatality rate for all the head injuries observed in 1944 was 8.1 percent, but during this year 24.4 percent of all deaths which occurred in these installations were attributed to cerebral injuries. Of the patients with penetrating head injuries who survived to reach general hospitals, another 3.7 percent died, chiefly as the result of complications of wound healing.

Scalp injuries, although they were associated with a low case fatality rate, presented numerous difficulties of management. Penetrating head injuries presented problems of the gravest import. Closed injuries of the head, except for the policy of early ambulation (p. 106), were treated according to standard policies.

SCALP WOUNDS

Scalp injuries, as just noted, comprised two-thirds of all head injuries observed in the Mediterranean theater and from the standpoint of numbers therefore represented a very important group. They were also of importance from the practical military standpoint that the majority of soldiers with such wounds, if they were correctly managed, could be promptly returned to duty. Complications were limited to cases in which the defect was large or in which underlying damage was overlooked because of inadequate exploration, usually in the belief that the wound was purely superficial.

Scalp wounds overlying penetrating wounds of the brain presented special problems which required prompt solution. Primary epithelization of such wounds is requisite to sound wound healing.

The type of incision employed in wounds of the scalp, whether or not they overlay deeper wounds, and their closure without tension were factors of great importance in the management of these cases. The limited elasticity of the tissues of the scalp often made it impossible to effect simple direct closure without undue tension. Closure under tension could lead to local dehiscence or even mild wound necrosis, and both could lead, in turn, to infections of real seriousness.

The technique of closure of scalp wounds was directly related to the type of incision used. Whenever practical, the simple vertical or curvilinear type of incision proved most satisfactory. Quite often, however, neither one of these incisions could be employed because extensive areas of tissue had been destroyed by the original incision or because extensive debridement had been required.

One or another of the following techniques was employed to overcome these difficulties:

1. Small defects were included in the center of a horseshoe flap or in one leg of a tripod incision, according to the technique devised by British surgeons in World War I. In an incision of this kind, the increased tension was distributed over a wider area than in simple vertical or curvilinear incisions.

2. The galea was undermined, to permit larger flaps to be mobilized and tissue to be stretched without undue increase in local tension.

3. Lateral relaxation incisions were frequently employed, though they proved useful only if they were generously long, so that tension was distributed over a wide area.

4. When loss of skin was particularly extensive, the rotation of one or more pedicle flaps was useful. In this technique, pericranium was left in situ to nourish the graft.

When relaxing incisions or pedicle grafts were employed, it was found essential to cover the defect at once with a split-thickness graft. The additional procedure did not greatly prolong the operating time and went far to insure primary healing. On a few occasions, failure to take this precaution is known to have resulted in local infections and discharging wounds which seriously compromised the primary incision.

The use of a free graft over the original scalp defect was found to be unwise, partly because of the likelihood of an incomplete take and partly because of the risk of faulty healing if reexploration of the wound should be required.

FRACTURES OF THE SKULL

The treatment of fractures of the skull produced by blunt force followed the policies usual in civilian injuries of the same sort. Fractures caused by missiles, however, presented a much greater variety of types than are ordinarily seen in civilian practice. The most common of these was the rounded perforation with blown-in fragments, in which the particles became, in effect, missiles themselves and usually traveled in directions other than those taken by the metallic fragments. Metallic fragments were likely to be broken into bits by the impact. Dirt, hair, bits of metal, and other foreign material were often driven into the bone edges and could be removed only by the rongeur.

If the missile struck the opposite wall of the calvarium with insufficient force to perforate it, there occasionally resulted a pavement fracture which was expressed instead of being depressed. Tangential or less forceful impacts on the calvarium resulted in the familiar type of depressed fracture in which the

dislocation of the inner table far exceeds that of the outer. The possibility of intracranial ricochet always had to be borne in mind and added materially to the gravity and complexity of the wound.

Fracture lines extending from the skull injury were either continuous or discontinuous. Continuous fracture lines might be as short as 1 to 3 cm., but in more serious injuries they were longer and embraced comminuted fragm overriding of which might be observed near the wound of entry or and of exit. The discontinuous type of fracture occurred at some di rom the perforation in the calvarium and was not connected with it or ny of its radiating tracts. The mechanism of discontinuous fractures wa er clearly elucidated, but they were a clear illustration of the important fact that injury to neither the skull nor the brain was necessarily confined to the track of the missile.

PENETRATING WOUNDS OF THE BRAIN

Selection of Cases for Surgery

Generally speaking, delay in operating on patients with head injuries was not responsible for many fatalities. The time element was naturally important, but it became apparent early in the Tunisian campaign that it was not a serious consideration, if any consideration at all, in most deaths from cerebral wounds.

The great majority of soldiers with penetrating head wounds which entailed irreparable damage to vital parts of the brain died on the field where they had been wounded. Had it been possible to treat all of these casualties at the level of the division clearing station, perhaps a few lives might have been saved. From the tactical standpoint, this was not a feasible plan, and its practical value was also open to question. Neurosurgeons assigned to field hospitals almost invariably found that the great majority of nontransportable patients with wounds of the brain who came under their observation had injuries which were primarily lethal, as proved by necropsy. It was also found that the majority of patients with such injuries who were transportable were not seriously affected by the ambulance ride to the evacuation hospital, even if the distance was long. If, as frequently happened, a patient with a serious head injury was rendered nontransportable by other wounds, the solution of the problem was to send a neurosurgeon forward from the nearest evacuation hospital to care for him.

In most of the primarily lethal head injuries, if death did not take place on the battlefield, it occurred soon after the casualty reached the hospital. Between 15 and 25 percent of patients with penetrating wounds of the brain who lived to reach evacuation hospitals were found to have suffered extensive injuries to the basal ganglia or the midbrain structures. At necropsy, it often appeared remarkable not that they had died but that they had survived for hours or even days and had not died instantly.

Operation was carried out in some of these potentially fatal cases, partly because there was a conscientious desire to give every wounded man any

chance of survival he might have, and partly because it was not always possible to identify hopeless wounds with absolute certainty (p. 335). Many profitless operations were performed because of this policy, and the surgical case fatality rates were correspondingly increased. In periods of great stress, it would have been of practical advantage to be able to identify mortally wounded men, so that surgical efforts could be more profitably directed toward men with an absolute, or a better, chance of recovery. It was found that decerebrate rigidity, fixed pupils, and profound disturbances of circulation and respiration were of the gravest import and usually indicated mortal damage to basal ganglia or midbrain structures, but it was also found that no single one of them was a certain harbinger of death and that operation could not justifiably be withheld on the basis of any one of them in itself.

In the absence of major injuries elsewhere in the body, shock was seldom marked in patients with penetrating head wounds which did not involve injury to the basal ganglia or the brain stem. Severe blood loss from the scalp or from a lacerated dural sinus was much less common than might have been expected. In grave injuries to the brain stem, the usual picture of clinical shock was often altered by discrepancies in vital signs. A high blood pressure, for instance, might accompany a rapid, thready pulse, or an elevation in temperature might accompany a clammy skin. It was a fixed rule that a patient in shock was not treated surgically until he had been resuscitated by the usual measures, on the principle that, if he could not be brought out of shock, he would not be likely to tolerate a major surgical procedure.

Hematoma Formation

One type of head injury proved the exception to the rule that postponement of surgery was not harmful in these cases. The patient with a hematoma did best under minimal transportation and prompt operation. If no neurosurgeon was within easy reach, the necessary surgery was done by a competent general surgeon who had had some special training in neurosurgery.

Clots were of variable size and extent. They were almost universally found in injuries of the cerebrum and cerebellum but were not common in the subdural and extradural spaces. The incidence varied widely, judging from the reports of the neurosurgeons in the theater, but hematomas were present in enough cases to make a search for them mandatory whenever debridement of the missile track failed to relieve intracranial tension. Distal intracerebral or intraventricular hematomas were notoriously responsible for persisting intracranial pressure and, if they were not evacuated, for a fatal outcome.

Wounds of the Ventricle

Wounds of the ventricle were associated with such a high immediate mortality that relatively few casualties in this category survived to reach hospitals. Associated injuries of the basal ganglia and brain stem were pres-

ent in a high proportion of cases, and the clinical picture often included deep coma, rapid, stertorous respiration, rapid pulse, high pulse pressure, and decerebrate rigidity or flaccidity. Diagnosis was not always simple. There was seldom a sufficient amount of cerebrospinal fluid drainage from the wound to indicate the location of the injury, while the course of the missile, once it had entered the body, could not always be traced, even when bone fragments were revealed by roentgenologic examination. If the patient with a suspected wound of the ventricle reached the hospital in condition for surgery, or could be resuscitated, the results were oftentimes unexpectedly good.

Cerebrospinal Fluid Fistula

Laceration of the dura adjacent to a fracture of the frontal or ethmoid sinus or the mastoid process was likely to result in a persistent leak of spinal fluid. In some instances, the fistula healed spontaneously, but if it occupied the site of a missile track, it was usually larger, and spontaneous healing was much less likely. The interposition of bits of sinus membrane, bone, or arachnoid tissue also tended to prevent or delay healing. Infection of the cranial cavity was always a possibility under the circumstances.

Diagnosis was often difficult. Fractures of the frontal sinuses were demonstrable in stereoscopic roentgenograms, but ethmoidal injuries usually were not. Not infrequently, it was impossible to be sure which side of the cribriform plate was perforated.[1] The presence of air in the subdural or subarachnoid spaces was considered almost diagnostic of fistula, as was the presence of air within the cerebrum or ventricular system. The escape of spinal fiuid was not always obvious. Even mentally clear patients did not notice it in a surprising number of cases, and careful observation as well as direct questioning was necessary to detect it.

How soon the dura was closed in ventricular injuries depended upon the patient's condition. If meningitis had developed before the fistula was diagnosed, or if for any reason operation had to be postponed, vigorous antimicrobial therapy was relied on to tide the patient over the intervening period. In meningitis, the patient's serious general condition, as well as the presence of edema of the brain, made operation hazardous. Penicillin was used in large doses intramuscularly and intrathecally, and an adequate blood level of sulfathiazole or sulfadiazine was maintained. As soon as the intracranial

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[1] Roentgenologic examination in head injuries in forward hospitals was not entirely satisfactory. Single anterior, posterior, and lateral exposures were made in all cases, but, without good stereoscopic views, it was often impossible to localize indriven bone fragments accurately or even to demonstrate their presence with certainty. Excellent work was sometimes done under field conditions, but, on the whole, the views made in head injuries were not so helpful as they should have been. Roentgenology in general hospitals in the base was excellent, as might have been expected.

(The difficulty described with roentgenograms taken under field conditions in the Mediterranean theater was not encountered in the European theater, probably because in the latter theater Col. Kenneth D. A. Allen, MC, Consultant in Radiology, personally supervised every army and communications zone medical installation to be certain that useful films were being produced. Many experienced neurosurgeons stated that some of the finest roentgenograms of the head which they had ever used were made in evacuation hospitals in which the only equipment was a portable X-ray unit and a stationary grid.—R. G. S.)

pressure was reduced and the patient's general condition was improved, dural repair was undertaken.

When there was no certainty which side of the cribriform plate was perforated, approach was through a coronal scalp incision, which permitted the cutting of small osteoplastic flaps on either side. Large dural lacerations, particularly those over the cribriform plate, usually required a patch of living fascia for firm closure. When the injury was adjacent to a frontal sinus, it was sometimes possible to operate extradurally. If it was over the ethmoid sinus, repair was much more satisfactorily achieved from within, because the dura in the ethmoid region strips poorly and traumatic enlargement of the opening was therefore a probability. The fascial patch had to be sufficiently large to overlap the margin of the defect by 5 to 10 mm. in all directions. German's method of utilizing dural flaps from the crista galli proved suitable for the repair of small rents in the adjacent dura.

Closure of the Dura

Closure of the dura when the existence of a cerebrospinal fluid fistula did not provide an absolute indication was originally a much disputed point in the management of penetrating wounds of the head. The majority of these injuries seemed to heal without difficulty, whether the dura was left open or was closed. The argument that it should be left open to permit spontaneous drainage if infection occurred did not prove valid; drainage was never satisfactory under the circumstances, and secondary debridement was usually necessary. In a number of instances, on the other hand, it was thought that deeper wounds had been effectively shielded from superficial infection when the dura had been closed by suture or graft, while in numerous wounds of the spinal dura, closure appeared to prevent inward extension of epidural infection. In deeply infected wounds of the brain, another possible advantage of dural closure was the prevention of fungi, or at least a delay in their formation.

When penetrating wounds of the brain involved the paranasal sinuses, every effort was naturally made to close the overlying dura at the initial operation, in order to prevent fistula formation and to minimize the risk of infection. It also became the general policy, as the war continued, to close the dura under other circumstances. When simple suture was not possible, a graft of living tissue (pericranium, temporal fascia, or fascia lata) was used and seemed to impose little additional burden on the wound.

Drainage

From the beginning of activity in the Mediterranean theater, it was the policy to close all head wounds without drainage after debridement, on the principle that a drain was of limited value in the evacuation of blood or serum while its presence was likely to predispose to the development of infection and of spinal fluid fistulas. The few surgeons who originally favored drainage soon ceased to employ it as its bad effects became evident.

REDEBRIDEMENT AND DELAYED DEBRIDEMENT

Superficial infections occurred in approximately 19 percent of the head injuries treated in the Mediterranean theater in 1943 and in approximately 16 percent in 1945. In this category are included infections which extended no deeper than the dura, wound dehiscence, mild degrees of necrosis, and any other result less than healing per primam. For the most part, these infections were strictly localized, and they occurred, as a rule, because closure had been accomplished under too great tension or because the silk used was too heavy. In many instances, removal of the sutures, sometimes supplemented by the application of warm compresses, was all that was necessary to control the infection, though subsequent healing was sometimes delayed. If the wound was large and, particularly, if debridement had not been done adequately, dirty, purulent granulation tissue was likely to develop, and weeks might be required for complete healing. If the dura had been left open in these cases, the consequences were sometimes serious.

Infections of a graver type were the greatest single hazard to recovery in head injuries. Most often, they followed incomplete debridement or inadequate hemostasis or both. The presence of clots and of pulped brain tissue offered an excellent medium for the growth of the bacteria which were always carried into the injury with the missile. If the infectious process was not too extensive and if bacterial contaminants were not too numerous or too virulent, spontaneous healing sometimes occurred. As a rule, however, active therapy of deep infections was necessary, because in the majority of cases they developed about retained bone fragments; this was true of 90 percent of the brain abscesses observed in the Mediterranean theater.

The introduction of redebridement of infected wounds of the brain and of late debridement of infected, neglected wounds represented the most important single advance in the management of head injuries in the Mediterranean theater. The policy was first employed toward the end of the Tunisian campaign, at the 21st General Hospital, by Lt. Col. Henry G. Schwartz, MC, and Capt. George Roulhac, MC, in a series of 130 cases. Redebridement of wounds elsewhere in the body, including those associated with compound fractures, had by this time come into use for cases in which early surgery had been omitted or had been inadequate, and the same principles were simply applied to head wounds. No arbitrary time limit was established within which debridement might be regarded as safe. In some cases observed at the 21st General Hospital, it was carried out as long as 51 days after wounding. The wound was then tightly closed, and no drainage was employed. The results were practically always good. While it sometimes seemed desirable to postpone operative intervention until definite abscess formation with encapsulation had occurred, this practice was soon discontinued. Encapsulation was frequently slow, and it was realized that in the interim a good deal of damage could be done by infection, including further destruction of brain tissue and the development of cerebral fungus and fatal meningitis.

When debridement was done late, or when redebridement was practiced, it was usually found that the track of damaged, devitalized tissue was sharply differentiated from healthy tissue. As a result, operation was sometimes simpler than in the first few hours after wounding. Soft, necrotic material could readily be removed by suction, and the limits of the healthy brain could be recognized without difficulty.

When the infected track was well encapsulated, the analogy between this type of infection and the civilian type of abscess was so striking that in the first months of the war, civilian methods (open drainage and marsupialization) were favored. As time passed and the effectiveness of debridement, whether performed as a primary or a secondary procedure, became more and more evident, tight closure of the wound was carried out, and drainage was omitted in both encapsulated and nonencapsulated cavities. For drainage to be omitted with safety, it was necessary that the wall of the cavity be gone over millimeter by millimeter with the sucker until all gray, shaggy exudate had been removed and healthy-looking pink granulation tissue became evident.

ADJUNCT THERAPY

Antimicrobial Therapy

At the beginning of the Tunisian campaign, it was the policy to dust sulfanilamide crystals into all wounds before applying the first dressing.[2] At the same time, a sulfonamide drug was given by mouth and was continued until after debridement. This policy was used in head wounds, with the warning that sulfonamides must not be permitted to come into contact with neural tissues. As time passed, less and less reliance was placed on local chemotherapy and more and more on thorough wound debridement. As was learned by experience everywhere, unless debridement had been well done, chemotherapy did not prevent infection, while if the operation were properly performed, healing per primam was the rule even when chemical agents were not used.

After penicillin became available, in the spring of 1944, it was administered routinely to all casualties by the intramuscular route, in dosages of 25,000 units every 3 hours. Sample studies suggested that this measure was helpful in preventing infection, but the apparent improvement which occurred at this time must be evaluated in the light of other factors, chiefly more complete debridement and a more generous use of whole blood, both of which were now the general policy.

Early Ambulation

Before the United States entered World War II, British neurosurgeons, following the example of German neurosurgeons, had begun to treat all head

[2] In some of the first casualties received at Walter Reed General Hospital, Washington, D. C., from North Africa, sulfathiazole had been used in wounds, because of shortages of sulfanilamide crystals. It caked in the wounds and had to be picked out with forceps.

injuries, closed as well as open, by early ambulation as soon as the patient was capable of the necessary movement. In the early days of the war, when overcrowded hospitals necessitated a rapid turnover of patients in Great Britain, the method was tested on a large scale, and it was found that early ambulation following cerebral concussion not only is safe but actually results in a lowered incidence of the posttraumatic syndrome.

Early in 1942, neurosurgeons in Army general hospitals in the Zone of Interior had begun to practice early ambulation in closed head injuries, but there was little opportunity overseas to test the method on a large scale until the North African campaign, in the fall of 1942. The first report on its use in this theater was made by Capt. (later Maj.) Edwin W. Shearburn, MC, and Lt. (later Capt.) Edwin H. Mulford, MC, from the 8th Evacuation Hospital in Casablanca. In 72 cases of acute head injury with cerebral concussion observed between 18 November 1942 and 6 March 1943, there was only 1 instance of posttraumatic syndrome following its use, against 4 instances in 18 cases which were observed over the same period and in all of which, for various reasons, early ambulation could not be used. No bad effects of any kind were observed in cases in which it was employed. After this time, the method became routine in the Mediterranean theater and was also adopted in the other theaters of operations.

COMPLICATIONS OF HEAD INJURIES

Cerebral Fungus

Cerebral fungi (herniation) fell into two groups, as follows:

1. The so-called benign type was manifested by local cerebritis associated with superficial wound infections. It was likely to occur in cases in which the dura had been left open. The fungus grew slowly, was seldom large, shaggy, or friable, and was not associated with high intracranial pressure, progressive neurologic changes, or deep infections. Treatment was limited to wet dressings, since, in most cases, the natural tendency was toward healing and retraction of the hernia. If bone fragments were present, they were removed. In the few cases in which benign fungi were excised, examination of the tissue showed a fibrotic process which suggested that the hernia would have subsided spontaneously in time.

2. So-called malignant fungi developed as a result of deep abscess formation, hematoma formation, or, less often, massive necrosis of brain tissue. They were associated with increased intracranial pressure, and, once they had extruded through the open dura, they grew rapidly, became shaggy, and bled easily. A hernia of this type occasionally was the sequel of intense cerebritis or of a virulent local wound infection, usually in an inadequately debrided wound.

In this type of fungus, progressive destruction of neural tissue was the rule, and early surgical interference was therefore essential, following pre-

operative localization of retained bone and metallic fragments by stereoscopic roentgenograms. Removal of the necrotic portion of the fungus often tended to reduce pressure locally and sometimes uncovered a sinus leading down to an abscess, which could be treated by appropriate means. Fatalities, which could usually be attributed to the presence of an unsuspected abscess or to its delayed evacuation, were fairly frequent.

Epilepsy

Convulsions sometimes occurred between the time of wounding and debridement, but when progress was satisfactory they seldom occurred within the first month after operation. Their earlier development, within the first week or two after operation, usually indicated the presence of an abscess, a hematoma, or a pocket of necrotic tissue in the cerebrum. The convulsions were most often Jacksonian in type and were likely to be associated with some degree of motor weakness.

DISPOSITION

The majority of patients with penetrating wounds of the brain in the Mediterranean theater were evacuated to the Zone of Interior because of skull defects, residual neurologic changes, or associated injuries. Patients with scalp wounds could usually be returned to duty after a relatively brief convalescence. In closed head injuries, disposition depended upon the extent of the injury and the outcome of treatment; in many instances, return to full duty was possible. Had appropriate materials been available, many skull defects which necessitated reclassification and disposition to the Zone of Interior could probably have been repaired overseas.

CHAPTER VII

The European Theater of Operations

R. Glen Spurling, M. D.

HEAD INJURIES BEFORE D-DAY

Except for head injuries sustained by Army Air Force personnel, all craniocerebral injuries observed in the United Kingdom before D-day were of the type encountered in civilian life. Patients with closed injuries were treated by conservative measures in the hospitals to which they were first admitted. In their management, a liberal policy was instituted, in contrast to the generally accepted policy in civilian practice, and large numbers were promptly returned to activity and later to full duty.

Patients with compound craniocerebral injuries admitted to station hospitals were transferred as soon as possible to the nearest general hospital for definitive treatment. The wound was protected with sterile dressings, the scalp was shaved over a large area surrounding the wound, sulfanilamide powder was sprinkled into it, and a sterile compression dressing and bandage were applied. Morphine was not given. No surgical interference was undertaken at any but a general hospital without consultation with the senior consultant in neurosurgery or his authorized representative. Treatment of open head injuries followed the principles laid down in Medical Bulletin No. 12, Office of the Chief Surgeon, Headquarters, ETOUSA, dated 1 November 1943. These principles were, in general, those followed in civilian practice before the war.

In Essential Technical Medical Data dated 22 August 1943, Col. Loyal Davis, MC, then the senior consultant in neurosurgery in the theater, reported the results of 147 head injuries treated according to these principles in the year ending on 30 April 1943. These patients were chiefly treated in three general and one evacuation hospital, and Colonel Davis commented that when patients were treated primarily in hospitals other than those in which officers with neurosurgical personnel were in charge, the occurrence of the posttraumatic syndrome was very high. He also commented on the precipitate decrease in the number of admissions for head injuries after the use of jeeps by enlisted personnel had been restricted to those on authorized missions.

There were no deaths in these 147 cases, and 88 of the 99 patients with closed injuries were returned to duty. In contrast, only 18 of 48 patients with open head injuries could be returned to full duty in the theater, and only 2 others to limited duty. Thirty-two of the forty-eight patients in the latter group developed the posttraumatic syndrome.

GENERAL POLICIES OF MANAGEMENT

In the Manual of Therapy, prepared in the European theater before D-day (p. 73), emphasis was laid on certain important considerations in the early management of combat-incurred head wounds. Among these considerations were the following: (1) That the sole objective of emergency treatment was to improve the condition of the patient sufficiently to permit transportation to an installation where competent neurosurgical care was available; (2) that high priority evacuation was always indicated in head injuries; (3) that the unconscious patient was to be transported on his side, with the foot of the litter elevated, to permit postural drainage; (4) that every scalp wound should be regarded as having serious implications until it was proved not to have; (5) that shock seldom resulted from uncomplicated head injuries and that its presence therefore demanded a search for other possible injuries; (6) that if shock were present, its treatment by the usual routine took precedence over the treatment of all other conditions; (7) that local measures should be reduced to a minimum; and (8) that morphine was not to be given unless other injuries demanded it, and then with great caution.

Special emphasis was placed upon the prompt recording of data essential for the efficient future treatment of the patient, including the time of injury, the state of consciousness when he was first seen, the pulse rate (counted for a full 30 seconds), the respiratory rate and rhythm, the blood pressure, and the presence or absence of paralysis in the extremities.

These policies were based on the recorded experiences in World War I (p. 3), the combat experiences of the British in the first years of World War II, and the United States experience at Pearl Harbor and elsewhere in the Pacific. They were promptly confirmed by the experience in the first weeks after D-day, which made clear the following important points:

1. Every wound of the scalp must be regarded as a possible penetrating wound until the presence of foreign bodies within the cerebrum is ruled out by roentgenograms.

2. Infection tended to occur in the wound of entrance rather than around the foreign body, which was usually sterile and which could be left in situ at debridement, for subsequent removal under ideal conditions. Bone fragments, not missiles, proved the chief cause of infection.

3. Patients with head injuries withstood transportation well for the first several hours after wounding but poorly for the first few days after operation. The best policy was therefore to move them back to hospitals in which definitive treatment could be carried out without undertaking any surgical measures other than debridement.

4. Subdural hematomas should be suspected in troops of armored units and in paratroopers.

5. Debridement could be safely undertaken even 5 or 6 days after injury. In the first days of the fighting on the Continent, many patients with compound head wounds were received in hospital installations in the United Kingdom

after delays of such length, having had no treatment other than first aid. All of them had had sulfonamide therapy, and most of them had had penicillin in adequate amounts. The wounds were in remarkably good condition, and debridement and delayed primary wound closure were usually successful, even at this late date.

6. The ceiling for air transportation of patients with head injuries was 3,500 to 4,000 feet. If they experienced breathing difficulties during the flight, the policy was to turn them on the side, face down, and elevate the foot of the litter; these measures were particularly important if there was fluid in the lungs.

On the basis of the experience immediately after D-day, the following broad principles of management were emphasized:

1. If penetrating wounds of the brain were seen soon after wounding, surgical debridement of the contused brain tissue, meninges, skull, and scalp, in that order, was the treatment of choice. Dural defects should be repaired with fascia from any available source. The wound should be frosted throughout with sulfanilamide powder (a policy that was later discontinued). Sulfathiazole should never be used locally (p. 106), nor should penicillin until more data on its possible irritating properties became available. Closure should be tight, without drains. Sulfonamide and penicillin therapy should be continued systematically after operation.

2. If penetrating wounds of the brain were observed late, the same plan of treatment should be adopted in the absence of infection. If infection were evident, the usual plan of care for infected wounds should apply.

Particularly in forward echelons, the treatment of head injuries occupied the major portion of the neurosurgeons' time and effort. The majority of the wounds were caused by penetration of missiles into the scalp, skull, and brain, but there was also a considerable number of closed head injuries.

WOUNDS OF THE SCALP

One of the important lessons learned by neurosurgeons in the European theater was the value of sliding flaps to cover large defects in scalp wounds. The use of this method permitted a more complete and more adequate debridement of the wound margins and at the same time provided a means of closing the wound without tension. Lt. Col. (later Col.) Eugene M. Bricker, MC, Senior Consultant in Plastic Surgery, Office of the Chief Surgeon, Headquarters, ETOUSA, prepared an article on closure of scalp defects by means of sliding flaps. This article, published in the Medical Bulletin, Office of the Chief Surgeon, helped materially to disseminate the advantages of the sliding-flap method of closure among neurosurgeons throughout the theater.

CLOSED HEAD INJURIES

Closed head injuries were for the most part treated by conventional methods. The most seriously injured casualties were evacuated as promptly as possible to fixed hospitals. Casualties who had suffered only cerebral

concussions, or even contusions, were kept within the Army area, since early experience had proved that if they once reached the level of the general hospital, an early return to duty was unlikely.

An important new concept in the treatment of this group of patients was early ambulation. The value of this method had been demonstrated earlier in the war in the German Army and the British Army, as well as in British civilian hospitals and United States Army hospitals in the Zone of Interior. It had become routine in the Mediterranean theater (p. 106) and was part of the original planning for head injuries in the European theater. Every effort was made to keep the patient out of the horizontal position. When consciousness was regained, he was kept in the semisitting position the entire time he was in bed, and he was encouraged to get out of bed as promptly as possible.

Depressed fractures of the skull without dural penetration, which were relatively common, furnished few therapeutic problems. If there was little evidence of underlying cerebral damage, debridement was followed, as soon as wound healing permitted, by cranioplasty with tantalum. A small number of patients successfully withstood tantalum repair of the cranial defect at the time of the original debridement. The one-stage operation was naturally advantageous, but it was attempted only when the wound was small and comparatively clean and when the casualty load did not require a rapid turnover of patients. Many patients treated by tantalum repair were returned to limited duty, and some to full combat duty, within the European theater.

PENETRATING WOUNDS OF THE BRAIN

Penetrating wounds of the brain, which comprised approximately a fifth of the neurosurgical casualties in the general hospitals in the European theater, made up the major portion of the neurosurgical load in the evacuation hospitals. In the first days of the European campaign, as already mentioned, initial debridement was often carried out in the neurosurgical centers in the United Kingdom. As the campaign progressed and more and more hospitals were transferred to the Continent, most initial neurosurgery was done in them, although secondary procedures were necessary in installations in the United Kingdom in about a quarter of all cases.

The soundness of the policies of complete debridement, repair of dural defects, and careful primary closure of scalp lacerations was established beyond dispute in this theater. When most of the initial operations were still being done in the United Kingdom Base, it was found that complete closure of the head wound was possible, with safety to the patient and usually with primary healing, after timelags from 6 up to 10 days. The chief indications for secondary surgery were the removal of bone chips or large missiles left in situ at the first operation; drainage or complete excision of brain abscesses; repair of cerebrospinal fluid fistulas; and excision of cerebral fungi, with subsequent closure of overlying cranial defects.

Most patients with penetrating wounds of the brain were returned to

the Zone of Interior as soon as possible after operation. Many had residual neurologic defects. Some developed convulsive seizures within a few weeks after injury. Some required cranioplasty for large cranial defects.

Closure of the Dura

In penetrating wounds of the brain produced by jagged metallic foreign bodies, the dura was usually badly fragmented, and simple suture was not possible. British neurosurgeons, with a large experience in earlier campaigns, had come to disregard dural perforations and to content themselves with thorough debridement, omitting plastic repair of the dural defect unless the injury was in the immediate vicinity of the accessory nasal sinuses. This policy was not in accord with practices in the United States, and United States Army neurosurgeons were instructed to close all dural defects when the original debridement was done unless valid contraindications existed. Accurate statistics are not available to confirm the value of this method, which was employed throughout the fighting in Europe, but it was thought significant that British neurosurgeons began to change their own policy and, by the close of the campaign in Europe, were repairing the dura with greater and greater frequency.

Methods employed to close the dural defect included the use of fascia lata, pericranium preserved in alcohol, human dura, and human fibrin film. The routine use of fascia lata for dural repair proved impractical, and most neurosurgeons learned to use pericranium unless the defect was too large. Fibrin film for dural repair had been developed in the Zone of Interior by Dr. Franc D. Ingraham of the Harvard Medical School, under a contract with the National Research Council, and it had been used with considerable enthusiasm in neurosurgical centers in the Zone of Interior.

Maj. Arthur D. Ecker, MC, made two important contributions to the problem of dural repair. The first was the demonstration that it is possible to dissect the pericranium and galea intact and thus to provide a thicker tissue for repair of the dura. Dissection of the two layers without separation is facilitated in this technique by the use of physiologic salt solution to distend the fatty layers of the scalp above the galea.

Major Ecker's second contribution was the demonstration that it is possible to reflect pericranial grafts into the region of the dural defect without detaching them at one margin, their viability thus being assured because part of their blood supply is preserved. This technique proved particularly valuable in tears around the anterior cranial fossa associated with cerebrospinal fluid leaks. Histologic studies made in some of the cases in which pericranial grafts were left attached at one margin established their viability and thus confirmed the feasibility of the method in infected wounds, in which free transplants of fibrous tissue of other types were likely to slough.

At the December 1944 meeting of the neurosurgeons of the United Kingdom,[1] the consensus was that grafts of fascia lata were most likely to slough

[1] Minutes, Meeting with Neurosurgeons, United Kingdom Base, 20 Dec. 1944.

and that the best results were likely to be obtained by the Ecker technique. Dissemination of this information through the efforts of the senior consultant in neurosurgery led to their general use and to the confirmation of their worth by a thorough clinical trial. In the interrogation of a German neurosurgeon in a prisoner-of-war camp some months after the December meeting, Colonel Spurling found that the German had used the Ecker technique for the past 2 years and that, prior to that time, he also had had poor luck with fascia lata transplants.

Management of Retained Foreign Bodies

Early in the war, British neurosurgeons had called attention to the danger of leaving bone fragments in situ in operations for penetrating wounds of the brain. They had shown conclusively that late brain abscess was of far greater frequency in incompletely debrided wounds than in wounds from which all foreign bodies, including bone fragments, had been completely removed. The only contribution of United States neurosurgeons to this problem was the demonstration (first in the Mediterranean theater, p. 105) of the safety of early reexploration in such cases, with excision of the gliotic tract containing foreign material, and simultaneous repair of the dura and of the skull defect with tantalum.

At the December 1944 meeting of the neurosurgeons of the United Kingdom Base, it was pointed out that the figure of 17 percent for retained bone fragments after primary operation, which appeared in the statistics collected to date by Colonel Spurling, was probably misleading, for two reasons. The first was that most of the wounds observed in neurosurgical centers, from which the data had been collected, concerned complicated wounds; patients whose wounds had healed primarily had already been evacuated from general hospitals to the Zone of Interior. The second reason was that the collected data covered only the patients treated in the United Kingdom. It was decided that these considerations should be taken into account thereafter and that an attempt would also be made to determine the situation in this regard in evacuation hospitals. Data collected in this manner showed a decided rise in the percentage of retained fragments, to 44 percent in the November 1944–January 1945 period, and to 35 percent from 1 February until V–E Day (table 5).

Before the end of the war, it was accepted practice in the European theater, when a shower of retained bone fragments was demonstrated in a wound, to reexplore it as soon as the patient's condition permitted, whether or not there was evidence of infection. After operation, the patient was returned to the Zone of Interior, with his wound cleanly debrided and with an essentially intact skull.

In the early days of the European campaign, not much attention was paid to metallic foreign bodies retained in the brain (p. 110). These missiles were often found at great distances from the contused track, as the result of scattering of bony fragments from the skull. The possible danger of the retention of

TABLE 5.—*Percentage data on 2,276 penetrating cerebral wounds operated on in United Kingdom hospitals*

Period	Cases	Case fatality rate	Average timelag	Primary dural repair	Retained bone fragments after primary operation	Infections				
						Superficial scalp infections	Subgaleal infections	Brain abscess	Meningitis	Total
D-day to 1 Aug. 1944	*Number* 494	4.3	*Days* 4	*Percent* 63.0	*Percent* 17.0	*Percent* 12.0	*Percent* 5.5	*Percent* 7.5	*Percent* 8.5	*Percent* 33.5
1 Aug. 1944 to 1 Nov. 1944	942	4.3	4	60.0	18.5	11.2	5.0	5.6	5.0	26.8
1 Nov. 1944 to 1 Feb. 1945	393	4.0	11	71.0	44.0	9.0	4.6	12.2	3.6	29.4
1 Feb. 1945 to V-E Day	447	5.1	8	55.0	35.0	10.1	6.9	13.0	17.0	47.0
Total	2,276	4.4	6.7	61.5	25.8	10.8	5.4	8.6	7.9	32.7

these fragments was realized, but it was originally thought that the danger of removal, which often introduced the risk of traversing essentially normal brain tissue, was greater than that of leaving them in situ. Later, the reverse was found to be true.

Neurosurgeons in the forward areas were the first to recognize that subdural or subcortical hematoma formation was likely to occur in traversing wounds of the brain in association with the presence of foreign bodies. They had observed that, when the patient's condition had failed to improve after the first operation, a hematoma was often found on later exploration for foreign bodies. The observation became so general that all neurosurgeons in forward areas soon learned to investigate the site of metallic foreign bodies by burrhole exploration. As experience increased, it became the routine, in the management of patients with traversing wounds of the brain, to explore the region of the metallic foreign bodies for a possible hematoma even before debridement of the wound of entrance. If a hematoma was demonstrated, its immediate evacuation usually so greatly improved the patient's condition that the more formidable procedure of debridement could be proceeded with in a more leisurely manner. This concept, which was entirely new, was presented by Capt. (later Maj.) Donald D. Matson, MC, in an article entitled "Hematomas Associated With Penetrating Wounds of the Brain." [2]

Complications

Brain abscess.—The first realization of the possible risk of infection associated with retained metallic foreign bodies came in an autopsy at the 160th General Hospital, on a patient who had arrived with an infected wound

[2] Matson, D. D., and Wolkin, J.: Hematomas Associated With Penetrating Wounds of the Brain. J. Neurosurg, 3:46-53, January 1946.

and a large brain abscess in the hemisphere, at the site of entrance of the missile. Although the abscess was excised successfully, his condition continued to deteriorate, and at autopsy a large abscess surrounding a metallic foreign body was found in the contralateral hemisphere. The wound of entrance had been on the left side in the frontal region, and the foreign body was in the same location on the right side; there had therefore been few clinical signs to indicate the proper management of the case. As a result of this observation, all neurosurgeons in the theater were instructed to explore the region of the foreign body in every traversing, penetrating wound of the brain if a cerebral abscess or hematoma could reasonably be suspected.

In the early phases of the campaign, neurosurgeons were permitted to treat brain abscess by whatever methods they had been accustomed to use in civilian practice. Conventional drainage was usually employed. Several surgeons later reported favorably upon the results of radical excision of the abscess wall, and the whole subject was discussed in detail at the first meeting of neurosurgeons in the United Kingdom, as part of the discussion of penetrating wounds of the brain.[3] It was difficult, it was pointed out, to distinguish between necrotic and infected brain, since in so many instances culture of the pus was sterile and since negative cultures were often obtained in the presence of active infection when the patient was being given sulfonamide drugs and penicillin. On the other hand, many surgeons present emphasized that the wall of an abscess, regardless of the report on cultures, always contained living organisms in great numbers and that a surgical procedure which did not deal with the wall was not a complete procedure.

In view of these observations a change of policy was decided upon. An anatomic definition of brain abscess was adopted; a brain abscess was to be indicated by microscopic or macroscopic pus present in the cavity. In cases in which there was profuse superficial infection of the scalp and the process was a cerebritis rather than a localized abscess, conservative methods were to be followed until localization had occurred, after which radical excision of the abscess wall was to be attempted. The wound was to be treated locally with sulfanilamide and penicillin, and both agents were to be given in large doses for several days after operation by the systemic route. The dura was to be closed tightly, with the aid of grafts if necessary, and the scalp wound was to be closed without drainage. If the inflammatory process was close to the ventricle or basal cisternae, penicillin by the spinal route was to be given for prophylactic purposes for several days after operation.

This method, which was originally suggested by the French neurosurgeons, Vincent and David, had previously been employed all over the world but only sporadically. It is believed that its use in the European theater was its first application on a large scale to the treatment of traumatic brain abscess.

Tube drainage methods were employed in cases in which the abscess re-formed after excision, as well as in cases in which the causative organisms

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[3] See footnote 1, p. 113

were found to be penicillin resistant. As this practice implies, bacteriologic studies were mandatory before radical surgery was undertaken.

During the 6-month period in which this method was used before the close of the European fighting, results of treatment of brain abscesses steadily improved. In addition to a decreased morbidity and the elimination of multiple operative procedures, it was thought that the incidence of convulsive seizures following brain abscess was materially reduced.

Meningitis.—The incidence of meningitis at the level of the general hospital, exclusive of cases of brain abscess complicated by active meningitis or meningeal irritation, was approximately 8 percent in the European theater. Most patients recovered under a combined regimen of penicillin by the intraspinous route and sulfadiazine by the systemic route.

Gram-negative bacillary meningitis, however, for a time threatened to become a serious problem. The first cases were observed in the summer of 1944, and the incidence steadily increased until, by the spring of 1945, the situation was grave. The causative organisms were, for the most part, *Bacillus pyocyaneus*, *Klebsiella friedländeri* and other members of the coliform group. Only a few of the patients went on to recovery in spite of massive doses of sulfadiazine and penicillin. This might have been anticipated, since all the causative organisms are penicillin resistant.

The solution of the problem was the demonstration by Major Ecker that a combination of urea (1 oz.) and sulfadiazine (2 gm.) in dextrose solution by vein every 4 hours produced almost immediate improvement. His first series of successfully treated cases was reported in the summer of 1944, and the method was brought to the attention of United Kingdom neurosurgeons at the conference in December. At the second conference in February 1945, the pooled experience showed 10 recoveries in 16 cases.[4] At the third conference,[5] in April 1945, it was again reported that most cases caused by the coliform bacilli, particularly *B. pyocyaneus*, had responded dramatically to the combined regimen of urea and sulfadiazine and that certain cocci resistant to penicillin and the sulfonamide drugs alone had also responded well, though most patients with meningitis caused by *K. friedländeri*, Type A, had died in spite of therapy. The high incidence of meningitis of this origin was undoubtedly responsible for the considerable increase in the neurosurgical case fatality rate observed in the United Kingdom hospitals between 1 February 1945 and V–E Day (table 5).

Cerebrospinal fluid fistulas and aeroceles.—Penetrating wounds of the frontal region of the skull were frequently associated with rhinorrhea and aerocele. In the past, these complications had had serious implications. With the advent of antimicrobial therapy, the incidence of complicating meningitis had been greatly reduced, though, in improperly handled cases, it was likely to occur after medication had been discontinued.

A decision concerning the repair of dural tears in this type of injury has

[4] Minutes, Meeting, United Kingdom Base Neurosurgeons, 15 Feb. 1945.
[5] Minutes, Meeting, United Kingdom Base Neurosurgeons, 18 Apr. 1945.

always been difficult. Many times, the leakage of cerebrospinal fluid ceases spontaneously. There is always concern, however, that the fistula may reopen or that infection may extend along the fistulous tract to the meninges.

The policy of early repair was followed in the neurosurgical centers throughout the European campaign. As soon as the patient's condition permitted elective surgery, the fistula was exposed through a unilateral or bilateral frontal bone flap, and plastic repair of the dura was accomplished by a pedicle flap of pericranium or a transplant of fascia lata. The operation was frequently long and tedious, because of the extensive damage to the dura associated with the injury, but closure of the leak was almost uniformly successful, and complications were practically nonexistent. Statistical data are not available, but the risks of the procedure can be assumed to have been remarkably low from the fact that in the final statistical analysis of 2,276 penetrating wounds of the brain treated in the United Kingdom Base it was found that only 4 percent of the deaths were caused by meningitis, including deaths from chemically resistant types of disease.

Fungus cerebri.—Fungus cerebri was relatively frequent in German prisoners of war with head injuries but was seldom seen in United States Army wounded because of the adequacy of their treatment in forward hospitals. This complication was observed, however, in a number of patients whose wounds became infected or who developed deep-seated brain abscesses.

When World War II began, little had been added to the treatment of this distressing condition since Cushing's contribution in World War I. The underlying principle of therapy had continued to be protection of the fungus from trauma and from the irritation of the endless dressings which were required. In many instances also, in spite of every precaution, the ventricle wandered into the structures of the fungus, and a ventricular fistula resulted. All in all, this complication was one of the most annoying and most difficult faced by neurosurgeons in the European theater.

At the second meeting of the neurosurgeons of the theater in the United Kingdom Base, Maj. Francis Carmichael, MC, presented an original plan of treatment for cerebral fungus. Accurate bacteriologic studies were first made. Then, if nonresistant chemical organisms were present, they were treated with applications of acetic acid and with other local measures until they had disappeared from the wound. When this had been accomplished, the fungus was trimmed with the electrosurgical unit until the presenting brain tissue could be tucked into the opening in the skull. This portion of the procedure could be facilitated by preoperative dehydration and spinal puncture. Finally, a tantalum plate fashioned to fit exactly over the skull defect was swaged into place, and the scalp was closed over the plate, either directly or by a swinging flap. No attempt was made to repair the dura; the presenting fungus was simply left to fit snugly against the smooth inner surface of the tantalum plate. Before operation, it was always necessary to exclude the possibility of an intracerebral abscess behind the fungus. This was accomplished by needle exploration or, particularly if a distant abscess was suspected, by ventriculography.

After operation, antimicrobial therapy was continued in maximum doses by the systemic and intraspinal routes until all signs of infection had completely subsided. The chief immediate postoperative problem, increased intracranial pressure, could usually be controlled by the use of a dehydrating agent, preferably sucrose or concentrated human serum albumin, and by daily lumbar punctures.

Major Carmichael's report at the February 1945 meeting consisted of three cases, to which Maj. (later Lt. Col.) Wesley A. Gustafsen, MC, added three other cases. In all of these cases recovery was without incident. Major Carmichael's report was later published.[6] In the ensuing months, equally good results were obtained in 25 cases treated by this technique in the neurosurgical centers in the United Kingdom.

INJURIES OF THE CAROTID ARTERY

Early in the European campaign, an occasional patient was observed without evidence of head injury but with a gunshot wound of the neck associated with concomitant contralateral hemiplegia. The only logical explanation of such a phenomenon was an injury to the carotid artery, with resulting ischemia of the ipsilateral hemisphere. In some cases in which there was no evidence of disruption of the carotid artery, arterial spasm was postulated as the explanation of the ischemia.

In a report on spasm of the internal carotid artery associated with gunshot wounds of the neck,[7] Major Ecker was able to demonstrate by pneumoencephalography that in the cases which had been under his personal observation atrophic changes had occurred in the ipsilateral hemisphere. In the following months, more and more such cases were encountered, and the observations described were confirmed. Treatment by early Novocain (procaine hydrochloride) block of the stellate ganglion was planned, but hostilities ended before the method could be tested fully. These observations opened up an entirely new field in traumatic neurosurgery, and further laboratory investigation of the mechanism of injury was considered to be indicated.

ANTIMICROBIAL THERAPY

Antimicrobial therapy was regarded in the European theater as of very great importance in the treatment of penetrating wounds of the brain, but only as an adjunct to early, adequate surgery. In most cases in which it failed to prevent infection, debridement was found to have been inadequate. Routines of therapy varied with individual surgeons, but penicillin and sulfadiazine were regularly administered by the parenteral route before and after operation and were continued until complete wound healing had occurred. Sulfanilamide and penicillin were usually used by the local route also.

[6] Carmichael, F. A., Jr.: The Reduction of Hernia Cerebri by Tantalum Cranioplasty. A Preliminary Report. J. Neurosurg. 2: 379–383, September 1945.

[7] Ecker, A.: Spasm of the Internal Carotid Artery. J. Neurosurg. 2: 479–484, November 1945.

Penicillin was used freely by the intraspinal and intravenous routes, according to the indications. It was always used as early as possible when the injury was complicated by meningitis or brain abscess. It was also used prophylactically before closure of cerebral spinal fluid fistulas.

STATISTICAL DATA

In Essential Technical Medical Data, ETOUSA, dated 17 August 1944, it was stated that wounds of the brain, spinal cord, and peripheral nerves comprised 10 percent of all surgical cases treated in the United Kingdom. Statistical data collected in Normandy by Colonel Spurling indicated that head injuries usually comprised at least 3 percent of all injuries treated in evacuation hospitals and that the proportion sometimes rose to 5 percent. The mortality rate in these hospitals for head injuries varied from 5 to 8 percent.

Relatively few penetrating wounds of the brain received primary definitive care in the United Kingdom Base, the majority of patients with these injuries being cared for in evacuation hospitals. A large proportion of the patients studied in the final statistical survey of neurosurgical data in the United Kingdom (table 5) are those who required secondary procedures because of complications, chiefly retained bone fragments, cerebrospinal fluid fistulas, and wound infections. The patients in this analysis were cared for in 9 specialized neurosurgical centers by approximately 20 neurosurgeons. In general, the regimen of management was the same in all centers; that is, the same indications for reoperation were observed, the same surgical technique was employed, and the critical analysis was made according to the same criteria.

Difficulties in collecting statistical data from the various field armies have been outlined elsewhere (p. 80). During the European campaign, however, an effort was made to make sample studies of penetrating wounds of the brain in the First and Seventh U. S. Armies. The material, while not precisely comparable, nonetheless illustrated the general situation in respect to these wounds in both Armies. The data were submitted to the Chief Surgeon, ETOUSA, 25 March 1945, and, after they had been approved by him, were distributed in mimeographed form to the neurosurgeons of the theater.

Data from the First U. S. Army (table 6) which were collected from the 13 evacuation hospitals which supported it include all penetrating wounds of the brain and all compound fractures of the skull without dural penetration in which surgery was done from D-day to 1 November 1944. Unfortunately, when these data were requested it was not specified that information was also desired on patients who had reached the hospital alive but who had died without surgery after admission. The data are therefore open to the criticism that they are somewhat selective and represent only favorable cases, as exemplified by the case fatality rates of 14.8 percent for penetrating wounds of the brain and 1.3 percent for compound fractures of the skull. The criticism, however, would not be precisely fair. It is true that any surgeon can keep his case fatality rate low if he operates only on favorable cases, while a surgeon whose

TABLE 6.—*Distribution of cases and postoperative deaths in 1,102 penetrating cerebral wounds and 390 compound skull fractures managed in 13 hospitals of the First U. S. Army*

Hospital	Penetrating cerebral injuries		Compound skull fractures	
	Cases	Deaths	Cases	Deaths
	Number	*Number*	*Number*	*Number*
A	135	34	31	1
B	77	27	13	0
C	97	12	21	1
D	84	14	15	0
E	95	5	54	0
F	69	12	32	1
G	43	5	12	0
H				
I	38	8	14	2
J	57	3	14	0
K	94	5	59	0
L	249	33	98	0
M	64	5	27	0
Total	1, 102	163	390	5

criteria of operability are broader—like a surgeon with poor judgment who selects cases unwisely—will show a higher rate. The cases in this series undoubtedly include many in which the casualty, when he was first seen, appeared to be a bad risk but in which he was transformed into a surgical possibility after proper resuscitative treatment for 12 to 14 hours.

It should also be emphasized, however, that the case fatality rates for penetrating wounds of the brain and compound skull fractures without dural penetration (14.8 and 1.3 percent, respectively) by no means represent the whole picture of such casualties in the First U. S. Army. In the early days of the campaign, a policy was decided upon by which, in times of stress, only the most serious head injuries would be operated on in evacuation hospitals, while patients whose general condition was satisfactory would be evacuated by air to the United Kingdom for primary definitive treatment. The figures must be read with this qualification in mind.

It will be noted (table 6) that two hospitals, designated as A and L, handled larger numbers of cases than any of the other units. This was the result of deliberate planning. The work in them was done by experienced neurosurgeons who had been given the responsibility of supervision of the neurosurgical work in evacuation hospitals in the early days of the campaign and who, at the same time, acted as neurosurgical consultants for the Surgeon, First U. S. Army.

The chief defect in First U. S. Army statistics, the failure to include in the group of penetrating head wounds the patients who had been admitted to hospitals but who had died before surgery, was borne in mind when data

from the Seventh U. S. Army were requested, and the figures collected from that source (table 7) are accurate in this respect. One hundred and ninety (16.2 percent) of all casualties with head injuries were so seriously injured that they died soon after hospitalization or could never be brought to a status in which surgery was considered practical.

These factors are reflected in certain of the data. Records of the individual hospitals show wide variations in the surgical case fatality rate. When it was lowest, the overall case fatality rate was likely to be high. The figures might, on superficial examination, be interpreted to mean that neurosurgeons of certain installations were timid. The truth is more likely to be that they showed the best possible judgment in the selection of cases for operation. The two most experienced neurosurgeons in the Seventh U. S. Army, who worked in units L and M, operated independently of each other but had remarkably consistent records. Their series of cases, furthermore, represent the largest and probably the most representative series of head injuries managed in Seventh U. S. Army hospitals.

TABLE 7.—*Essential data in 1,176 penetrating cerebral wounds managed in 14 hospitals of the Seventh U. S. Army*

Hospital	Total cases	Total deaths		Surgical cases	Surgical deaths	
		Number	Percent		Number	Percent
A	84	20	23. 8	51	4	7. 8
B	96	33	34. 4	84	21	25. 0
C	34	9	26. 5	29	4	13. 8
D	76	34	44. 7	52	12	23. 1
E	63	14	22. 2	52	3	5. 8
F	87	16	18. 4	76	5	6. 6
G	72	13	18. 0	66	7	10. 6
H	39	11	28. 2	31	3	9. 7
I	95	27	28. 4	80	12	15. 0
J	67	30	44. 8	52	17	32. 7
K	19	4	21. 1	15	0	0
L	174	41	23. 6	133	16	12. 0
M	165	41	24. 8	143	19	13. 3
N	105	34	32. 4	85	14	16. 5
Total	1, 176	327	27. 9	949	137	13. 7

CHAPTER VIII

The Management of Acute Craniocerebral Injuries Due to Missiles

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GENERAL CONSIDERATIONS OF PENETRATING HEAD INJURIES

Early in World War I, surgical mortality in acute penetrating missile wounds of the brain averaged 50 to 60 percent. During the war, this rate was greatly reduced, principally by the use of operative techniques introduced by Harvey Cushing. At one casualty clearing station, during a 3-month period in 1917, Cushing's own surgical team operated on 133 patients with dural penetration. The case fatality rate for the first 44 cases was 54.5 percent. For the next 44 cases, it was 40.9 percent, and for the third set of cases (45), the rate was reduced to 28.8 percent.[1] In World War II, during a 5-month period in the European Theater of Operations, United States Army, the surgical case fatality rate in two field armies averaged about 14.5 percent.[2]

This improvement cannot be attributed to any single innovation in therapy. The basic principles of operative treatment were not significantly altered, though the equipment to carry them out was vastly improved. The following factors are believed to be the most important in the lowered mortality: (1) The placement of trained neurosurgical personnel in forward hospitals, in which there was adequate specialized equipment; (2) the rapid evacuation of casualties to these hospitals, which permitted early definitive debridement and repair; (3) the availability of blood in large amounts in the forward areas for replacement therapy; and (4) the universal employment of sulfonamide and penicillin therapy in all compound injuries.

In World War II, the line of battle was seldom fixed for any length of time, and it was therefore as necessary for expert medical care in forward areas to be mobile and adaptable to changing tactical situations as it was for combat and supply units to meet these requirements. In this war, the proportion of injuries due to high-explosive shell fragments, aerial-bomb fragments, and land-mine

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[1] (1) Cushing, H.: Notes on Penetrating Wounds of the Brain. Brit. M.J. 1: 221-226, 23 Feb. 1918. (2) Cushing, H.: A Study of a Series of Wounds Involving the Brain and its Enveloping Structures. Brit. J. Surg. 5: 558-684, April 1918. (3) Hanson, A. M.: Management of Gunshot Wounds of the Head and Spine in Forward Hospitals, A. E. F. *In* The Medical Department of the United States Army in the World War. Washington: Government Printing Office, 1927, vol. XI, pt. I, pp. 776-794. (4) Horrax, G.: Observations on a Series of Gunshot Wounds of the Head. Brit. J. Surg. 7: 10-54 July 1919.

[2] Essential Technical Medical Data, European Theater of Operations, for March 1945. Inclosure 8, subject: Statistical Analysis of Penetrating Wounds of the Brain From Army Evacuation Hospitals.

fragments to those due to small-arms fire was greatly increased over that of World War I. In the series of injuries upon which this chapter is based, as well as in Maltby's [3] series, the ratio was about 1 gunshot wound to 9 wounds caused by fragments of various types of high-explosive missiles.

The helmet was modified early in the war to give added protection to the frontal, occipital, and temporal areas. This precaution undoubtedly reduced the morbidity and case fatality rates from wounds of the head, particularly by the deflection of small shell fragments and the transformation of many potential penetrating brain wounds into simple gutter and tangential wounds of the skull and scalp alone. When, however, large shell fragments or machine-gun or rifle bullets hit the helmet at a direct 90° angle to its curved surface, they often penetrated it and carried fragments of it into the brain.

The task of primary, definitive operative treatment of head injuries, particularly the time-consuming, tedious debridement of penetrating wounds of the brain, was a tremendous one, and the global distribution of the fighting necessitated the services of a large number of surgeons. Although, for this reason, there were many minor variations in operative techniques and methods of supportive therapy, this chapter attempts to record, in a general way, the problems faced, the results achieved, and the lessons learned.

OBJECTIVES OF TREATMENT

The treatment of acute penetrating wounds of the brain caused by all types of high-explosive missiles was directed primarily toward four ends as follows:

1. *The immediate saving of life.*—Actually, this was an uncommon accomplishment of early operation. Very occasionally, persistent bleeding from torn dural sinuses or cortical vessels could be controlled only by operative intervention. Cerebral compression which occasionally occurred from intracranial hematomas, extradurally, subdurally, or in the depths of a wound track could be relieved by surgery, with dramatic improvement in the patient's general condition.

2. *The prevention of infection.*—This was perhaps the most important duty of the neurosurgeon in the treatment of acute head injuries. The most common cause of infection was inadequate primary debridement, debridement here meaning 100-percent surgical excision of all tissues which had been contaminated, macerated, or severely contused by the destructive agent, or which showed enough evidence of ischemia to presage subsequent necrosis. This type of debridement implied direct inspection of each layer of the wound, including skin, galea, pericranium, cranium, dura, leptomeninges, cerebral cortex, subcortical tissues, and, if necessary, the interior of the ventricular system itself. It also meant removal of foreign substances carried into the

[3] Maltby, G. L.: Penetrating Craniocerebral Injuries, Evaluation of Late Results in a Group of 200 Consecutive Penetrating Cranial Wounds. J. Neurosurg. 3: 239-249, May 1946;

brain. In addition to the surgical procedure, the early and adequate use of chemotherapy was a major responsibility of the operating surgeon.

3. *The preservation of nervous function.*—The key to this primary aim of early treatment is found in the work of Penfield and his associates,[4] who stated:

> There is a startling difference between the result of direct brain laceration where injured cerebral tissue is left in place, and the result of clean excision which leaves no destroyed cerebral tissue behind. In the former case a rich plexus of new-formed vessels appears and a core of mixed connective tissue and astrocytes is formed. This is surrounded by a zone of intense fibrillary gliosis in which the most robust glial fibers point toward the contracting cicatrix. * * * The excision of cerebral tissue, however, results in little or no new vessel formation and only a very small amount of gliosis which is apt to be found in a thin and inconstant layer in the grey matter * * * while there may be none in the white matter. Thus the excision results in a fluid-filled space almost free from gliosis, while laceration results in intense glial and connective tissue proliferation.

Clean removal of devitalized cerebral tissue, relief of cerebral compression, and repair of dural defects all served, therefore, to minimize formation of meningocerebral scars and to permit the maximum recovery of neurophysiologic defects.

4. *The restoration of anatomic structure.*—Accurate closure of the dura and scalp at the time of primary debridement of the wound always provided the best opportunity for satisfactory anatomic reconstruction.

ESSENTIALS OF TREATMENT

The Neurosurgical Team

The majority of acute penetrating craniocerebral injuries were cared for in evacuation hospitals, under the supervision of a neurosurgeon assigned to the hospital or under the direction of a mobile neurosurgical team (usually from an auxiliary surgical group or mobilized from an inactive general hospital) temporarily attached to it. For satisfactory care of any significant number of injuries, a team trained for this particular type of work proved imperative. The actual debridement of the wound of the brain was the problem of the operating surgeon, but complete management of the patient and of the operating equipment required the coordination of all members of the team.

When there were many craniocerebral injuries, there were usually large numbers of other wounds as well; as a result, only one operating table per team was usually available. This shortage necessitated foresight in the preparation of the patient and of the operating equipment, which was carried out as completely as possible by personnel not needed in the operation. With proper organization, a single neurosurgical team could often treat from 5 to 8 craniocerebral wounds, depending upon the severity of the injuries, on a 12-hour shift. When large numbers of casualties were concentrated at one hospital during heavy fighting, it was advantageous to have two neurosurgical teams available,

[4] Penfield, Wilder (ed.). Cytology and Cellular Pathology of the Nervous System. New York: P. B. Hoeber, Inc., 1932, vol. II, p. 458.

so that each could work a 12-hour shift and thus maintain uninterrupted surgical treatment.

For continued effective operation, it was found that the following personnel should be assigned to a neurosurgical team, whether the team was an integral part of a medical installation, such as an evacuation hospital, or a mobile team temporarily attached to the hospital:

1. A trained neurosurgeon or a well-qualified general surgeon who had received special instruction in the technique of handling nerve tissue, to serve as the responsible operator and chief of the team.
2. Another surgeon or a capable, well-trained enlisted surgical technician.
3. A medical officer or trained nurse-anesthetist, who was needed even when local anesthesia was used because it was necessary that a responsible person be present to observe the patient and give supportive treatment.
4. A surgical nurse or an enlisted surgical technician trained in the handling of neurosurgical equipment, to serve as scrub nurse.
5. A trained enlisted man, not on the operating team, who would be available at all times to move the patient, arrange lights, shave the next patient, do catheterizations, clean instruments, and procure supplies.

Equipment

Certain specialized equipment was regarded as essential for definitive primary operation on craniocerebral injuries. As emphasized by Cushing,[5] this is all-or-nothing surgery, and operation should not be attempted unless complete debridement and repair can be carried out. While adequate surgery was undoubtedly performed occasionally with insufficient equipment, the following items, in addition to standard surgical supplies, were considered to be necessary for satisfactory work:

1. A portable electric suction unit or other means of providing reliable strong suction.
2. A portable Bovie electrosurgical unit.
3. A headlight, narrow lighted retractors, a small malleable light, or other lighting suitable for illumination of deep, narrow wounds.
4. Pressed cotton, dental roll, or long-fiber bulk cotton, which could be made into moist pledgets for sponging and for protection of normal brain tissue.
5. Special instruments, such as a hand drill with cranial perforator and burr, sharp periosteal elevator, narrow malleable retractors, ventricular cannulas, silver clips, and small curved round needles for dural sutures.
6. No. 8 to No. 10 French soft rubber catheters, each with a hole in the side near the tip for irrigation of the wound.
7. No. 00 or 000 silk, for accurate approximation of the dura and scalp, or cotton or nylon of similar size.

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[5] (1) See footnote 1 (1) and (2), p. 123. (2) Cushing, H.: Organization and Activities of the Neurological Service, American Expeditionary Forces. *In* The Medical Department of the United States Army in the World War. Washington: U. S. Government Printing Office, 1927, vol. XI, pt. I, pp. 749-758.

8. Bone wax.

9. Fibrin or gelatin foam, which are efficient absorbable hemostatic agents and were of great value when they were available.

Records

It was the duty of every surgeon doing primary debridement and repair of craniocerebral wounds to make a record of his operative findings and manipulations. Since treatment could seldom be continued for any length of time after operation by the surgeon who did the original operation, it was imperative that an accurate record of the operation and of all other therapy accompany the patient when he was transferred from one medical installation to another in the necessary chain of evacuation. The data needed by those responsible for secondary care and eventual reconstructive surgery were best provided by a brief but comprehensive operative note written or dictated by the operating surgeon himself immediately following operation. Experience showed that the operative note should include statements concerning (1) the size and location of the cerebral wound and an estimate of the amount of cerebral tissue lost, (2) the removal of intracerebral bone fragments, (3) the removal of metallic foreign bodies, (4) the type and amount of local chemotherapy used, (5) the absorbable hemostatic agents used, (6) the size of the dural defect and the type of closure, (7) the type of scalp closure, (8) drainage, and (9) complications which had occurred, such as unusual bleeding or involvement of the dural sinuses, ventricular system, paranasal air sinuses, or mastoid.

All these data could be included in a short paragraph. A mimeographed diagram of the head and brain on which the operative findings could be sketched quickly was used by some surgeons, and it was found very useful not only by them but also by others who treated the patient subsequently.

In addition to the operative note, it was important that records made to accompany the patient on transfer include the following data: Positive neurologic findings and the patient's general condition before operation, complications of the postoperative period, changes in the neurologic picture, total amounts of chemotherapy and other medications, additional operative procedures, and transfusions.

Recording this pertinent information was an integral part of the care of the patient; it was found to be especially important in highly mobile warfare, when there were frequent and unforeseen transfers from one hospital to another.

FIRST AID TREATMENT

Initial treatment was usually given by battalion aidmen either on the field or at a nearby battalion aid station. No reliable figures are available for the time interval between wounding and first aid treatment. It naturally varied considerably with the character of the fighting but was usually prompt and was often provided almost immediately after wounding.

Early in the war, first aid treatment consisted of dusting powdered sulfanilamide into the wound [6] followed by the application of a firm, dry, sterile dressing. Occasionally, hair around the wound was clipped or shaved in first aid stations, but this was not the rule. Conscious patients often were given a dose of a sulfonamide by mouth. Later in the war, penicillin therapy was begun in the battalion aid station or clearing station, and most casualties, when they arrived at field or evacuation hospitals, had already had 20,000 to 40,000 units intramuscularly.

It was the general policy not to give morphine sulfate to patients with head injuries on the field and in aid stations, though the rule was occasionally violated when the patient was in extreme delirium, was agitated, or had painful injuries elsewhere.

The maintenance of an adequate airway in unconscious patients throughout first aid treatment and transportation was of vital importance.

PREOPERATIVE MEASURES

The Management of Shock

Traumatic shock was treated by the intravenous administration of plasma on the field or in aid stations when it was thought necessary to support the patient for evacuation to a hospital. Upon his arrival at an evacuation or other fixed hospital, the treatment of shock was given preference over all other therapy except the maintenance of an adequate airway. Intensive therapy for patients with only craniocerebral injuries was seldom necessary. Usually, they responded quickly to the removal of wet clothing, warmth, sedation, rest, and one or two transfusions of plasma or whole blood. Oxygen was administered by intranasal catheter or Boothby-Lovelace-Bulbulian mask, and small doses of caffeine were often used to advantage.

A more serious problem was encountered in patients with craniocerebral injuries who also had thoracic or abdominal wounds, or extensive compound wounds of the extremities with excessive pain and loss of blood. In these patients, shock was likely to be profound, and rapid infusion of large amounts of whole blood was often a lifesaving measure. Blood was not withheld when it was needed, though it was thought at times to be dangerous in that it would increase cerebral edema.

Concentrated plasma (2 units of plasma made up with 1 unit of distilled water) was frequently used for comatose patients in shock. Physiologic or other salt solutions were not administered intravenously. Fifty percent dextrose in amounts of 50 to 100 cc. occasionally was given, with good results, to patients with depressed or irregular respiration who were receiving shock therapy and awaiting operation.

It should be emphasized that excessive manipulations for neurologic examination, roentgenographic examination, shaving the head, and all other proce-

[6] WD TB MED 147, March 1945

dures which did not contribute to the patient's immediate welfare were delayed until shock was alleviated.

Neurologic Examination

An attempt was made to carry out a brief neurologic examination on every patient with an injury of the head as soon after his admission to the hospital as his general condition warranted. The policy was never to omit this examination before operation. In periods of heavy fighting, and under the stress of long hours of operating with insufficient rest, this often seemed difficult to the neurosurgeon, but an examination, thorough enough to plan proper surgical treatment, could usually be carried out in less than 5 minutes, with no more equipment than a flashlight, a reflex hammer, and a pin. The practice of seeing a patient with a craniocerebral injury for the first time after he was on the operating table, with his head shaved and a compound wound presenting for debridement, was strongly condemned. The surgeon who permitted such shortcuts did not do justice to his patient or himself.

Observations of the following points were found to be the most significant in estimating intracranial damage and in planning the time and type of operative treatment:

State of consciousness.—If the patient could understand and answer questions readily, no further tests were needed. If he failed to respond, the simplest measure of his level of consciousness was his response to painful stimuli, which was tested by pinprick, by pulling hair on the trunk or extremities, or by firm pressure over the supraorbital notch. Serial observations at intervals by the same person were especially valuable. When the depth of coma increased in spite of supportive treatment, it almost invariably meant that intracranial hypertension was increasing from diffuse or intraventricular bleeding or that direct injury to the brain stem or basal ganglia had been severe.

In estimating the level of consciousness, care was taken to note the amount of sedation previously given, as well as the status of the peripheral circulation. Since morphine or barbiturates were occasionally given subcutaneously to patients with head injuries in the forward areas, the Field Medical Record was checked for evidence of this medication as part of the examination.

It was shown repeatedly that when shock was present, morphine or sedatives given by subcutaneous injection were not absorbed and consequently were not immediately effective. It was necessary, therefore, to exercise particular caution in giving additional medication before the efficiency of the circulation had been restored by appropriate shock therapy. In addition, the state of consciousness was sometimes masked by shock itself in the absence of any sedation. A patient in this condition who appeared unresponsive, restless, and apparently comatose was often found alert and cooperative as soon as his circulation had improved.

Respirations.—The rate and character of the respirations constituted one of the most reliable guides to the extent of injury to the central nervous system.

Slow, deep, stertorous breathing and irregular respirations of the Cheyne-Stokes variety indicated a grave prognosis. In the absence of sedation, respirations below 10 to 12 per minute almost invariably meant massive intercranial hemorrhage.

Rapid, shallow breathing and labored respirations were seldom due to lesions of the central nervous system alone. When they were present, there was usually trauma to the chest or encroachment upon the airway itself.

Eye signs.—The presence of fixed, dilated pupils was invariably an ominous sign. A patient who presented this sign when first observed after a penetrating wound of the brain almost never recovered. It did not matter what the other vital signs were; if both pupils were dilated and fixed, the patient almost always succumbed, usually within 48 hours, and whether or not operation was performed.

A unilateral dilated, fixed pupil usually accompanied extensive cerebral damage on the same side, but the patient sometimes recovered. Direct injury to the orbit or to the eye itself often accounted for a fixed pupil or a blind eye.

In conscious patients, gross testing of the visual fields by confrontation proved of great value. Partial or complete homonymous hemianopsia, which was often seen in compound depressed fractures of the occipital region, indicated a high priority for surgical treatment because of the frequency of recovery of lost vision following early operation.

Paralysis.—Although examination of the extremities was occasionally hampered by splinted fractures of the long bones, in almost every instance, satisfactory appraisal of motor function could be obtained. A complete flaccid paralysis of the arm and leg associated with a central type of facial palsy almost always indicated a large loss of substance from the contralateral cerebral hemisphere. Significant early recovery seldom took place in these cases. On the other hand, an early spastic type of hemiparesis associated with a cortical wound usually indicated a much less extensive loss of tissue, or none at all. In these cases, there was a greater likelihood that the dysfunction was due to local contusion or to pressure from hemorrhage, edema, or foreign bodies. This type of hemiparesis often showed early improvement after debridement.

Isolated monoplegias were almost never seen. Spastic paraplegia was seen with wounds of the sagittal sinus and with parasagittal wounds. Spasticity of all four extemities invariably indicated a poor prognosis. It was usually seen only in patients already moribund.

Aphasia was usually associated with a complete flaccid hemiplegia of the right side. All types and degrees of aphasia were seen with various lesions in the left frontotemporal region. When aphasia was partial in the acute stage of injury, or when complete motor aphasia existed with partial hemiplegia or in the absence of any paralysis, it was usually found to improve rapidly after operation.

Reflexes.—In unconscious patients, whose motor function was difficult to appraise, examination of the reflexes was especially valuable. Total areflexia

indicated extremely profound coma and was an ominous sign. With extensive subanachroid hemorrhage, significant findings included stiffness of the neck, generally hyperactive deep-tendon reflexes, and positive Babinski responses bilaterally. Lateralization of pathologic changes not readily apparent from examination of the wound itself was often made possible by comparison of the superficial- and deep-tendon reflexes of the two sides.

Cranial nerve palsy.—Patients with penetrating injuries which directly involved the base of the brain did not often survive long enough to reach a neurosurgeon. Occasionally, fractures radiated from a point of injury to involve the base of the skull. In these instances, cranial nerve palsies were sometimes seen, particularly palsies of the third, sixth, seventh, and eighth nerves. In penetrating wounds or radiating fractures through the mastoid, a peripheral type of seventh-nerve palsy was common and was often accompanied by at least a partial lesion of the eighth nerve.

No attempt was made to examine all the cranial nerves in every case, but when cranial nerve palsies were apparent, the examiner's attention was directed toward possible complicating injuries of the base of the skull.

Summary.—It was the practice that every patient with a penetrating craniocerebral wound should have an early preoperative examination and evaluation, not only of his general status and the nature of his external wound but also of significant neurologic manifestations. Positive observations were noted on the record for comparison with findings at subsequent preoperative and postoperative examinations. Experience proved that these observations should include (1) the state of consciousness, (2) the character of the respirations, (3) the size and activity of the pupils, (4) the nature and severity of paralysis, (5) abnormalities of the reflexes, (6) the presence of cranial nerve palsies, and (7) the character and frequency of convulsions.

Roentgenologic Examination

Second in importance to clinical examination of the patient during preoperative preparation was roentgenologic examination of the skull. In addition to the examination of patients with obvious penetrating wounds, roentgenograms were made on all patients with scalp wounds, no matter how small, and on all patients who were unconscious or who had positive neurologic signs, even if no wound of the head was visible. Trifling scalp wounds were often found to conceal extensive cerebral injury.

Posteroanterior and lateral roentgenograms of the skull were made routinely. Stereoscopic films were not available in the forward areas, though they would have been of great value. Unavoidable factors often prevented pictures of high technical quality. It was usually possible, however, to make satisfactory posteroanterior and lateral films, which were usually sufficient to permit fairly accurate localization of intracranial metallic foreign bodies and to postulate the structures through which the foreign bodies must have passed from the wound of entry.

The visualization of indriven bone fragments provided an even more accurate guide to the location of the cerebral wound track. Often the actual pathway of the missile could be outlined in the film by a trail of bone fragments of various sizes. High-velocity missiles, in striking the rounded skull and dura at various angles, were often deflected in extremely bizarre patterns. Roentgenograms were an invaluable aid to clinical examination in these instances in clarifying the pathologic state before operation. For instance (fig. 4), a metallic foreign body might pass in one direction from the point of entry, or ricochet across the skull, while bone fragments were driven in another.

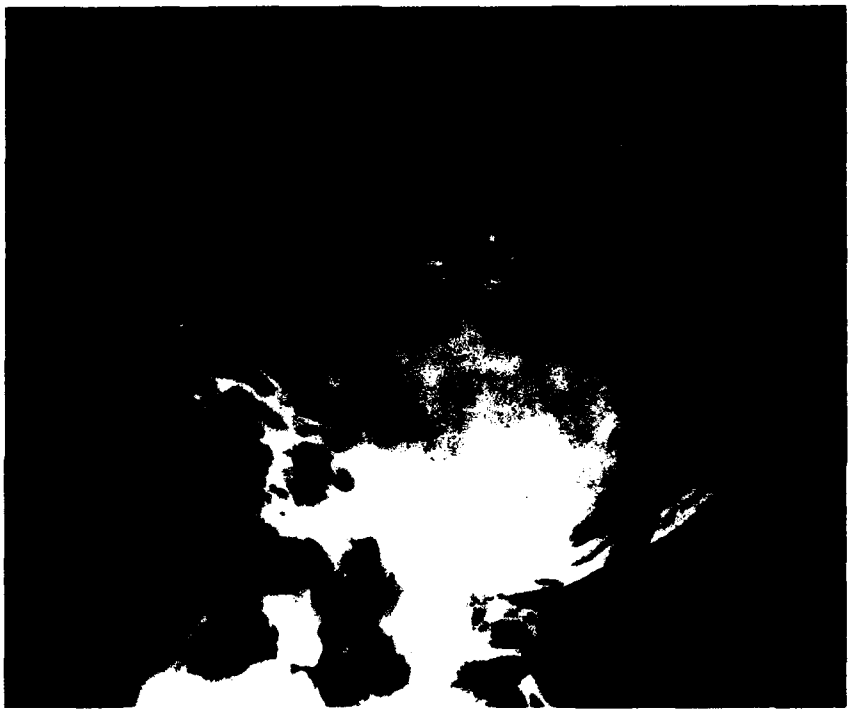


FIGURE 4.—Lateral roentgenogram of penetrating wound of frontal bone. An accurate preoperative estimation of the direction and depth of the wound track in the brain is gained by studying the position of the intracerebral bone fragments.

The actual number of intracerebral bone fragments could frequently be counted (fig. 5). As these fragments were removed at operation, they were saved, counted, and compared with the roentgenograms in the operating room. Obvious discrepancies in the amount of the indriven bone visualized in the roentgenograms and that recovered at operation were thus immediately apparent.

Sometimes the indriven bone was seen in clusters of small comminuted fragments. The individual fragments could not be counted in such a case, but a comparison of the total mass of intracerebral bone removed at operation with that seen in the roentgenograms was of value (fig. 6).

The path taken by a penetrating foreign body was occasionally indicated by the presence of intracerebral air. A small collection of air in the temporal

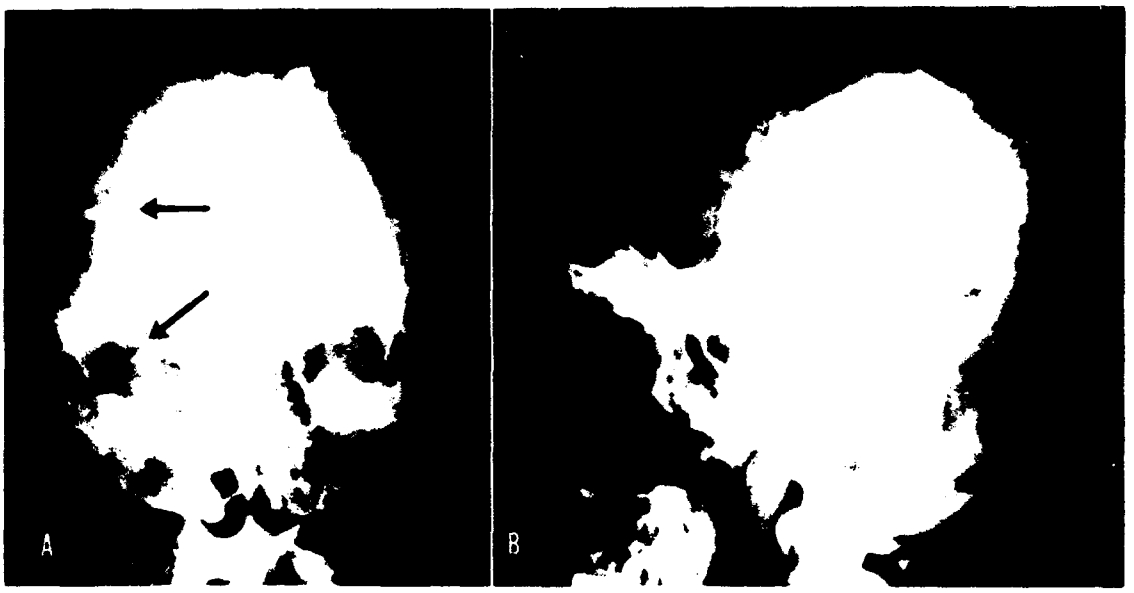


FIGURE 5. Roentgenograms showing penetrating shell-fragment wound of frontal area. A. Anteroposterior view. The lowest arrow points to the wound of entry. The middle arrow points to bone fragments driven into the right frontoparietal region. The upper arrow points to the shell fragment which has crossed the midline to the left parietal cortex. B. Lateral view, showing position of bone and shell fragments.

lobe, for instance, might indicate the path of a missile which actually had entered through the mouth and then passed through the antrum and the floor of the middle fossa.

Because of the importance of good roentgenograms, considerable attention was directed toward making satisfactory films, but no attempt was made to take roentgenograms on patients in shock. The head dressing was examined



FIGURE 6. Roentgenograms showing intracerebral bone fragments. A. Anteroposterior view, showing position of fragments. B. Lateral view. Note that the number of fragments is now seen more clearly.

to be sure no radiopaque materials were present. If the hair contained mud, gravel, or other foreign bodies, they were removed before the films were made. Figure 7 shows the confusing type of picture obtained when dirt and small stones were present in the scalp wound. Whenever possible, films were made with the side of the wound of entrance nearest the film.



FIGURE 7.—Lateral roentgenograms giving false impression of number of intracerebral bone fragments because of mud and gravel in patient's hair and in scalp wound. A. Roentgenogram before head was shaved. B. Roentgenogram repeated after head was completely shaved. Arrow points to simple depressed fracture of vertex.

Roentgenograms taken of irrational, delirious, uncooperative patients were usually of no value. In such patients, the period of effective preoperative medication could be used opportunely to take roentgenograms, with ample time for development and interpretation of the films while preoperative preparations were being completed. If roentgenograms were poor because of faulty technique or other avoidable causes, additional roentgenograms were made whenever possible.

Postoperative roentgenograms in the forward hospitals were indicated only when significant amounts of indriven bone could not be located during the original debridement or when there were convulsive seizures of the Jacksonian type during the first few postoperative days. There were no indications for ventricular air studies in treatment of these acute injuries.

PRIORITY FOR SURGERY

Ideally, it was desirable to operate on every patient with a compound craniocerebral injury as soon as possible. In practice, it was found that a period of stabilization of 1 or 2 hours, and even up to 10 to 12 hours, was usually beneficial, whether or not the patient was in actual shock. Beyond the time taken to improve the patient's general condition, there was no virtue in further delay, and there was sometimes definite hazard.

Unfortunately, there was seldom an even distribution of work in the forward areas. A sudden flood of casualties occurred with every major engage-

ment, and the backlog of surgery mounted quickly. This meant that some system of priority for the available surgical facilities had to be established. To set up an effective system required the most efficient use of the neurosurgeon's skill and experience, in cooperation with the activities of those in charge of shock therapy and other preoperative measures.

In general, any wound which penetrated the dura was considered more serious than a nonpenetrating compound depressed cranial fracture. In rush periods, patients with the latter type of injury were usually evacuated to a medical facility farther to the rear. Before evacuation, an attempt was made to shave the area about the wound, dust sulfanilamide into it (a practice discontinued later in the war), apply a firm dressing, and administer systemic chemotherapy.

Patients found to require the highest priority for surgical intervention included—

1. Those with active bleeding which could not be controlled by application of a firm dressing or by placing a hemostat or ligature on an artery in the scalp. Bleeding from lacerated cortical vessels or dural sinuses usually stopped spontaneously after the formation of a clot under a firm dressing, though occasionally it persisted until operative intervention was carried out.

2. Those with transventricular wounds. These patients deteriorated rapidly, sinking deeper into coma with increasing respiratory failure. This was particularly true if large amounts of spinal fluid were being lost through the wound or if the ventricular system contained old blood and macerated brain. Hyperthermia was very often seen. Haynes [7] observed that patients in this group were usually moribund within 8 to 12 hours after injury. The earliest possible operative intervention, therefore, was thought to offer them their best chance for recovery.

3. Those with large herniations of cerebral tissue. Such herniations were often due to intracranial hypertension secondary to a large hematoma in the depths of the wound of the brain. Patients with this type of injury could be saved only by early operation.

4. Those showing progressive failure of consciousness or increasing neurologic signs.

When two patients had wounds of approximately the same seriousness, the one who had been wounded first was operated on first. Contrary to all previous observations, in World War II the duration of the wound at the time of operation, at least up to 48 to 72 hours, was found to make little difference in the incidence of infection or the case fatality rate. Criteria other than the length of time after wounding were therefore used to determine priority for operation during this period, provided that the patient was receiving adequate supportive therapy and chemotherapy.

Lowest priority for surgical intervention was given to patients with wounds at both extremes of severity. If the patient's general condition was

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[7] Haynes, W. G.: Transventricular Wounds of the Brain. J. Neurosurg. 2: 463–468, November 1945.

good and his neurologic examination negative, simple compound depressed fractures without dural penetration were never considered urgent. At the other extreme, patients who were obviously in extremis, with bilateral dilated, fixed pupils, Cheyne-Stokes respiration, or generalized rigidity were not operated on if other patients with craniocerebral injuries needed the neurosurgeon's attention.

PREPARATION FOR OPERATION

During the entire preoperative period, systemic chemotherapy was continued at regular intervals. Original dressings, which had usually been applied in an aid station or on the field, were apt to be adherent to herniated cerebral tissue. Unless they were removed with the greatest gentleness, clots were pulled away and bleeding was started.

Local preparation.—As soon as possible after the patient's admission to a unit in which definitive treatment could be carried out, his entire head was clipped with ordinary mechanical or electric clippers or was sheared with scissors. Clipping or shearing was often difficult when there were clotted blood, dirt, and macerated cerebral tissue in the hair. In such instances, cutting the major part of the hair off first with scissors was of help. When the hair was matted with dried blood, it was helpful to wash the head first with a mixture of soap and hydrogen peroxide.

After the entire head had been clipped, it was shaved cleanly for a distance of at least 3 inches from all wounds, or, preferably, completely shaved. It was usually necessary to finish this preparation after the patient was on the operating table under a good light. Preparation of the head was often a tedious and time-consuming process, and, insofar as possible, it was carried out with the patient on a litter or separate operating table, in order not to delay the operating team. Shaving about the wound edges was often very painful and could be completed satisfactorily only after premedication or even surgical anesthesia had been administered. For shaving around craniocerebral wounds, the most satisfactory implement proved to be the ordinary safety razor blade held with a straight or curved half-length clamp.

After the scalp was shaved, it was thoroughly washed with warm water and soap. If there was old dried blood about the wound edges, the addition of hydrogen peroxide to the soapy water was advantageous. This mechanical scrubbing with soap and water applied with gauze sponges was considered the most important step in the preparation of the field and was never neglected. The scrubbing was followed by additional cleansing of the scalp with gauze sponges soaked in 70 percent alcohol alternated with sponges soaked in bichloride of mercury. Painting the scalp with 2.5 percent iodine solution or tincture of Merthiolate (thimerosal), followed by alcohol, also constituted a satisfactory preparation. When local anesthesia was used for field block, the skin was washed again with alcohol or repainted with antiseptic solution after the injection.

Positioning on operating table.—Great care was taken in getting the patient into satisfactory position on the operating table, especially if local analgesia was to be used. All clothing was removed, and he was covered with sheets or blankets according to his condition and the environmental temperature. Gentle immobilization was accomplished by the use of a broad piece of canvas across the thighs. The arms were loosely restrained at the sides or on padded armrests. The operating table was usually broken or tilted, so that the head was slightly elevated.

The most satisfactory support for the head proved to be a soft sandbag or a firm rubber-covered pillow about 6 to 10 inches square and 2 to 3 inches in thickness. All lesions in the frontal areas and vertex could be managed easily with the patient lying on his back. If the lesion was in the temporal or parietal region, he was rotated toward the opposite side, and a pillow or sandbag of sufficient size to give adequate support was placed under the shoulder on the side of the lesion. If he was conscious and restless, he was allowed to lift and turn his head slightly from time to time during the operation.

For wounds in the occipital and postparietal areas, the classical cerebellar or face-down position was used. For this purpose, a well-padded horseshoe-shaped headrest was employed, which carried the weight of the head on the forehead and malar regions and allowed the anesthetist free access to the patient's nose and mouth. In this position, especial care was taken to elevate the chest from the operating table by sufficient support under the shoulders to permit free respiratory exchange. A tightly rolled blanket or a narrow, firm pillow or sandbag placed diagonally under each shoulder served admirably for this purpose. When no operating table was available, an elastic-bandage sling fastened to the handles of the standard litter was used to support the forehead.

Draping.—The simplest possible system of draping with sterile towels and sheets was used to exclude completely from the operative field everything except the prepared area of scalp. It was satisfactory to follow the standard civilian practice of using a Mayo stand immediately above and caudad to the operative field, to serve the double function of elevating the drapes from the face and supporting a tray for the most frequently used instruments. A generous area of the scalp was usually exposed in draping for cranial operations, because of the frequent necessity for extending wounds and doing plastic procedures to achieve satisfactory closure.

ANESTHESIA

Three types of anesthesia were commonly used in the primary debridement and repair of acute penetrating brain injuries in World War II: (1) Local infiltration of procaine hydrochloride, combined with various types of preoperative medication; (2) intravenous injection of Pentothal Sodium (thiopental sodium); and (3) intratracheally administered nitrous oxide-

oxygen-ether. Avertin (tribromoethanol) was available to a few surgeons and was used by them to good advantage.

The selection of anesthesia varied with the general condition of the patient, the location and type of his injury, the presence of complicating wounds elsewhere in the body, the anesthetic facilities available, and the individual preference of the surgeon.

While certain problems demanded a specific type of anesthesia, such as intratracheal ether for craniofacial wounds, the great majority of penetrating injuries of the brain could be handled satisfactorily under local anesthesia alone, under local anesthesia plus intravenous thiopental sodium, or under thiopental sodium alone. Patients who were deeply comatose or moribund or whose respirations were depressed or irregular were operated on under local infiltration alone. When there were complicating wounds of the extremities, it was common practice to care for the craniocerebral injury first, under local infiltration, and then put the patient to sleep with thiopental sodium for the other procedures. When penetrating injuries of the chest or abdomen were present with craniocerebral injuries, they naturally took precedence for surgical intervention. Often the injury of the brain could be debrided subsequently under the same anesthesia. If the injury of the brain was extensive or if the patient's condition militated against further prolongation of general anesthesia, the wound of the head was usually operated on 24 to 36 hours later, under local infiltration of procaine hydrochloride.

Local anesthesia.—One percent procaine hydrochloride, to which 3 to 5 drops of 1:1,000 solution of epinephrine per ounce (30 cc.) were added, was used routinely for local infiltration and field block. While it was seldom necessary to use more than 100 cc. of procaine hydrochloride, as much as 150 cc. of 1 percent procaine hydrochloride was often used in extensive wounds without ill effects.

Procaine hydrochloride was best injected in normal tissues, away from, rather than through, contaminated wound edges. Bleeding, particularly from the scalp, was always less when local infiltration was used. A cooperative patient under local anesthesia could sometimes aid in the debridement of his own wound by straining and coughing gently under the surgeon's direction and thus forcing devitalized tissue and debris from the depths of the wound.

When local anesthesia was used, preoperative sedation was always important. Pentobarbital sodium (0.09 to 0.39 gm. by mouth) or Sodium Amytal (amobarbital sodium) (0.39 to 0.58 gm., given subcutaneously 1 hour before operation), together with 11 to 16 mg. of morphine sulfate intravenously at the time the head was being prepared, was ordinarily found to constitute satisfactory preparation for local anesthesia. Paraldehyde, in doses of 12 to 20 cc. by rectum or 4 to 7 cc. intramuscularly, proved an excellent substitute for the barbiturates, especially in overactive, restless patients. Complications from the use of intravenous morphine in patients with open wounds of the head were almost never observed provided that the morphine was not used in the presence of respiratory irregularity or depression. The effect of 16 mg. of intravenous

morphine was invariably prompt and usually lasted from 1½ to 2 hours. If the drug was given just before local procaine hydrochloride infiltration was begun, its effect lasted throughout the average craniocerebral debridement. It was never used in patients with severe closed injuries of the head.

Pentothal Sodium (thiopental sodium) anesthesia.—One of the outstanding advances in the primary operative treatment of all casualties in World War II was the availability of thiopental sodium for intravenous anesthesia; this was true for craniocerebral wounds as well as for other types. Some surgeons used it for as high as 85 percent of their craniocerebral operations. It proved to be an ideal form of anesthesia for restless, overactive, and uncooperative patients, who would not tolerate local anesthesia alone. Complete muscular relaxation was not needed for this type of operation. It was necessary only to keep patients quiet and free from apprehension and pain. When the drug was given slowly and carefully in combination with local infiltration of procaine hydrochloride, it was usually possible to keep patients asleep for 2- to 3-hour procedures with from 1.5 to 2.0 gm. of thiopental sodium. Thiopental sodium was not given in solutions stronger than 2.5 percent to patients with craniocerebral injuries. Induction was slow, and the danger of apnea and laryngospasm was over before surgical manipulations were begun.

For long procedures, an intravenous needle was inserted and kept open by a slow drip of suitable fluid, so that additional thiopental sodium could be given as needed. A skillful anesthetist soon learned to give it just before it was needed. Some surgeons preferred a steady but adjustable drip of from 0.5- to 1-percent thiopental sodium solution. Oxygen, when needed, was most satisfactorily administered through a nasal catheter. Atropine sulfate, from 0.43 to 0.65 mg. subcutaneously, was used routinely before thiopental sodium anesthesia.

Thiopental sodium proved especially valuable for wounds around the ear and mastoid and about the orbit, where local infiltration was difficult. It was also admirably suited for patients with associated injuries which could be repaired at the same time or immediately following the repair of the cranial injury. It was contraindicated in patients with craniocerebral wounds involving the nasopharynx, paranasal sinuses, or the floor of the mouth, in patients with respiratory depression or irregularity, and in deeply comatose patients.

Intratracheal ether anesthesia.—The primary indication for this type of anesthesia was the presence of wounds involving the frontal, ethmoid, sphenoid, or maxillary air sinuses, or the nasopharynx. In these areas, it was essential that a clear airway, as well as adequate relaxation, be maintained throughout long operative procedures. It was particularly important that anoxia be avoided during induction and maintenance of inhalation anesthesia because of its rapid reflection in increased intracranial pressure. Thorough aspiration of the trachea when the tube was first inserted and again before it was removed was very useful; if there was persistent respiratory obstruction, bronchoscopy was occasionally necessary.

CLASSIFICATION OF CRANIAL INJURIES

In World War I, Cushing [8] described nine different categories of head wounds due to missiles. More recently, classification of these wounds has been into three general groups: (1) Lacerations of the scalp alone, (2) compound linear and depressed fractures of the skull without dural penetration, and (3) compound fractures with penetration of the dura and brain. The last-named group of wounds may be usefully subdivided into the gutter type of wound in which depressed bone fragments have been driven into the substance of the brain but no foreign body is present, penetrating wounds in which a missile is retained in the brain, and through-and-through wounds in which a metallic foreign body has passed completely through a portion of the intracranial chamber.

Certain additional complicating injuries could occur in any one of these three categories of penetrating brain wounds, such as involvement of the ventricular system, fractures and wound tracks passing through the paranasal air sinuses or orbit, laceration of the dural venous sinuses, and large intracranial hematomas.

The operative management of wounds of the scalp, skull, dura, and brain will be described in turn. After the general principles for handling each of the tissues involved has been considered, attention will be devoted to the various special types of injuries.

DEBRIDEMENT

Wounds of the Scalp

It was repeatedly shown in World War II that when scalp wounds were debrided early, with adequate preparation and supportive chemotherapy, wide block excision of skin around penetrating wounds was unnecessary. Indeed, as experience increased, it became more and more evident that a minimal excision of skin from the wound margins was the procedure of choice. Surgeons who removed a centimeter or more of skin around penetrating wounds very often found closure impossible without extensive plastic procedures. On the other hand, the surgeon could not allow his plans for closure to influence the thoroughness of the debridement. If there was reasonable doubt about the blood supply of any area of scalp, that area was removed.

All devitalized tabs of skin were removed routinely, together with any dirt, hair, bits of bone, particles of headgear, or other foreign bodies in the wound. Each layer of the scalp was inspected separately. Often the deeper layers were more damaged than the superficial. As narrow a margin as could be excised feasibly with a scalpel or sharp scissors was then trimmed from the wound margins (fig. 8). It was repeatedly observed that extensive loss

[8] See footnote 1 (2), p. 123.

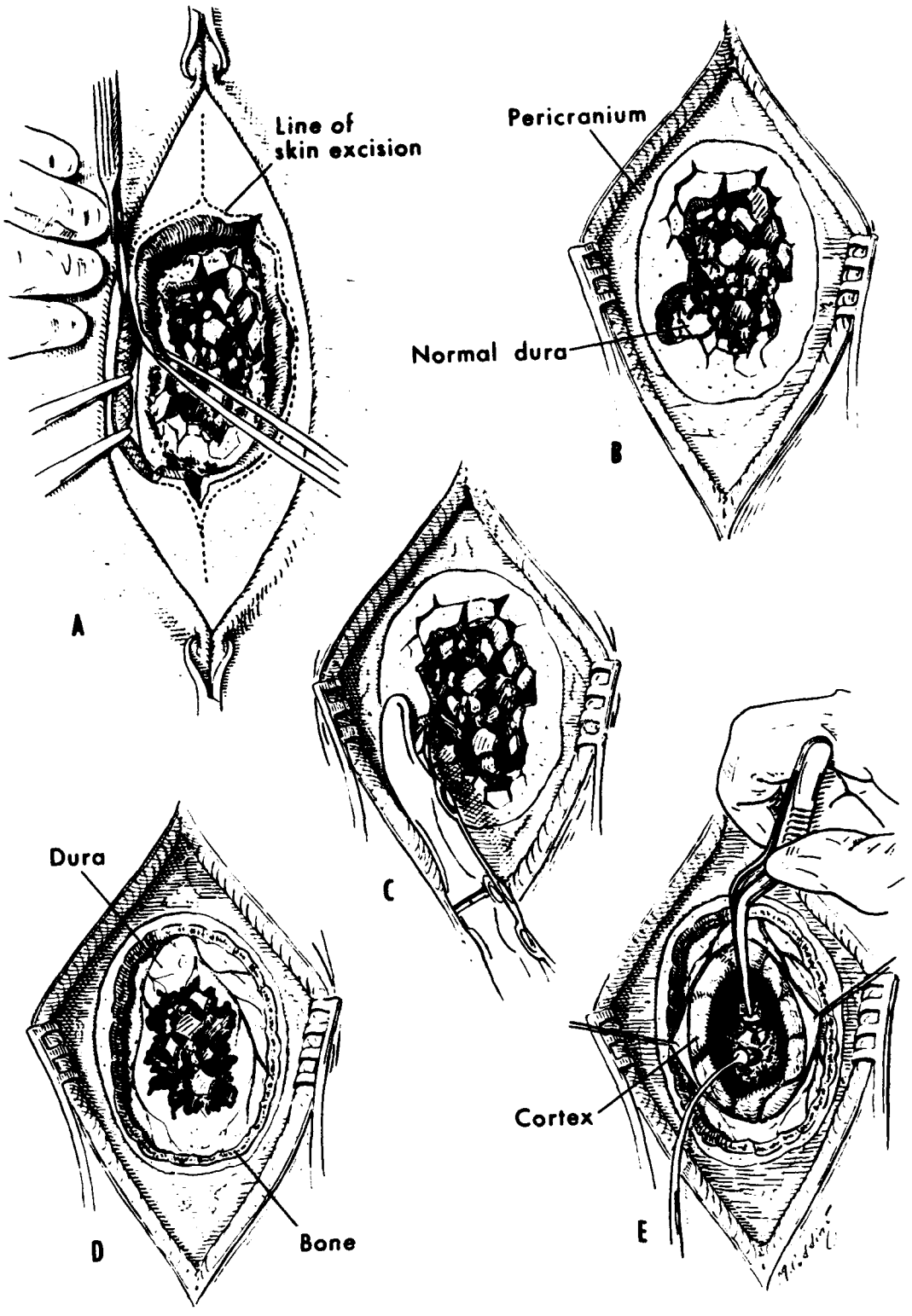


FIGURE 8.—Steps in debridement of scalp, pericranium, skull, and dura.

of blood could occur from the scalp if hemorrhage was not controlled by pressure and retraction of the galea as debridement proceeded.

In contrast to minimal excision of the scalp, wide removal of devitalized muscle in the temporal and occipital regions was carried out.

When debridement of the scalp had been completed, the instruments used for this purpose were discarded. Self-retaining retractors were introduced, or hemostats were applied to the galea to separate the wound margins. If the underlying cranial wound was not adequately exposed, the scalp wound was immediately extended. If it was readily apparent that there had been a loss of skin sufficient to prevent closure, it was often wise to anticipate subsequent plastic procedures at this time.

Wounds of the Skull

Not until debridement of the scalp had been completed and the wound margins walled off was attention directed to the bone (fig. 8). All contaminated bone was removed, whether it lay loose in the external wound or deep within the cerebral substance or remained in continuity with the rest of the cranium. In addition, of course, it was essential to remove sufficient bone to give adequate exposure to an underlying wound of the meninges and cerebrum.

All comminuted fragments were removed, even if they did not appear contaminated, because of the risk of infection plus the unsatisfactory cosmetic result which almost invariably followed attempts to replace them. With modern methods, it usually proved just as easy to perform a large cranioplasty as a small one, and wide removal of bone was therefore regarded as the method of choice.

During World I, block resection of an area of bone surrounding the cranial wound was carried out by drilling four or more burrholes and connecting them with a saw or rongeur of the Montenovesi type. In World War II, this technique was no longer thought necessary or desirable. A more direct approach was found to be quicker, easier, and just as effective.

The method of debridement of the bone varied with the type of wound. If large, loose fragments were present or had been completely driven through the dura, it was usually possible to start removal of bone, with rongeurs, at one margin of the defect. Removal was carried back at this point until a margin of about 1 cm. of normal dura was exposed (fig. 8). Starting at this point, and keeping the instrument always over normal dura, it was possible to rongeur the bone quickly and safely entirely around the defect (fig. 8). This technique was always much easier than nibbling at the edge of the defect all the way around because frequently the dura was damaged and retracted underneath the bony margin. There was also less danger of contaminating new areas, as well as less bleeding associated with debridement, when operation was carried out from normal bone and dura toward the defect.

If the wound in the bone was small, if fragments were wedged tightly in the defect, if there was a comminuted depressed fracture with no exposure of

the dura, or if the depressed fracture was over a dural sinus, debridement was carried out as follows:

A burrhole was made adjacent to the wound of the bone through an area of undamaged bone, to expose normal dura. Starting at this point, the surgeon rongeured toward the defect and around its margin, again keeping the instrument over normal dura. In this way, the danger of starting bleeding from an unexposed site in the dura or brain wound was greatly decreased.

In removal of bone, care was taken to preserve the pericranium over undamaged areas as far as possible, since it was often needed for subsequent repair of a dural defect. When flaps of scalp were swung, it was especially important to leave the pericranium intact as a bed on which to place a free-skin graft.

Control of bleeding from the bone was no particular problem as there was usually no increase in vascularity. Bone wax was used for diploic hemorrhage. Thin strips of fibrin foam were occasionally of value in controlling persistent ooze from underneath a bony margin. Sutures placed from the dura to the galea were sometimes used to hold the dura tightly to the bony margin for the same purpose.

Wounds of the Dura

Extensive excision of dura around a traumatic defect was neither necessary nor desirable. The entire dural defect was exposed, after which, under direct vision, the edges were carefully examined. Only obviously necrotic tags and badly shredded areas were removed. If there was visible gross contamination of dural margins which could not be readily wiped or washed away, the involved area was excised as frugally as possible. Often, no dura at all was removed. The importance of tight dural closure was considered to be so great that considerable attention was devoted to the preservation of the dural edge as the debridement proceeded. The dural opening was never enlarged unless it was necessary to control cortical bleeding or remove a large superficial foreign body.

Wounds of the Brain

Special techniques.—Adequate debridement of penetrating injuries of the brain followed the general principles of wound debridement elsewhere in the body. It was necessary, however, to make use of some of the specialized methods and equipment of the neurosurgeon in order to carry it out with thoroughness and facility. These specialized methods had to do principally with the control of bleeding in the central nervous system and with the protection of normal cerebral tissue. The necessary special equipment has already been described.

No attempt was made to debride a wound track in the brain until adequate exposure had been obtained. Debridement of the scalp, bone, and dura was completed before attention was directed to the wound of the brain proper. This procedure not only gave the best possible exposure but also minimized the danger of introducing new contamination into the depths of the wound.

Wherever possible, each step in the debridement of the brain wound was carried out under direct vision. Upon completion of dural debridement, it was frequently advantageous to place one or more traction sutures on each margin of the dural defect to facilitate the exposure (fig. 8). Debridement

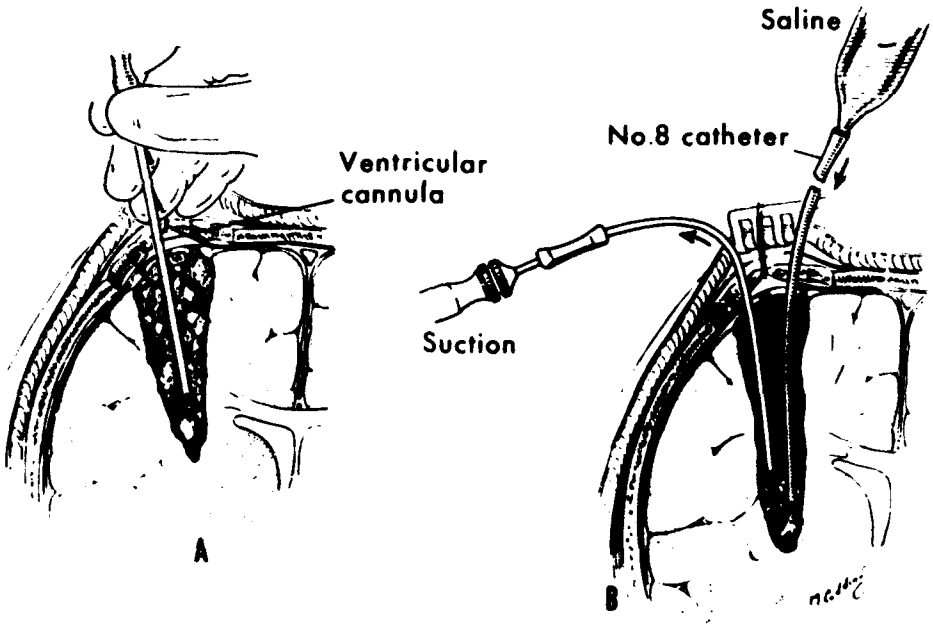


FIGURE 9.—Debridement of cerebral wound. A. Gentle probing in entrance wound with catheter or ventricular cannula to help confirm direction and depth of track before starting debridement. B. Removal of macerated cerebral tissue and clotted blood by irrigation and suction.

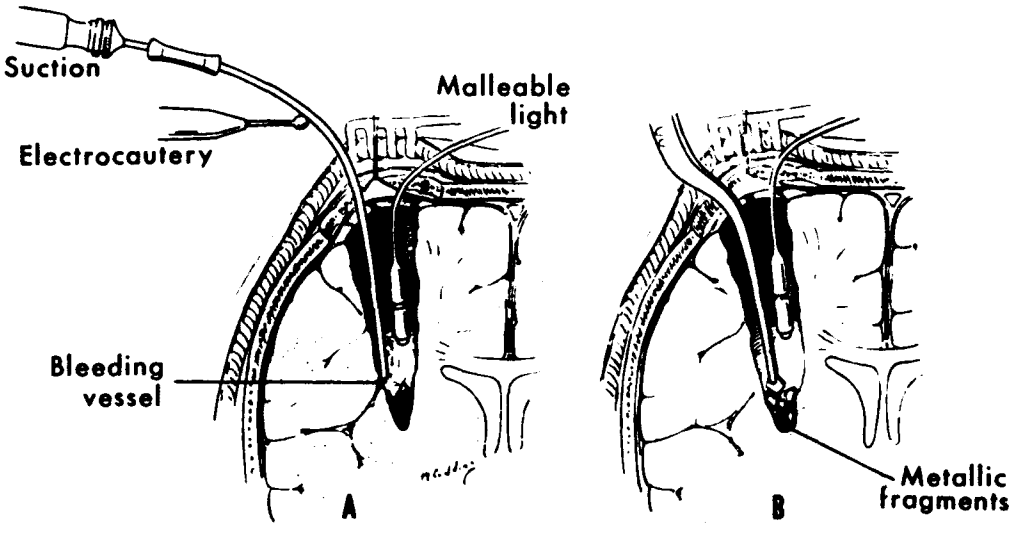


FIGURE 10.—Debridement of cerebral wound. A. Hemorrhage in depths of wound controlled by application of electrocautery to metallic suction tip held against bleeding vessel. B. In deep, narrow wound tracks, too small to permit use of ordinary retractors, a small malleable light is inserted to permit suction under vision.

was then continued as a combination of various maneuvers (figs. 9, 10, and 11), depending upon the characteristics of the particular brain wound.

Before starting debridement of the brain wound proper, the roentgenograms were carefully reviewed to note the position of any indriven bone fragments and metallic foreign bodies in relation to the position of the wound of entrance. Gentle probing in the entrance wound, with a catheter or ventricular cannula (fig. 9A), then helped to confirm the direction and depth of the track before debridement was started.

Most of the removal of macerated cerebral tissue and clotted blood was accomplished by judicious use of suction (fig. 9B). Fragments of bone, hair,

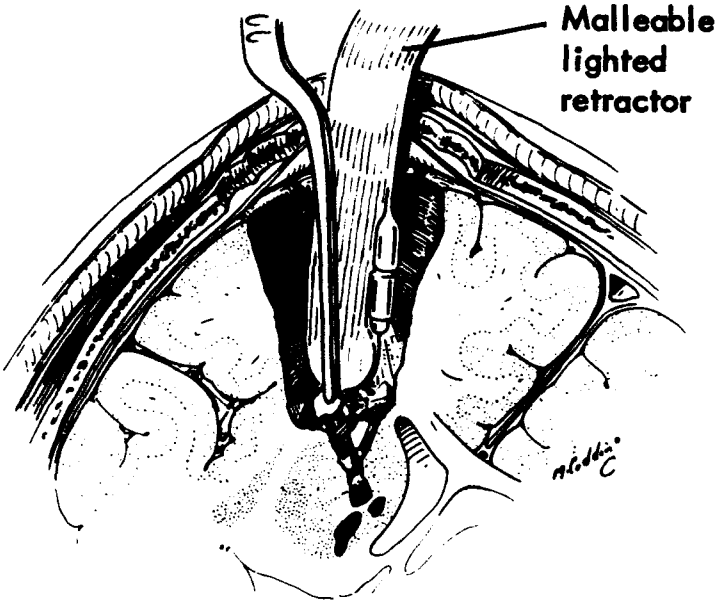


FIGURE 11.—Debridement of cerebral wound. Bone chips and metallic foreign bodies are removed with forceps, under direct vision, with the aid of a malleable lighted retractor.

clothing, and headgear, and the missiles themselves, were removed gently with forceps as they were exposed. Since suction should never be employed except when the point of the sucker is under direct vision, this technique was not used in the depths of a narrow track unless a lighted retractor or small malleable light could also be introduced or the depths of the track could be illuminated adequately by overhead light.

Gentle irrigation of the brain wound with warm physiologic saline solution was often of considerable aid in the removal of macerated tissue (fig. 9B). Irrigation and suction were used simultaneously. The surgeon had to be careful never to force saline solution into the depths of a narrow wound, because this might disseminate contaminated material into uninvolved tissues. Ascroft [9]

[9] Ascroft, P. B.: Treatment of Head Wounds Due to Missiles: Analysis of 500 Cases. Lancet 2: 211-218, 21 Aug. 1943.

emphasized the dangers of irrigation in deep wounds, and Munro [10] expressed the same opinion in discussing the care of compound craniocerebral wounds in civilian practice. Careful irrigation of the walls and floor of the wound track under direct vision proved, however, a safe and useful procedure.

As debridement proceeded, an endeavor was made to keep bleeding under control at all times, both to insure the best visibility and to prevent unnecessary loss of blood. Many cortical vessels which had thrombosed were necessarily reopened during debridement. They were coagulated with the electrosurgical unit (fig. 10). Silver clips were applied to larger vessels. Generalized oozing could usually be controlled with warm, moist cotton packs, with fibrin foam, or with muscle stamps. The troublesome deep bleeding which was occasionally encountered around the falx or in the distant surfaces of a hemisphere was controlled by whatever technique was most appropriate.

Fragments of bone driven into cerebral tissue were extracted with extreme caution and always under direct vision if this was at all possible (fig. 11). Bone fragments were extracted along the same path by which they had entered, to avoid damage to normal brain.

Management of foreign bodies and bone fragments.—The primary aim in debridement of a wound of the brain was always the removal of all bone fragments and other organic foreign material, as well as aspiration of macerated cerebral tissue and old blood. When deep infection was discovered at the general hospital level, it was almost invariably found to be associated with retained bone fragments. Campbell [11] discussed this problem in a report based upon 100 cases of compound comminuted skull fractures produced by missiles, in 94 of which there were indriven bone fragments. There were 20 deep infections in 49 cases in which only part or none of the fragments were removed, while in 45 cases in which thorough debridement was done, there were only 2 deep infections. Rowe and Turner [12] reported from a neurosurgical center in the communications zone that brain abscess developed within 6 weeks in 25 percent of their patients with retained bone fragments.

It was the general policy to remove metallic foreign bodies at debridement whenever this was possible without damage to normal tissue. If, however, removal meant an approach through uninjured tissue or necessitated an exposure considerably greater than that necessary for debridement of the wound, the metallic foreign body was left in place. It was removed later if it was very large or near the surface or if it was giving rise to any signs or symptoms.

The chief reason the metallic foreign body was exposed, if this was at all feasible, was to permit the exposure and removal of small bone fragments and other contaminated material that might have been carried all the way in ahead of it. When the metal had penetrated considerably deeper than the

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[10] Munro, Donald: Cranio-Cerebral Injuries, Their Diagnosis and Treatment. New York, Toronto, and London: Oxford University Press, 1938.

[11] Campbell, Eldridge H., Jr.: Compound, Comminuted Skull Fractures Produced by Missiles; Report Based Upon 100 Cases. Ann. Surg. 122: 375–397, September 1945.

[12] Rowe, S. N., and Turner, O. A.: Observations on Infection in Penetrating Wounds of the Head. J. Neurosurg. 2: 391–401, September 1945.

bone fragments, the track usually had sealed behind it. The infrequency of infection around unremoved metallic foreign bodies suggests that any contaminated material carried in on the foreign body must have been sterilized by the heat of the missile.

In very narrow, deep tracks, it was not always possible to visualize the depths of the wound well enough, even with a narrow malleable retractor, to remove bone fragments under direct vision. Under these circumstances, fragments could often be located by gentle probing in the track with a rubber catheter or with a blunt ventricular cannula. When the fragments were located, an attempt was made to visualize them and pick them out with forceps. When the point of entrance of a wound track in the brain was wide enough, gentle palpation of the walls and depths of the track with the gloved index finger was of great aid in locating small fragments of bone. The finger was never forced into a wound and was never used for dissection but was useful in cautious palpation of the base of wide, shallow wounds.

No attempt was made to do block excisions of wound tracks by electrosurgical dissection, as had been suggested in the early days of the war.[13] This practice proved unnecessary and impractical. Instead, debridement was carried out by keeping within the wound track and removing loose, necrotic, and macerated tissue in all directions until normal cerebral tissue was reached. When bleeding was well controlled and visibility good, the margins of the wound track were usually easy to determine.

LOCAL CHEMOTHERAPY AND ANTIBIOTIC THERAPY

There is no question about the important part played by chemotherapy and antibiotic therapy in the reduction of the morbidity and mortality rates in craniocerebral injuries in World War II. Controlled observations of the importance of chemotherapy are difficult because no large group of patients was treated without it. Several reports, however, are available which compare the incidence of infection in patients treated with sulfonamides alone in early campaigns and in patients treated with sulfonamides and penicillin in later campaigns. In Martin and Campbell's [14] series, the respective rates of infection were 23 and 13 percent. In Haynes' [15] series, they were 21.1 and 6.8 percent. In Slemon's [16] experience in the British Army, they were 31.2 and 9.2 percent. In evaluating the incidence of infection, it should be remembered that the period when penicillin and the sulfonamides were used jointly was also a period during which surgical experience in the management of war wounds had materially increased.

Though some form of local chemotherapy was almost universally employed, individual surgeons applied it by many different methods. A common method

[13] Horrax, G.: A Proposal for the More Radical Treatment of Gunshot Wounds of the Brain. Canad. M. A. J. 43: 320–324, October 1940.

[14] Martin, J., and Campbell, E. H., Jr.: Early Complications Following Penetrating Wounds of the Skull. J. Neurosurg. 3: 58–73, January 1946.

[15] Haynes, W. G.: Penetrating Brain Wounds; Analysis of 342 Cases. J. Neurosurg. 2: 365–378, September 1945.

[16] Slemon, H. V.: Forward Neurosurgery in Italy. J. Neurosurg. 2: 332–339, July 1945.

was to dust all layers of the craniocerebral wound with a powder made by adding 1 gm. of sulfanilamide to each 5,000 units of penicillin. Walker and his coworkers [17] found that penicillin introduced subcortically was a convulsive agent but that this was true only when the quantities used were much larger than required for therapeutic purposes. As much as 20,000 units of penicillin was used repeatedly by many surgeons in various types of acute cerebral war wounds without clinical convulsive manifestations.

In deep, narrow tracks, which could not be dusted satisfactorily with a powder, 10,000 units of liquid penicillin diluted with saline solution was usually instilled before closure of the dura. In the United States Army, sulfathiazole was not used in wounds of the brain, though the British reported its local use without the convulsive complications noted earlier in the laboratory and in civilian practice.[18] In injuries near the motor cortex, a maximum of 10,000 to 15,000 units was not exceeded.

Munslow [19] reported no difference in the incidence of infection when penicillin was used locally in the Mediterranean theather in the later campaigns of the war. In a well-debrided wound, there seemed to be little indication for local application of a sulfonamide, provided that penicillin had been used and that adequate systemic chemotherapy was maintained. No amount of local or systemic chemotherapy was regarded as a substitute for careful, complete, surgical debridement.

REPAIR OF THE DURA

In the past, there had been no general agreement about closure of the dura at the time of primary debridement. Early in World War II, British neurosurgeons thought it should never be closed but should be left open for drainage of contaminated material.[20] With added experience, however, dural closure became the procedure of choice, especially among United States Army neurosurgeons.

The dura is the most effective natural barrier to the entrance of infection to the brain from outside. Cairns [21] stated that in the majority of brain wounds, infection developed in the superficial layers and spread to the deep layers. The dura is also the most effective barrier against cerebral herniation and escape of spinal fluid. In addition, a smooth, well-healed dura is the best prophylaxis against the formation of an adherent cerebral scar at the point of injury.

A tight dural closure was therefore thought to be of paramount importance in the management of every acute craniocerebral wound, and considerable

[17] (1) Walker, A. E., and Johnson, H. C.: Convulsive Factor in Commercial Penicillin. Arch. Surg. 50: 69–73, February 1945. (2) Walker, A. E., Johnson, H. C., and Kollros, J. J.: Penicillin Convulsions; Convulsive Effects of Penicillin Applied to Cerebral Cortex of Monkey and Man. Surg., Gynec. & Obst. 81: 692–701, December 1945.

[18] (1) See footnote 9, p. 145. (2) Ascroft, P. B.: Neurosurgical Experience in Egypt and Libya. Arch. Neurol. & Psychiat. 21: 295–296, March 1944. (3) Eden, K. C.: Mobile Neurosurgery in Warfare; Experiences in the Eighth Army's Campaign in Cyrenaica, Tripolitania, and Tunisia. Lancet 2: 689–692, 4 Dec. 1943.

[19] Munslow, R. A.: Penetrating Head Wounds; Experiences From Italian Campaign. Ann. Surg. 123: 180–189, February 1946.

[20] See footnote 18 (2) and (3).

[21] Cairns, H.: Penicillin in Head and Spinal Wounds. Brit. J. Surg. 32: 199–207, July 1944.

time was often devoted to this part of the procedure. Whenever possible, the margins of the dural laceration were brought together directly with fine silk sutures. When this could not be accomplished, as it could not be in well over half of the dural penetrations which form the background of these observations, some form of graft was employed to fill in the defect. Partial closure of large dural defects by direct suture was frequently possible, leaving a residual gap that could be closed by a relatively small graft. Often the dura was found split for some distance beyond a defect, without actual loss of substance. These splits needed only cleansing and direct suture.

After debridement, it was infrequent to have a dural defect larger than 4 to 5 cm. in diameter which required grafting. The reason was that the greater the loss of dura, the larger, as a rule, was the loss of the underlying cortical tissue. Consequently, by the time debridement of the brain wound had been completed, there was likely to be enough relaxation of the dura to bridge the defect, at least partially. This was especially true in long, ragged dural lacerations associated with gutter or tangential wounds. On the other hand, when a missile had penetrated the brain more nearly at right angles to the surface, there was apt to be a punched-out defect of the dura, with a deep and narrow wound of the brain. There was no relaxation of dura in this type of wound after debridement, and a graft was therefore necessary.

The dura was repaired with free grafts of temporal or occipital fascia, with fascia lata taken from the lateral aspect of the thigh, with free grafts of pericranium, or with a pedicle flap of pericranium rotated from adjacent bone (fig. 12). In general, pericranium used in the form of a free graft was found to be the most satisfactory material available. By undermining and elevating the galea from the pericranium, a large patch could almost always be obtained, without further enlargement of the scalp wound, from bone adjacent to the defect. Measurable contraction was usually found to have occurred after separation of the pericranial patch from the bone. A generous patch of pericranium was therefore excised, usually one-third to one-half as large again as the dural defect to be repaired. The pericranial graft was held in place with multiple fine silk sutures or, occasionally, with silver clips.

In some instances, it was possible to use a pedicled pericranial graft, as recommended by Ecker.[22] The advantage of this method presumably was in the maintenance of a more adequate blood supply to the graft. By this method (*fig. 12 A and B*), *a pedicle of pericranium* was outlined, so that a wide base was left attached to bone and the graft could be rotated and sutured to the margins of the dural defect.

Pericranium was usually more readily available than fascia and also was nearer the thickness of dura. Fascia lata was used by some surgeons only in large dural defects on the undersurface of the frontal lobes, in which communication into the nose and the paranasal sinuses was present. Here it was

[22] Ecker, A. D.: Tight Dural Closure With Pedicled Graft in Wounds of the Brain. J. Neurosurg. 2: 384-390, September 1945.

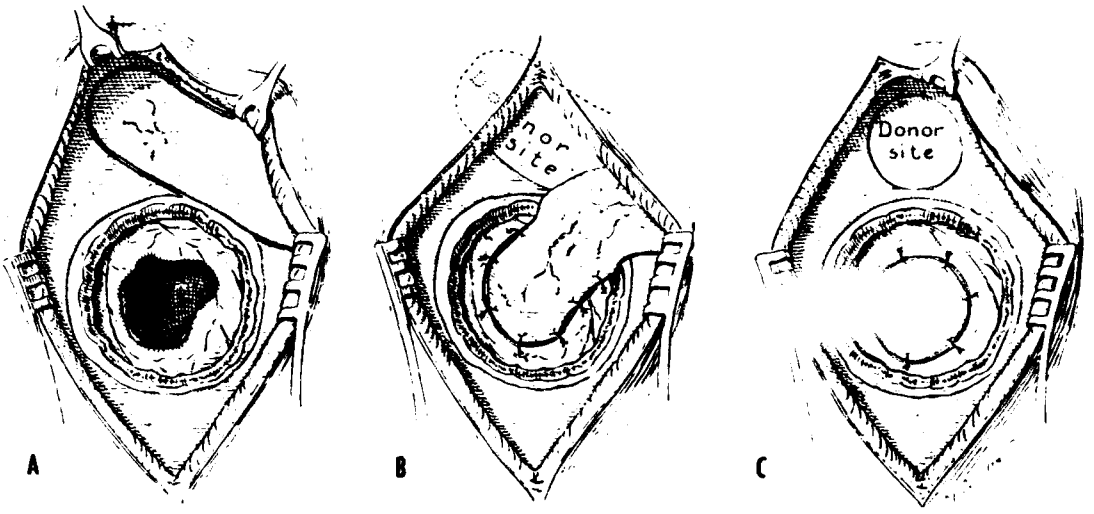


FIGURE 12.—Techniques of repair of dural defect. A. The scalp on one side of the debrided wound has been undermined by separating the galea from the pericranium, and a pedicle flap of pericranium has been outlined. B. The flap has been rotated and sutured tightly to the margins of the dural defect. C. A free graft of pericranium has been taken from adjacent bone and sutured over the dural defect. Free grafts of temporal fascia or fascia lata are used in the same manner.

considered desirable to achieve as watertight a closure of the dura as possible and also to give some support to the brain in the absence of the underlying bony structures. Shearburn and Mulford [23] used preserved dural grafts in 56 cases but subsequently abandoned this method.

Fibrin film was not available in the forward areas during the major campaigns, and its use in acute penetrating wounds due to missiles therefore did not receive an extensive trial. It should be valuable, however, as a dural substitute in these injuries, especially underneath a pericranial or fascial graft, for the prevention of adherent cicatrices. Ingraham, Bailey, and Cobb [24] recommended its use in uninfected wounds when the dura could not be closed completely.

REPAIR OF THE SCALP

Scalp wounds were extended, relaxing incisions made, and skin flaps rotated or advanced in such a manner as to effect tight closure of wounds without tension. The most universally applicable and satisfactory type of incision was a generous S-shaped one (fig. 13). A curvilinear incision was made in one direction from one end of the long axis of the wound, and a similar curvilinear incision was made in the opposite direction from the other end. Undermining of the scalp was always carried out in the areolar layer since the galea, with skin firmly attached, is freely movable over the pericranium in this layer. Extensive mobilization of the scalp for two-layer closure in a

[23] Shearburn, E. W., and Mulford, E. H.: Penetrating Wounds of the Brain; Management in Evacuation Hospital. Am. J. Surg. 72: 441–447, September 1946.

[24] Ingraham, F. D., Bailey, O. T., and Cobb, C. A., Jr.: The Use of Fibrin Film as a Dural Substitute and in Prevention of Meningocerebral Adhesions; Further Studies and Clinical Results. J. A. M. A. 128: 1088–1091, 11 Aug. 1945.

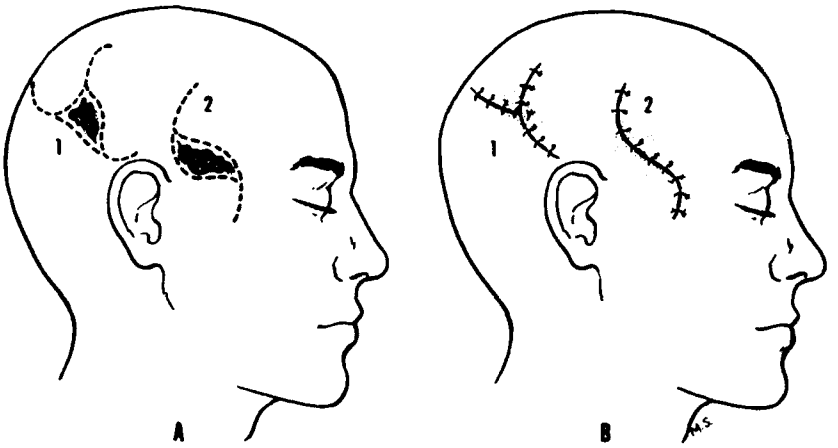


FIGURE 13.—Techniques of repair of scalp. A1. Tripod incision outlined around triangular-shaped wound; all arms of the incision are curved in the same direction. A2. S-shaped incision outlined around transverse wound in temporal area. B1. Closure of tripod incision leaves weak point at junction of the three arms directly over the bony defect. B2. After closure, the S-shaped incision assumes a gentle curve without tension.

gently curved line then became possible under minimum tension and with minimum distortion of the blood supply. Often a simple, elliptical incision with linear extensions was sufficient.

Surgeons in World War II generally agreed that the tripod and "Isle of Man" incisions recommended after World War I [25] should be avoided whenever possible. If they were used, the arms of the incision were always curved rather than straight and were all curved in the same direction (fig. 13). If it was necessary to make an extension away from the long axis of a wound, it was found wise to make it as nearly as possible at a right angle to the line of closure of the wound (fig. 14). Sharp points of skin were avoided, especially when the base of these points was away from the source of blood supply.

Relaxing incisions (fig. 15) were seldom necessary for closure of scalp wounds and were avoided whenever possible. It was almost always preferable to elevate a flap of scalp and slide or rotate it across a defect rather than to attempt to relax one or both margins of the defect and approximate them directly over the underlying wound. When relaxing incisions were used, it was important that they should not be placed across the normal blood supply, since almost certain ischemia of the strip between the wound margin and the relaxing incision would follow. A relaxing incision was never sutured; it ceased to be a relaxing incision if it was closed.

The method of choice for closure of large scalp defects proved to be the use of the sliding or rotation flap (figs. 16, 17, 18, 19, and 20). It was important that the base of the flap always be directed toward the blood supply and that it be curved gently to avoid leaving sharp points at the angles. The

[25] See footnote 1 (1) and (3), p. 123.

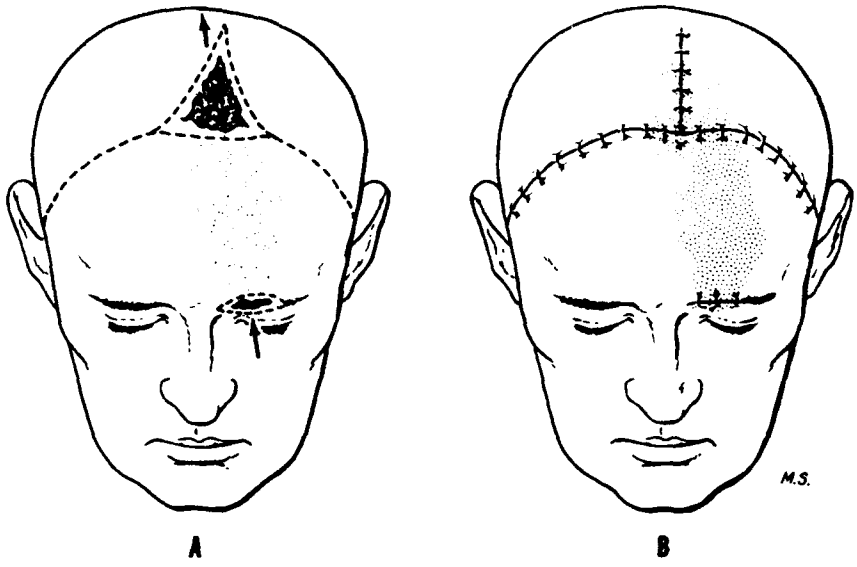


FIGURE 14.—Techniques of repair of scalp. A. Through-and-through wound with entrance in left orbital rim and exit at vertex. Coronal incision is outlined with posterior extension around exit wound. B. The entrance wound is closed separately and the coronal flap reapproximated. Note that the posterior extension is closed at right angles to the coronal incision.

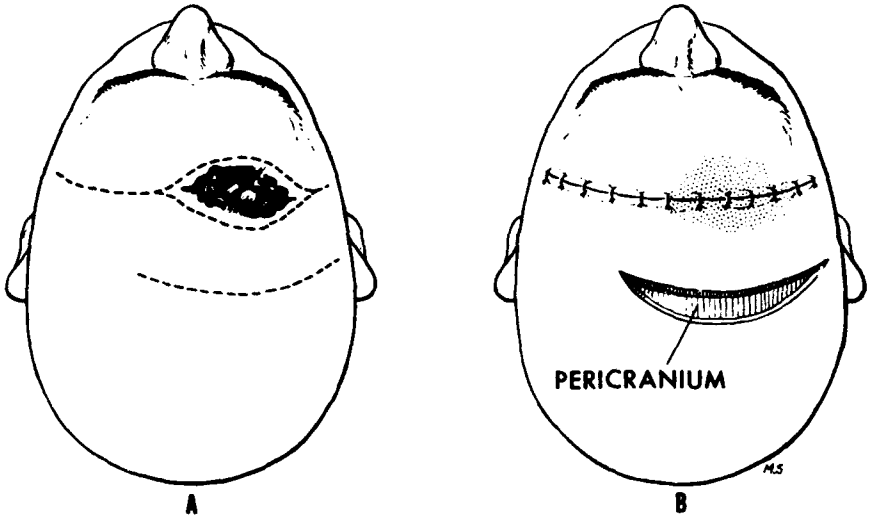


FIGURE 15.—Techniques of repair of scalp. A. Large frontal wound at hairline outlined by coronal incision. A relaxing incision is outlined parallel to the line of closure. B. The scalp is advanced to effect closure of the wound defect. Note that the relaxing incision is considerably longer than the scalp wound itself and is not resutured.

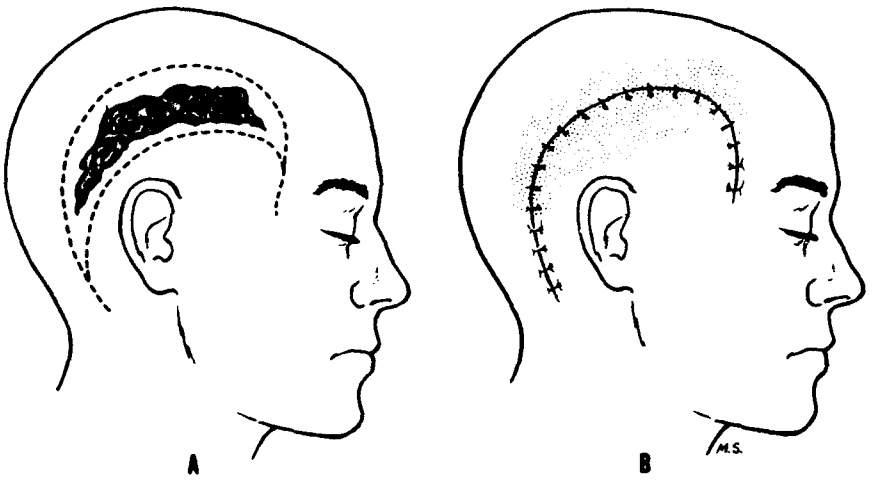


FIGURE 16.—Technique of repair of scalp. A. Large transverse wound in parietal region outlined by sliding flap with its base toward blood supply. B. Smooth curved wound closure without tension after advancement of scalp flap.

flap was made large enough to swing across the defect and permit a two-layer closure without tension; it was usually about three times the size of the defect to be covered. When it was necessary to swing a flap the base of which was at an angle to the blood supply, as in transverse wounds with large loss of tissue just above the ear, the base was made particularly wide (fig. 19).

Occasionally, sufficient mobilization of scalp was possible to permit primary closure of the donor site of a flap. If not, a split-thickness graft was laid directly on the exposed pericranium or fascia (fig. 19). It was useful to

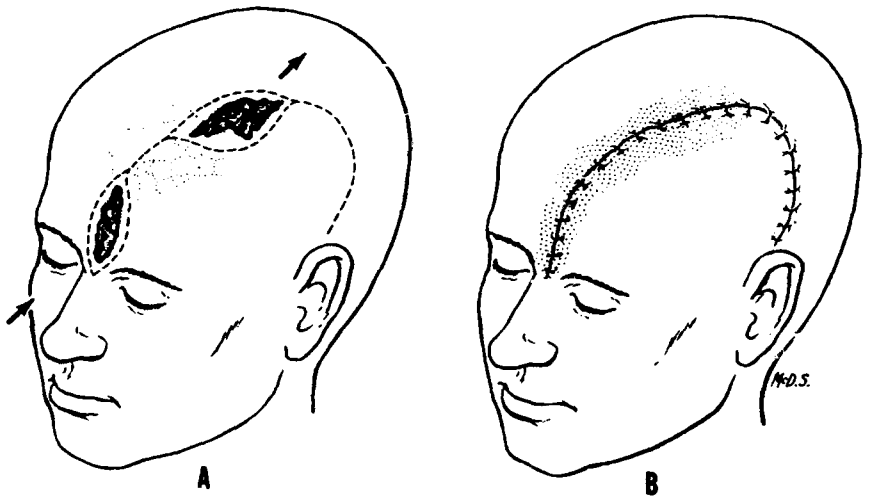


FIGURE 17.—Technique of repair of scalp. A. Wounds of entrance and exit in perforating wound connected and extended to outline large frontoparietal-temporal scalp flap. B. Flap advanced upward and forward to cover huge bony defect. Wide undermining of the scalp has thus been made possible, and primary closure is possible without tension.

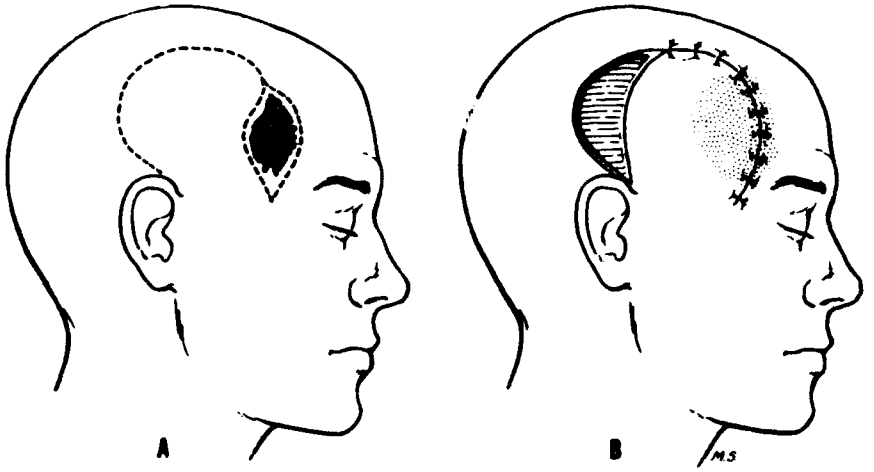


FIGURE 18.—Technique of repair of scalp. A. Generous temporoparietal scalp flap outlined for closure of frontal defect. B. Flap rotated forward, leaving scalp defect to be grafted well posterior to underlying craniocerebral wound.

have another surgeon cut the graft from an accessible donor site, such as the thigh, while the debridement was in progress. The graft could then be sutured into position while the repair was being completed without unduly prolonging the operative procedure. If immediate grafting was impossible or if it was undesirable because of the patient's general condition, it could be done at a later date. Grafts were never placed directly over debrided wounds at the time of primary operation if it was possible to swing full-thickness grafts of uninjured scalp over the defect and then graft the donor site. In very extensive

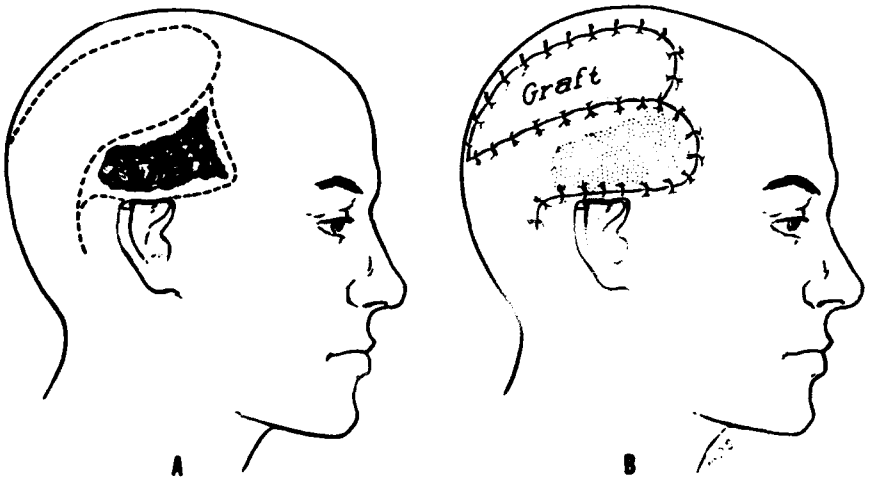


FIGURE 19.—Technique of repair of scalp. A. Low transverse wound in mastoid area with avulsion of upper part of ear. A pedicle flap with a wide occipital base is outlined. B. Flap rotated and sutured to margins of scalp defect including posterior surface of external ear. The donor site has been covered with a primary split-thickness graft.

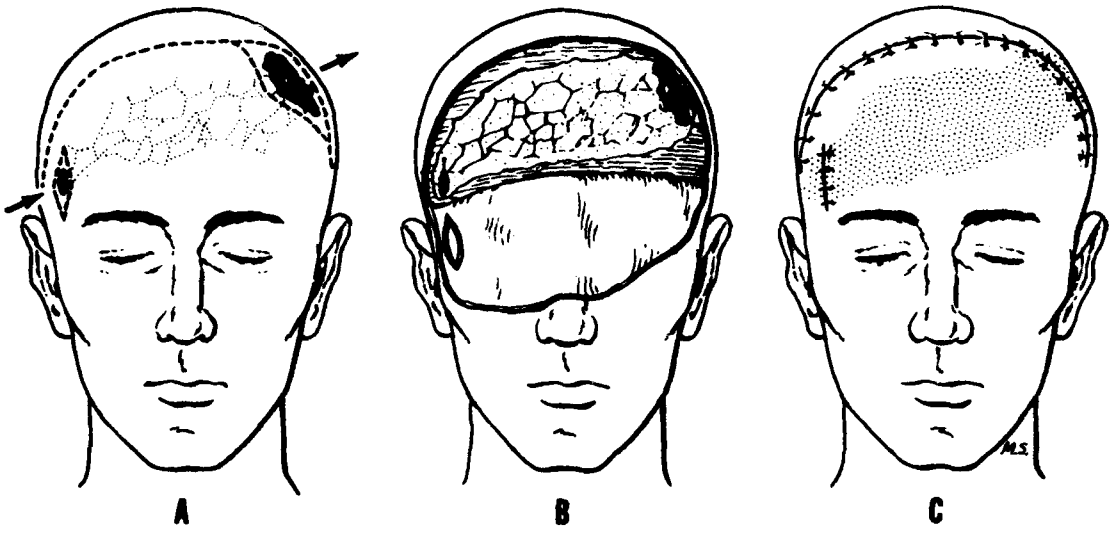


FIGURE 20.—Technique of repair of scalp. A. Through-and-through frontal wound with extensive comminution of bone. A coronal incision is outlined to include the exit wound. The entrance would is debrided separately. B. Wide exposure of entire craniocerebral wound obtained after reflection of coronal flap. C. Closure of entrance wound and reapproximation of coronal flap behind hairline.

avulsed wounds of the scalp, there was sometimes insufficient skin available to cover the defects; in such instances, split-thickness grafts could be laid directly on the pericranium, the dura, or even the exposed cortex itself if necessary.

Probably the most important single measure in the prevention of cerebral fungus formation with all its distressing sequelae was a tight two-layer closure of the scalp without tension. A great deal of attention was therefore devoted to this apparently simple procedure. A group of suitable incisions and the resulting closures achieved, all based on actual cases, are illustrated in figures 13, 14, 15, 16, 17, 18, 19, and 20.

Interrupted sutures of fine silk were used throughout for separate closure of the galea and skin (fig. 21). Continuous sutures were never to be used in

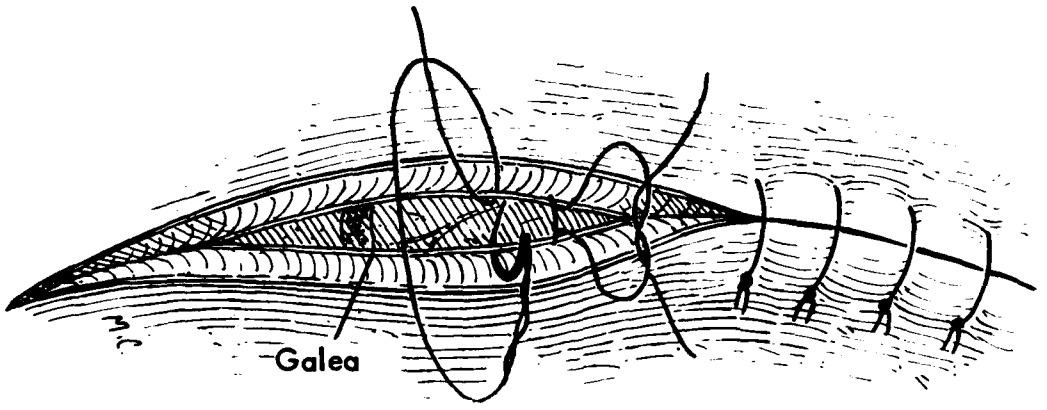


FIGURE 21.—Closure of scalp. The galea is closed with inverted, interrupted sutures of fine silk. The skin is always approximated in a separate layer, also with interrupted silk sutures.

these contaminated wounds. Black silk, No. 00 or 000, was used routinely by most surgeons. If the galea and skin could not be brought together without tension by using silk of this size, the closure was regarded as too tight and a plastic procedure of some kind was indicated. Pulling superficial layers of the scalp together with mattress sutures of heavy silk or catgut simply to get a defect covered almost invariably resulted in necrosis of skin margins and failure of primary healing.

DRESSINGS

Dressing of craniocerebral injuries in the acute stages was often a major problem. Many types of dressings were used, depending on the circumstances. If careful hemostasis was carried out during the debridement, postoperative extradural or subgaleal hematomas were not frequent, and drains were seldom necessary. In the majority of instances, there was therefore no need to disturb the dressing for 3 or 4 days.

For the average patient, who was not to be transported early, a simple pressure dressing of gauze was held in place by a melon-type dressing over the entire head made of ordinary rolled gauze bandage 2 to 4 inches in width. This was anchored by several long strips of adhesive tape, placed in crisscross fashion over the vertex and held by circular strips.

When elastic bandages of 3- and 4-inch widths were available, they were admirably suited for the melon-type dressing of the entire head to hold in place either a scanty or a voluminous dressing. Additional security could be gained by a chin strap of gauze or soft felt attached at both temporal regions.

Light plaster-of-paris headcaps were used frequently in the forward areas. Some surgeons used them routinely. Others thought them neither necessary nor desirable except for the following three categories of patients: (1) Those with extensive skull defects, especially when there were associated radiating fractures and marked comminution (the so-called eggshell or bursting type of fracture); (2) those who were extremely restless, uncooperative, and belligerent and who repeatedly pulled loose other types of dressings; for example, patients with injuries of the frontal or temporal lobe, who, particularly in the first few postoperative days, often would leave no other kind of dressing in place unless they were kept under the heaviest sedation; and (3) those who could have only limited observation and nursing care because tactical circumstances necessitated early evacuation after operation.

Plaster caps were never applied when drains were used or when it was thought that early inspection of the wound for the detection of a hematoma or other complication might be desirable. When used, they were well padded to insure protection of the scalp from pressure necrosis if there should be increased edema postoperatively. An outline of the underlying wound and bony defect was drawn on the outside of the cap and the date of application was added to guide other surgeons in subsequent removal of the cap.

SPECIAL TYPES OF PENETRATING CRANIOCEREBRAL WOUNDS

The operative treatment of a variety of specialized types of craniocerebral injury due to high-explosive missiles will be considered in the sections to follow. The general principles of wound management which have been outlined applied in every case, in addition to the particular features which will be mentioned.

Compound Linear and Depressed Fractures of the Skull

Compound linear and depressed fractures of the skull without dural penetration were seen frequently, though they were only about one-third as frequent as penetrating wounds of the brain. During periods of great activity, patients with these simpler lesions were usually not operated on in forward areas but were evacuated to hospitals farther from the frontlines. When time permitted, they were operated on as soon after injury as possible.

No attempt was made to excise fissured fractures without depression or comminution. They were treated surgically in the same manner as simple laterations of the scalp, and systemic chemotherapy was used. A burrhole was made, however, and the dura was opened in all linear fractures, and even in scalp wounds without fracture, if there were localizing neurologic signs.

When depressed fractures of the skull were associated with contaminated wounds, no effort was made to elevate fragments and leave them in position, as was frequently done in closed injuries of the head. After debridement of the scalp and excision of contaminated pericranium, a burrhole was drilled in normal bone at one side of the depressed area. The hole was always made with caution because depression of the inner table usually extended well beyond that of the outer. Starting at the burrhole, bone was rongeured toward the depressed area and around it until all interlocked and contaminated fragments had been removed.

Ascroft stated that fracture of the outer table never occurred alone and that the inner table was always more extensively involved than the outer. The inner table, in fact, was occasionally found depressed, with underlying dural contusion and subdural hematoma, without any visible fracture in the outer table (fig. 22A). There was also sometimes only slight depression of the outer table, with actual comminution of the inner table and dural penetration (fig. 22B). In every case, bone was removed from both tables until a smooth-edged defect was achieved all the way round.

The dura underlying depressed fractures was frequently torn without penetration of the brain. Simple dural suture was carried out in such cases. If the dura was intact but appeared markedly contused or discolored, it was opened; this procedure would often release a small local subdural hematoma and sometimes macerated cerebral tissue as well. After removal of all clots and devitalized brain tissue by gentle irrigation and suction, the dura was sutured tightly. When depressed fractures were accompanied by neurologic

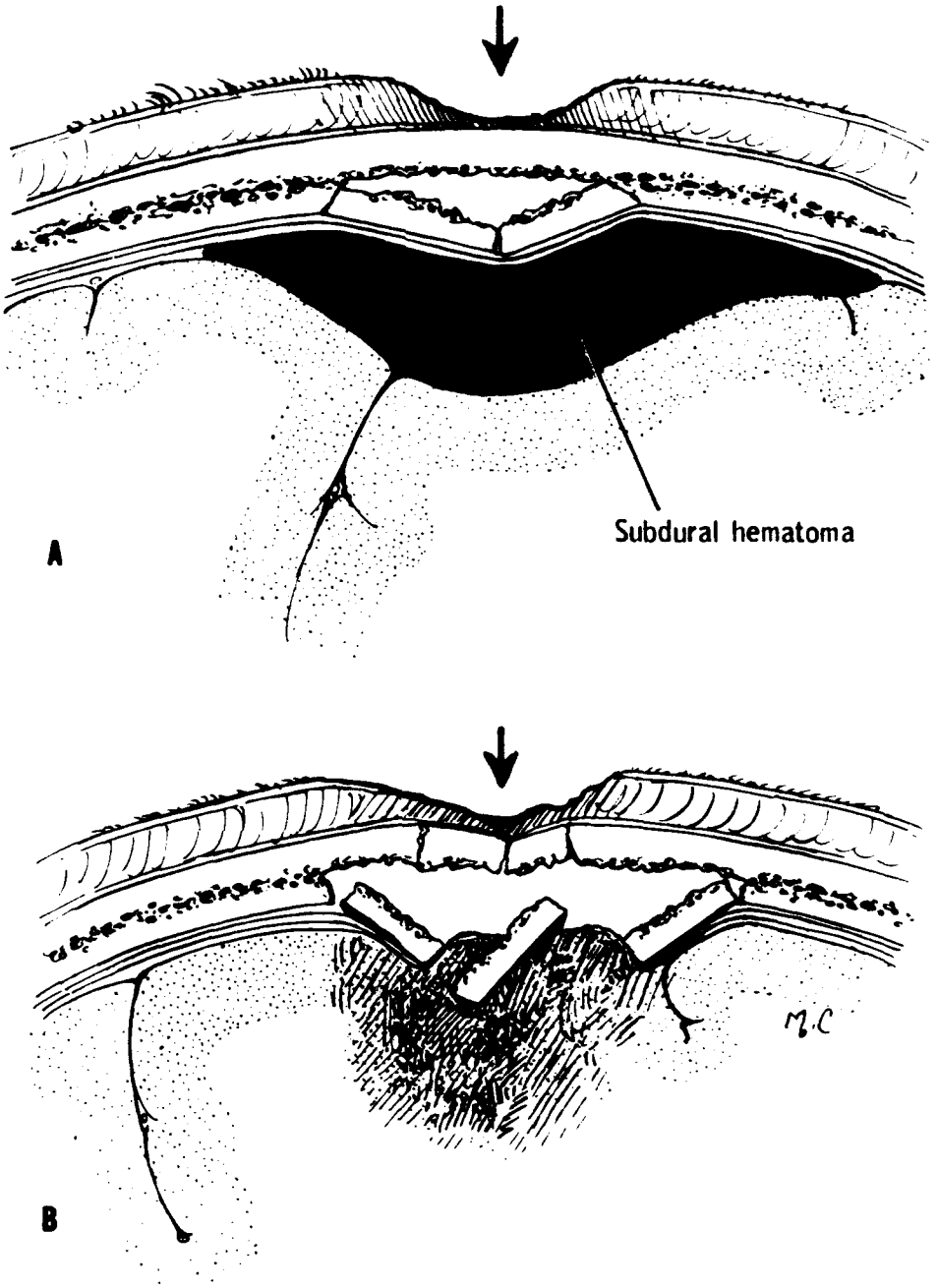


FIGURE 22.—Compound depressed fractures due to nonpenetrating missile wounds. A. Outer table of skull intact while inner table is fractured and depressed. The underlying dura may be intact but also depressed. Beneath this area, localized subdural clots are common, and the brain may be contused or even macerated. B. Linear crack with slight depression in outer table, while inner table is comminuted and a loose fragment has penetrated the dura and cerebral substance.

signs referable to the underlying cortex, such as aphasia, paresis, and hemianopsia, the dura was always opened, to be certain that all subdural clots and devitalized brain tissue had been removed. If the neurologic examination was negative and the dura was not discolored, it was not opened.

Some of the most satisfactory immediate results of head surgery were seen in the prompt disappearance of neurologic deficits following early operation on depressed fractures without dural penetration. Striking improvement of aphasia within 24 hours was often noted in fractures of the left frontotemporal area. Similar rapid improvement in homonymous visual field defects was observed after operation on depressed fractures over the occipital cortex, as in the following case:

A staff sergeant was admitted to an evacuation hospital in Belgium on 4 October 1944 with a lacerated wound, 2 cm. in length, of the right occipital region, which he had sustained 2 days before. He was in excellent general condition, and neurologic examination was negative except for a left homonymous hemianopsia. Roentgenograms revealed an occipital depressed fracture on the right side.

After excision of the scalp wound and removal of all depressed bone fragments, the dura was found intact but depressed and contused. The dura was opened because of the hemianopsia. There was a small amount of dark liquid subdural blood; the cortex was contused but not macerated. The dura and scalp were closed tightly.

There was marked improvement of vision within 24 hours, and by the fourth postoperative day, when the patient was evacuated, the visual fields were almost normal. The wound healed primarily.

Wounds Involving the Dural Sinuses

Craniocerebral wounds which involved laceration of the dural venous sinuses constituted one of the more difficult technical problems for the surgeon responsible for the treatment of patients with acute injuries. Operative treatment of patients with these wounds should have been undertaken only when special neurosurgical equipment was available and when whole blood for transfusion was already in the operating room or nearby.

In Matson's experience, the longitudinal sinus was most frequently involved (16 times) and the transverse and sigmoid sinus next (11 times). The straight sinus was involved in only one instance. The problems in management of these lesions were essentially the same, no matter which sinus was involved.

Sinus injuries could be predicted before operation from the location of the visible wound and examination of the roentgenograms (fig. 23). In such cases, preparations were made to cope with possible vigorous hemorrhage. The head was elevated during debridement to reduce the pressure in the large venous channels unless a falling blood pressure contraindicated this position. It was extremely important that these patients should remain quiet during operation and should not strain or groan. It was even more important that a good airway be maintained at all times. Therefore, if the patients were at all restless or uncooperative, general anesthesia, with intravenous thiopental sodium, or intratracheal ether was always used.

The scalp wound was debrided in the usual fashion. Exposure usually revealed depressed fragments of bone turned on edge and driven through the dura in the region of the venous sinus, or a mass of clotted blood, macerated brain, and comminuted bone fragments overlying the sinus (fig. 24). Since

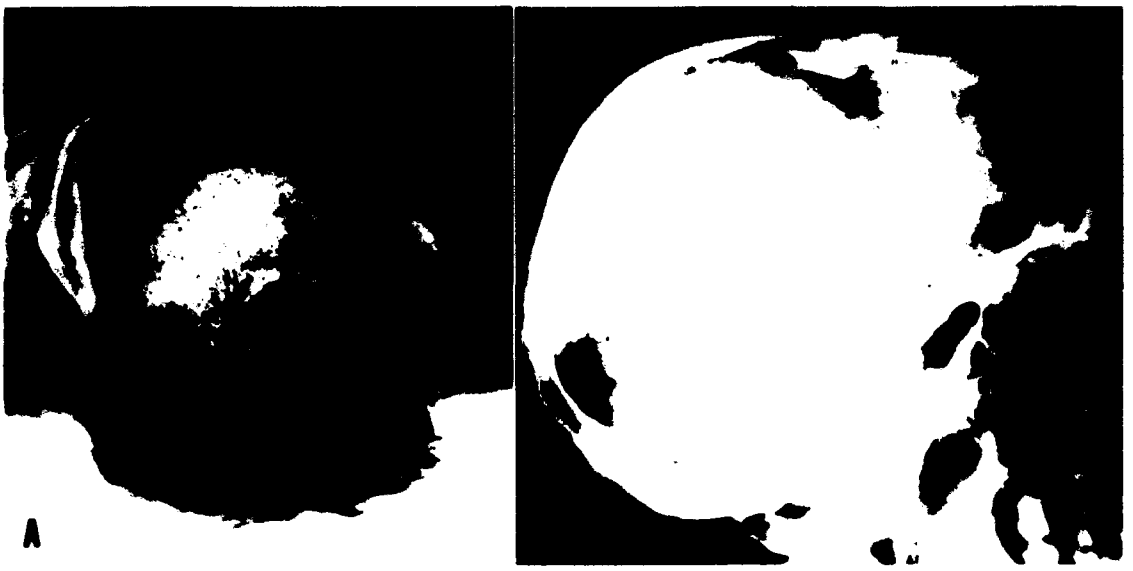


FIGURE 23. Injury of longitudinal sinus. A. Position of wound which should alert surgeon to investigate possible involvement of longitudinal sinus. B. Lateral roentgenogram showing marked depression of both tables of skull through area of longitudinal sinus. Film further warns surgeon that sinus is almost certainly involved.

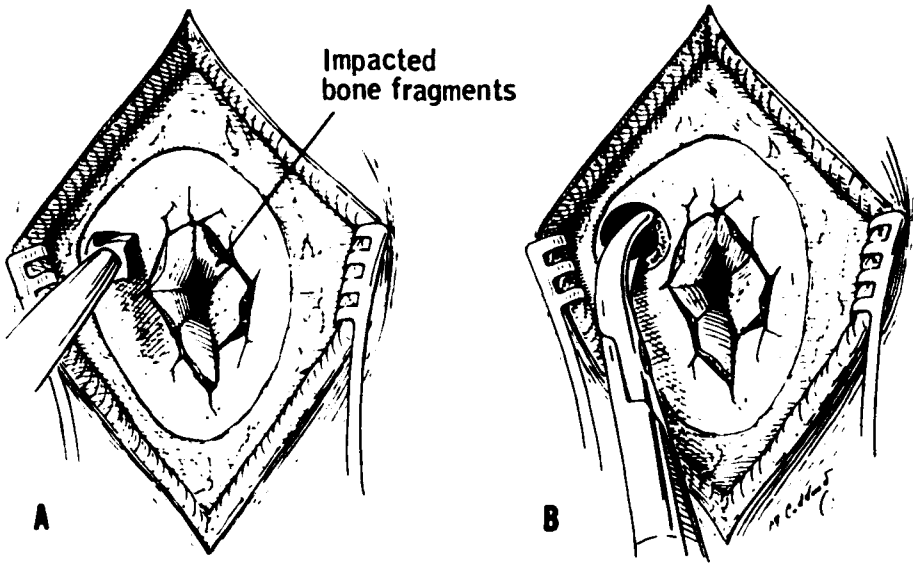


FIGURE 24. Debridement of compound comminuted depressed fracture with impaction of bone fragments. A. The scalp and periosteum have been debrided. A burrhole is drilled through normal bone adjacent to the depressed area. B. Starting at this burrhole, and keeping always over normal dura, bone is rongeured until all comminuted and depressed fragments of both outer and inner tables have been removed.

the cardinal principle in the management of sinus injuries is not to disturb the point of laceration of the dural sinus until the area is well exposed, the removal of a generous amount of bone from the surrounding area was usually required.

Figure 25 illustrates the steps in the management of a typical penetrating wound with depressed fracture through the longitudinal sinus. One or more burrholes were made in normal bone adjacent to the lesion but well away from the sinus (fig. 25A). Starting at these holes, bone was removed with a rongeur,

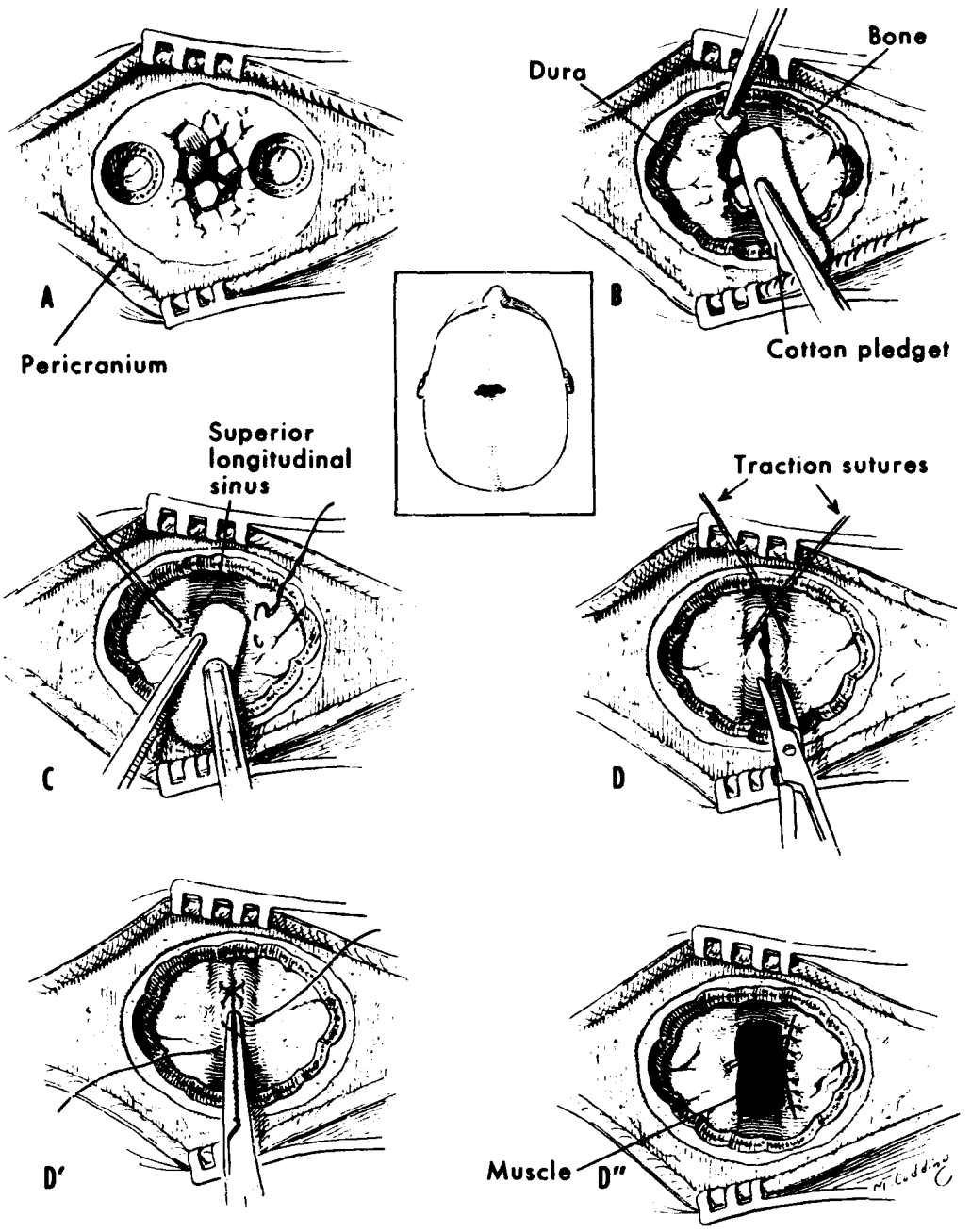


FIGURE 25.—Management of penetrating craniocerebral injuries involving longitudinal sinus Insert shows position of entrance wound.

working toward the point of depression into the sinus but not disturbing it if it could be avoided. If bleeding from the sinus started, it was controlled with pressure on cotton pledgets and suction until exposure of the surrounding bone had been completed (fig. 25B). Before removal of bone fragments or foreign bodies in the sinus itself was attempted, it was often found useful to place one or more stay sutures through the dura on each side of the sinus (fig. 25C). Depressed bone fragments in the sinus were finally gently teased free and removed. It was necessary that strong suction be available at this time.

When this method of approach was used, it was often possible to visualize the actual tear in the sinus and close it with silk sutures or with silver clips (fig. 25D). If the bleeding was too profuse or the margins of the lacerated sinus could not be brought together, bleeding was best controlled by the application of a muscle stamp or fibrin-foam pledgets soaked in thrombin. To prevent rapid loss of large amounts of blood, the fibrin foam or muscle was prepared ahead of time, not after the bleeding from the sinus had begun. If the laceration in the sinus was large, sutures previously placed were tied over the muscle stamp to hold it snugly in position. Once bleeding had been controlled by fibrin foam or muscle, care was taken not to disturb these substances.

Bone fragments depressed into the sinus underneath an intact bony margin were not removed, since bleeding, which could be exceedingly difficult to control, was almost certain to result. When small tears in the dural sinuses were covered by clotted blood which was not contaminated and which did not contain comminuted bone fragments, the clots were usually left alone.

If a dural sinus had been completely divided, there was usually clot formation in the severed ends. Such a wound could sometimes be debrided without dislodging the clots. Usually, however, it was necessary to use some method of occluding the sinus. When there was active bleeding from a completely divided sinus, it usually seemed wiser to close the sinus with a silk ligature, or with silver clips if possible, than to use fibrin foam. In one case in which bleeding from a completely severed longitudinal sinus was controlled readily with fibrin foam soaked in thrombin, the patient did well for 2 days, then suddenly lapsed into profound coma and died within a few hours. At post mortem examination, although no local hemorrhage was evident, the longitudinal sinus was found completely filled with a thrombus from the point of application of the fibrin foam in the frontal region back to the torcular Herophili. When simple tears in the dural sinuses were present, therefore, even though they were large, the use of fibrin foam was recommended. When the flow of blood had been interrupted by complete division of the sinus, bleeding was better controlled by ligation.

When bleeding from a sinus occurred underneath an intact bony margin and further debridement was unnecessary, hemorrhage was best controlled by fibrin foam or muscle tucked between the dura and the overlying bone. The dura was then held up snugly against the bone by silk sutures, placed as close as possible to the bony margin, from the dura to the galea.

An example of a dural sinus injury follows:

A soldier was admitted to an evacuation hospital in Belgium on 31 January 1945. He was semiconscious and in mild shock; the neurologic examination was negative except for an homonymous hemianopsia on the right. A jagged wound of the scalp in the left occipital region was filled with blood clot. Roentgenograms revealed a comminuted fracture of both the occipital bone and the left parietal bones, with bone fragments driven 5 or 6 cm. into the left occipital lobe.

After treatment of shock, with good results, debridement was carried out under 1 percent procaine hydrochloride field block anesthesia. As comminuted, depressed bone fragments were removed to expose the dural defect, furious bleeding was encountered from a tear 1.5 cm. long in the longitudinal sinus about 2 cm. proximal to the torcular Herophili. It was controlled with fibrin foam and three figure-of-eight silk sutures. One and one-half to two ounces of pulped brain tissue were then aspirated from the wound track, which crossed the occipital horn of the lateral ventricle. Bone fragments were removed from the lateral ventricular lumen and from beneath the falx. The straight sinus, which was also lacerated, was closed with two silver clips. Ten thousand units of liquid penicillin were instilled in the ventricle, and the dura and scalp were closed tightly. One thousand cubic centimeters of whole blood were given during and immediately after operation.

The postoperative course was uneventful and the wound healed primarily. At the time of evacuation, the patient was ambulatory and afebrile, but there was no improvement in the hemianopsia.

Intracranial Hematomas

Hematomas of varying size were usually found with penetrating wounds of the brain. As a rule, they were small and of no significance as far as the patient's general condition was concerned. Often, large hematomas formed in the superficial layers of a wound underneath the first aid dressing. In fact, what appeared to be huge cerebral herniations frequently turned out to be chiefly hematomas (fig. 26).

Of great importance were the sizable intracortical hematomas found at points along the tracks made by small missiles or by indriven bone fragments. Hematomas of this kind were often found at the deepest point of the wound track, where an explosive type of lesion frequently occurred. In cases of this sort, treatment was usually very satisfactory, since there was likely to be rapid improvement in both the neurologic deficit and the patient's general condition after evacuation of the clot.

An illustrative case is cited:

A private was admitted to an evacuation hospital in Belgium on 15 January 1945, semistuporous and aphasic but not in shock. There was a wound of entrance, 1 cm. in diameter, in the left eyebrow, without herniation of brain tissue. Roentgenograms showed a depressed fracture of both plates of the left frontal sinus and a metallic foreign body, 1 cm. in diameter, deep in the left parietal area.

Through an eyebrow incision, all depressed fragments of bone and the entire mucosa of the left frontal sinus were removed, revealing a hole, 1 cm. in diameter, in the dura. As the entrance of the brain track was exposed, 40 to 50 cc. of dark clotted blood were extruded under pressure, leaving a large cavity within the frontal lobe. Debridement of the track was completed to a depth of 8 cm., and 10,000 units of liquid penicillin were instilled into the bottom of the wound. The foreign body was not removed. The dura and skin were closed tightly.



FIGURE 26. Severe gutter wound of vertex with compound depressed fracture through longitudinal sinus.

The aphasia and level of consciousness improved dramatically after operation, and the patient was ambulatory and afebrile when he was evacuated on the fifth postoperative day.

Another important variety of intracranial hematoma was that which occurred on the side opposite from the wound of entrance, as described by Matson and Wolkin.[26] When a metallic foreign body was visualized by roentgenogram near the cortex of the side opposite the side of entrance, there was no way of knowing whether its position marked the end of the wound track or whether the foreign body had struck the opposite side and bounced back. If the missile had approached the opposite surface, the likelihood of hemorrhage into the subdural or subarachnoid space, or subcortically, was great (fig. 27). When such a hemorrhage occurred at a distance from the wound of entrance, the blood could not escape and a space-occupying hematoma was formed. As a result, the pathologic physiology assumed the nature of that seen in closed injuries of the head in which, in addition to actual damage at the moment of trauma, subsequent additional injury due to cerebral compression becomes an even more important factor.

When a foreign body lay near the surface at a distance from the point of entrance (fig. 27), it was the policy to place a burrhole as nearly as possible over it. If no extradural or subdural hematoma was found but if the brain was softened or contused, a ventricular cannula was directed toward the loca-

[26] Matson, D. D., and Wolkin, J.: Hematomas Associated With Penetrating Wounds of the Brain. J. Neurosurg. 3: 46-53, January 1946.

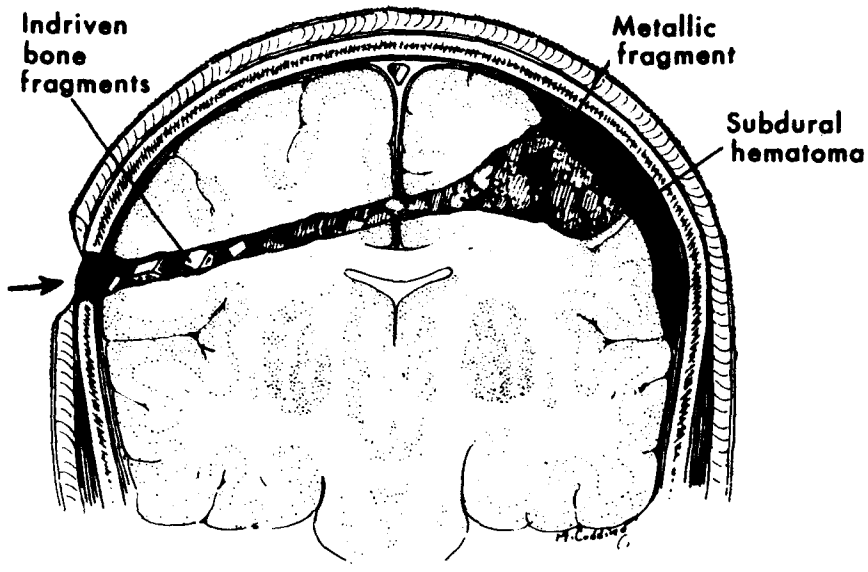


FIGURE 27.—Coronal section illustrating penetrating wound in which high-explosive shell fragment is seen close to surface at point distant from wound of entry.

tion of the foreign body. It was sometimes possible to retrieve the foreign body itself; the attempt was made if it was of any size or was readily located. More important, however, was evacuation of any hematoma and macerated brain tissue in its neighborhood or in its path through the brain. If the clinical picture was that of cerebral compression out of proportion to the extent of the visible wound, preliminary exploration over a distant foreign body proved advantageous before attacking the wound of entrance.

An example of this type of injury follows:

A corporal was admitted to an evacuation hospital in Belgium on 3 January 1945. He was stuporous, with a blood pressure of 180 systolic and 50 diastolic, and a pulse of 48. A wound 10.0 by 2.0 cm. in size in the left frontotemporal region was associated with herniation of brain tissue. Right hemiparesis was present. Roentgenograms showed numerous intracerebral bone fragments in the left temporal area and 2 pea-sized metallic foreign bodies close to the midline of the left occipital region about 2 cm. beneath the bone.

Under local anesthesia, a burrhole was first made over the expected site of the metallic fragments, after which the dura was quickly opened. A minimal amount of subdural blood was present, but the brain was bulging. When a ventricular needle was inserted, about 60 cc. of dark subcortical clot was evacuated and the foreign bodies were removed. The patient's general condition improved considerably, and routine debridement, with temporal fascia repair of the dural defect at the entrance wound, could be accomplished.

The patient was evacuated on the eighth postoperative day with a right hemiparesis and right visual field defect. He was fully conscious and eating well, and his wounds were well healed.

Transventricular Wounds

In World War I, Cushing [27] reported from a forward aid station that when missiles crossed the ventricular system, the wounds were 100 percent fatal;

[27] See footnote 1 (2), p. 123.

when bone fragments alone entered a ventricle, the case fatality rate was almost 50 percent. In the same war, Horrax [28] reported from a base hospital a case fatality rate in transventricular wounds of 57.1 percent. In World War II, ventricular wounds again were among the most serious, but, in reports by Haynes [29] and other studies, the fatality rate in surgical cases was reduced to about 30 to 40 percent. With strong suction and facilities for electrosurgery available, as well as blood for replacement therapy, early, thorough debridement of intraventricular wounds became much more feasible. This procedure not only reduced the damaging central effects of old blood (fig. 28), macerated brain tissue, and other debris in the ventricles but also reduced the liability of subsequent infection because of early removal of the sources of contamination. The incidence of ventriculitis and meningitis, the chief causes of death in patients with ventricular wounds who survived the immediate injury, was also greatly reduced by the use of local and systemic chemotherapy.

Examination of the roentgenograms and of the position of the wound of entrance almost always indicated whether a missile or bone fragments had entered the ventricles. If spinal fluid was seen to leak from the wound of



FIGURE 28.—Coronal section through brain in fatal transventricular wound. Note complete cast of clotted blood throughout ventricular system.

[28] See footnote 1 (4), p. 123.
[29] See footnotes 7, p. 135; and 15, p. 147

entrance when the patient strained, no further evidence was necessary. When it had been established that a transventricular wound was present, or when such a wound was suspected, the highest priority for operation was given unless the patient was moribund.

In the operative management of ventricular wounds, adequate exposure and light were especially important (fig. 29). The preliminary debridement of

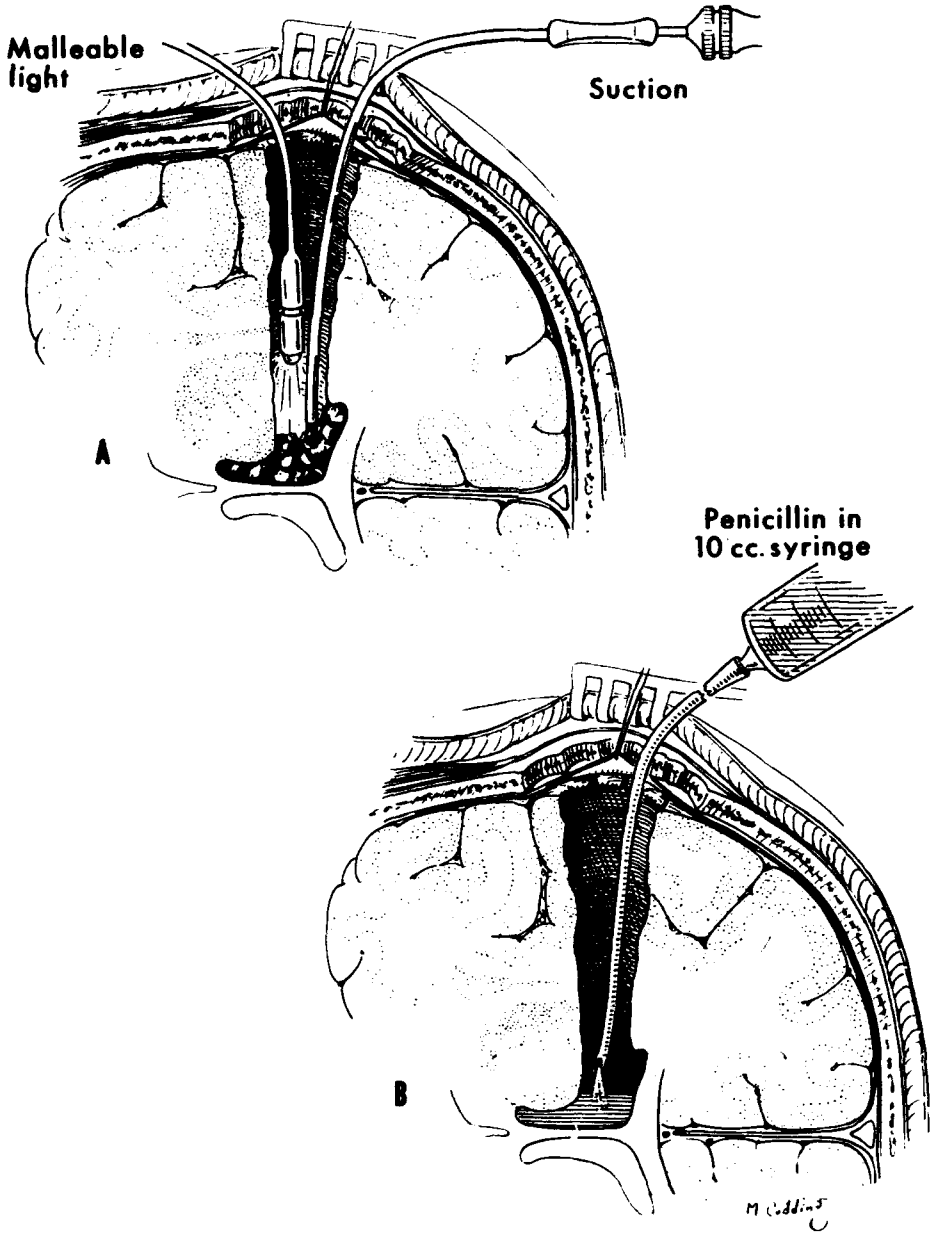


FIGURE 29.—Debridement of intraventricular wound. A. Gentle suction of macerated cerebral tissue, bone chips, and old blood within the ventricular lumen is carried out under direct vision with the aid of a small, malleable light. B. From 10,000 to 20,000 units of diluted liquid penicillin are instilled directly into the ventricle and the depths of the wound before closure of the dural defect.

the scalp, cranium, dura, and cerebral wounds was carried out as already outlined. Whenever possible, the wound in the brain was debrided all the way down to the ventricle under direct vision. With the aid of a lighted retractor or a small, malleable light (fig. 29A), it was often possible to visualize the ventricular wound itself and to remove bone fragments and blood clots from the lumen (fig. 29 B). Gentle irrigation with warm physiologic salt solution facilitated removal of macerated cerebral tissue and debris. When the walls of the wound track collapsed or the track was not wide enough for the operator to see directly into the ventricular lumen, gentle irrigation and suction of old blood and debris were carried out through a ventricular cannula introduced blindly into the ventricle. As much of the debridement as possible down to, and inside of, the ventricle was always performed, however, under direct vision. In wounds which crossed the midline, debridement of the opposite lateral ventricle could occasionally be carried out through the foramen of Monro and the third ventricle. When this was not possible, a burrhole was placed on the opposite side, and the lateral ventricle on this side was irrigated through a ventricular cannula.

When debridement had been completed, from 10,000 to 20,000 units of liquid penicillin were instilled into the ventricular system. The penicillin was well diluted with physiologic salt solution, to insure good circulation throughout the ventricles and to lessen the likelihood of local irritation. Studies by Walker and his coworkers [30] showed that not more than 20,000 units should be introduced directly into the ventricles at any one time. In all transventricular wounds, it was Matson's policy to give 10,000 units of penicillin intrathecally by the lumbar route daily for at least 5 days after operation; penicillin was given systemically in full dosage for at least another 10 days.

The dural repair in transventricular wounds received particular attention, for in them a watertight closure was especially desirable to lessen the possibility of formation of a cerebrospinal fluid fistula or the development of ventriculitis from outside contamination. If dural grafts were used, they were made large enough to allow suture without tension. Catheters or drains were never left beneath the dura.

Foreign bodies loose in the lateral ventricle were removed successfully on several occasions. It was thought that the attempt should be made if the foreign body was of any size. A very small metallic foreign body in the third or the lateral ventricle, not easily visualized at the time of debridement, was left alone. Attempts to remove small intraventricular foreign bodies through new transcortical exposures were not warranted at primary operation.

When a missile crossed one of the lateral ventricles or the third ventricle or drive bone fragments into the ventricular lumen, the possibility of intraventricular hemorrhage was always present. If hemorrhage was massive, filling the entire ventricular system, the patient usually did not survive long enough to reach a hospital in which definitive surgery could be carried out. When patients with such conditions did reach the hospital, they were usually mori-

[30] See footnote 17, p. 148.

bund, with dilated, fixed pupils, irregular, moist respirations, and generalized rigidity. When this clinical picture was present, surgical intervention was invariably of no avail.

The following procedure was attempted on 3 or 4 such patients with small wounds of entry: Debridement of the wound down to the lateral ventricle, with removal of as much clotted blood as possible from the lumen, followed by the placement of a burrhole on the opposite side, the insertion of a ventricular cannula into this lateral ventricle, and the irrigation of clotted blood from the lumen of the ventricle on this side. There was no clinical improvement in any case, however, and at post mortem examination it was seen that very little of the total amount of clotted blood in the ventricular system had actually been removed (fig. 30).



FIGURE 30.—Coronal section through brain in fatal through-and-through wound showing cast of blood clot filling entire ventricular system.

One of these cases is described:

An officer admitted to an evacuation hospital in Germany on 10 March 1945 was comatose but not in shock. Respirations, which were 20 per minute, were quiet and regular. The pulse was 76 and the blood pressure 120 systolic and 70 diastolic. Both pupils were dilated

and fixed to light. The patient did not respond to painful stimuli. There was a wound of entrance, 1.5 cm. in diameter, in the left frontotemporal region. Roentgenograms revealed a metallic fragment, 1.0 cm. in diameter, deep in the right cerebral hemisphere.

At operation, the wound of entrance was excised, and 1 to 2 oz. of pulped brain tissue and blood clot under pressure were removed from a narrow wound track which entered the left lateral ventricle. Dark liquid blood, as well as clotted blood, was removed from the ventricle by irrigation. A burrhole was then made on the opposite side, and a ventricular needle was introduced into the right lateral ventricle. Again dark blood, both liquid and clotted, was removed in small amounts by irrigation. Tight closure of both wounds was carried out.

The patient went progressively downhill and died about 18 hours after operation. Post mortem examination revealed a solid cast of blood clot throughout the ventricular system, including the aqueduct and fourth ventricle. The operative efforts had not significantly altered the total amount of intraventricular blood clot present.

If hemorrhage into the ventricular system was extensive but solid clotting throughout the ventricles had not taken place, it was occasionally possible to evacuate large amounts successfully and for the patient to recover. Such a case is cited:

A sergeant was admitted to an evacuation hospital in Germany on 1 March 1945, in very poor condition. He was in moderate shock and in deep coma, with slow respirations. Reflexes were all markedly depressed and the extremities flaccid. The right pupil was dilated and fixed; the left was dilated but reacted to light. There was a wound, 2.0 by 3.0 cm., in the right frontal region with herniation of brain tissue. Roentgenograms revealed a fracture in the right frontal region with indriven fragments of bone, a small metallic foreign body in the midline, and a larger foreign body about 1 cm. from the surface low in the left postparietal region.

Under local anesthesia, the right frontal wound was debrided. About 50 cc. of macerated brain tissue and numerous indriven bone fragments were removed from a track which entered the frontal horn of the lateral ventricle. It was possible to lift considerable clot from the lateral ventricle and also from the third ventricle through the foramen of Monro. The dural defect was repaired with a pericranial graft, and the scalp was closed. A burrhole was then placed over the site of the foreign body on the left side. No subdural hematoma was present. The cortex was contused, and a ventricular needle dropped easily into a track from which an estimated 15 to 20 cc. of dark fluid and clotted blood were evacuated. The track was debrided down to the ventricle on this side, which was irrigated gently, in order to remove the old blood.

After operation, the patient's condition was immediately improved, and it continued to improve slowly until he was evacuated on the tenth postoperative day. He was still semistuporous, but he was taking fluids by mouth.

When the lateral ventricles were involved by small missiles without hemorrhage, there was often very little reaction. There seemed no reason why recovery should not follow adequate debridement and repair of such injuries. When there was gross damage to the ventricular wall, even without massive bleeding, the prognosis was poor. Haynes,[31] in reporting 100 transventricular wounds, thought that maceration of more than one-third of the wall of a lateral ventricle was incompatible with life.

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[31] See footnote 7, p. 135.

Through-and-Through Wounds of the Brain

It has long been appreciated by neurosurgeons that, when a missile passes completely through a portion of the intracranial chamber, there is more destruction of soft tissues and more extensive comminution of the skull at the wound of exit than at the wound of entrance (fig. 31). Because a large number

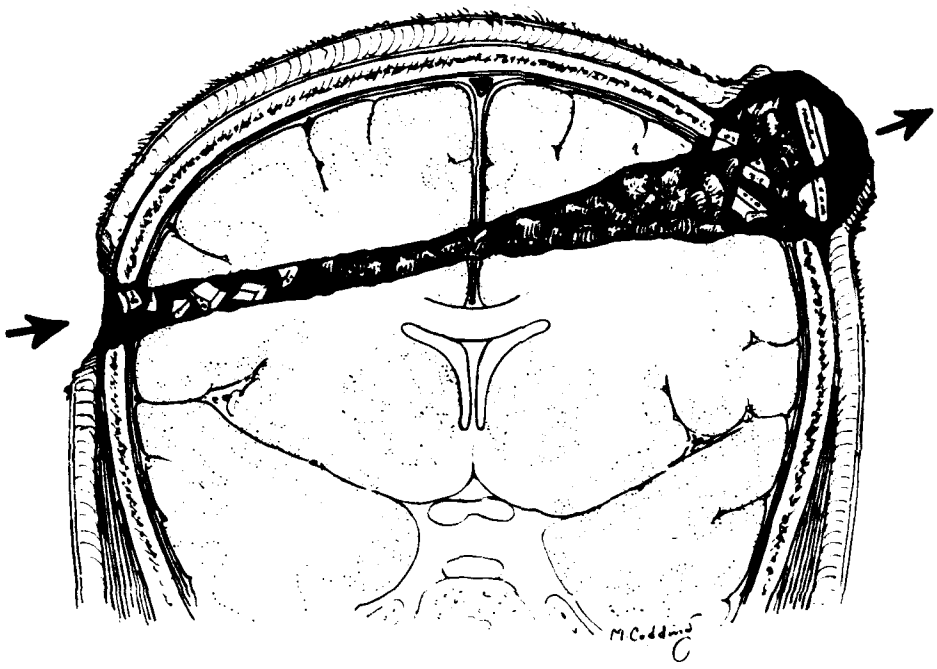


FIGURE 31.—Coronal section illustrating perforating wound due to high-explosive shell fragments. Small indriven bone fragments are characteristically found at the wound of entrance. At the point of exit, a larger, explosive type of lesion, with a bursting fracture, hematoma formation, and cerebral herniation, is commonly seen.

of cortical vessels are apt to be lacerated at the site of exit, hematomas which may be extradural, subdural, or intracortical are often associated with the exit wound. This statement does not refer to the ordinary maceration of brain with small hemorrhages which occur all along the course of every brain wound, but rather to the large hematomas usually located at or near the exit of the wound track from the brain.

These findings led to the suggestion that in through-and-through brain lesions, if the two wounds were to be approached separately, the wound of exit should always be explored first.[32] Rapid evacuation of a hematoma, in a situation otherwise too precarious to permit much surgical intervention, sometimes resulted in sufficient improvement in the patient's general condition to permit thorough debridement and primary repair of both exit and entrance wounds. If necessary, attention to the wound of entry could be delayed for 24 hours or more after preliminary debridement of the exit wound, and supportive treatment

[32] See footnote 26, p. 164.

could be given until the patient's general condition warranted definitive repair. Chemotherapy and antibiotic therapy made such delay possible without significant added danger of infection. Whether or not a hematoma was found, debridement of the more extensive exit wound first was thought to be the procedure of choice.

When roentgenograms revealed comminution of the bone between the points of entrance and exit in through-and-through wounds near the surface limited to one side of the head or across the frontal or occipital areas, exposure was best achieved by connecting the two wounds with a linear incision or by raising a scalp flap so as to include both wounds (fig. 17).

A case illustrative of improvement following preliminary debridement of the exit wound in a through-and-through injury is cited:

A German prisoner of war was admitted to an evacuation hospital in Germany on 28 March 1945, in very poor condition. He was in moderate shock and profound coma and gave no response to painful stimuli. Left facial paralysis and left hemiplegia were present. The respirations were Cheyne-Stokes in type, and oral secretions drained from the mouth. The right pupil was larger than the left. There was a wound of entrance, 1.0 cm. in diameter, low in the right occipital region with herniation of brain tissue, and a wound of exit, 2 cm. by 2 cm. in size, in the right frontal region at the hairline, also with herniation of brain tissue. Roentgenograms revealed defects in the skull corresponding to these scalp wounds. No metallic foreign body was evident.

Under local anesthesia, the wound of exit was first excised. After removal of comminuted fragments of the frontal bone, 15 to 20 cc. of extradural clot was found. When the brain wound was uncovered, at least 50 cc. of clotted dark blood emerged under pressure from the brain track. After debridement of the track, the dural defect was repaired with a pericranial graft. It was noted that the patient's breathing at once became more regular and his pulse of better quality. The wound of entrance in the occipital region was then debrided and repaired in the routine manner.

Postoperatively, there was steady improvement. The patient was afebrile after the third day. He began to move his right side and respond to pain on the first postoperative day, and by the fourth day he took fluids by mouth and responded accurately to questions. The left hemiplegia remained complete. The wounds healed primarily.

Craniofacial Wounds

Penetrating wounds of the craniocerebral tissues which also injured adjacent structures of the face deserve particular attention. In this category are included wounds involving the paranasal air sinuses, orbit, middle ear, and mastoid. From the technical standpoint, they probably constituted the most difficult injuries handled by the neurosurgeon in forward areas. They were less serious injuries in one respect, however, in that usually only silent areas of the brain were involved.

In World War I, craniofacial wounds were considered among the most serious, and the case fatality rate from infection was extremely high. At that time, most of these wounds were left open; cerebral herniation and fungus formation resulted, and meningitis almost invariably followed. With chemotherapy, however, and the availability of blood for replacement therapy at

the time of primary debridement, a much more extensive and definitive primary operation became possible in World War II.

Debridement.—The objective of treatment of this type of injury was the complete removal of all contaminated, necrotic, and macerated tissue, followed by primary repair, which would isolate the brain and subarachnoid spaces from contaminated areas as completely as possible. In addition, the surgeon had to preserve structures needed for plastic reconstruction of the face, so that the best possible cosmetic result would be obtained.

Complete debridement of an extensive compound wound involving one or both orbits, the frontal and ethmoid sinuses, and the nasal and facial bones in addition to the frontal lobes of the brain, the floor of the frontal and middle fossae, and perhaps the calvarium, was a time-consuming and formidable procedure. It was Matson's firm belief that such wounds should not be operated on in stages or be left open. Instead, immediate, complete debridement and repair should be carried out by the neurosurgeon with the assistance or advice of the otolaryngologist and ophthalmologist. If the surgeon was not prepared to do this, or if circumstances were such that sufficient time could not be devoted to the case, then it was better to evacuate the patient farther to the rear, to a center where complete primary treatment could be undertaken. The disadvantages of an additional delay of even 48 hours or more did not outweigh the advantages of proper primary management of these wounds. Webster, Schneider, and Lofstrom [33] expressed the same opinion in reporting 40 cases of orbitocranial wounds seen in a general hospital, in 5 of which redebridement was necessary.

The debridement and repair of the average craniocerebral wound took between 1 and 2 hours. A craniofacial wound, on the other hand, often required as long as 3 to 4 hours. The surgeon had to be prepared to spend this amount of time, if necessary, and proper supportive measures had to be available. Inhalation anesthesia through an intratracheal tube was essential. Local anesthesia and intravenous Pentothal Sodium anesthesia were contraindicated because blood and mucus in the pharynx or running down into the respiratory tract might encroach upon the airway during operation and precipitate laryngospasm or complete respiratory obstruction. Only after induction of inhalation anesthesia, insertion of an intratracheal tube, aspiration of all blood and mucus from the nasopharynx and trachea, and packing off of the pharynx had been completed was debridement of a craniofacial wound started.

These injuries were usually handled through the wound of entrance. To obtain the best exposure, it was found that debridement of the intracranial and facial components of the wound should be carried out simultaneously. There was usually marked comminution of the orbital, nasal, zygomatic, sphenoid, and frontal bones. The contents of the orbit were frequently completely macerated, and the globe was often collapsed or even unrecognizable. When the paranasal air sinuses were involved, the mucosa was usually lacerated

[33] Webster, J. E., Schneider, R. C., and Lofstrom, J. E.: Observations Upon the Management of Orbito-Cranial Wounds. J. Neurosurg. 3: 329–336, July 1946.

and at least partially separated from the bone, and the lumen was filled with macerated cerebral tissue, blood clot, and comminuted bone fragments. In Matson's series, the frontal sinuses were most commonly involved (26 times), the ethmoids next (17 times), and then the maxillary antrums (12 times). Whenever there was a communication between the intracranial chamber and a paranasal sinus, the entire mucosal lining of the sinus was removed, in addition to the debris found within the lumen.

Removal of comminuted fragments of the supraorbital plate and greater wing of the sphenoid was particularly helpful in making it possible to visualize dural lacerations on the undersurface of the frontal lobe. The dura was frequently torn loose from the floor of the frontal fossa except for its attachments at the sphenoid wing and the crista galli. Freeing these dural attachments often resulted in sufficient mobilization of the dura for closure of defects without grafting.

Closure of dura.—In craniofacial wounds, watertight repair of dural defects was particularly important. Since these wounds communicated directly with the nasal sinuses or the nose itself, continuity with a contaminated area continued, in spite of complete debridement of the sinuses and removal of the mucosa. At least partial closure of the dural defect was almost always feasible, and a graft could then be used to complete the closure. A free graft of temporal fascia or pericranium was most commonly used. Particular care was taken to suture the graft in position as tightly as possible, by means of silk sutures placed close together.

If the dural defect extended posteriorly on the undersurface of the frontal lobe toward the optic chiasm and cavernous sinus, it was not always possible to reach the margins of the dural defect. In these instances, care was taken to remove any bone fragments protruding through the dura. A generous graft of temporal fascia, fascia lata, or pericranium was then prepared and sutured in place as far posteriorly as possible, after which the free graft was pushed backward to lie over the margins of the dural defect. A piece of temporal muscle could sometimes be wedged beneath the graft to hold it up in position. Fibrin foam was used for the same purpose.

Plastic surgery.—In debridement of wounds of the face, orbital region, and about the ear, all the skin possible was preserved for subsequent plastic procedures. Plastic operations were carried out at the time of debridement and repair of the brain wound if they were necessary to accomplish primary skin closure. Simple undermining of the scalp and skin of the face often permitted sufficient mobilization for closure. If not, the wound was extented, or scalp flaps were swung to effect primary closure. When the skin could not be closed primarily, Webster, Schneider, and Lofstrom recommended the use of a temporary fascial graft to the dural defect and a petrolatum-impregnated gauze pack, followed by a split-thickness skin graft in about 10 days. This should seldom be necessary if debridement of skin and the primary plastic closure has been carried out wisely.

Drainage.—In many of these wounds, a large dead space remained when debridement had been completed, particularly when the contents of the orbit and paranasal air sinuses had been removed. If this cavity was not too large, it was simply drained by a rubber-tissue drain through the lateral aspect of the wound closure. If the cavity was extensive and involved the maxillary antrum and nose, a petrolatum-impregnated gauze pack was placed loosely in the cavity and brought out through the nose. It was gradually removed during the first 4 to 5 days after operation.

Antimicrobial therapy.—In all wounds involving the paranasal sinuses, sulfanilamide and penicillin powder, or penicillin solution, was left in all parts of the wound as closure was carried out. In addition, 10,000 to 20,000 units of penicillin were usually injected into the wound every day for the first 4 or 5 postoperative days. The patients were also given both sulfadiazine and penicillin systemically for at least 10 days after operation. When a cerebrospinal fluid fistula was present, chemotherapy was continued until the leakage of fluid stopped.

An example of primary treatment of a severe craniofacial wound is presented:

A soldier was admitted to an evacuation hospital in Germany on 23 March 1945. He was conscious but in mild shock and was confused and disoriented. There was a huge lacerated wound of the right frontotemporal-orbital region with herniation of brain tissue and maceration of the orbital contents (fig. 32A).

After the patient had been treated for shock and roentgenograms had been taken, nitrous oxide-oxygen-ether anesthesia was induced and maintained through an intratracheal tube.

The skin edges were debrided, herniated cerebral tissue and blood clot were aspirated, and multiple comminuted fragments of the frontal, temporal, zygomatic, orbital, ethmoid, nasal, and maxillary bones were removed. Both frontal and both ethmoid sinuses were completely exenterated. The entire right bony orbit and its devitalized contents were removed. The superior plate of the antrum was fractured but was left in place. One and one-half ounces of pulped brain tissue were then removed from the right frontal lobe and about one-half ounce from the left frontal pole. The dura was mobilized from the floor of the frontal fossa and sutured across the midline, and a residual dural defect underneath the right frontal pole, 2.5 by 5.0 cm., was closed tightly with a free graft of pericranium. Penicillin and sulfanilamide powder were frosted throughout the wound, and a rubber-tissue drain was brought out the lateral angle. It was possible to mobilize the skin of the cheek and the forehead sufficiently to carry out a two-layer closure without tension. Five hundred cubic centimeters of whole blood and 500 cc. of Alsever's solution were given during the 3½-hour operative procedure.

Convalescence was remarkably smooth, with no leakage of spinal fluid. The drain was removed in 24 hours. Sutures were removed on the fourth postoperative day, at which time the photograph shown in figure 32B was taken. When the patient was evacuated on the seventh postoperative day, he was afebrile, and the neurologic examination was negative.

POSTOPERATIVE MANAGEMENT

The general principles of good postoperative care for all major surgical problems were applicable to penetrating injuries of the brain, with additional attention to certain special requirements. It was desirable to assign attendants



FIGURE 32. Severe shell-fragment wound with destruction of soft tissues of face and scalp; comminuted fractures of right maxillary, zygomatic, sphenoid, nasal, ethmoid, frontal, and temporal bones; penetration of both frontal, both ethmoid and the right maxillary sinuses; and laceration of both frontal lobes of brain with herniation of cerebral tissue. A. Patient prepared for operation. Intratracheal anesthesia has been induced. B. Same patient shown in view A 4 days after complete primary debridement and plastic closure.

familiar with the particular problems involved to the special head wards whenever possible. Constant shifts of personnel were undesirable.

Position. The optimum postoperative position for the patient with a craniocerebral injury was with the head elevated. Venous congestion in the head was thereby reduced, and bleeding was lessened. Elevation of the head by placing pillows or folded blankets underneath it was usually unsatisfactory and uncomfortable. It was better to have the patient raised from the waist upward, rather than just from the neck. The most satisfactory method, especially with patients who were not cooperative and alert, was to raise the head of the cot or bed by placing it on shock blocks, a chair, boxes, or whatever else was available. The patient could then lie at full length and turn readily, but the head was still maintained higher than the rest of the body. An invaluable aid to the comfort and management of patients placed in this position was a footboard fitted to the end of the cot, to prevent continual sliding downward.

This position was best for all patients during the early postoperative period with two important exceptions: namely, patients who were vomiting and patients who were in such deep coma that they had lost their pharyngeal reflexes and were therefore unable to take care of their own secretions. Whether these conditions were the result of a recently administered general anesthesia or of the injury itself, the maintenance of a clear airway and the prevention of aspiration of mucus or vomitus demanded that the patients lie flat on one side or the other and that they be turned frequently. If necessary, the foot

of the bed was raised to promote drainage of excessive bronchial secretions, but the danger of increased venous congestion in the cerebral circulation had to be considered carefully before this was done. A much more satisfactory plan was to leave the patient flat and aspirate the pharynx and mouth frequently with mechanical or electric suction.

Sedation. No stated formula for the use of analgesics and sedatives was universally satisfactory, and the surgeon often had to use all his own judgment and ingenuity. In general, patients with simple uncomplicated wounds of the vault had little pain. Craniofacial wounds, however, were often very painful, and patients with injuries of the head often had severe painful wounds elsewhere in the body.

Acetylsalicylic acid, alone or in combination with small doses of codeine or with phenacetin and caffeine sodium benzoate, was given freely and was usually sufficient to control the ordinary headache and postoperative discomfort. Morphine sulfate in doses of 0.008 gm., 0.01 gm., or occasionally of 0.016 gm. was given to patients with penetrating wounds of the head for relief of pain provided that they were under good supervision and that there was no preexisting respiratory depression or irregularity. Barbiturates given in small doses 2 or 3 times during the day, and in doses up to 0.2 gm. at bedtime, provided sufficient relaxation for sleep.

For the control of the restless, delirious, uncooperative patient (as so many with frontal and temporal lobe injuries proved to be), paraldehyde was the most useful and effective drug. It was often continued regularly for a number of days without harm. It was administered by rectum, preferably in doses of 15 to 25 cc., and could be repeated every 3 to 4 hours. It was also occasionally given by mouth, in doses of 10 to 15 cc., though it was less well tolerated by this route. Intramuscular injection was used for quicker action, and when the oral and rectal routes were impractical; 4 to 7 cc. were usually given deep in the gluteal muscles. When immediate action was necessary in unmanageable, delirious patients, 1 to 3 cc. might be given intravenously. Chloral hydrate also proved a useful sedative.

Fluid intake.—Ordinarily, there was no particular problem in the postoperative administration of fluids to patients with penetrating craniocerebral injuries. Fluids were not restricted. As in other surgical situations, the criterion was maintenance of adequate hydration as determined by the volume of the urinary output and the moisture of the skin and mucous surfaces.

In conscious patients, fluids were given freely by mouth unless vomiting was frequent. Then fluids given orally in small amounts at frequent intervals were supplemented by parenteral fluids. Since all these patients were receiving sulfonamide as well as penicillin therapy, an attempt was made to maintain a 24-hour fluid intake of 2,500 to 3,000 cc. When fluid was given by the parenteral route, not more than 1,000 cc. of the total was given as physiologic salt solution. Blood plasma and whole blood were counted as saline solution.

Transfusions of whole blood were given during the postoperative period for replacement of red cells as needed. In the forward areas, if the patient's

general condition was good, blood was not usually given unless the red cell count was less than 3,000,000 per cubic millimeter. Plasma transfusions were used frequently to sustain falling serum protein levels.

If, at the end of 48 to 72 hours after operation, the patient was still unable to take any fluids by mouth, a Levin tube was passed for administration of nourishing fluids. Though this was not always feasible in forward areas, it was carried out whenever possible.

Chemotherapy and antibiotic therapy.—The principles of chemotherapy and antibiotic therapy, once debridement had been completed, were in general the same as for contaminated wounds elsewhere in the body. Chemotherapy was usually well under way by the time operation had been completed, because, as a rule, preoperative treatment with sulfonamide, with or without penicillin, had been started at the first station at which the casualty had been seen and had been continued during the preoperative and operative periods.

All patients with penetrating craniocerebral injuries continued to receive sulfadiazine and penicillin after operation. In general, a daily dosage of 6 to 8 gm. of sulfadiazine, given in divided doses at 4-hour intervals by mouth, or 2 or 3 times daily intravenously, provided an adequate blood level for prophylaxis, that is, 8 to 15 mg. per 100 cubic centimeters. In the presence of actual infection, the dosage was increased to raise the blood level to 15 to 20 mg. per 100 cc. A fluid intake of 3,000 cc. per 24 hours was maintained after the desired blood level was obtained, and the urine was checked frequently for sulfadiazine crystals.

Penicillin was usually administered intramuscularly every 3 hours in doses of 20,000 to 30,000 units. When wound debridement had been delayed beyond 48 to 72 hours in the presence of actual infection, as well as in transventricular wounds and in injuries involving the paranasal air sinuses, this dose was increased to 40,000 units. The administration of penicillin intramuscularly was continued for 3 to 5 days postoperatively in simple compound depressed fractures without dural penetration, for 5 to 7 days in simple penetrating wounds of the brain; and for at least 10 days in wounds treated after delay, transventricular wounds, and craniofacial injuries. Penicillin was given for an even longer time in the presence of any inflammatory reaction about the wound or of persistent rhinorrhea or otorrhea.

Because of the failure of penicillin to penetrate the subarachnoid space in significant amounts, efforts were made to treat the subarachnoid space directly when it had been contaminated grossly. Therefore, in addition to introducing 10,000 to 20,000 units of penicillin directly into the lateral ventricles at operation in transventricular injuries, it became Matson's practice to instill 10,000 units by the lumbar subarachnoid route daily for 4 to 5 days after operation. This prophylactic intrathecal penicillin was also given occasionally to patients with craniofacial injuries when dural defects had been difficult to close.

When the cell count in the spinal fluid was increased or clinical meningeal signs were present, the intrathecal dose of penicillin was increased to 10,000 units, 2 or even 3 times daily. If there was actual gross infection, as much as

30,000 to 40,000 units of penicillin were given intrathecally. Ordinarily, however, doses of over 10,000 units at one ti[illegible]re not used. It was necessary that penicillin used in this fashion be freshl[illegible]d, clear light yellow, and well diluted with spinal fluid or physiologic s[illegible]olution (1,000 units per cubic centimeter) and that it be introduced slowly. Repeatedly, spinal fluid with counts of 500 to 5,000 white blood cells per cubic millimeter, associated with clinical meningeal signs, was observed to clear dramatically after 1 or 2 doses of penicillin introduced intrathecally by lumbar puncture.

Lumbar puncture.—Postoperative lumbar punctures were not done routinely in patients with penetrating craniocerebral injuries but were performed only for the following reasons: (1) Diagnosis and treatment of associated massive subarachnoid hemorrhage; (2) prophylaxis and treatment of meningeal infection, as just described; (3) drainage of sufficient fluid to reduce an elevated pressure to approximately half its initial value, a procedure very useful occasionally for persistent, severe headache unrelieved by ordinary medication or when there were signs of increasing intracranial hypertension; and (4) diagnosis of persistent fever, coma, or unexplained complications of recovery.

Dressings.—There was usually no need to change the dressing or examine the wound before the third or fourth postoperative day if the patient was doing well. At this time, skin sutures were removed if a tight galeal closure had b[illegible] accomplished. The wound was carefully palpated to detect any subgaleal collection of fluid; if fluid was present, it was aspirated.

If drainage had been instituted, the dressing was changed earlier than usual (in 24 to 36 hours), and the drain was removed. Drainage was almost never necessary if all precautions were taken to achieve complete hemostasis as repair was carried out.

Bladder drainage.—In unconscious patients, bladder drainage was usually instituted immediately after operation. This prevented distention, made it possible to measure the urinary output, and also aided materially in the care of the skin and in general management by keeping the bed dry. Chemotherapy was continued as long as constant urinary drainage was continued.

Irrational, uncooperative patients, who were often difficult problems because of incontinence, frequently refused to leave an inlying urethral catheter in place unless their hands were restrained. Frequent changes of linen and blankets were necessary in these cases to prevent the skin from breaking down.

Management of convulsions.—Convulsions were not common during the acute phase of penetrating cerebral injuries but were occasionally seen. The occurrence of repeated convulsions of the Jacksonian variety during the first few days after operation suggested that debridement had been incomplete or that a postoperative hematoma was present in the track of the brain wound or in the subdural or epidural space.

Generalized convulsions also occurred occasionally during the postoperative as well as the preoperative period. During such generalized seizures, care had to be taken to keep the patient's airway clear and to prevent further injury from violent involuntary movements.

Seizures were usually well controlled by the use of barbiturates or paraldehyde. Slow administration of a 2.5-percent solution of thiopental sodium intravenously until relaxation occurred was found to be the most satisfactory method of stopping severe convulsions. A regular prophylactic regimen of sedation was initiated in every patient who had any postoperative seizures.

EVACUATION

A standard schedule for evacuation of patients with craniocerebral injuries from forward hospitals was frequently impossible because of the rapidly moving line of battle and the lack of transportation facilities because of weather. In general, patients with penetrating wounds of the brain traveled better before operation than they did in the first 3 to 5 postoperative days. An attempt was made, therefore, to move them to relatively fixed installations before operation and then keep them there during the early postoperative period. Patients with compound depressed fractures without dural penetration and those with simpler penetrating cerebral wounds were usually evacuated within 2 to 5 days; those with more extensive injuries were kept in forward hospitals for 7 to 10 days if possible.

Unless it was absolutely necessary, patients were not evacuated until they were taking fluids by mouth in adequate quantity. Comatose, highly febrile, or failing patients were not evacuated unless the tactical situation demanded it. All patients with transventricular wounds, cerebrospinal fluid leaks, and usually contaminated wounds were not evacuated for at least 7 days, to insure uninterrupted chemotherapy throughout this period. Whenever circumstances made early postoperative evacuation necessary, every effort was made to apply an especially secure headdressing and to arrange for administration of fluids and chemotherapy during transportation.

Patients with craniocerebral injuries tolerated air evacuation very well at the altitudes ordinarily traveled from the forward areas to hospitals in the communications zone. No hesitation was felt in evacuating them by this means if they were at all ready for transportation.

CASE FATALITY RATES

The case fatality rate of acute wounds of the brain is difficult to determine with any accuracy in military surgery because of the varying circumstances under which these wounds were treated. The rate varied according to the echelon in which surgical intervention was carried out, the type of warfare, the terrain, the speed of evacuation, the availability of trained medical personnel, and the length of lines of communications. When neurosurgical teams functioned close to the scene of combat and received casualties very early (2 to 6 hours after injury), they often operated on patients with wounds of the head who would not have survived to reach a hospital farther to the rear (6 to 24 hours after injury). The surgical fatality rate in the group oper-

ated on early was naturally higher than in those who survived to reach a hospital farther to the rear.

When there was a rush of casualties into one hospital during heavy fighting, patients often succumbed without surgery who might otherwise have been operated on. Also, in spite of the fact that the surgeon's aim should always be the reduction of the overall fatality rate, individual surgeons differed over what constituted operable injuries and in their enthusiasm for tackling extensive lesions.

By far the greatest number of patients with wounds of the head were operated on in evacuation hospitals in all theaters, and it is surprising how uniform the operative fatality rate was in the statistics available.

During a 5-month period in 1944, 12 hospitals in the First U. S. Army admitted 1,236 patients with penetrating wounds of the brain who were operated on; the postoperative fatality rate was 14.1 percent.[34] In a 5-month period, 14 hospitals in the Seventh U. S. Army admitted 949 patients with penetrating wounds of the brain who were operated on, with a postoperative fatality rate of 14.4 percent.[35] In unpublished observations and in reports of individual surgeons (Haynes,[36] Gaynor and Gurwitz,[37] Munslow,[38] Weaver and Frishman,[39] Shearburn and Mulford,[40] Tinsley,[41] Ascroft,[42] and Slemon [43]) from evacuation hospitals, the postoperative case fatality rate ranged from 10.8 to 14.7 percent. These figures are not based on similar periods of followup observation, but, in general, they include all deaths up to the time of evacuation from the hospitals in which the operations were done, which is an average period of observation of 5 to 10 days.

The majority of postoperative deaths occurred within the first 24 to 48 hours. Most of the patients who died within these periods exhibited the syndrome of progressive respiratory depression and irregularity, hyperthermia, deepening coma, and terminal pulmonary edema. At post mortem examination, they were found to have either massive intraventricular hemorrhage (figs. 28 and 30), injury of the brain stem or basal ganglia, or multiple small hemorrhages and areas of contusion throughout the brain. The clinical course was almost certainly very little influenced by the operative procedures carried out.

Death from meningitis in the early postoperative period was almost unheard of in patients whose wounds were properly debrided before clinical evidence of infection. Some of the postoperative deaths occurred in patients who might have recovered from their craniocerebral injuries but for the added

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[34] Annual Report, Surgeon, First U. S. Army, 1944.

[35] See footnote 2, p. 123.

[36] See footnote 15, p. 147.

[37] Gaynor, W. C., and Gurwitz, J.: Experiences With 156 Penetrating Wounds of the Head. Ann. Surg. 122: 12-22, July 1945.

[38] See footnote 19, p. 148.

[39] Weaver, T. A., Jr., and Frishman, A. J.: A Report on the Treatment of Craniocerebral Wounds in an Evacuation Hospital. J. Neurosurg. 3:148-156, March 1946.

[40] See footnote 23, p. 150.

[41] Tinsley, M.: Penetrating Wounds of the Head. Surgery 19: 535-542, April 1946.

[42] See footnote 9, p. 145.

[43] See footnote 16, p. 147.

insult of severe wounds elsewhere in the body, particularly penetrating wounds of the chest and abdomen.

The overall case fatality rate in wounds of the head deserves just as strict an analysis as the postoperative case fatality rate. Martin and Campbell reported that a survey of 14,000 battle casualties treated in the Fifth U. S. Army in Italy in 1944 disclosed that 6.17 percent of the wounds involved the head and that one-third of these were intracranial; 24.4 percent of all deaths in field and evacuation hospitals were attributed to head injuries. These observers also reported 1,000 autopsy examinations made on soldiers who succumbed before reaching aid stations (studied by Tribby [44]); 133 had head wounds only, and 401 had head wounds of sufficient severity to have caused death in association with other wounds.

The overall case fatality rate in patients with penetrating injuries of the brain who reached evacuation hospitals alive averaged about 25 percent. The Seventh U. S. Army (14 hospitals) during an approximately 5-month period in 1944 had 1,176 patients with penetrating injuries of the brain; the overall case fatality rate was 27.8 percent.[45] Surgeons in three evacuation hospitals reported overall case fatality rates of 26.3, 20.7, and 29.0 (Gaynor and Gurwitz, Weaver and Frishman, and Shearburn and Mulford).

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[44] Tribby, William W.: Examination of One Thousand American Casualties Killed in Action in Italy. 1945. 6 vols. [Official record.]

[45] See footnote 2, p. 123.

CHAPTER IX

Penetrating Wounds of the Cerebral Ventricles

Henry G. Schwartz, M. D., and George E. Roulhac, M. D.

HISTORICAL NOTE

In 1918, in a report of 30 battle-incurred wounds of the ventricle confirmed by operation, autopsy, or both, Cushing [1] pointed out that one of the most serious complications of penetrating wounds of the brain is the opening or the traversing of the ventricle by a missile or by bone fragments. Operation under these circumstances in World War I consisted of debridement with blind irrigation of the track of the missile. The dura was not widely opened. The average interval between injury and operation in this series was 25 hours, the range being from 5 to 72 hours.

Eight of the 30 casualties recovered. Death was attributed to infection in 12 cases, to trauma and hemorrhage in 9, and to intercurrent causes in 1. Further analysis of the individual case reports, in which there is some overlapping of mortality factors, reveals that in 5 cases, the primary cause of death was hemorrhage, which was variously subdural, intracortical, or intraventricular. When the 12 cases in which death was due to infection are excluded, the remaining 5 fatalities could be more directly ascribed to the effects of extensive damage or pneumonia.

Haynes,[2] in 1945, reported a series of 100 ventricular injuries sustained in World War II in the Mediterranean and European Theaters of Operations, United States Army. Operation was performed in 77 cases, with a case fatality rate of 33.7 percent and a 10.3-percent incidence of infection. Penetrating wounds were classified on the basis of the size of the missile and the damage incurred. Wounds with narrow tracks, produced by small missiles, were treated by blind suction and irrigation through either the brain needle or a long glass aspirator until the ventricle was reached.

CLINICAL MATERIAL

This chapter is based on an analysis of 50 consecutive battle-incurred wounds of the head with ventricular penetration, all observed in the Mediterranean theater in World War II and all verified by inspection of the ventricular opening at either operation or autopsy. One patient died before oper-

[1] Cushing, H.: A Study of a Series of Wounds Involving the Brain and Its Enveloping Structures. Brit. J. Surg. 5: 558-684, April 1918.

[2] Haynes, W. G.: Transventricular Wounds of the Brain. J. Neurosurg. 2: 463-468, November 1945.

ation, 24 hours after he was wounded. All of the others were operated on. There was no selection of cases, and no patient was denied operation because he was moribund or for any other reason.

In 36 of the 49 surgical cases, primary surgery was performed in forward hospitals. In 31 of the 36 cases, debridement was inadequate, and secondary operation was necessary at the 21st General Hospital, because of infection or retained foreign bodies. There were 10 deaths in this group. The remaining 13 patients had their primary operations at this hospital. There were 4 deaths in this group.

Primary Operation in a Base Hospital [3]

The 13 patients who underwent primary debridement at the 21st General Hospital were seen at intervals varying from 3 to 96 hours after injury. The average interval was 39 hours, but more than half of the patients were seen within 24 hours. All four deaths in this group, as demonstrated by autopsy, could be attributed to damage to vital centers.

Infection developed in only two cases. In one, the scalp was closed under too great tension, and an abscess developed in the wound track. In the other, an abscess formed around a metallic foreign body and leaked into the ventricle. Both patients recovered promptly after adequate surgery. Details of these cases follow:

Case 1.—A 29-year-old soldier, struck in the right parietal region by a mortar-shell fragment on 14 November 1944, was comatose when he was admitted to the 21st General Hospital 17 hours later. The left side was spastic and pathologic toe signs were present bilaterally. Brain and blood clot protruded through a wound in the right parietal region 3 cm. in diameter. Roentgenograms showed a fracture in the right parietal bone, with multiple indriven bone chips, and a large metallic foreign body in the midline at the level of the lateral sinus (fig. 33).

Operation was performed 18 hours after injury. The right parietal wound was debrided. A forked track filled with necrotic brain, blood clot, and bone fragments was followed under direct vision through the posterior portion of the right lateral ventricle and the occipital lobe to the tentorium. Necrotic cerebellar tissue extruded through a hole in the tentorium, and there was considerable bleeding from the posterior fossa. Suboccipital craniotomy was done, and the macerated anterior lobe of the right cerebellum was sucked away.

A track led from the hole in the tentorium to the rostral part of the vermis. The metallic foreign body seen in the roentgenogram was found directly in the midline, just caudal to the aqueduct, and was removed.

Both wounds were closed tightly, though because of loss of skin in the parietal region, it was necessary to fashion and rotate a skin flap to effect closure. By further undermining anteriorly, it was possible to close the donor site as well. This proved an error, since it resulted in too much tension on the suture line.

Chemotherapy was begun immediately after operation. The patient's condition improved, and he began to respond. The cerebellar incision healed per primam. The parietal wound, however, became macerated at the central portion several days after operation, because of too great tension at the suture line, and aspiration beneath the skin flap showed slightly purulent fluid. Examination of this fluid revealed numerous pus cells and

[3] The 21st General Hospital acted as a base hospital as well as a forward hospital at various stages of the campaign in the Mediterranean, particularly in Italy.

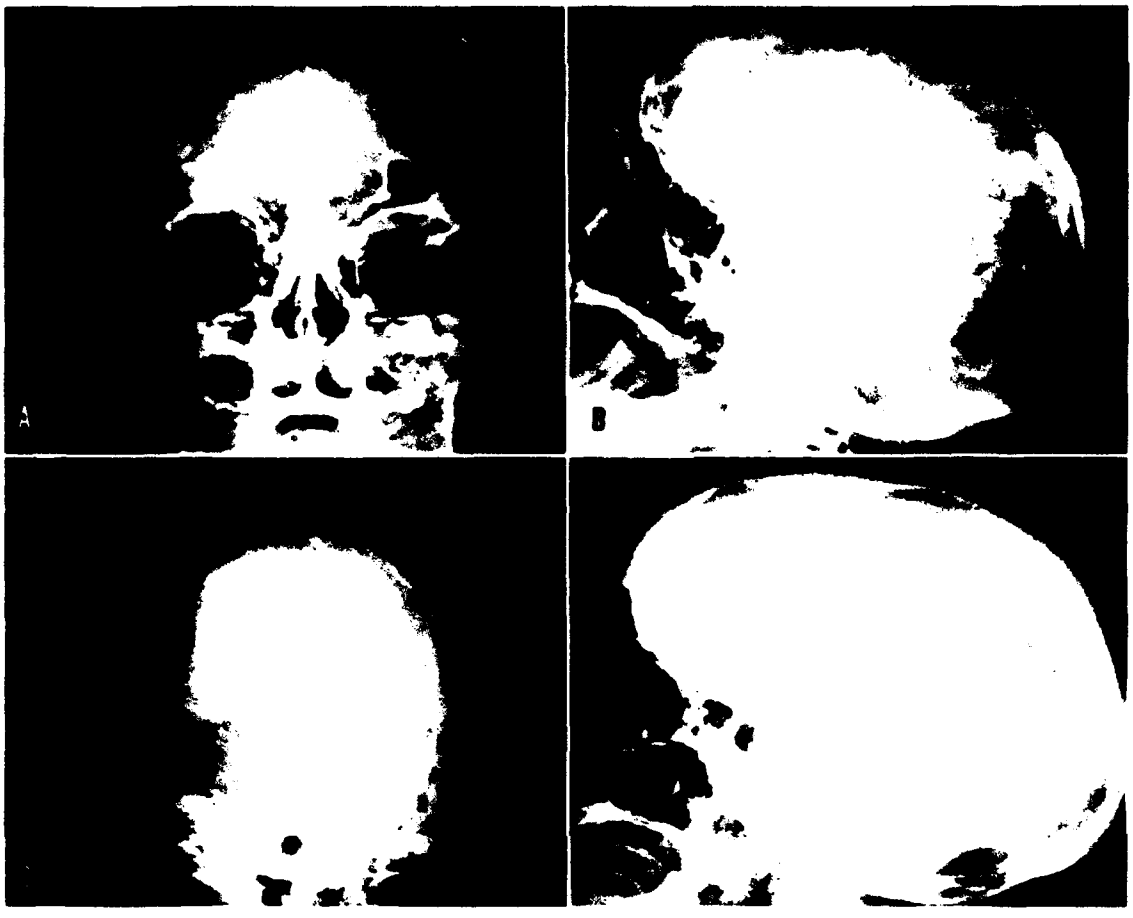


Figure 33. Penetrating wound of right parietal region. A. Anteroposterior roentgenogram of skull before operation, showing bone fragments and metallic foreign body in parietal lobe. B. Lateral roentgenogram before operation. C. Towne's projection of skull after operation, showing clean removal of bone fragments and metallic foreign body. D. Lateral roentgenogram after operation.

gram-positive rods, which, on culture, were thought to be *Bacillus subtilis*. Penicillin was injected locally.

Six days after operation, the patient's condition began to deteriorate. His neck became stiff, and there was a frank leakage of cerebrospinal fluid through the parietal wound. The skin edges were spread apart, and penicillin was injected through the opening in amounts of 10,000 units every 6 hours. Forty-eight hours later, when infection was evidently increasing, secondary craniotomy was done. The wall of the ventricle, after the infected tract was sucked out, was found to be covered with exudate, which was removed. A pocket of foul-smelling pus communicating with the ventricle was also evacuated. Penicillin was instilled before the wound was closed. The donor area of the rotation flap was not sutured, and good closure was obtained over the wound, without tension. Five days after the secondary craniotomy, the donor area was covered with a full-thickness skin graft. Healing occurred without further difficulty.

From the neurosurgical standpoint, recovery thereafter was smooth, but evacuation was delayed until 7 February 1945, because the patient developed otitis media and laryngeal diphtheria. Examination at this time showed left homonymous hemianopsia, bilateral nystagmus, marked ataxia of the right arm and leg, staggering gait, and considerable witzelsucht.

Case 2.—A 21-year-old officer was struck by mine fragments on 15 May 1944. Evacuation was delayed, and he received no definitive treatment. When he was admitted to the 21st General Hospital on 19 May 1944, he was drowsy, and his neck was extremely rigid. There was marked papilledema on the left side. The right eyelids were swollen and shut. On the right frontotemporal region was an extensive area of contusion and laceration. The left arm was weak. A wound of the left foot had not yet been debrided.

Roentgenograms (fig. 34) showed a fracture of the right orbital ridge and a defect at the sphenosquamosal suture line. Several small metallic fragments were seen in the right temporal region. A larger fragment lay just anterior to the right mastoid, and another piece of metal was seen in the occipitoparietal region near the midline.

At operation, which was performed shortly after admission, a defect in the right orbit, which did not extend intracranially, was debrided down through a tear in the orbital capsule. After the temporal muscle had been retracted, a defect was visualized in the anterior part of the squamous bone, just above the zygoma. It was enlarged, and retractors were inserted

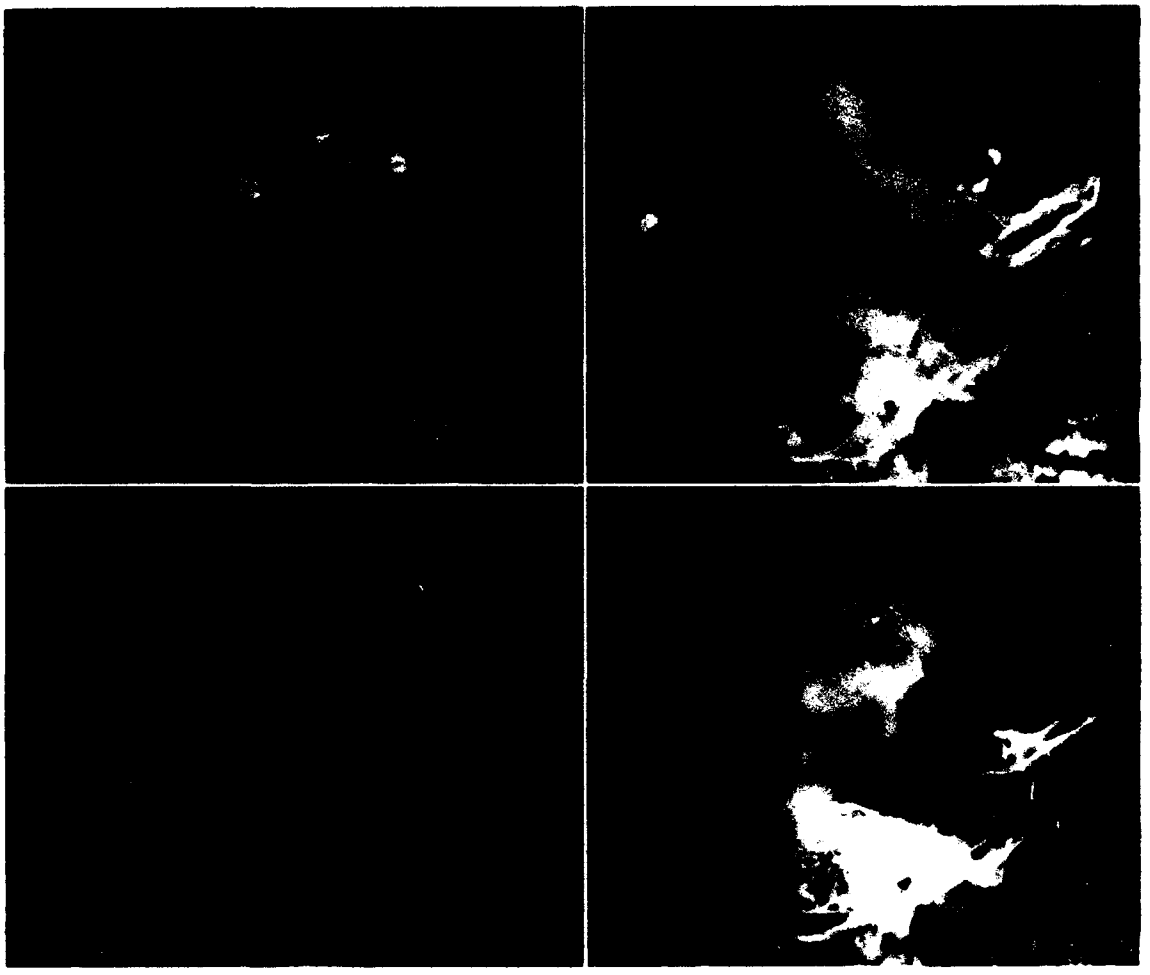


FIGURE 34.—Penetrating wound of right frontotemporal region. A. Anteroposterior roentgenogram of skull before operation, showing metallic fragments in situ, including one large fragment just anterior to right mastoid area (a) and fragment in parieto-occipital region near midline (b). B. Lateral roentgenogram before operation. C. Posteroanterior roentgenogram after operation, showing clean removal of metallic objects in right mastoid and parieto-occipital regions. Metallic objects now present are silver clips used for hemostasis. D. Lateral roentgenogram after operation.

into the mouth of a track which extended posteriorly and medially and from which necrotic brain and blood clot were sucked out. This track was followed into the temporal horn of the ventricle. When the opposite ventricular wall was visualized, it was found to be bruised by the course of one of the metallic fragments. No attempt was made to go beyond the far wall of the ventricle or to remove the fragment in the occipitoparietal region. The wound was closed tightly after hemostasis had been secured. Sulfadiazine was begun at once.

The patient improved after operation, and the head wound healed well. A mild otitis media which developed on the right cleared up after removal of the large metallic fragment from the posterosuperior portion of the auditory canal. On the fifth postoperative day, the patient began to complain of severe headache, and examination showed that the temporal defect was tense.

On 24 May, the wound of entry was reopened. The track leading to the ventricle was grossly clean, and smears and cultures were later reported negative. This wound was therefore closed, and an opening was made with a perforator in the right parieto-occipital region, at a point thought to be near the retained foreign body. Very foul pus was aspirated from an abscess located at a depth of 3 cm. Gram-positive rods were seen on smear. Two days later, following repetition of the roentgenograms to secure better localization, the fragment was removed. It lay in a pocket containing a small amount of pus and was surrounded by necrotic brain tissue. The perforator opening was closed.

After again doing well for 5 days, the patient became drowsy and again showed signs of increased intracranial pressure. On 31 May, following ventriculography, the right occipitoparietal burrhole was reopened. A large quantity of pus at once gushed out. Retractors were inserted through the previously created dural defect. The walls of the track, which communicated with the lateral ventricle, were covered with thick exudate. The track was sucked out, and 10,000 units of penicillin were instilled into it. The wound was closed with silk sutures. A narrow corrugated rubber drain was placed in the wound and left in situ for 48 hours. This was contrary to the usual practice in contaminated head wounds, but at this stage of the war, some hesitancy was still felt about tight closure if clostridial infection was suspected. Bacteriologic examination revealed *Clostridium sporogenes*. The drain was removed in 48 hours, and healing was complete 10 days after operation.

The patient's course following the third operation was entirely satisfactory, though convalescence was delayed by a wound of the left foot, which was closed secondarily on 18 June. Ambulation was then permitted. Examination at this time showed a temporal field defect in the left eye; the central field was intact. Vision in the right eye, because of retinal detachment and vitreous hemorrhage, was limited to light perception. Hearing on the right was 6/20 and on the left 20/20. There was slight weakness of the left arm, but fine hand movements were present on this side.

The patient was evacuated on 14 July 1944. He was followed up by letter for 20 months. During this time, there was no recurrence of infection, vision improved so greatly that he could play as good a game of golf as before his injury, and, when last heard from, he was leading an active life on his farm, just as he had before he entered service.

Two patients returned to combat duty, their reassignment being dictated by the gravity of the tactical situation at the time. Their histories follow:

Case 3.—A 25-year-old French-Arabian soldier, wounded on 13 May 1944, was admitted to the 21st General Hospital in a stupor 18 hours after injury. Examination disclosed a penetrating wound of the right temporal region, and roentgenologic examination revealed a bone defect 3 cm. in diameter; a shower of bone fragments; two small metallic fragments deep in the right parietal lobe; and a metallic fragment in the left parietal lobe just beyond the midline.

At operation, after retraction of the right temporal muscle, a dural defect 1.5 cm. in length was seen, through which a mass of necrotic brain tissue and blood clot gushed. Retractors were inserted into the missile track, which traversed the body of the right

ventricle, and bone chips and all damaged brain tissue were removed. The group of bone fragments was removed from the ventricle. The track was followed for a depth of 7 cm. The two small metallic fragments in the right parietal lobe were removed. It was not thought that any indication existed to go through to the opposite side of the brain, to remove the metallic foreign body in the left parietal lobe. As debridement progressed, the previously pulseless brain began to pulsate freely.

After operation, there was temporary weakness of the left arm and the face, which cleared completely within 2 weeks. The patient developed a low-grade pneumonia but otherwise had a smooth convalescence. He was out of bed on the ninth day. Because of the current tactical situation and his own great desire to return to his regiment, he was permitted to return to combat duty with his outfit on 1 June 1944, 18 days after wounding.

Case 4.—An Arab soldier was admitted to the 21st General Hospital on 30 January 1944, 48 hours after he had sustained a tangential wound in the left frontoparietal region. He presented weakness of the right arm and face and was aphasic. Operation consisted of debridement with removal of 15 indriven bone fragments (fig. 35). The missile track extended into the anterior horn of the left ventricle. The falx was visualized at the end of the track. Tight closure was effected, after mobilization of the scalp by means of a modified Isle of Man incision.

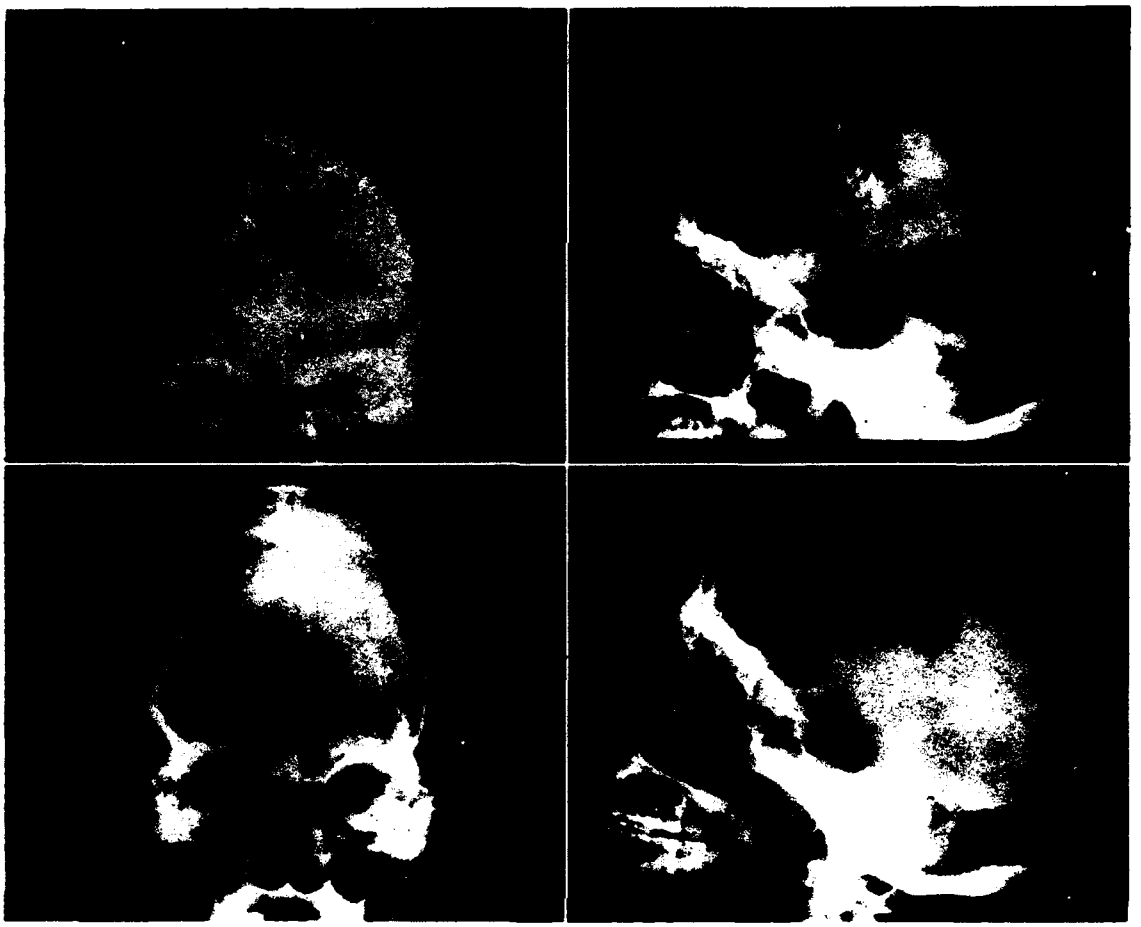


FIGURE 35.—Penetrating wound of left frontoparietal region. A. Posteroanterior roentgenogram of skull before operation, showing numerous indriven bone fragments. B. Lateral roentgenogram before operation. C. Anteroposterior roentgenogram of skull after operation, showing clean removal of bone fragments. D. Lateral roentgenogram after operation.

The wound healed per primam. Sensory aphasia disappeared within 48 hours and weakness of the right arm after 2 weeks. Motor speech returned at the same time. The patient returned to full duty on 25 February, 27 days after wounding.

Primary Operation in Forward Hospitals

Of 36 patients with ventricular injuries who reached the 21st General Hospital after primary debridement in forward hospitals, 31, as already mentioned, required secondary surgery. In this group were 12 patients with abscess, 3 with meningitis, and 14 with meningitis and ventriculitis. Two other patients had retained bone chips but presented no clinical evidence of infection. The original operations had been performed from 3 to 29 hours after injury, the average being 15 hours. Ten of the thirty-six patients died, infection being responsible for nine deaths and brain damage for the tenth.

PATHOLOGIC PROCESS

The pathologic process in wounds involving the ventricles needs little comment, since it is essentially the same as was seen in other wounds produced in World War II by high-explosive shell fragments or bullets. The first effect of the injury was a shattering of the skull, the extent depending upon the size of the missile involved and the angle at which it struck. The extent of the dural tear, which was always jagged, depended upon the size and velocity of the missile, the comminution of the skull, and the number of indriven bone chips and foreign bodies. The missile track was usually filled with pulped brain, blood clots, and bone and metallic fragments. The size of the opening into the ventricle depended upon the size of the missile and the angle at which it had traversed the ependyma. The ventricle might be filled with bright red blood or blood clot and with bone chips, hair, and other material, or the opening might be only a tiny slit which had sealed off under pressure of surrounding edematous brain.

If debridement was incomplete and an abscess developed in the missile track, pus might leak into the ventricle to produce ventriculitis. If the ventricle itself was not cleaned out, infection might develop primarily within the ventricular system.

Failure to effect hemostasis in a bleeding choroid plexus might produce clots in the entire system, or on a single side, with a block of the foramen of Monro. Similarly, infection within the ventricles which produced a shaggy exudate over the ependyma and choroid plexus might extend throughout the system and might lead to foramina block.

DIAGNOSIS OF VENTRICULAR PENETRATION

It did not prove possible to diagnose ventricular penetration on the basis of symptomatology. Signs present in any given case depended upon the area

of cerebral involvement rather than on penetration of the ventricular wall. The patients were usually seriously and acutely ill, as was to be expected from the amount of cerebral tissue which must be damaged before a missile or bone fragments could reach the depth at which the ventricles lie. On the other hand, a few patients in whom the track of brain damage was narrow and hemorrhage was slight might present a deceptively good general appearance.

Ventricular penetration was suspected whenever roentgenologic examination revealed a track outlined by bone chips leading toward the normal position of the ventricle, though it was not always found to exist under these circumstances. The diagnosis could be made with reasonable certainty whenever films showed, as they occasionally did, a radiopaque foreign body which shifted with movements of the head.

Routine anteroposterior and lateral stereoscopic roentgenograms of the skull were found to be of particular value when ventricular penetration was suspected. Their diagnostic aid was twofold: (1) They gave a dimension of depth to foreign bodies, and (2) they also assisted in the detection of shifts of position. Furthermore, accurate visualization of the pattern of a group of bone fragments often confirmed the suspicion of ventricular penetration.

MANAGEMENT

Treatment of ventricular wounds did not differ from the treatment of other penetrating wounds of the brain, though the procedure was more serious because of the depth at which the ventricles lie.[4] As experience accumulated during the war, radical surgery with closure became the routine practice in almost all cases, even when gross evidence of infection existed.

Operation

Patients suspected of having ventricular injuries were given first priority for surgery, because the longer the interval between wounding and operation, the greater was the risk of infection or clots in the ventricles. Preliminary treatment for shock or dehydration was begun promptly, and operation was performed as soon thereafter as possible.

The crux of treatment of penetrating ventricular wounds was adequate, thorough, aggressive debridement of the entire missile track, including the ventricle, under direct vision. It was soon found unsafe to assume that adequate removal of all pulped brain, clot, hair, bone, and metal could be done without visualization of these foreign materials. Debridement was inadequate without visualization, and there was always danger of producing extensive damage outside of the missile track and of causing further hemorrhage. Following the track was seldom difficult. As narrow malleable retractors were placed deeper and deeper and pulped brain and clot were removed, tension

[4] Schwartz, H. G., and Roulhac, G. E.: Craniocerebral War Wounds, Observations on Delayed Treatment. Ann. Surg. 121: 129–151, February 1945.

fell rapidly, and the track could readily be spread, without danger of additional damage to the brain.

Radical, complete debridement with primary closure was equally essential in late secondary cases with abscess and ventriculitis. Since the cure of ventriculitis could not be expected until the source of contamination was removed, the abscess was evacuated under direct vision, and the shaggy exudate was thoroughly sucked off the walls of the track. The ventricle was then inspected, and blood clot and fragments of bone were removed, as were metallic foreign bodies if they could be readily reached, without inflicting additional trauma. Metallic foreign bodies usually proved innocuous, but they occasionally required removal by drastic means.

Closure of the dura was accomplished in most cases, whether operation was primary or late secondary, by grafts of pericranium, temporal fascia, or fascia lata. The danger of loss of a graft through infection was negligible in a thoroughly debrided ventricular wound, just as it was in uncomplicated penetrating brain wounds. On the other hand, the presence of a watertight dural closure minimized the danger of a ventricular fistula if a scalp wound which had been closed under too much tension gave way. If the dura was not closed, there was a tendency for ventricular fluid to collect under the widely mobilized scalp, thus inhibiting galeal-pericranial apposition and keeping the suture line constantly under tension. If great care was not taken, the wound was likely to spread from tension, and a fistula might develop, with disastrous consequences from infection.

Fresh pericranium or fascia proved the material of choice for watertight closure. In two cases in this series in which the ventricles were widely opened, fibrin film was used as a dural substitute. The technique was not satisfactory; fluid continued to accumulate beneath the galea until the film was removed and was replaced with fascia.

Chemotherapy and Antibiotic Therapy

There is no doubt that complete debridement was the factor in World War II which chiefly decreased the morbidity and mortality of penetrating wounds of the brain. The role played by chemotherapy and antibiotic therapy, while it was undoubtedly extremely important, is difficult to evaluate precisely because these agents were so often given indiscriminately.

Sulfadiazine and penicillin were used parenterally, alone or together in various combinations, in wounds of the ventricles, and it is believed that they were effective. In addition, in penetrating ventricular wounds, it was the usual practice to instill 10,000 to 20,000 units of penicillin into the ventricle before closure. This treatment proved adequate in cases in which signs of ventriculitis were not present. If definite ventricular infection was found at operation, penicillin, in amounts of 20,000 to 50,000 units was administered daily by the intrathecal route until all signs of infection had disappeared. If there were some contraindication to intrathecal administration, such as internal hydro-

cephalus, the penicillin was injected directly into the ventricles through the bone defect or through perforator openings made especially for this purpose. No deleterious consequences were observed to follow the intraventricular injection of penicillin.

FACTORS OF MORTALITY

Analysis of the material in this series and of comparable material reported by other neurosurgeons revealed three factors of paramount importance in wounds of the ventricles: (1) the introduction of infection with resultant ventriculitis; (2) hemorrhage into the ventricular system; and (3) associated damage to vital centers.

Infection With Resultant Ventriculitis

The introduction of infection has, properly, always been a greatly feared complication of ventricular penetration, and its serious potentialities were evident in these cases. Treatment was directed toward removal of potential sources of infection, or toward the eradication of infection if it had already occurred. The following case reports are illustrative:

Case 5. —A 26-year-old soldier, wounded in the left frontal region by a shell fragment on 5 March 1944, was submitted to craniotomy 24 hours later at an evacuation hospital. A missile track 7 cm. long was cleaned out, sulfanilamide was introduced, and the wound was closed with a drain. Chemotherapy was instituted immediately after operation. When the drain was removed on the following day, leakage of cerebrospinal fluid occurred.

When the patient was admitted to the 21st General Hospital on 11 March 1944, he had a defect in the lower part of the wound through which sanguinopurulent material was draining. Compression of the upper portion of the wound caused gas to escape through the defect. Cultures of the pus were reported positive for staphylococci and streptococci. Roentgenologic examination (fig. 36A and B) revealed a large frontal defect involving the supraorbital ridge and the left frontal sinus, with extensive linear-fracture lines. In the left frontal region, in a position corresponding to the anterior horn of the ventricle, was a large nest of retained bone fragments. Lumbar puncture revealed the pressure elevated to 310 mm. H_2O. The fluid was cloudy and contained 250 cells per cubic millimeter.

At secondary craniotomy on 14 March 1944, a large amount of pus and necrotic brain tissue was removed. Retractors were inserted through a track 8 cm. long and 1.5 cm. wide, which led directly to the anterior portion of the lateral ventricle. Four bone chips were removed from the depth of this track. The wound was closed without drainage.

The patient improved steadily for 5 days. Then, when the lower end of the wound was spread, 10 cc. of pus escaped. Purulent drainage steadily increased, and the patient became aphasic. Repeated smears and cultures showed gram-positive rods. Roentgenograms revealed that the nest of bone chips previously seen in the left frontal region had been removed (fig. 36C and D). Another exploration revealed an abscess just beyond the limits of the previous exploration.

Thereafter the patient's course was steadily downhill. Signs of meningitis progressed. Bilateral postparietal burrholes were made on 4 April, and 10,000 units of penicillin were injected daily into each ventricle, a total of 155,000 units being administered. Sulfadiazine and penicillin were also given parenterally. Death occurred on 12 May 1944. Autopsy revealed extensive encephalomalacia of the left hemisphere, basal ganglia, and thalamus; marked left frontal cerebritis; and lateral ventriculitis.

A review of this case indicates that the infection was the result of incomplete debridement with retention of bone fragments. A very careful reexamination of the roentgenograms (fig. 36) showed a barely perceptible bone chip which lay in the posterior parietal region and which had been overlooked in the concentration of attention on the prominent nest of chips in the anterior horn. When the preoperative and postoperative films were compared, it could be

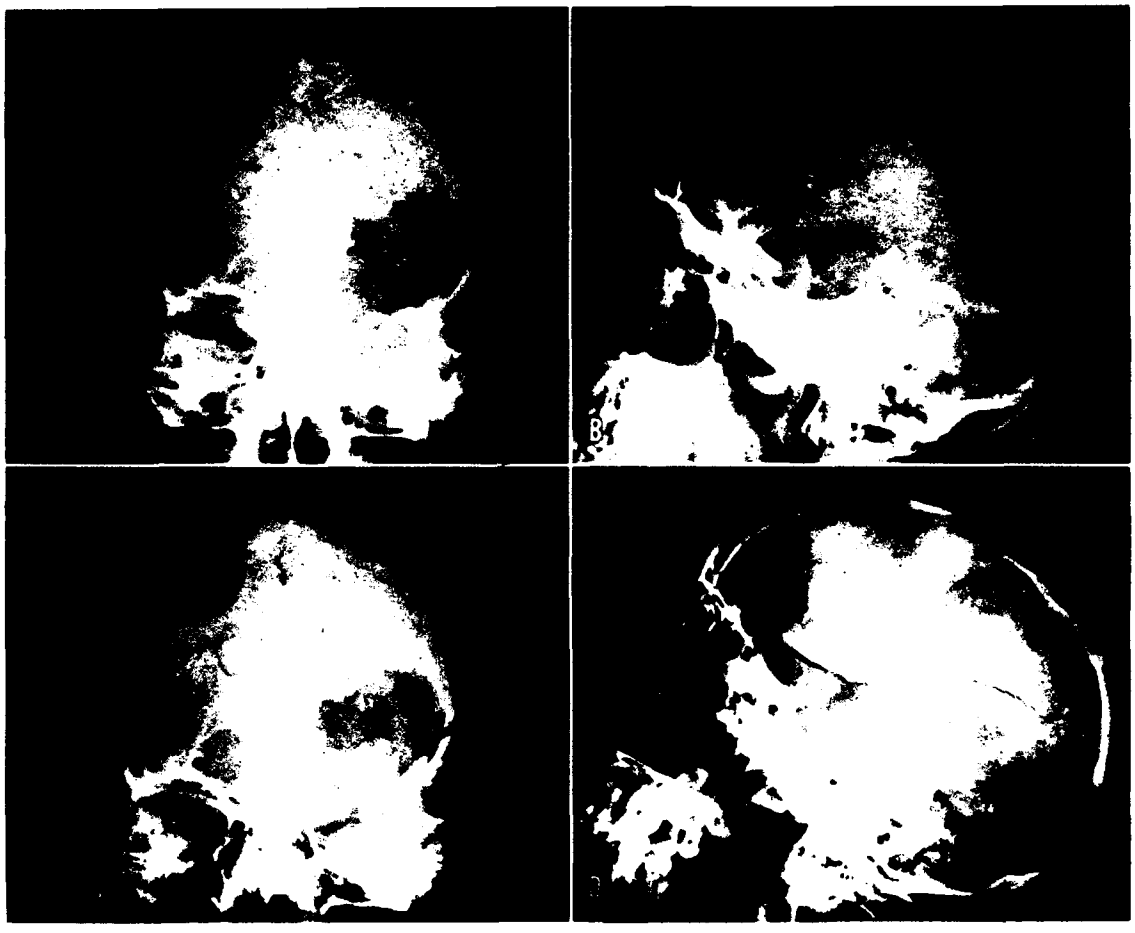


FIGURE 36. Penetrating wound of left frontal bone. A. Anteroposterior roentgenogram of skull after primary operation. Note cluster of retained bone fragments overlooked at this operation. Note also silver clips close to midline. B. Lateral roentgenogram after primary operation. C. Anteroposterior roentgenogram of skull after secondary debridement. D. Lateral roentgenogram after secondary operation. Silver clips are still in situ. All bone fragments have now been removed, except for one small fragment in occipital horn of lateral ventricle.

seen that the chip lying in the posterior parietal region, though it had shifted its position, was still present. This chip obviously lay in the occipital horn. Had it not been overlooked, it is possible that its removal through a separate approach might have altered the outcome. No penicillin was available during the patient's first days in the hospital, but it is doubtful that the omission of antibiotic therapy at this time had anyting to do with the final outcome.

Case 6. A 20-year-old French soldier received a midline parieto-occipital wound on 15 November 1944, for which operation was performed at an evacuation hospital 25 hours later. Because of bleeding from the lateral sinus, debridement could not be completed, and the wound was packed with gauze. Five days later, without further attempt at debridement, secondary closure was carried out with a transverse skin-relaxing incision.

When the patient was received at the 21st General Hospital on 25 November, his neck was stiff, he was disoriented, and he presented complete amaurosis. The transverse wound over the occiput was broken down and infected, and the relaxing incision was also infected. There were bilateral hernias of the occipital lobe. Roentgenograms (fig. 37A and B) revealed numerous indriven bone fragments. Sulfadiazine therapy was begun.

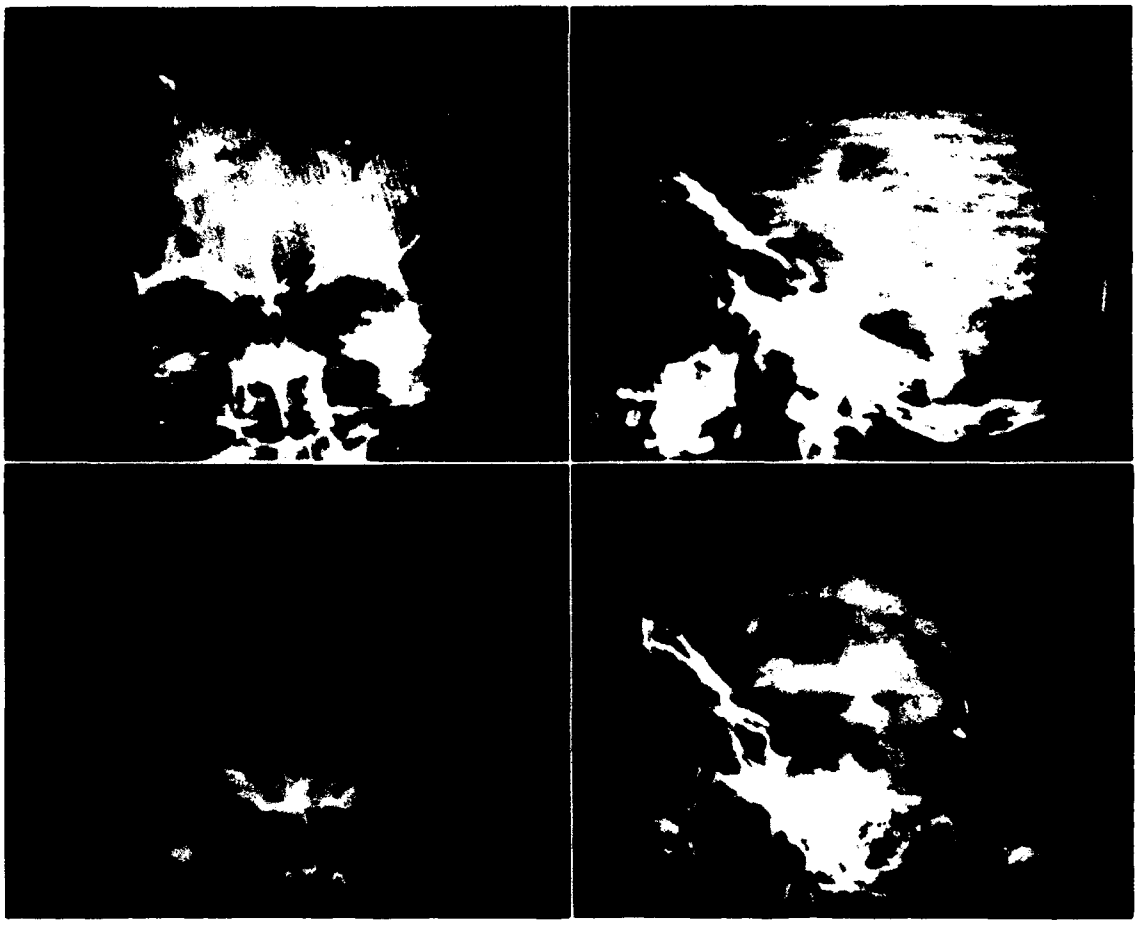


FIGURE 37.—Penetrating wound of parieto-occipital region in midline. A. Posteroanterior roentgenogram of skull after primary operation. Arrow indicates residual bone fragments. B. Lateral roentgenogram after primary operation. C. Posteroanterior ventriculogram after definitive surgery, showing clean removal of fragments. Note air in ventricular system, resulting from opening of occipital horn of left lateral ventricle. D. Lateral roentgenogram after definitive operation.

A severe cellulitis of the scalp was somewhat improved by the use of wet dressings. On 28 November, the wound edges were excised and spread apart. The bone defect was enlarged, and the longitudinal sinus was exposed; it had been completely destroyed just anterior to the torcular Herophili for a distance of 2 cm. Two large bone fragments were removed from the stump of the sinus, and bleeding was controlled. Two tracks in the left occipital fungus were cleaned out to a depth of 3 cm. each. The wound was left open because the

procedure, on account of the overwhelming scalp infection, had been planned merely to remove bone chips and establish adequate external drainage.

The patient steadily improved on sulfadiazine therapy until 9 December, when the temperature became elevated and cervical rigidity returned. Penicillin was given intrathecally. The scalp became much cleaner under local treatment, and another operation could be performed on 16 December. A track was found, just lateral to the left occipital fungus, which led directly into the occipital horn of the lateral ventricle. After the track had been debrided, the body and temporal horn were well visualized. The ventricular fluid was cloudy, and the wall was smooth and glistening. The choroid plexus was coagulated, clipped, and excised. It was thought wiser not to close the dura with a graft at this time. A single pedicled flap of scalp was mobilized to cover a defect 12 by 4 cm., and the scalp was closed after 10,000 units of penicillin had been instilled into the ventricle.

In spite of daily intrathecal injections of 20,000 units of penicillin, signs of meningitis persisted. Spinal fluid cultures were consistently positive for *Staphylococcus albus* (*Micrococcus pyogenes* var. *albus*). On 28 December, the patient became much worse. Lumbar puncture did not affect the tension of the occipital defect, but puncture of the right ventricle through an anterior burrhole readily decompressed it, which indicated that a block existed at or below the aqueduct.

Penicillin was injected into the ventricle daily, and by 6 January, the block had disappeared, and the patient was greatly improved. Large amounts of fluid, however, continued to collect below the scalp defect, and the wound was reopened on 16 January. The porencephalic cavity in the left occipital lobe measured 60 cc., and there was an opening into the occipital horn 8 mm. in diameter (fig. 37C and D). The track was clean except for a small amount of exudate at the ventricular opening. The dura was closed tightly with a fascia lata transplant, and the scalp was closed in layers with silk sutures.

The patient showed progressive improvement following this last operative procedure, though his course was still interrupted by repeated flareups of staphylococcic meningitis. Penicillin therapy was continued, and the fluid was permanently clear after 10 February. By this time, the patient was able to get about, the wound was solidly healed, and there was light perception in both eyes. When he was evacuated to a French hospital on 7 March, neurologic examination was negative except for visual loss.

Hemorrhage Into the Ventricular System

Aggressive efforts were always made to arrest or prevent hemorrhage and to remove as much blood and blood clot from the ventricles as possible. Ventricular hemorrhage was a less frequent complication than infection in these cases, probably because patients with serious hemorrhage died soon after injury and did not reach rear hospitals. The infrequency of hemorrhage in this series of cases of course in no way lessens the importance of this complication, nor did it in any way modify the practice of removing a blood clot in the ventricle under direct vision.

A typical history of hemorrhage follows:

Case 7.—A 27-year-old soldier, who had received a penetrating wound in the right midparietal region on 22 December 1944, suffered immediate paralysis of the left arm and leg but was able to talk on admission to an evacuation hospital, where operation was performed 26 hours after injury. Pulped brain and 200 cc. of blood clot were removed, together with bone spicules, and the wound was closed after the instillation of 50,000 units of penicillin. On the sixth postoperative day, meningitis developed, and intrathecal treatment with penicillin was instituted.

When the patient was admitted to the 21st General Hospital on 5 January 1945, he was aphasic, and there was complete left hemiplegia, with loss of fine discriminatory sense and position sense on the left. He also presented bilateral papilledema. The wound had broken down, and there was an area of infection 3 cm. in diameter near the center. Roentgenograms showed several large bone fragments deep in the parietal lobe, with one fragment shifting its position (fig. 38 A, B, and C

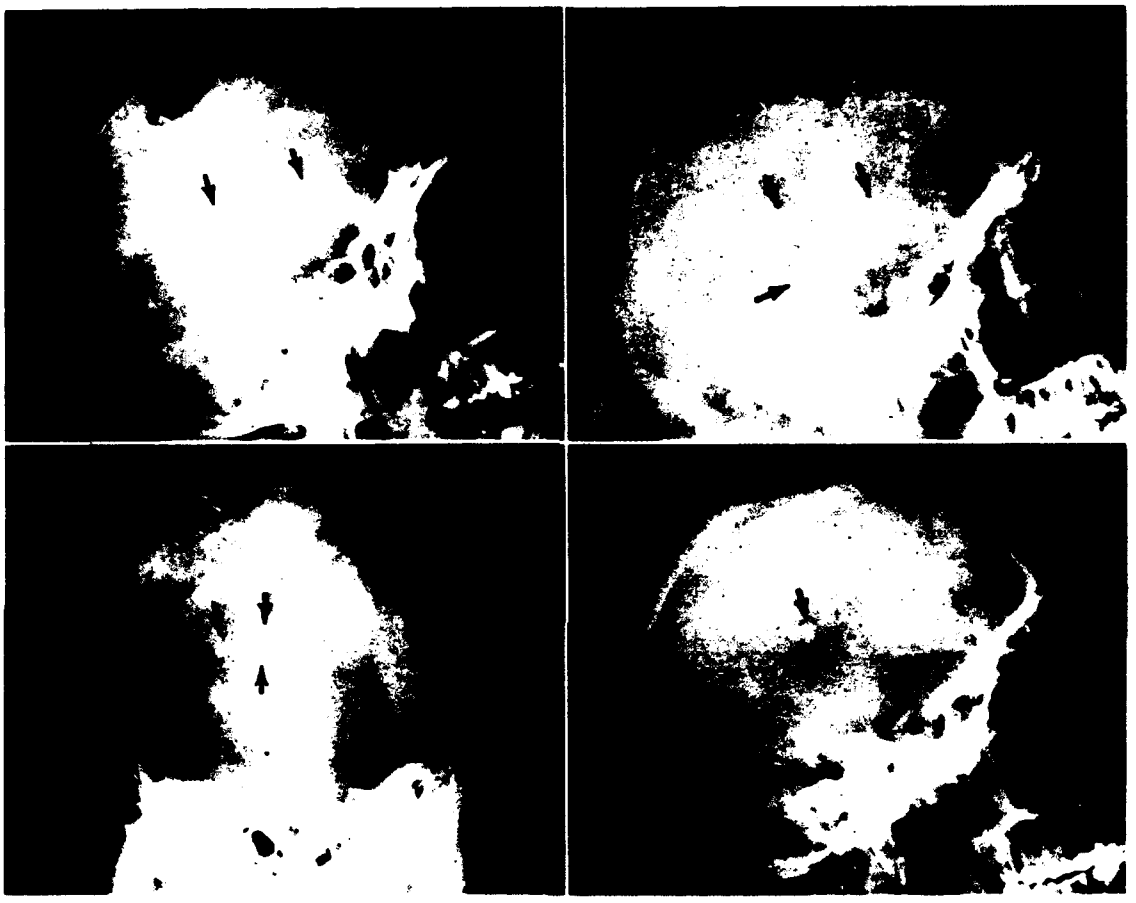


FIGURE 38. Penetrating wound of right midparietal region. A. Lateral roentgenogram of skull after primary operation, showing three groups of retained bone fragments (indicated by arrows). B. Same. C. Anteroposterior roentgenogram of skull before operation. D. Lateral roentgenogram of skull after secondary operation, showing retained bone fragment which has shifted its position from the temporal horn to the body of the lateral ventricle.

Secondary craniotomy was performed on 7 January 1945. After excision of the infected scalp and removal of a dural graft, the entrance of a missile track was found in protruding brain. Retractors were inserted, and a large mass of old clot and necrotic brain tissue was excised. The resulting cavity, which was 60 cc. in volume, communicated, at a depth of 5 cm., with a very large defect in the superior lateral wall of the right lateral ventricle. This ventricle was very large and was distended with an organized clot, on removal of which the entire ventricular body could be visualized and explored through the large lateral opening. Four bone fragments were found lying in the ventricle, and a larger fragment was found adjacent to the foramen of Monro. It was thought, however, that the dilatation of the ventricle was caused by the large clot present rather than by blockage of the foramen by the bone chip. The dura was closed with a facial transplant

after instillation of 20,000 units of penicillin. The scalp was closed by means of a rotation flap, and the donor area was covered with a full-thickness skin graft.

The wound healed per primam, papilledema rapidly cleared, and the patient's improvement was steady. Roentgenograms, however, revealed a retained bone fragment which shifted its position between the temporal horn and the body of the ventricle (fig. 38D). Since cultures made at the first operation had been positive, it was thought best that this fragment be removed. This was done on 20 January, through a temporal approach; operation had to be delayed because the patient had contracted laryngeal diphtheria.

All wounds healed promptly, though about a month after the first operation in the base hospital, the parietal wound became puffy at one point, and exploration revealed a small cotton pledget beneath the galea. It was removed without difficulty. Aphasia began to clear in a week after the last operation. The patient was then able to report that as a child he had been left handed. When he was evacuated on 21 March 1945, the eye grounds were normal, though there was left homonymous hemianopsia, and his speech was improved, though it was by no means normal. The left arm was still paralyzed, but considerable power had returned in the left thigh.

Associated Damage to Vital Centers

Most series of ventricular injuries, like this one, make it clear that a fairly large proportion of fatalities can be expected in this type of wound from associated damage to vital centers. This is a factor which is not particularly amenable to improvements in therapy. The fact that there were only six deaths from this cause in this series (including the death which occurred before operation) is explained by the seriousness of such damage; most patients did not live long enough to permit operation. On the other hand, some patients, as the following case history shows, may survive long enough to undergo extensive surgery only to die days or even weeks later. This particular patient withstood the effects of intraventricular hemorrhage but succumbed to a lesion of the brain stem.

Case 8.—A 23-year-old soldier sustained penetrating wounds of the left frontal and parietal regions on 31 May 1944 (fig. 39A and B). He lost consciousness immediately and remained comatose. Debridement was undertaken at an evacuation hospital 24 hours after injury, but the patient's condition was so poor on the operating table that a complete operation had to be abandoned. The scalp was closed.

When he was received at the 21st General Hospital on 4 June 1944, he was still in coma. Examination revealed a sutured scalp wound, 6 cm. long, in the left parietal region. Both wounds were tense and did not pulsate. Roentgenograms showed bone defects underlying both wounds. Two small bone chips were seen in the parietal lobe, and a metallic fragment was seen in the midline near the base (fig. 39C). After removal of the skin sutures, the scalp wounds began to gape, and the parietal wound broke down and discharged foul pus.

On 7 June, the parietal wound was opened widely, and the purulent cerebral hernia was amputated. A necrotic track was followed for 3 cm. During this procedure, the brain remained quite tense. It began to pulsate, however, when a ventricular needle was introduced and a large quantity of blood clot and old blood was aspirated. Because of the patient's poor condition, it was thought best to attempt to tide him over with ventricular punctures rather than to explore the ventricle under direct vision. Cultures on both aerobic and anaerobic media showed staphylococci.

The patient's condition remained poor. When he again showed evidence of pressure, ventricular puncture was done on 10 June, and 40 cc. of old brown blood mixed with clot

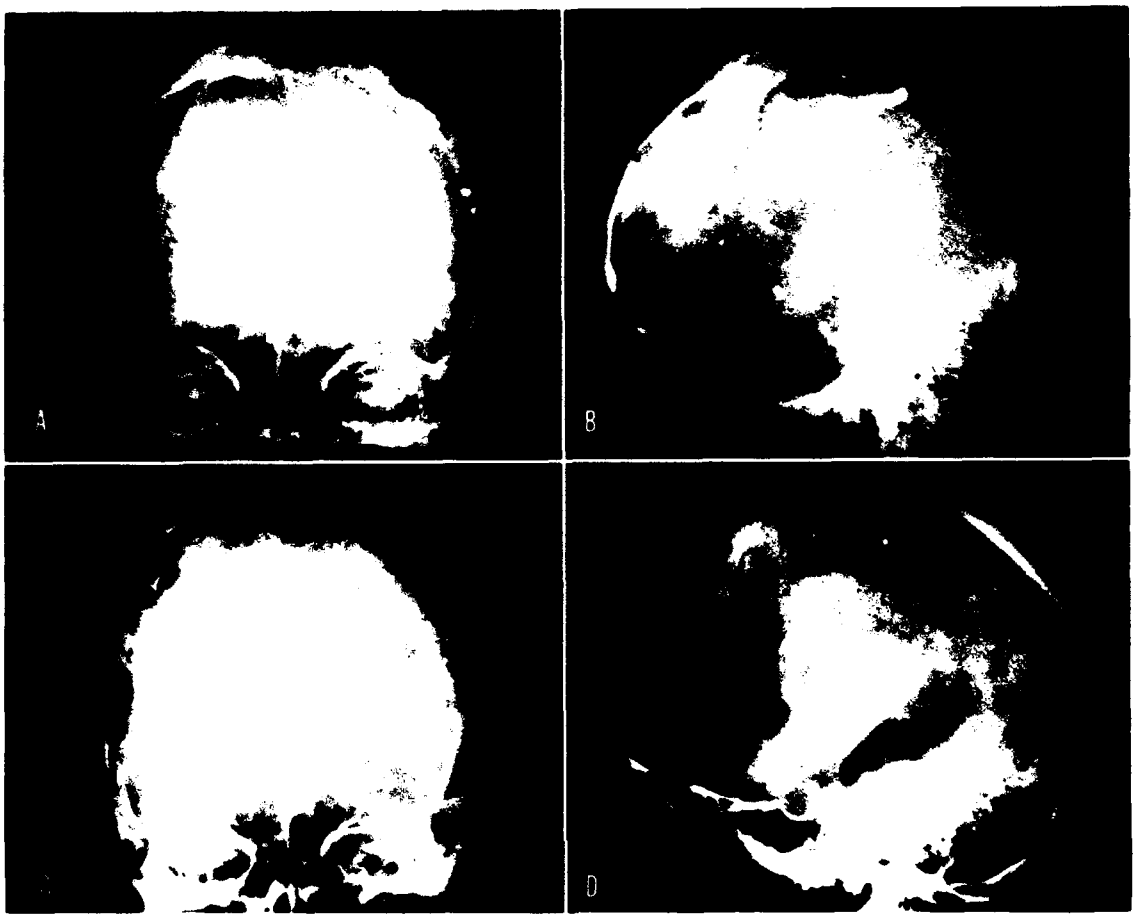


FIGURE 39. Penetrating wounds of frontal and parietal regions. A. Anteroposterior roentgenogram of skull before operation, showing multiple bone and metallic fragments. B. Lateral roentgenogram before operation. C. Posteroanterior roentgenogram of skull after incomplete primary operation. Note retained metallic foreign body, probably in vicinity of brain stem. D. Lateral ventriculogram after primary operation. Incomplete filling is due to retained intraventricular blood clot.

were evacuated. The brain remained tense, and further ventricular aspiration seemed futile, since a good deal of clot was thought to be still present. A ventriculogram (fig. 39D) showed a large defect in the dilated left ventricle, and it was obvious that removal of the clot was necessary, in spite of the patient's precarious condition. Retractors were therefore introduced down to the ventricle, which was found dilated by a large old clot surrounded by a thin membrane attached at several points to the choroid plexus. A large piece of clot, separate from the main mass, was found plugging the foramen of Monro. After removal of the clot and membrane, the walls of the track through which retractors had been introduced fell away, leaving a cavity 3.5 cm. wide and 6 cm. deep, which communicated freely with the dilated left ventricle. The dura was closed with a fascia lata transplant.

The patient failed to improve after this operation, and death occurred on 13 June. At autopsy, the missile track was clear, and the left ventricle, which was collapsed, was found free of clot and exudate. Extending through the brain substance on the left side from the mesencephalon into the pons was a hemorrhagic area 1 cm. in diameter and 5 cm. long. The metallic fragment visualized by roentgenogram was found in the center of a necrotic area 2 cm. in diameter in the very center of the pons.

SUMMARY AND CONCLUSIONS

This chapter has as its background 50 consecutive penetrating wounds of the ventricle, in all of which ventricular penetration was verified by direct inspection at operation or at post mortem examination.

Fifteen of the fifty patients died, including one who died before operation could be carried out. In 36 cases in which primary operation was carried out in forward hospitals, there were 10 deaths, 9 due to infection and 1 to brain damage. In 31 of these cases, secondary operation at the base hospital was necessary. There were 4 deaths, all due to brain damage, in 13 cases in which primary operation was done at the base hospital.

In case of ventricular injury in which survival is sufficiently long for operation to be done, infection is responsible for fatalities at least as often as is brain damage. Associated injuries are responsible for a small proportion of deaths, the number not being greater because death usually occurs promptly.

Prompt, thorough debridement of the entire missile track, including the ventricle, is the most effective means of lowering the morbidity and mortality rates in penetrating ventricular injuries. It must be carried out under direct vision. The same treatment is indicated in infected cases, with removal of debris, retained bone fragments, and pus. Wounds should be closed primarily, without drainage.

Chemotherapy and antibiotic therapy, no matter how they are administered, are effective only when they are combined with complete surgery.

CHAPTER X

Infections Following Acute Gunshot Wounds of the Brain

Stuart N. Rowe, M. D.

The immediate fatalities which follow penetrating wounds of the brain, as well as the fatalities which occur shortly after wounding, are a result of destruction of vital areas of the brain. Infection is the principal cause of death in casualties who survive the initial effects of cerebral wounds only to die later. Much of the treatment carried out from the time of wounding until final recovery must therefore be directed toward the prevention of infection or, if it has already occurred, toward its control.

In World War I, a discouragingly high toll was taken by infection following acute gunshot wounds of the head.[1] Between World Wars I and II, great strides were made in the prevention and treatment of bacterial diseases, and it is therefore not surprising that in World War II fatalities from infections following penetrating wounds of the head should have been reduced to a figure slightly less than 4 percent. Reports from different surgeons in different theaters of operations revealed case fatality rates well in accord with this overall figure.

The incidence of infection varied with the level of the hospital. According to Haynes,[2] infection occurred in 12.6 percent of the injuries of the brain observed in evacuation hospitals. Webster, Schneider, and Lofstrom[3] and Maltby,[4] reporting from general hospitals, found the incidence to be 16 percent and 23.5 percent, respectively.

PREVENTION OF INFECTION

The most important single measure in the prevention of infection was careful, complete debridement, which implied the removal of all necrotic brain tissue, all blood clots, and all fragments of bone, as well as the achievement of a very dry field before closure.

[1] (1) Cushing, H.: A Study of a Series of Wounds Involving the Brain and Its Enveloping Structures. Brit. J. Surg. 5: 558–684, April 1918. (2) Kerr, H. H.: A Statistical Analysis of Gunshot Wounds of the Head. *In* The Medical Department of the United States Army in the World War. Washington: Government Printing Office, 1927, vol. XI, pt. I, pp. 841–847.

[2] Haynes, W. G.: Penetrating Brain Wounds. Analysis of 342 Cases. J. Neurosurg. 2: 365–378, September 1945.

[3] Webster, J. E., Schneider, R. C., and Lofstrom, J. E.: Observations on Early Type of Brain Abscess Following Penetrating Wounds of the Brain. J. Neurosurg. 3: 7–14, January 1946.

[4] Maltby, G. L.: Penetrating Craniocerebral Injuries: Evaluation of the Late Results in a Group of 200 Consecutive Penetrating Cranial War Wounds. J. Neurosurg. 3: 239–249, May 1946.

Closure of the scalp wound was an essential part of debridement, but it was equally essential that the closure be effected without tension. The pressure of patients awaiting surgery and the comparative ease with which the wound edges could be pulled together with heavy silk sutures sometimes encouraged the surgeon to use this method of closure, to save time. When the patients reached neurosurgical centers in the communications zone, the damaging effects of this technique were frequently apparent. The sutures had cut through the skin. The wound edges had separated. Healing was unsatisfactory. The end result was almost invariably secondary infection.

The course of events just described was entirely unnecessary. Curved scalp incisions, wide undermining, rotation of flaps, and sometimes relaxing incisions all were feasible and all permitted closure with two layers of interrupted silk sutures, without excessive tension.

Early surgery.—The policy of early surgery in evacuation hospitals was practiced whenever it was possible. Some surgeons believed that, in order to prevent infection, it was essential to operate promptly after wounding, but, as the war progressed, it was found that patients with head injuries who were sent to general hospitals for initial surgery usually did well. When the operation had been done in a forward hospital, the patient was evacuated to a general hospital as soon as his general condition permitted, without regard to whether or not the sutures were still in situ. The risk of contamination was increased by repeated wound dressings, but, if the initial debridement had been adequate, this increase was minimal and could be disregarded.

Antimicrobial therapy.—Antimicrobial therapy was employed prophylactically in all penetrating wounds of the brain. Sulfonamides were employed systemically until penicillin was introduced, after which it was used routinely.

Early in the war, it was standard practice to use sulfanilamide locally immediately after wounding. In numerous instances, wounds seen 4 to 6 days after they had been incurred, with the first dressing still in place, showed no evidence of infection, and only occasionally did serious infections occur when these wounds were debrided, even though, as just mentioned, initial wound surgery had been deferred for these and longer periods.

How much local chemotherapy had to do with these good results is open to question. Subsequent observations suggested that the effects of this technique were offset by the presence of exudate or devitalized tissue and also indicated that the added trauma which accompanied the local application of sulfanilamide powder more than counterbalanced any benefit which might be derived from it. In the European theater, in 1944 and early 1945, local chemotherapy was thought to be of value in the prevention of infection, but the circumstances did not permit carefully controlled studies, and, before the end of the war, this method was discontinued as both a prophylactic and a therapeutic measure.

MANAGEMENT OF INFECTED WOUNDS

Surgical management.—The general management of an infected cranial wound followed the same principles at all levels of medical care. The details of management depended upon a number of considerations, including the adequacy (or inadequacy) of the original debridement; the closure of the dura (or failure to close it); the type of closure of the scalp wound; the causative organisms and their susceptibility to penicillin; the degree of infection (superficial or invasive); the general condition of the patient; the severity of concomitant injuries; and the response to simple drainage and to antimicrobial therapy.

If the original debridement was known to have been complete and the infection did not seem deep seated, the first necessity was to provide adequate open drainage for the escape of the exudate, to prevent its intracranial spread. This was usually accomplished by reopening the wound, or a portion of the wound, or, occasionally, by instituting drainage through a stab wound.

When the initial operation was known, or suspected, to have been inadequate, radical secondary debridement was required. The same policy was followed when it was clear that infection was already established. The margins of the scalp wound were excised, and the wound was opened sufficiently to permit a comprehensive view of the entire involved area. Granulation tissue was excised, and all foreign material, including silk sutures and bone wax, was removed.

If the infection involved underlying brain tissue, clean excision of all devitalized and infected tissue was undertaken along the entire track of the wound.

These procedures were in keeping with the basic principles advocated for all wound infections. In many instances, the response to simple drainage supplemented by antimicrobial therapy was rapid and satisfactory, and more radical surgical intervention was not required.

Adjunct therapy.—Every effort was made to promote wound healing by correction of low hemoglobin levels, hypoproteinemia, vitamin deficiencies, and inadequate fluid and nutritional intakes. In addition, the powerful weapons of modern antimicrobial therapy were employed promptly and vigorously.

The general plan was to use sulfadiazine in doses of 4 to 6 gm. every 24 hours and penicillin intramuscularly in doses of at least 20,000 units every 3 hours. Cultures and smears were made routinely, and an effort was also made to determine the penicillin sensitivity of the causative organisms.

Penicillin was rather widely used locally, partly on the basis of experience in United States Army hospitals and partly on the basis of the British experience. It was often the practice, to cover the period over which a high systemic level was being built up, to apply a concentrated dose to the infected area immediately after drainage had been instituted. Two techniques were employed as follows:

1. A small Dakin tube was placed in the wound and carried out through

the dressing. Sometimes a sterile medicine dropper was inserted into it. Penicillin was instilled into the tube in small amounts, every 2 hours, by a nurse.

2. The wound was dressed daily, and penicillin was instilled directly into it with a syringe and needle. This method resulted in less continuous application of the penicillin but also offered much less opportunity for secondary contamination of the wound.

If the tube method was used, the tube had to be left in place long enough to permit the delivery of effective doses of penicillin, but, at the same time, it had to be removed before secondary infection became a possibility.

The decision for or against using penicillin intrathecally had to be made in each individual case. Unless the spread of micro-organisms to the meninges seemed imminent, this route was not employed, in view of occasional local reactions in the subarachnoid space.[5] On the other hand, if there was any suspicion that meningeal infection might be developing, a prophylactic dose of about 20,000 units of penicillin was usually given promptly by the intraspinal route, in the hope that it might halt the beginning involvement of the meninges and prevent the development of full-blown meningitis.

MENINGITIS

Meningitis, fortunately, was not of very frequent occurrence. The incidence varied both with the location of the hospital [6] and with the timelag between wounding and initial wound surgery.

Sources of infection.—There were, obviously, numerous possible sources of infection of the meninges after penetrating cranial injuries. A very large break might have occurred in the natural barriers between the exterior of the body and the cerebrospinal fluid. Wounds produced by large shell fragments or by multiple smaller fragments (figs. 40 and 41) sometimes opened a very wide portal for the invasion of the cerebrospinal fluid by micro-organisms on the scalp or the helmet or on the missile itself. Wounds of the gutter type offered the same opportunity. If these large openings remained patent for any length of time or if debridement of the wound was not possible within a relatively short time, even the most vigorous antimicrobial therapy was likely to prove useless.

Even if the wounds were less extensive, it was still possible that organisms might be harbored by metallic fragments, particularly if they were of considerable size, and by bone fragments. Bacteria were also harbored in necrotic cerebral tissue and blood clot. A gradual spread of infection from any of these

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[5] (1) Walker, A. Earl, and Johnson, Herbert C.: Penicillin in Neurology. Springfield: Charles C Thomas, 1946. (2) Erickson, T. C., Marsten, M. G., and Suckle, H. M.: Complications of Intrathecal Use of Penicillin. J. A. M. A. 132: 561–565, 9 Nov. 1946.

[6] (1) Weaver, T. A., Jr., and Frishman, A. J.: A Report on the Treatment of Craniocerebral Wounds in an Evacuation Hospital. J. Neurosurg. 3: 148–156, March 1946. (2) Campbell, E. H., Jr., and Martin, J.: Cerebral Fungus Following Penetrating Wounds. Surgery 19: 748–755, May 1946.

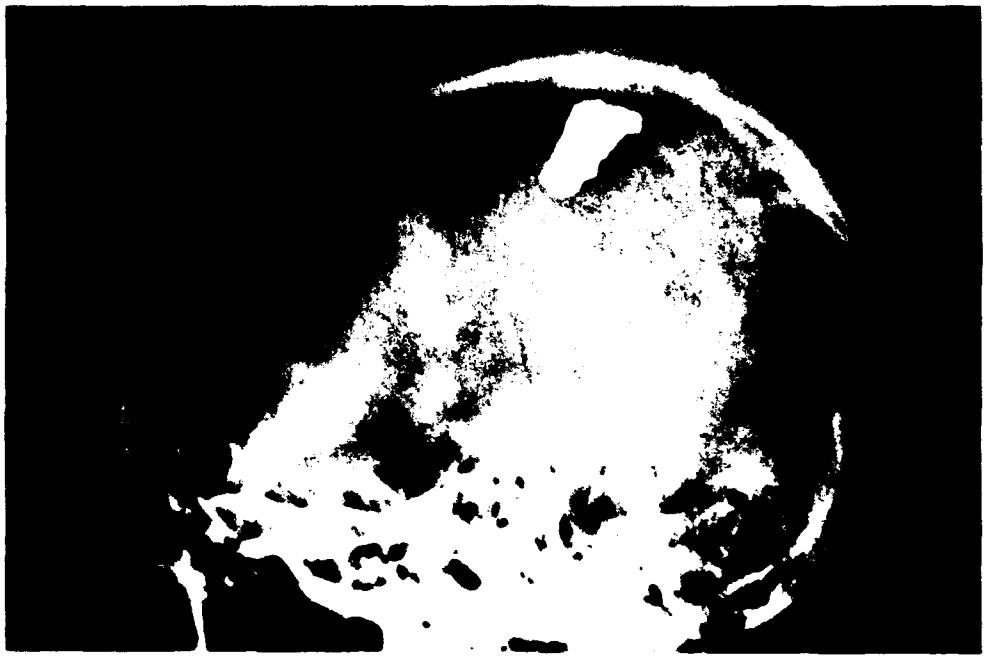


FIGURE 10. Lateral roentgenogram showing large shell fragment which entered left frontal region and came to rest in right parietal area. It was removed at the same time that a secondary debridement was carried out, with closure of a leaking wound at the site of entrance.

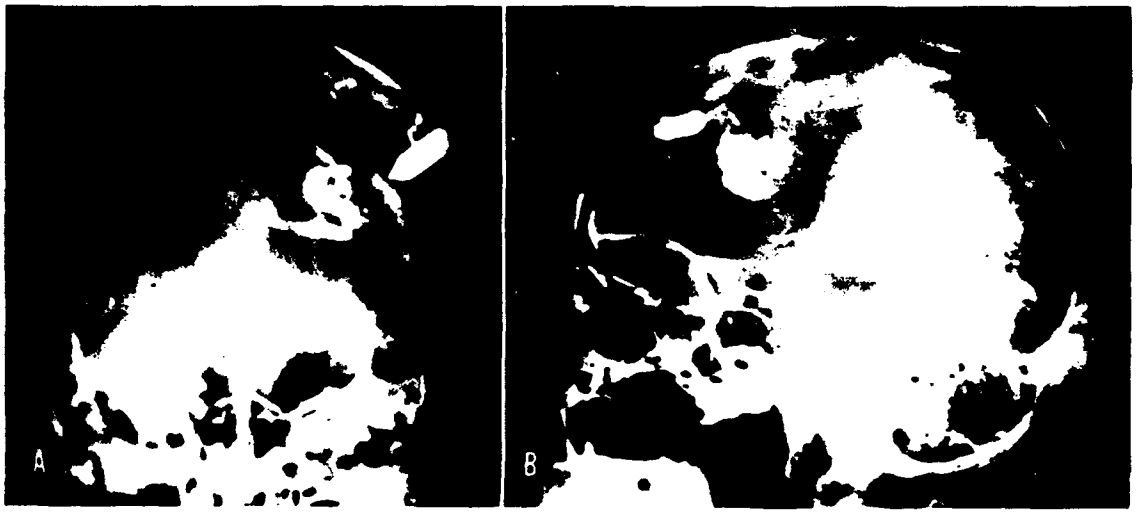


FIGURE 11. Posteroanterior (A) and lateral (B) roentgenograms of skull, showing initial films taken in evacuation hospital, following penetrating gunshot wound of right parietal region. The bone fragments were driven into the right lateral ventricle and, following the original debridement, a cerebrospinal fistula developed through the wound. At secondary operation in a neurosurgical center in a general hospital, a large fascia lata graft was sutured in place to close the dural defect; the wound healed satisfactorily. Deep transventricular wounds often gave rise to the problem of cerebrospinal fistula, particularly if it was not possible to carry out a complete dural closure at the time of the original operation.

foci, if it was not interrupted by surgery, eventually involved the spinal fluid and meninges.

It was observed relatively early in the war that the point of entrance of a missile, as well as its course within the head, was of considerable importance in relation to the possible development of both meningitis and brain abscess. Thus, a missile passing through one of the paranasal sinuses or the orbit was much more likely to carry infection than a missile which entered the intact scalp above the sinuses.

Another source of meningitis that developed later was the presence of a persistent passageway or fistula between the exterior of the head and the subarachnoid space, with resulting rhinorrhea or otorrhea, or the presence of a chronic cerebrospinal fluid fistula through the wound itself.

A delayed wound infection, spreading inward, might also give rise to meningitis. In an occasional case, in which there was no direct opening between the exterior and the subarachnoid space, meningitis apparently developed on an embolic basis.

Bacteriology.—The micro-organisms responsible for meningitis were usually streptococci or staphylococci. Occasionally, gram-negative organisms, including the colon group or *Klebsiella friedländeri*, were the offenders. Meningitis of meningococcic origin was very uncommon.

Diagnosis.—The diagnosis of meningitis was not always easy. A certain degree of meningismus followed all head injuries, and an increase in the number of white blood cells in the spinal fluid, as a result of hemorrhage at various points along the track of the bullet, was almost invariably present for some days, or even for weeks, after injury. Fever was also frequently present for some time.

Meningitis occasionally began insidiously, with relatively little febrile reaction, partly, perhaps, because the patient was receiving antimicrobial therapy at the time. In general, the diagnosis depended upon evidences of wound infection; the usual systemic signs of spreading infection, such as a rise in temperature, a chill, or a toxic mental change; and signs of meningeal irritation. The final diagnosis rested upon spinal puncture, the finding of an increased cellular reaction in the fluid, and the finding of organisms on smear or culture. The clue to the diagnosis, however, was the constant suspicion that infection might be present in the meninges.

Management.—Once the diagnosis was established or strongly suspected, prompt and vigorous systemic chemotherapy and antibiotic therapy with both pencillin and a sulfonamide was instituted. Even though the causative organisms were not yet identified, it was standard practice, whenever the spinal fluid appeared grossly cloudy, to instill penicillin into the subarachnoid space at the time of the original diagnostic lumbar puncture. The amounts used varied widely, depending upon the source of the infection, the condition of the patient, and the surgeon's estimate of the gravity of the situation. On the average, doses of 50,000 units every 12 to 24 hours were regarded as ample, though doses up to 100,000 units were sometimes employed.

One problem which immediately arose concerned the possible damaging effects of the presence of penicillin in these amounts in the subarachnoid space. Some brands of penicillin were found to be extremely irritating at the time of the injection. Other brands were not, and, in general, few aftereffects were reported when treatment extended over only a few days. When, however, large amounts of penicillin were used intraspinally over long periods (7 to 14 days), there were sometimes clear-cut evidences of diffuse arachnoiditis, particularly in the spinal canal. In most cases, fortunately, treatment was not necessary for long periods of time or in excessively large doses, and no late manifestations could be attributable to the presence of penicillin within the spinal fluid.

Although penicillin was injected at diagnostic lumbar puncture whenever the spinal fluid appeared suspicious, prompt identification of the organism and determination of its sensitivity to penicillin were regarded as essential. If the invader was gram positive, intensive systemic chemotherapy was instituted immediately with sulfadiazine, which had been shown to penetrate the subarachnoid space more effectively than any other sulfonamide drug. The efficacy of penicillin by the systemic route was debatable, but there was some evidence to suggest that a low concentration might be built up in the spinal fluid when the meninges were inflamed. It was not the custom, therefore, to withhold penicillin, even though there was uncertainty concerning its reaching the spinal fluid. Penicillin was always used intramuscularly, of course, if an infectious process was present in the wound.

When the invading organisms were gram negative, the problem was more complicated. Large doses of a sulfonamide drug, supplemented by large doses of urea, seemed to offer the most effective therapy.[7] Some patients recovered under this regimen, but this type of infection was frequently fatal, and it accounted for many late deaths from infection. Had streptomycin been available, the story might have been different.

When a spinal-subarachnoid block was suspected, the prompt use of intraventricular chemotherapy was occasionally lifesaving. No evidence of serious toxic cerebral effects from this method of treatment was accumulated during the war.

Fistulas associated with rhinorrhea or otorrhea, which usually resulted from penetrating wounds involving the cribriform plate, the paranasal sinuses, or the petrous ridge, provided, as already mentioned, ready sources of infection and gave rise to meningitis in a fairly large number of cases. Prophylactic doses of the sulfa drugs were used routinely in the hope that spontaneous sealing off of the cerebrospinal fluid fistula would occur. If it did not, a resort to surgical management always had to be considered.

The timing of surgery was another serious problem. The patient's condition was not always ideal for extensive surgical intervention, and delay for several weeks was usually justified unless there were obvious signs of impending meningeal infection. On the other hand, relatively few cerebrospinal fluid

[7] Ecker, A. D.: Tight Dural Closure With Pedicled Graft in Wounds of the Brain. J. Neurosurg. 2: 384–390, September 1945.

fistulas closed spontaneously after penetrating head wounds, and operation was usually necessary.

The decision to operate and when to operate was made on the basis of the findings in each case. In general, persistence of rhinorrhea or otorrhea for more than 3 weeks was thought to be a sufficient indication for operation. A large craniotomy was employed to permit adequate access to the involved area, an intradural approach being used whenever it was practical. The use of fibrin foam and of large fascia lata grafts held in place by as many sutures as possible offered the best chance of success. Repeated operations were sometimes necessary before closure of the fistula was obtained.

Adjunct therapy.—Of all supportive measures, the maintenance of an adequate fluid and caloric intake and provision of adequate rest seemed of the greatest importance. Transfusions were given as necessary to maintain the hemoglobin level. Feedings through the Levin tube were helpful in the stuporous patient. Restlessness was controlled by regularly administered doses of barbiturates, paraldehyde, bromides, or chloral hydrate. Measures to relieve discomfort by control of the increased intracranial pressure included elevation of the head of the bed, free catharsis, and, in occasional cases, spinal or ventricular drainage.

Results.—The case fatality rate from meningitis averaged about 36 percent. Most of the fatalities were the result of gram-negative infections, which were likely to be 100 percent fatal. Ecker, however, reported 3 survivals in 10 cases.

BRAIN ABSCESS

Sources of infection.—Brain abscesses developed from several sources following gunshot wounds of the head. The usual foci were small collections (clusters) of indriven bone fragments, which were commonly encountered when initial debridement has been incomplete. These bone fragments, which were present in about 20 percent of the head injuries observed in general hospitals, came to be recognized as such a constant source of brain abscess that they were regarded, per se, as an indication for prophylactic secondary debridement when cranioplasty was done. This policy did not mean that all isolated deep fragments were routinely extirpated or that wide, traumatizing explorations were undertaken in patients whose clinical progress was satisfactory. Nests of bone fragments in or close to the missile track (fig. 42) were, however, carefully removed in most cases to prevent the possibility of brain abscess.

Large metallic intracerebral fragments also provided a nidus for abscess formation (fig. 43), particularly if the path of entrance led through the paranasal sinuses, the orbit, or the mastoid cells. Infection might develop along the track of the missile, around the fragments, or at both sites. In one case, for instance, in which a left frontal wound involved the sinus, a left frontal abscess was successfully extirpated. When the patient died suddenly a week later, a right frontal abscess was found to have developed about a shell fragment in the right frontal lobe.

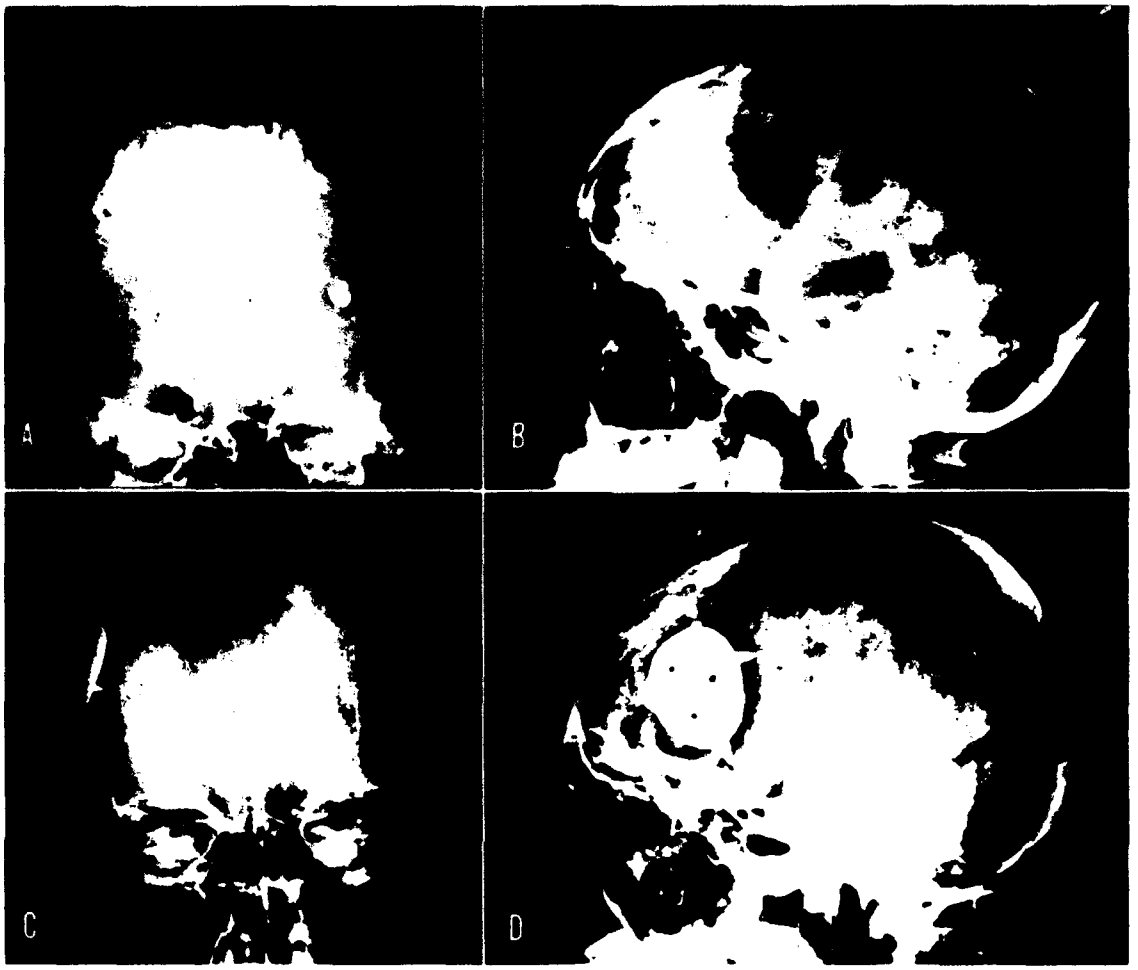


FIGURE 12. Serial roentgenograms of patient with penetrating wound of left frontotemporal region. A. Posteroanterior roentgenogram of skull before operation. B. Lateral roentgenogram before operation. C. Posteroanterior roentgenogram of skull after secondary debridement and cranioplasty with application of tantalum plate over skull defect. Note metallic fragment still in situ in right hemisphere. D. Lateral roentgenogram after operation.

Multiple abscesses were not uncommon in the terminal stages of overwhelming infection and also occurred in gram-negative infections which resisted the antimicrobial therapy then available. Almost all proved fatal, usually from the rupture of a deep abscess into the ventricular system at some point.

Abscess formation sometimes followed wound infection, but this was not a frequent development, meningitis being the greater hazard under these circumstances. Similarly, while metastatic brain abscesses sometimes followed scalp infections, this was not a frequent development. Osteomyelitis was itself uncommon, and, in the few cases observed, no underlying intracerebral abscesses were encountered.

Pathologic process. Abscesses of the brain encountered in military surgery differed in some respects from those seen in peacetime. In general,

the micro-organisms seemed somewhat less virulent, perhaps because of the widespread employment of antimicrobial therapy, including penicillin after it was introduced. Another possible reason for the apparently lesser virulence of the micro-organisms was the youthful age and the general physical fitness of the patients. Gram-negative organisms were found more frequently than in civilian practice. Brain abscesses observed in combat casualties were usually poorly encapsulated, undoubtedly because of the trauma sustained in the area.

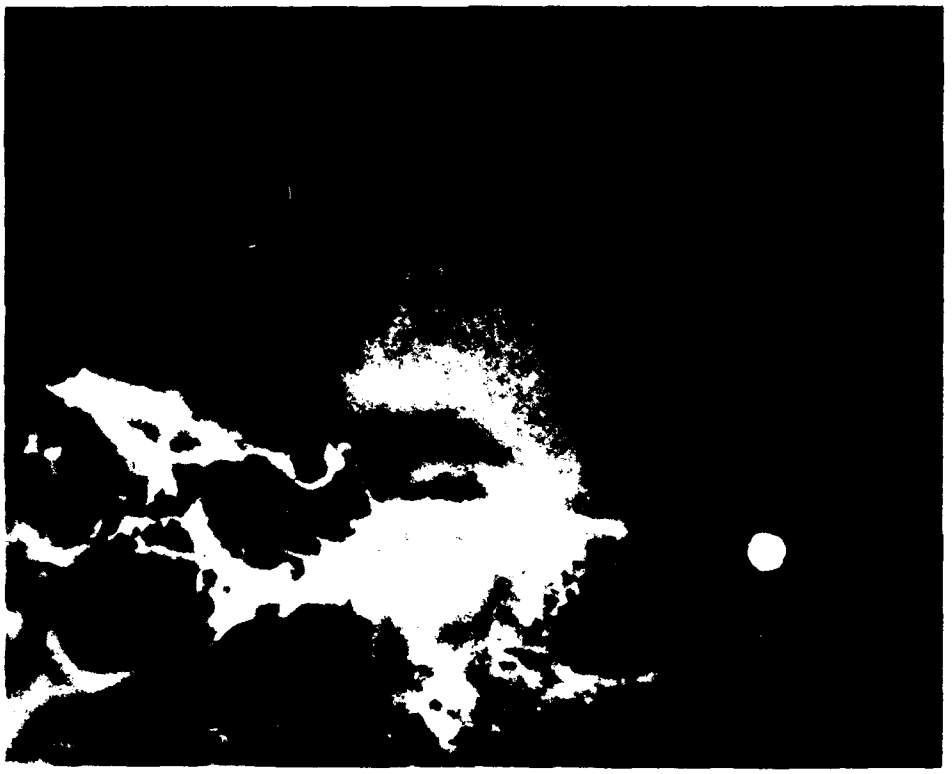


FIGURE 43. Lateral roentgenogram of skull of patient with machinegun wound in left frontal region. The missile track extended nearly through the hemisphere. After the original debridement, the bullet could be seen lying on the tentorium in the occipital region. This missile was subsequently removed through a small craniotomy in the left occipital area, since missiles of this size were regarded as definite sources of late brain abscess. The patient recovered considerably from his right hemiplegia and alphasia and was ambulatory when he was transferred to the Zone of Interior.

Clinical picture and diagnosis.— The clinical picture of brain abscesses in military surgery was much less definite and of far more insidious onset than was true of abscesses seen in civilian practice, and diagnosis was correspondingly difficult. Previous trauma not only provided a reasonable explanation for headaches, bulging wounds, or some increase in the intracranial pressure but also usually obscured any focal neurologic changes for some time. The presence of a purulent collection might therefore be unsuspected until the onset of stupor or convulsions heralded the spread of the infection. This was so likely to be the course of events when large foreign bodies were present, whether they

were fragments of missiles or bone fragments, that the policy of removing them for prophylactic reasons was instituted.

The most certain evidence of the development of a local abscess was increasing intracranial pressure. Protrusion of the scalp flap or of the wound, with the diminution of local pulsation, was suggestive. Other reasons for suspicion were persistent headaches, a slow pulse, early choked disks, and elevated spinal fluid pressure. The progression of neurologic signs, spreading wound infection, persistent fever, and a rise in the white blood cell count were all indications for a search for possible abscess.

Less tangible changes in the clinical course also furnished grounds for suspicion. These changes included mild mental disturbances, particularly irritability, a tendency to drowsiness, and perhaps some slight personality change.

An alteration in the character or location of the headache from which the patient had already been suffering was significant. The slow pulse characteristically found in the brain abscesses seen in civilian practice was less helpful diagnostically since bradycardia was usually present already in penetrating wounds of the head and remained unaltered if a purulent collection developed.

Important findings on spinal puncture included increased pressure and an increase in the number of white blood cells, particularly lymphocytes. In the interpretation of these findings, however, allowance had to be made for the pleocytosis usually present in head injuries.

If the presence of an abscess could not otherwise be established or excluded, the policy was to explore the suspected area either through the intact skin, with a lumbar puncture needle, or through a nearby burrhole, with a brain cannula. If these exploratory measures proved negative, they were practically always innocuous; when they were positive, the experience was that the suspected abscess was recognized and located more quicly than if diagnosis had been delayed until the clinical picture of a full-blown intracerebral abscess had been awaited.

Management.—The management of brain abscesses following penetrating cranial injuries varied widely during the war, just as it had varied in peacetime practice. Almost all of the accepted neurosurgical methods were tried, including continuous drainage, repeated aspiration, and radical removal. Grant,[8] Northfield,[9] and Meredith[10] advocated simple aspiration. King[11] and Bucy and Haverfield[12] advocated some type of continuous tubal drainage. As the war progressed, there was a gradual tendency to a more radical attack on the lesion.[13]

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[8] Grant, F. C.: End-Results in One Hundred Consecutive Cases of Brain Abscess. Surg., Gynec. & Obst. 75:464–467, October 1942.

[9] Northfield, D. W. C.: The Treatment of Brain Abscess. J. Neurol. & Psychiat. 5: 1–9, January–April 1942.

[10] Meredith, J. M.: Recognition and Management of Brain Abscess. Am. J. Surg. 63: 10–15, January 1944.

[11] King, J. E. J.: Management of Abscess of Otorhinogenic Origin. Laryngoscope 50: 1146–1158, December 1940.

[12] Bucy, P. C., and Haverfield, W. T.: Cranial and Intracranial Complications of Acute Frontal Sinusitis. J. A. M. A. 115: 983–991, 21 Sept. 1940.

[13] (1) Groff, R. A., and Grant, F. C.: The Surgical Treatment of Brain Abscess by Exposure and Enucleation. Ann. Surg. 111: 925–941, June 1940. (2) LeBeau, J.: Radical Surgery and Penicillin in Brain Abscess. J. Neurosurg. 3: 359–374, September 1946.

With any of these methods, it was possible to visualize the lesion roentgenologically by the use of some contrast medium at the time of the original drainage. Air was occasionally used. When thorotrast was available, small quantities were injected. This medium had the great advantage of permitting some visualization of the capsule, so that an estimate could be made of the tendency of the abscess to refill merely by repeating the roentgenograms. Kahn's [14] technique, the use of thorotrast with overlying decompression, which permitted control of any herniation of the lesion toward the surface, was not employed in any of the head injuries treated at the 160th and 184th General Hospitals in England.

The simplest of all methods of abscess management, namely, repeated aspiration followed by the injection of penicillin, was not successful in any of the cases in which it was used at these hospitals. Although culture of the pus was frequently negative, the organisms apparently lingered within the wall of the abscess, and the lesion recurred even after it had been completely drained and large doses of penicillin had been employed.

Continuous drainage of the abscess by the use of an indwelling tube was occasionally effective, but recurrence of the purulent exudate was not infrequent because of the persistence of organisms within the abscess wall.

The most effective method of dealing with an abscess of the brain after a penetrating gunshot wound of the head proved to be its radical removal. This technique was perfectly feasible. For one thing, the lesion was usually close to the surface. For another, abscess formation in these circumstances occurred in a previously traumatized area of the brain, and radical extirpation therefore produced no additional crippling neurologic aftereffects. In cases in which the abscess was completely removed and the wound tightly closed, operation was performed without difficulty; the wounds healed promptly, and the lesion did not recur.

The technique devised by Webster and his associates of treating abscesses in the frontal area by suction removal, followed by closure of the dura with a temporary fascia lata graft, was intended to prevent leakage of the cerebrospinal fluid and fungus formation. At a later operation, the temporary graft was removed, and the defect was covered with a split-thickness graft. At the 160th and 184th General Hospitals, this method was thought to have certain disadvantages, including possible early tension on the involved portion of the brain, increased difficulty in carrying out a satisfactory cranioplasty, and, finally, more widespread scar formation over the cortex. For these reasons, this procedure was not employed in any of the cases treated at these hospitals.

Cerebral fungus was not encountered frequently. When this complication did occur, the fungus was usually found to contain one or more brain abscesses, which it was essential to drain before any other procedure was undertaken.[15] After drainage, prompt closure of the skull by the use of a tantalum plate ap-

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[14] Kahn, E. A.: The Treatment of Encapsulated Brain Abscess with Visualization by Colloidal Thorium Dioxide. Univ. Hosp. Bull. 4: 17-19, March 1938.

[15] See footnote 6 (2), p. 204.

plied directly over the fungus was usually successful. If necessary, the fungus could be reduced to the level of the skull margins by spinal drainage repeated daily, as necessary, for a number of days. In most cases in which this method was used, the scalp was closed completely over the tantalum plate when cranioplasty was done.

This technique prevented further infection of the fungus, expedited the healing of the wound by weeks, and minimized the neurologic damage which would otherwise have resulted from the protrusion of the edematous and often infected brain.

Adjunct therapy.—Antimicrobial therapy was used vigorously in the treatment of a brain abscess, whether or not it was treated surgically. Local chemotherapy with both penicillin and a sulfonamide was employed in most cases in addition to systemic therapy.

All of the usual supportive measures were also employed, with particular emphasis upon the maintenance of a high hemoglobin level, by transfusion of whole blood, and the prevention of hypoproteinemia by special attention to the protein intake.

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CHAPTER XI

Blast Concussion and Cerebral Injuries Due to Explosion Waves

Fritz Cramer, M. D.

DEFINITION AND TERMINOLOGY

Cerebral Concussion, Airborne and Solidborne

"Blast concussion" was the term commonly used in World War II to describe the condition in which traumatic alterations of the substance and function of the brain were caused by shock waves from nearby explosions which passed to the central nervous system without the interpolation of a solid blow to the head. Fabing,[1] who employed the term "cerebral blast syndrome" for this condition, defined it as follows:

> The blast injury syndrome is that morbid condition which results from the nearby explosion of one or more agents, and causes the following tetrad of symptoms, (1) unconsciousness with retrograde amnesia for the sound of the explosion and of varying anterograde duration, but persisting an hour in the usual case, (2) protracted nonspecific *headache*, (3) *tinnitus* which is usually nonpersistent, and (4) diffuse *anxiety* symptoms.

Under the latter syndrome are included the vertiginous sensations and the extreme intolerance to noises of which the majority of patients complain. In thus defining this sometimes moot entity, Fabing used the generic definition for the syndrome of cerebral concussion and the postconcussional state.

Denny-Brown [2] deplored the use of the term "blast concussion" because it implies a mechanism of production which differs from the mechanisms usually comprehended as the causes; namely, acceleration-deceleration of the skull and compression. This type of concussion, however, presents a challenge which must be met because it does have special limitations of incidence to the traumas of war, because the mechanism of its production is in fact unique, even though not unfathomable, and because it constitutes a syndrome which is frequently manifest and is inexorable.

The term "blast concussion" (cerebral blast concussion) is used in this chapter because it is in general use and has behind it well-established facts concerning the mechanism by which it can be caused [3] and also concerning

[1] Fabing, H. D.: Cerebral Blast Syndrome in Combat Soldiers. Tr. Am. Neurol. A. 71: 29-33, June 1946.

[2] Denny-Brown, D.: Cerebral Concussion. Physiol. Rev. 25: 296-325, April 1945.

[3] (1) Scott, W. W.: Physiology of Concussion. Arch. Neurol. & Psychiat. 43: 270-283, February 1940. (2) Walker, A. E., Kollross, J. J., and Case, T. J.: The Physiological Basis of Concussion. J. Neurosurg. 1: 103-116, March 1944. (3) Webster, J. W., and Gurdjian, E. S.: Acute Physiological Effects of Gunshot and Other Penetrating Wounds of the Brain. J. Neurophysiol. 6: 255-262, July 1943.

the organic cerebral changes which have been demonstrated in experimental cerebral concussion of all degrees of severity.[4] It is granted that there is no uniformity of definition of the term "concussion" as applied to the brain. Most writers attempt to restrict its meaning to a very circumscribed condition characterized chiefly by a short period of posttraumatic unconsciousness with which, it is presumed, no organic changes in the brain, or only reversible organic changes, are associated. The attempt at restriction is unsuccessful, however, because of many factors, not the least of which is that these writers themselves, in actual usage, employ the term to include not only concussion but the postconcussional syndrome and postconcussional encephalopathy.

Because of the tendency toward the indiscriminate use of terms, the following definitions are essential:

Cerebral blast injury implies traumatic cerebral changes identifiable by neurologic sequelae and by tests such as encephalography.

Cerebral blast concussion (blast concussion) indicates the prime symptoms generic to cerebral concussion, with emphasis on the pattern of symptoms seen in this particular method of production, which implies indirect compression. There may or may not be persistent neurologic findings. There may be molecular changes within the nerve cells and nuclear groups, as well as possible injuries to the blood vessels. Gross lesions, including cerebral contusion and laceration, may be responsible for persistent symptoms in the postconcussional syndrome.

This use of the term "cerebral blast concussion" is general. Symonds[5] used the term "concussion" to indicate not only the effects of concussion on the brain cells, or true commotio cerebri, but also, inevitably, some trauma to the blood vessels. This trauma may be manifested by free blood in the spinal fluid but need not be. Loss of consciousness is regarded as a prime symptom, but merely as a single symptom, and not as an infallible index of the degree of concussion.

Air-blast concussion in the sense in which the term is used by Schwab[6] is not included in the definition of blast concussion. He described his personal experiences with what is also technically known as muzzle blast, which is not usually traumatic except under circumstances other than those related by him. What he described was in essence merely an intensely strong wind occurring in the neighborhood of gunfire. It should not be confused with the term "shock wave" and the accompanying air blast from a high-explosive missile.

In this chapter, the term "blast" is used in the strict sense to designate the shock wave itself moving through air, fluid, or solids, including the living body. On the other hand, the term "concussion" is used in the broad sense, to designate

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[4] (1) Groat, R. A., Magoun, H. W., Dey, F. L., and Windle, W. F.: Functional Alterations in Motor and Supranuclear Mechanisms in Experimental Concussion. Am. J. Physiol. 141: 117–127, March 1944. (2) Windle, W. F., and Groat, R. A.: Disappearance of Nerve Cells After Concussion. Anat. Rec. 93: 201–209, October 1945. (3) Windle, W. F., Groat, R. A., and Fox, C. A.: Experimental Structural Alterations in the Brain During and After Concussion. Surg., Gynec. & Obst. 79: 561–572, December 1944.

[5] Symonds, C. P.: Concussion and Contusion of the Brain and Their Sequelae. *In* Injuries of the Skull, Brain and Spinal Cord, edited by Samuel Brock. Baltimore: Williams & Wilkins Co. 1940.

[6] Schwab, R. S.: Cerebral Airblast Concussion. J. A. M. A. 131: 101–103, 11 May 1946.

a temporary posttraumatic state and its sequelae, whose chief, but not only, physiologic characteristic is loss of consciousness and whose morbid anatomy is characterized by intracellular and interstitial cerebral changes, as well as by imperceptible or minor gross injuries to the cerebral vascular system.

Blast Concussion and Shellshock

Since the introduction into warfare of missiles of high velocity and high-explosive quality, men have noted and reported a morbid condition of the central nervous system which in most respects is comparable to cerebral concussion and the postconcussion syndrome. For its causation, no other trauma than the wave of the explosion or the compressive wave created by the missile in flight could be ascribed. Various references in the medical literature concerning World War I indicate that nervous manifestations, specifically concussion, were long considered to have occurred at times from the violent forces existing immediately about a flying missile, called the wind of the bullet [7] or the wind of the cannonball.[8]

In more ancient times, before the use of high-explosive shells, the actual passage close by of a nonexploding cannonball was believed capable of causing death. The "wind" of a round shot was believed to cause fatalities by the compression of the air. It is not the purpose in this chapter to elaborate on these earlier observations and interpretations except to note that the "wind of the explosion" [9] and shell concussion or shellshock [10] were some of the terms used in World War I to describe the same pathologic and clinical phenomena which have been described under the term "blast concussion" and allied terms in World War II.

Controversial Aspects

The term "shellshock" soon became abused and fell into disrepute because of its confusion in the minds of clinicians, as well as of the lay public, with the psychogenic disturbances of warfare, chiefly the anxiety neuroses, hysteria, and malingering. There was also equivocation, notably on the part of Mott,[11] who described pathologic lesions in cases of this type. He questioned whether other factors, including carbon monoxide poisoning, might have played a part in producing the cerebral petechiae which were described, and he discussed some of the psychiatric aspects of the problem. It should be emphasized, however, that Mott's thesis is, in essence, that true concussion and injury to the brain were due to the effects of blast as we now speak of it. He considered that the possible association with carbon monoxide poisoning, from the accumulation

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[7] Nervous Manifestations Due to the Wind of Explosives (editorial). Lancet 2: 348-349, 14 Aug. 1915.

[8] The Pathology of Shell Concussion (editorial). Brit. M. J. 2: 264-265, 14 Aug. 1915.

[9] Stevenson, W. F.: Note on the Cause of Death Due to High-Explosive Shells in Unwounded Men Brit. M. J. 2: 450, 18 Sept. 1915.

[10] Tulloch, A. B.: Shell Shock (letter to the editor). Lancet 2: 575, 4 Sept. 1915.

[11] Mott, F. W.: The Effects of High Explosives Upon the Central Nervous System. Lancet 1: 331-338, 12 Feb. 1916.

of carbon monoxide in the dugouts in which the cases occurred, was a distinctly secondary factor and not a primary cause of the syndrome.

For these reasons, in World War II there was a cautious approach to the problem, which inevitably presented itself. Not only was there variance of opinion whether the symptoms of cerebral disturbance were in fact, or in part, due to organic cerebral damage, but Denny-Brown [12] has even stated his frank disbelief that these symptoms are due at all to concussion by the primary explosion waves.

The reasons underlying these variances in opinion may be simplified as follows:

1. There has been a fundamental paucity of autopsy reports and of microscopic studies of the brains of persons dying from the primary effects of explosion waves.

2. There is a paucity of complete, minute, objective records of neurologic examinations by trained neurologists.

3. By contrast, there has been a preponderantly psychiatric approach to the problem, which has overbalanced the neurologic approach.

4. There were administrative fallacies in World War II, as in World War I.

In this connection, it seems worthwhile to quote Crichton-Miller's [13] rather adroit summary:

> After the war [World War I] a commission was appointed to investigate the problem, and reached a conclusion which may not unfairly be formulated thus: "We cannot deny that there is such a condition as true shell-shock, but it has been so much abused that we must never again admit its existence." * * * the naïf conviction that a symptom can be eliminated by refraining from allowing it a name had its inevitable result. The symptom appeared, a designation had to be found for it, and so the clinicians—as opposed to the ministerial authorities—described it as blast-concussion. It is to be hoped that this name will not be exploited in the way in which shell-shock was exploited; but if it should be the fault will lie not in the name, but in the situation.

In World War II, no forensic furor was raised over the term "blast concussion" as there was over shellshock in the previous world war. On the contrary, such great stress was placed on the purely psychiatric aspects of the problem, and such caution was used in publicizing the organic aspects, that the net error in diagnosis is probably much too low for blast concussion and much too high for the neuroses and allied conditions. For example, it was reported at a meeting of Allied psychiatrists at a base hospital overseas in June 1943 [14] "that all cases of blast injury were diagnosed as exhaustion for administrative reasons and to avoid the diagnosis of 'shellshock.' "

This administrative policy would vitiate the statistical value of many records, as would the opposite policy of labeling combat neurosis as blast effect to spare the individuals or the organization the stigma of a psychiatric

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[12] Denny-Brown, D.: The Effect of High Explosives on the Post-Traumatic Syndrome. Tr. Am. Neurol. A. 69: 98-102, 1943.

[13] Crichton-Miller, H.: Somatic Factors Conditioning Air-Raid Reactions. Lancet 2: 31-34, 12 July 1941.

[14] Hanson, F. R.: Recent Experience of War Neurosis. Report of Societies: Meeting of Allied Psychiatrists at a Base Hospital Overseas June 5, 1943. Brit. M. J. 2: 336-337, September 1943.

diagnosis. The probability that this practice occurred has also been reported.[15] Aside from errors introduced by such policies, the fact that combat exhaustion neurosis actually existed in many men who received concussion by blast, as discussed by Swank and Marchand,[16] has added another source of error and has caused many cases of blast concussion to be hidden under the symptoms of purely functional origin.

Because of the exigencies of warfare, few data are sufficiently complete for scientific evaluation in the sense that such evaluation is required in controlled experiments. Conversely, it must be said that there are not sufficiently numerous or intricate and extensive laboratory experiments in this field to permit a true comparison with the clinical syndrome observed in human beings, or to disprove that the condition exists.

It is evident that those who have investigated this problem specifically and have reported their findings have made great efforts to establish the facts, ruling out those cases in which the diagnosis was suspect. Fortunately, there are available eyewitness observations and estimates of the distances involved, the relative position of the injured to the explosion, the relative protection afforded by foxholes, and early medical observations, including autopsy reports, even though they are not abundant.

The Nature of Blast

By blast is meant the wave or shock or pressure transmitted through the air, from the detonation of a high-explosive shell or bomb (one which is brisant or capable of blowing things to bits). Its effects will be considerable in a limited area which is just outside of its zone of brisance. Within this area, everything is blown to bits. Beyond this zone, and before it loses its traumatic effectiveness and becomes merely a sound wave, the shock wave, or explosion pressure in air, as Corey [17] proposes that it be called, has the power to kill outright or to injure by concussion. These phenomena are considered in detail by Corey, Sutherland,[18] and Robinson,[19] as well as others.[20] The shock wave has the characteristic of passing through the surrounding medium without moving the medium, whether the latter be air, water, a solid body, an animal, or a human being (figs. 44, 45, and 46). The wave has a pressure phase, followed immediately by a vacuum phase, and an oscillatory period. The compression phase is of about 0.006 second's duration and of hundreds of pounds per square inch of pressure. The suction phase is of about 0.03 second's duration and is equivalent to a pure vacuum. The velocity and duration of either

[15] Rottersman, William, and Peltz, William: History of the Neuropsychiatric Work in the Western Pacific Base Command, 14 April to 31 August 1945, p. 5. [Official record.]

[16] Swank, R. L., and Marchand, W. E.: Combat Neuroses: Development of Combat Exhaustion. Arch. Neurol. & Psychiat. 55: 236–247, March 1946.

[17] Corey, E. L.: Medical Aspects of Blast. U. S. Nav. M. Bull. 46: 623–651, 5 May 1946.

[18] Sutherland, M. W.: The Physics of Blast. Lancet 2: 641–642, 23 Nov. 1940.

[19] Robinson, Clark Shove: Explosions: Their Anatomy and Destructiveness. New York City: McGraw-Hill Book Co., 1944.

[20] (1) Bernal, J. D.: The Physics of Air Raids. Nature 147:594–596, 1941. (2) Wakeley, C. P. G.: Effect of Underwater Explosions on the Human Body. Lancet 1: 715–719, 9 June 1945.

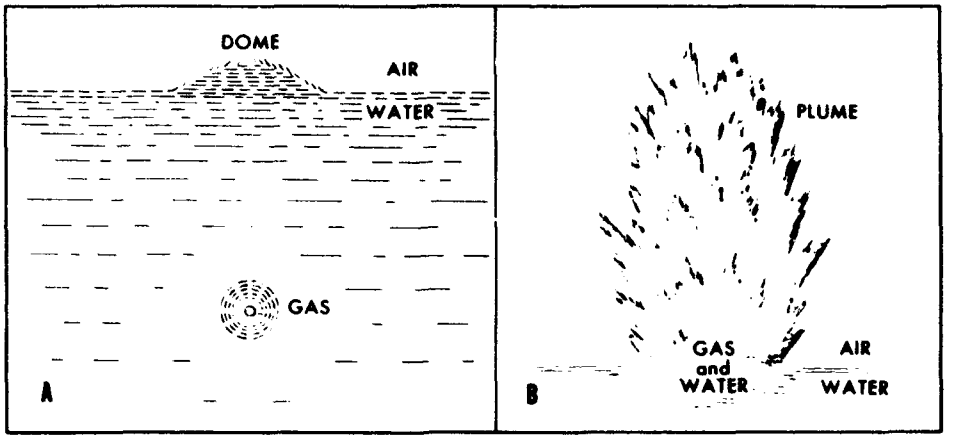


FIGURE 44.—Effects of underwater explosion. The shock wave travels through fluid at nearly 5,000 feet per second. A. At the surface of the fluid and gas, a disruption of the particles occurs. In the human body, shock is transmitted through the integument and then through the solid and fluid portions, according to definite physical principles. Surfaces within the gas-bearing organs, such as the alveoli of the intestine, sustain the greatest damage. Hemorrhages also occur in other organs. The most serious damage occurs to the parts most directly exposed to the shock wave. B. Plume resulting from underwater explosion. This formation, which represents the mixture of gases and water thrown up turbulently after the shock wave has passed, may hurl about any objects in its path, like the rush of wind which follows an explosion in the air. Secondary damage may be caused by these objects. It is the preceding shock wave which produces primary trauma in blast injuries. (Modified from Wakeley.)

phase are such that the body of a man would be completely immersed for an instant in a wave of almost uniformly altered pressure. It is, presumably, the pressure phase of the wave which produces the injuries of medical importance, and these injuries occur only relatively close to the source of the explosion. The oscillatory period is important to walls and buildings, which may be concussed at great distances from the detonation.

The Shock Wave of Air Blast

Air blast, which is to be differentiated from its misnomer airblast or muzzle blast, is the turbulent wall of moving air which is propelled for a limited distance by the momentum of the shock wave and the enormous expansion of gases colliding with the immediately adjacent air. It may travel at 20,000 miles per hour for a short distance, carrying everything in its path, but its force is quickly dissipated. This air blast may hurl bodies before it or lift them high into the air, thus producing secondary injuries. The air blast close to the explosion, being in essence a mass of compressed air, acts almost like a solid body blown out from the explosion. Being of lighter weight than the solid bodies which are loosened or hurled by the explosion, it travels ahead of the latter for a short distance, practically simultaneously with the shock wave. For a limited distance, the air blast and the shock wave run an identical course, but the latter quickly outdistances the former. The term "blast," however, is the

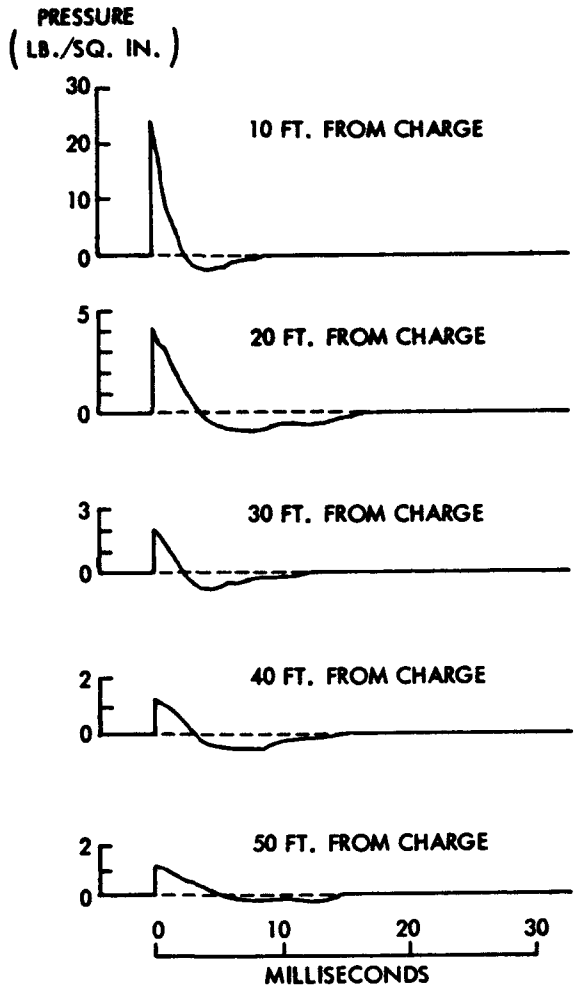


FIGURE 45.—Initial high peak and gradual fall of pressure of high-pressure wave. Peak pressures of about 100 lbs. per square inch, or 6 atmospheres, are required to endanger human beings. These pressures occur only close to explosions. The shock wave is always breaking, as the free front portion loses energy, and is always dying down, most quickly nearest to the explosion. Ultimately, the shock wave degenerates into an ordinary sound wave. (From Bernal.)

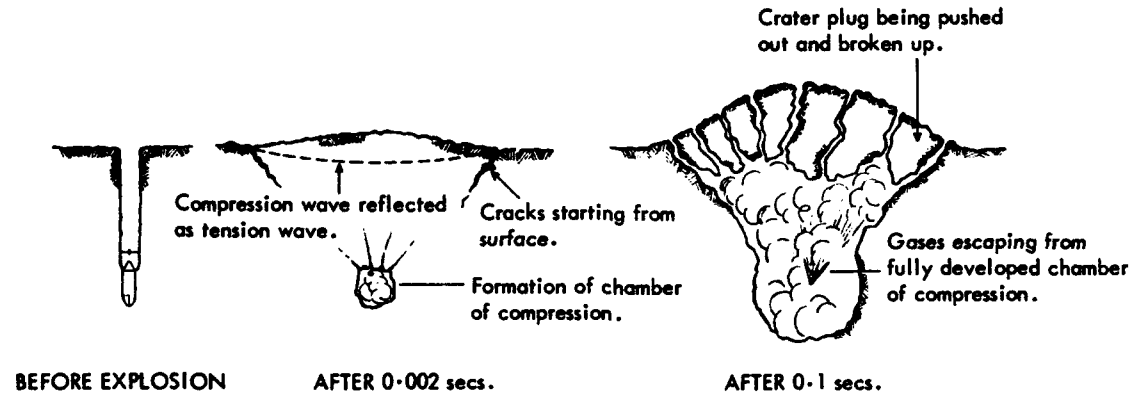


FIGURE 46.—Formation of crater in underground explosion. (From Bernal.)

one which should be applied to the shock wave, whether it be traveling in air, water, or solid mediums.

Barrow and Rhoads [21] discussed the characteristics of the blast waves and the reasons why persons or structures standing next to each other, and therefore

[21] Barrow, D. W., and Rhoads, H. T.: Blast Concussion Injury. J. A. M. A. 125: 900-902, 29 July 1944.

presumably within the same effective range of an explosion, may nevertheless, suffer fantastically different results. They have cited examples of the unpredictability or capriciousness of the wave, as follows:

1. B stands between A and C. A is thrown 42 feet and is killed. C is thrown 15 feet into the air and is seriously injured. B receives only a tympanic injury.

2. B stands behind A, resting his chin on A's shoulder. A is blown to bits. B has only a fractured maxilla.

The authors observed that standing in the second row offers protection except for the tympanum.

The physical characteristics of the blast wave are as follows:

1. Near the source, the outline is irregular, with many eddies in the periphery. Therefore a man in the periphery, sheltered in such an eddy, might escape serious injury.

2. Like sound waves, the blast waves are reflected from surface to surface and may be reinforced or neutralized by meeting similar waves in the same, or the opposite, phase.

3. Pressures vary as the square or cube of the distance from the source; this difference in critical pressures may be represented by a few feet.

The difference between the shock wave and the air blast can be easily exemplified by comparing the two phenomena to two essentially equivalent phenomena which occur in underwater explosions. Figure 44 illustrates how the shock wave from an underwater explosion travels to the surface, there raising a dome almost immediately, while the more striking plume caused by the eruption of the gases from the detonation does not occur until moments later. It is from the compression by the shock wave, as well as by secondary reflectance from the surface of the water, that trauma ensues to individuals or bodies of other types floating in the water.

THE INCIDENCE OF BLAST CONCUSSION

Studies During the Early Clinical Course

Two groups of investigators have made a special study of blast concussion in the early stages.

Von Storch[22] and his collaborators set up a mobile unit in a forward evacuation hospital in Italy. The unit included a self-contained mobile electroencephalographic laboratory and was staffed by a neuropsychiatrist, a psychologist, two technicians, and a driver. These observers made a comparative analysis of various types and degrees of cerebral concussion as observed soon after injury. The course of the disorders was followed during the patients' stay in an evacuation hospital, and, in some cases, in hospitals in the rear as well.

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[22] Von Storch, T.: A Study of Cerebral Concussion. *In* Annual Report, 33d General Hospital, 1945.

The patients were studied from a few hours to 16 days after injury, the first preliminary observations being obtained as soon as they arrived in the hospital, often while they were still in shock or while they were unconscious. A history was obtained of the circumstances of the injury, its presumed severity, and the immediate and subsequent results, symptoms, and sequelae. A general physical, psychiatric, and neurologic examination was made. A thorough psychometric study was done in as many cases as possible, and an electroencephalogram was obtained as soon as possible. It was demonstrated that the electroencephalograph could be transported over difficult terrain under combat conditions and could be used effectively in forward areas.

Of 75 cases of closed head injury, 33 were caused by blast, of which 16 (21.3 percent of the total number) were considered to be instances of true concussion; that is, the patients had been definitely unconscious at the onset. Most of these injuries were considered to be cases of mild concussion. The high percentage of these diagnoses made by definitive methods in a forward echelon is of very special interest and importance. The farther such patients move to the rear, the less apt is the diagnosis to be made or confirmed, unless the symptoms and signs be severe and persistent and the accompanying record unequivocal.

Relationship to artillery explosions.—Fabing[23] undertook a special study of blast concussion in the European theater, in a general hospital sufficiently close to the front to have received patients as early as 45 hours after trauma. Over 200 cases were studied, but only 80 could be reported because of the loss of the first 130 records as a result of enemy action. Of the total number of 1,009 neuropsychiatric battle casualties observed between late January and May 1945, 80 cases (7.9 percent) were diagnosed as due to blast. Fifty-six of the diagnoses were made in forward echelons and twenty-four in the rear.

Fabing pointed out many interesting facts regarding the incidence of blast concussion. It varies directly, he found, with the magnitude of the tactical situation and is in all likelihood an index of the effectiveness of the enemy's artillery. It occurs regardless of rank, and the incidence is spread evenly over the categories of new replacement troops and combat troops of 3 to 4 months' experience alike.

Those cases in which blast injury (blast concussion, wounded in action, shell blast, etc.) was the first diagnosis made in the medical evacuation chain constituted 70 percent of the total in which this diagnosis was corroborated in subsequent studies, while in only 30 percent was this diagnosis made for the first time in the special hospitals.

Schwab[24] gave a dissenting opinion regarding the incidence. While not denying that air-blast concussion occurs, he stated that over 90 percent of these diagnoses in Naval installations in the Pacific area were incorrect and that extreme hysterical-anxiety states were produced in men who were ex-

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[23] See footnote 1, p. 215.

[24] See footnote 6, p. 216.

hausted or otherwise sensitized to this form of trauma. He believed that only 3 cases out of well over 300 were correctly diagnosed as blast concussion.

Schwab's communication, however, is not sufficiently detailed to be of value for analysis and comparison with the others cited. Furthermore, he has introduced an element of confusion. He describes his own experiences on shipboard in the neighborhood of muzzle blast, in which the rush of wind tore open his shirt, blew off his spectacles, and produced discomfort in the eyes and ears without endangering him. The inference that this type of blast is similar or equivalent to exposure to the atmospheric blast wave, that is, the high-explosive shock wave from an exploding missile, should be avoided.

Comparative Early and Later Studies

Aita and Kerman [25] made a critical study, including neurologic, psychiatric, and electroencephalographic examinations, of patients treated later in the course of their head-injury syndromes, in an Army neurologic-neurosurgical center. They received their cases from 50 to 370 days (a median lapse of 144 days) after trauma. Of 400 cases of closed head injury, 52 (13 percent) had been diagnosed previously as blast concussion, blast encephalopathy, and similar conditions. Of these 52 cases, 34 were considered due to blast trauma, and 18 probably due to other causes (8 to solidborne concussion, 8 to the nervous strain of combat, and 2 to cerebral embolism). In all, therefore, 8.5 percent of all closed head injuries seen in late stages were, in the critical judgment of these observers, considered as due to blast concussion.

The standards of selection, testing, and evaluation of findings for the acute injuries in Von Storch's group, and those for the late sequelae in Aita and Kerman's group and in a group studied by Cramer, Paster, and Stephenson [26] are very similar and permit comparison. The great difference between the two (early and late) groups, other than the time interval, is that in Von Storch's group, which he estimates as 21.3 percent of all early closed head injuries, there was no selection of cases other than in respect to etiology. The patients doubtless represented many men who were later returned to some type of duty and did not require evacuation to the Zone of Interior because of persistent symptoms following their cerebral trauma. In the two studies made of late clinical material, on the contrary, there had been a previous clinical and administrative selection, since these patients had all required evacuation from the battle zones because the severity or persistence of their symptoms, or both, had rendered them ineffectual for active duty.

One may therefore summarize the data, from the studies made by Fabing, Von Storch, and the Aita and Kerman group, respectively, as follows:

1. The incidence of blast concussion in all cases of neuropsychiatric battle casualties in the battle zone was 7.9 percent.

[25] Aita, J. A., and Kerman, W. Z.: The Closed Head Injury Syndrome Due to Blast. Bull. U. S. Army Med.Dept. 6: 411–427, October 1946.

[26] Cramer, F., Paster, S., and Stephenson, C.: Cerebral Injuries Due to Explosion Waves—"Cerebral Blast Concussion." A Pathologic, Clinical and Electroencephalographic Study. Arch. Neurol. & Psychiat. 61: 1–20, January 1949.

2. The incidence of blast concussion in acute head injuries in the battle zone was 21.3 percent.

3. The incidence of blast concussion in the subacute and chronic closed head injury syndrome in the Zone of Interior was 8.5 percent.

THE RECOGNITION OF BLAST CONCUSSION

Fulton [27] and Pollock [28] discussed the blast-concussion syndrome of World War II in relation to the shellshock (concussion) syndrome of World War I and included references to articles on, and names of, the observers of this generic phenomenon in that period. With the exception of the tactical variants of these two conflicts, and the increase in size and power of many of the missiles employed, there may be said to be little essential change in the origins of the problem, the clinical syndrome, or the treatment.

Mott, as already mentioned, raised the question of the possibility of carbon monoxide poisoning as a cause of the petechial hemorrhages found in the brains of soldiers who died from the primary effects of bomb explosions without evidence of a blow to the head by any solid object. Lest that possibility be seriously cited again as the cause of the syndrome under discussion, it seems desirable to clarify the issue and state the following facts which refute this possibility:

1. High explosives do not depend upon the oxygen content of the air but carry the oxygen required in chemical combination.

2. Although the carbon monoxide content may be increased in the atmosphere of an area under intensive bombardment, it is quickly dissipated in the open.

3. The effect of the blast itself is instantaneous, whereas if carbon monoxide poisoning were to be a factor, it would become operative only after a (comparatively) greatly delayed period.

4. In those instances in which death occurred as a result of the blast, the span of life between the time of the explosion and death was only a fraction of the time which would be required to produce death from carbon monoxide poisoning.

The origin of shellshock in World War I was the battlefield. In World War II, the sources of blast concussion and other cerebral blast injuries were chiefly from the field, but there were also numerous observations of the condition among civilians, as well as among soldiers not in actual combat, because aerial bombing so greatly extended the zone of action.

Therein lies some possibility of a differentiation between the organic or physiogenic and the neurotic or psychogenic aspects of this clinical syndrome. There will be a discernible difference as the syndrome is encountered in the soldier who becomes a casualty from the effect of explosion waves under the conditions of combat and the soldier or civilian who is injured by an explosion

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[27] Fulton, J. F.: Blast and Concussion in the Present War. New England J. Med. 226: 1-8, 1 Jan. 1942.

[28] Pollock, L. J.: Blast Injuries of the Central Nervous System. Illinois M. J. 83: 165-168, March 1943.

while under conditions less fraught with horror and privation. The fighting soldier will, or may, have been exposed to constantly recurring fire for days or weeks; to hunger, starvation, thirst, and sleeplessness; to physical discomfort of every sort; to the intense emotional trauma produced by the loss of close friends and others from his military detachments; and to the sights and sounds of a shambles. He will therefore experience the symptoms and exhibit the objective picture germane to all soldiers who have suffered the excessive and successive and cumulative psychosomatic traumata from the effects of battlefield stimuli. If, superimposed upon this definite pathologic emotional and intellectual state, he has a cerebral concussion, there will be no obvious and unequivocal signal which will differentiate the moiety of the syndrome due to postconcussional encephalopathy from that of functional origin. Systematic study is required to effect this differentiation.

Comparison of Battle Zone and Civilian Casualties

Swank and Marchand, observing combat exhaustion and combat neuroses during the Normandy campaign, noted the relation of these conditions to blast concussion. A soldier with combat exhaustion usually continued in battle until an acute and severe incident, such as a near miss from artillery or mortar fire, or a heavy artillery barrage, precipitated a breakdown. The soldier became wild and confused and amnesic for that period. He exhibited tremors; fears, particularly provoked by the noises associated with combat; stupor; and confusion. In veteran combat troops, amnesia was very seldom seen, if at all, except in men who were confused and emotionally exhausted or in men suffering from a blast concussion. It seemed certain that practically all infantry soldiers suffered from a neurotic reaction eventually if they were subjected to the stress of modern combat continuously and long enough. About 70 percent of those who were evacuated because of a neurosis had been exposed to an acute incident; that is, direct attack by small-arms fire, bombardment of various types and degrees, or both. A much stronger explosion was required to precipitate the abnormal cerebral state in the veteran soldier than in the untried soldier. It was estimated that a much larger number of veteran troops broke down under the psychic stresses than under the physical stress of a nearby explosion.

In soldiers suffering concussion, there was considered to be either a physiologic or an anatomic injury to the nervous system and its exteroceptive nerve endings, as well as to other parts of the body. They were often reported to have been unconscious or dazed immediately after the explosion, and they remained so for a varying period, for which they were completely amnesic. On recovering consciousness at a battalion aid station or farther back of the line, their hearing was impaired, they had a constant buzzing in one or both ears, and they complained of headache and dizziness. Many bled from the ears, nose, mouth, or all of these orifices. Pains in the chest and other symptoms due to blast might have been present, and frequently one or both tympanic membranes had been ruptured. As the slow recovery from the effects of blast

occurred, over the course of 1 to 3 or 4 weeks, the symptoms of combat exhaustion usually became increasingly evident. In a few cases, the symptoms of blast lasted for as long as 12 months; in these, anatomic damage to the nervous system was probable. Some men with symptoms of severe concussion due to blast exhibited no evidence of combat exhaustion.

PATHOLOGIC CHANGES OF BLAST CONCUSSION

General Effects

Abdominal and pulmonary lesions.—In 1940, Atkins [29] reported cases of immersion blast injury, emphasizing the occurrence of perforation of the intestine, most commonly adjacent to the ileocecal valve, but stressing petechial hemorrhages and lacerations of the brain and lungs as primary causes for death.

Abdominal lesions found at operation and at post mortem were reported by Cameron, Short, and Wakeley,[30] but they failed to mention examinations of the central nervous system, even though several of their patients had been unconscious and some had sustained spinal paralysis.

Most of the reports on the pathologic processes in blast injuries deal with the lesions in the abdominal and thoracic viscera, since, although all organs and parts of the body may be affected, the hollow or air-containing viscera sustain the greatest damage. Injuries to the ears,[31] retinal detachment [32] and even isolated fractures of the first rib associated with blast forces [33] have been reported. When sudden or early death has ensued, it has usually been considered due either to massive disruptive, contusive, hemorrhagic lesions of the viscera or to an indefinable effect upon the body as a whole, in which no single lesion could be held accountable for the death. Hooker [34] observed that there might be complete absence of pathologic evidence of the cause of death in animals subjected to lethal air-blast forces and attributed the phenomenon to primary shock, characterized chiefly by a complete collapse of the blood pressure. He found that the severity of the lesions in the lungs bore little if any relationship to the severity of the gross damage to the brain. He decried his inability to present the results of microscopic studies of the brains of his animals, which were carried out by others and were apparently not published.

Effects on central nervous system.—Evidences of involvement of the central nervous system have actually been more frequently noted than they have been emphasized, and there are, unfortunately, few complete studies which

[29] Atkins, H. J. B.: Lesions from Dover. Guy's Hosp. Gaz. 54: 192-195, 29 June 1940.

[30] Cameron, G. R., Short, R. H. D., and Wakeley, C. P. G.: Abdominal Injuries Due to Underwater Explosion. Brit. J. Surg. 31: 51-66, July 1943.

[31] Bettington, R. H. B.: Injuries to the Ear Due to Blast. M. J. Australia 2: 210-211, 18 Aug. 1945.

[32] (1) Stallard, H. B.: Retinal Detachment: A Series of 78 Cases in the Middle East Force. Brit. M. J. 2: 330-333, 9 Sept. 1944. (2) Evans, J. J.: Organic Lesions From Shell Concussion. Brit. M. J. 2: 848, 11 Dec. 1915.

[33] Gutman, P. E.: Isolated Fractures of the First Rib Associated With Blast Forces. Am. J. Surg. 65: 408-412, September 1944.

[34] Hooker, D. R.: Physiological Effects of Air Concussion. Am. J. Physiol. 67: 219-274, January 1924.

include microscopy of the brains. The chief reason for this lack is that conditions of combat do not permit such studies. In most of the reports of post mortem examinations, the examination of the nervous system is not mentioned, and the inference is that it was not performed. In many other reports, this was specifically stated.

Wilson and Tunbridge [35] reported the autopsy findings in 12 persons killed outright by blast. A 500-pound aerial bomb had burst at one entrance to a deep rock shelter, and, in the opinion of experts, the concrete roof at the opposite entrance had been blown off by the blast. All persons within the shelter were stunned. Those who were later able to go home were dazed and confused. Autopsies were performed on those bodies on which there was no external evidence of trauma, but, because of a delay of 36 hours, the brains were not examined. The principal macroscopic findings were extensive bilateral pulmonary hemorrhage in all cases, hemoperitoneum in 6 cases, and mediastinal hemorrhage in 3 cases. The pulmonary hemorrhage occurred throughout the lungs; there was no constant location of the subpleural hemorrhage. Microscopically, there was seen widespread congestion of the pulmonary capillaries, red blood cells in the alveoli and alveolar epithelium, and occasional emphysema with ruptured alveoli. Other factors which might have caused death, such as burns, noxious gases, or suffocation, were ruled out in these cases.

Wilson [36] observed that the blast injuries in the lungs occurred primarily in the parenchyma immediately adjacent to the intercostal spaces, indicating that the trauma occurred directly to the chest wall. Although the injuries to the lungs were the most severe and the most demonstrable, he did not believe that they were the immediate cause of death. His opinion was that the vital centers had been damaged by the shock wave.

Cerebral Atmospheric Blast Injuries

Citation of cases.—Ascroft [37] reported the case of a gunner, aged 26 years, who was injured accidentally at Benghazi by a small grenade of enemy origin. There was no knowledge of his immediate posttraumatic state of consciousness. When he was examined 2 hours later, he was conscious but apathetic and was slow in answering questions, which had to be repeated. No detailed neurologic examination was carried out, but he was observed to move his limbs. He was operated on for injuries to the hands. He had also sustained many external local lesions in the left chest above the seventh rib, as well as a penetration of the left eye without injury to the skull. He did not recover consciousness and had cyanosis and a bloody froth.

Autopsy revealed hemorrhages in the lung due to the blast (inasmuch as no fragments entered the thorax) and fluid in the pleural cavity. The skull

[35] Wilson, J. V., and Tunbridge, R. E.: Pathological Findings in a Series of Blast Injuries. Lancet 1: 257–261, 27 Feb. 1943.

[36] Wilson, J. V.: The Pathology of Closed Injuries to the Chest. Brit. M. J. 1: 470–474, 17 Apr. 1943.

[37] Ascroft, P. B.: Blast Injury of the Lungs With Curious Lesion of the Cerebrum. Lancet 1: 234–235, 20 Feb. 1943.

and dura were normal. There were evidences of increased intracerebral pressure, in that the gyri were flattened and there was a small cerebellar cone and tentorial grooving. There was also subpial bleeding over wide areas, and the cortex was superficially lacerated in two areas, (1) on the undersurface of the left frontal lobe, behind the olfactory bulb, and (2) in the left temporal region, behind the sylvian point. The left precentral and postcentral gyri were swollen and soft. The gyri and the posterior two-thirds of the right parietal lobe were lilac pink. The junction of damaged cortex and underlying white matter was very discrete. Histologic examination revealed vast numbers of recent capillary hemorrhages in the cortex. No serious change was observed in the nerve cells. There were no fat emboli. Ascroft considered that blast alone was responsible for the lesions in this case.

Bell [38] reported the case of a man who was repairing the roof of a cinema and who lay down on the tiles when a bomb fell 100 yards away. He caught the full force of the blast on the left side of the body. On admission to the hospital, he was confused and irrational. His confusion increased, and he died 2 days later, with signs of consolidation of the left lung. Necropsy showed gross contusion in the left side of the brain. Microscopy showed blood cells among the brain tissue and in most of the alveoli of the lung, in which many of the alveolar walls were torn.

Wood and Sweetzer [39] reported a case of blast injury with hemorrhagic lesions in the brain. A 20-year-old Marine was transferred from the beachhead with the diagnosis of *atmospheric blast concussion. No further history* was available. He was semiconscious when received, did not regain consciousness, and died on his fourth hospital day. There were no localizing neurologic signs. Necropsy revealed contusions of the anterior and lateral thorax. The lungs were heavy, and the lower lobes were red and contained frothy fluid. Small hemorrhages were encountered in the parietal pleura and peritoneum, both surfaces of the diaphragm, and the epicardium. There were lacerations of the liver and spleen.

The scalp, the calvarium, the base of the skull, and the dura mater were intact. The pia-arachnoid vessels were engorged with blood, and the cerebrospinal fluid was blood tinged. The brain contained punctate hemorrhages which were principally in the white matter and were symmetrically located. The body of each caudate nucleus and the midbrain near the substantia nigra were involved. A few hemorrhages were also found in the white matter of the cerebellum, in the brainstem, and in the upper part of the spinal cord.

Microscopy revealed the following: (1) The majority of the hemorrhages surrounded a capillary or a venule; (2) the extravasated red blood cells were well preserved; (3) some of the involved capillaries were thrombosed; (4) there was no instance of fat globules; and (5) the white matter was rarefied in the foci of bleeding, but no phagocytic glial cells were present. In some of the

[38] Bell, R. C.: Analysis of 259 of the Recent Flying Bomb Casualties. Brit. M. J. 2:689–692, 25 Nov. 1944.

[39] Wood, H., and Sweetzer, H. B., Jr.: Punctate Cerebral Hemorrhage Following Thoracic Trauma. U. S. Nav. M. Bull. 46: 51–56, January 1946.

hemorrhagic zones, a ring of astrocytes surrounded the central capillary. No hemorrhagic infiltration was seen around arterioles. Some arterioles and small veins were surrounded by a rarefied zone filled with edema coagulum. In several sections of the white matter, a single fat globule was demonstrated within a capillary, but there was no bleeding around that vessel. The cerebral capillaries were engorged with blood, but there were no fat globules present.

Wood and Sweetzer's explanation of the mechanism of the injury is considered elsewhere (p. 253).

Stewart, Russell, and Cone [40] observed a pheasant found 90 feet away from the craters of two large bombs and reported that, although the bird had no external evidences of direct trauma, it stood about with eyes closed, exhibiting no fear and offering no resistance, and remained in any position in which it was placed "like a catatonic." When the bird was killed and examined, gross hemorrhages were found in the lungs and brain. The cerebral hemorrhages, which were in the forebrain, extended back as far as the third ventricle, surrounding which there was a striking hemorrhage. Microscopic examination revealed the capillaries to be congested throughout. Hemorrhages of various sizes in both forebrains occurred chiefly in small veins and, to a lesser extent, in the capillaries.

Ganado [41] described post mortem examinations on two girls who were killed outright by blast injury. In these cases, there were no secondary traumatic factors and no external evidences of trauma to the skull. There was the usual congestion of the viscera and hemorrhages in the lungs. The brains were found to be very large, edematous, and congested, with flattened convolutions, but no hemorrhages were present. Ganado, who had examined many corpses in which blast injuries had occurred in combination with other injuries, considered these two girls to have been affected by "pure blast," although he proffered no exact cause of death. He considered, however, that the brains were sufficiently abnormal to have accounted for cerebral symptoms had they lived. He stated as follows: "The condition of the brain recalled the clinical observation that many wounded exposed to blast had presented * * * all the signs that are usually associated with cerebral concussion—unconsciousness or drowsiness, amnesia, irritability, headache, etc."

Cohen and Biskind [42] reported autopsy findings in 11 cases of atmospheric blast injuries, considered to be authentic, from the Army Medical Museum. Nine of these casualties had sustained damage to the central nervous system. Pronounced hyperemia of the leptomeninges and the brain was a constant finding. Four cases showed diffuse leptomeningeal hemorrhage, severe in one case and moderate in the other three. Another case showed extensive hemorrhage and a horizontal laceration in the vermis and both cerebellar hemispheres, as well as many small punctate hemorrhages in

[40] Stewart, O. W., Russell, C. K., and Cone, W. V.: Injury of the Central Nervous System by Blast. Lancet 1: 172-174, 8 Feb. 1941.

[41] Ganado, W.: Blast Injuries (correspondence). Brit. M. J. 2: 118, 24 July 1943.

[42] Cohen, H., and Biskind, G.: Pathologic Aspects of Atmospheric Blast Injuries in Man. Arch. Path. 42 12-34, July 1946.

the cerebrum. Still another case showed multiple, discrete, red, punctiform spots involving 1 to 2 cm. of cortex in the right temporal lobe. Three cases showed a few hemorrhages, seen microscopically, in the perivascular spaces. No special cytologic studies were done.

A summary of the symptoms referable to the central nervous system in these 11 cases and the pathologic findings in the brain in 9 cases of the 11 are given in table 8.

TABLE 8.—*Summary of symptoms referable to the central nervous system and pathologic findings in the brain in 9 of 11 cases of atmospheric blast injuries (adapted from Cohen and Biskind* [1])

Case No.	Symptoms and signs	Pathologic findings	
		Leptomeninges	Brain
1	Pronounced restlessness	Hyperemia	Hyperemia.
2	Excitability, irrationality, mania.	No gross lesions	No gross lesions; no special cytologic studies done.
3	Patient dead on arrival at hospital.	Diffuse, moderate hemorrhages.	Hyperemia; no hemorrhages.
4	None mentioned in abstract	do	Focal punctate hemorrhages grossly visible in right temporal lobe.
5	do	Hyperemia	Hyperemia; occasional microscopic and perivascular hemorrhage.
6	Stupor, then extreme restlessness and irrationality.	Hyperemia; focal microscopic hemorrhages.	Hyperemia; focal microscopic hemorrhages.
7	Case omitted by authors		
8	None mentioned in abstract	Hyperemia	Hyperemia.
9	Unconsciousness, disorientation, excitability, hyperactive reflexes, bradypnea.	Diffuse, severe hemorrhage.	Punctate hemorrhages in cerebrum. Large horizontal tear and massive cerebellar hemorrhage.
10	Case omitted by authors		
11	None mentioned in abstract	Hyperemia	Hyperemia; occasional microscopic and perivascular hemorrhage.

[1] Cohen, H., and Biskind, G.: Pathologic Aspects of Atmospheric Blast Injuries in Man. Arch. Path. 42: 12-34, July 1946.

Blast Trauma With Intracerebral Hematoma

In the opinion of Rogers,[43] structural changes have occurred in the brain after blast injury more often than is realized. In support of this opinion, he cited the following case:

A 19-year-old seaman was standing by a 4-inch gun when it discharged.

[43] Rogers, L.: Blast Injury of the Brain. M. J. Australia 2: 209-210, 18 Aug. 1945.

He experienced a sudden stiffness and numbness of the left hand and arm, from which he recovered in 10 minutes. Three weeks later he had the first of a series of attacks affecting the left extremities in a Jacksonian march, beginning with the hand, and gradually giving way to a spastic hemiplegia. During this period, the diagnosis of a possible hysteria was made. The patient, however, contended: "All my trouble started when the big guns fired." After a sufficiently detailed preoperative study, an exploratory craniotomy disclosed subcortical hematoma with an extensive cavity.

This case should more properly be regarded as due to muzzle blast, or air blast, than to blast concussion in its more typical form.

Grunnagle [41] reported a case of intracerebral hematoma resulting from blast injury, without skull fracture. The patient, a sergeant, had previously been observed in the same hospital in August 1944, for treatment of a fecal fistula following appendectomy. He was observed to be an extrovert; he was cheerful and not in the least depressed by his experiences in combat missions during his 30 months' service in the Southwest Pacific Area. During convalescence, he participated actively in the performance of duties about the ward. Because of a severe weight loss, he was evacuated from the hospital 2 weeks after admission, for further convalescence before return to duty.

He was readmitted to the same hospital 2 months later, on 27 October 1944, with a history of having been rendered unconscious by the blast from an enemy knee-mortar shell on 20 October 1944, during the Leyte invasion. He had suffered a minor laceration of the scalp in the left anterior frontal region from a small shell fragment. When questioned, he stated that he thought he had lost consciousness for a few minutes. Accompanying records stated that he been drowsy, listless, and irrational at intervals during the first 3 days after injury. He then had become more alert and complained frequently of frontal headache.

Examination on admission found him listless and disinterested in his surroundings. No information was volunteered, and the history had to be elicited by direct questioning, to which only monosyllabic answers were given. Headaches had been almost constant since the injury, but other complaints could not be elicited; the patient was apparently not aware of the change in his personality. When he was visited by the ward surgeon under whose care he had been during his previous hospitalization, his attitude was one of indifference.

The temperature, pulse, and blood pressure were normal. There was a healed scalp laceration about 2 cm. in length in the left temporal region. Roentgenograms of the skull were negative for fracture. There was a small metallic foreign body 2 mm. in size in the scalp, in the left temporal region.

Neurologic examination revealed slight resistance on testing for neck rigidity. Kernig's sign was negative. There was no evidence of aphasia, although the patient limited his conversation. The nasal borders of the optic disks were moderately blurred, and there was some engorgement of the retinal

[41] Grunnagle, J. F.: Traumatic Intracerebral Hematoma. J. Nerv. & Ment. Dis. 103: 298-300, March 1946.

veins, but there was no measurable elevation of the optic disks. Visual fields appeared normal to gross testing. There was weakness of the right lower face. Otherwise, examination of the cranial nerves was not remarkable. Upon extension of the arms, slight wandering of the right arm occurred, and dysdiadochocinesia was also noted on the right side. No difference in motor power and no other abnormal neurologic findings could be detected.

Spinal puncture, performed on 31 October 1944, revealed a pressure of 260 mm. H_2O and deeply xanthochromic fluid. The Pandy reaction for globulin was 3 plus. No cells were seen. Repetition of the procedure on 5 November 1944 revealed an increase in the pressure to 330 mm. H_2O. The fluid itself was of clear consistency and xanthochromic. The Pandy reaction was 3 plus, and there was one cell per cubic millimeter.

The patient's general condition remained unchanged, but, during the following 2 weeks, he became more drowsy. His personal habits deteriorated, his bed being constantly dirty with cigarette ashes, butts, and particles of food. He became incontinent of urine and used foul language in the presence of the nurses. The facial weakness became more pronounced, and the optic disks became elevated one diopter.

A diagnosis of subdural hematoma or left frontal intracerebral hematoma was considered, and on 10 November 1944 under local procaine infiltration, an exploratory trephine opening was made in the left frontal region. The exposed dura was of normal color. When it was opened, however, the cerebral cortex was found brownish yellow and bulging through the dural opening. There was no evidence of subdural fluid or blood clot, but at a depth of 3 cm. approximately 1 ounce of dark, liquid, old blood was found and evacuated.

The patient rapidly became alert and interested in his food. He requested reading material. He volunteered that he had no headache. Forty-eight hours after operation, he became continent of urine. He was permitted out of bed on the third day after operation and was soon fraternizing with other patients. His reactions 1 week later resembled those on his first admission, before his blast trauma, and his personal appearance and habits again became neat and clean. When he was evacuated on 27 November 1944, the papilledema had subsided to only slight haziness of the disk margins. The remainder of the neurologic examination was negative except for a minimal lag of the right lower face on spontaneous smiling.

Subdural Hematoma Associated With Blast Injuries

Among the patients discussed in a symposium on immersion blast injury was one reported by Hamlin [45] with a subdural hematoma. This man, while floating in the water, was rendered unconscious for a short period by an underwater explosion. He then vomited and was in shock. Severe injury to the abdominal organs from the blast caused death 8 days later. Meanwhile, the patient was wildly excited, delirious, and disoriented. The subdural hematoma

[45] Hamlin, H: Neurological Observations on Immersion Blast Injuries. U. S. Nav. M. Bull. 41: 26-32, January 1943.

found post mortem was over the left temporal region and measured 8 by 4 by 6 cm. Hamlin surmised that the subdural hematoma had occurred as a result of a direct blow to the head, but the detailed notes in the other portions of the symposium regarding this patient would seem to rule out any basis for this surmise. The case exemplifies the occurrence of both cerebral concussion and subdural hematoma, both of which may have contributed to the abnormal mental state.

Abbott, Due, and Nosik [46] observed a number of blast injuries, both of the atmospheric and immersion types, in which post mortem examinations revealed multiple hemorrhages throughout the body, varying from petechiae to frank bleeding. They reported in detail 3 out of a total of 10 cases in which subdural hematomas producing the typical syndrome were found and relieved by surgical procedures. In five of these cases, the hematomas were bilateral and several patients had had subdural effusions.

Blast Concussion With Delayed Posttraumatic Cerebral Hemorrhage

In discussing cases of concussion in which there had been no solid blows to the skull, Grinker and Spiegel [47] described two instances of delayed posttraumatic hemorrhage which had come to their attention. These patients had been exposed to nearby explosions, but the type of explosive and the distance of the explosions from the patients were not documented in the report. Both were concussed and had had symptoms persisting for several weeks after their battle experiences and consisting of anxiety, insomnia, and restlessness. Because of the absence of any sign of external injury, the patients had been treated for neurosis. At a later date, one of them had a sudden fall to the ground, fractured his skull, and died of bleeding from a ruptured meningeal blood vessel. Autopsy revealed, in addition, old hemorrhages within a severe laceration of the frontal lobe. The second patient died after severe frontal headache and coma had developed. Autopsy revealed extensive old injury to the frontal lobes, into which fresh hemorrhages had taken place. Grinker considered these cases as examples of blast concussion. He stated in a personal communication: "Perhaps some of our discharged patients with anxiety states die in other hospitals with terminal hemorrhages into lacerated brains. It is unfortunate that followup studies cannot be made; yet our opinion is that such cases are infrequent."

A case in point is reported from the study by Cramer, Paster, and Stephenson.[48] It was an instance of cerebral concussion due to the blast from a nearby mortar-shell explosion, with prolonged unconsciousness and amnesia. The injury was followed by posttraumatic headache, dizziness, irritability, and in-

[46] (1) Abbott, W. D., Due, F. O., and Nosik, W. A.: Subdural Hematoma and Effusion as a Result of Blast Injuries: First Preliminary Report. J. A. M. A. 121: 664–666, 27 Feb. 1943. (2) Abbott, W. D., Due, F. O., and Nosik, W. A.: Subdural Hematoma and Effusion as a Result of Blast Injuries: Second Preliminary Report: Diagnosis by Psychiatric Examination. J. A. M. A. 121: 739–741, 6 Mar. 1943.

[47] Grinker, Roy R., and Spiegel, John P.: War Neuroses of North Africa: The Tunisian Campaign (January–May 1943). New York: Josiah Macy, Jr. Foundation, 1943.

[48] See footnote 26, p. 224.

tolerance to noises; death, from delayed posttraumatic cerebral hemorrhage from an area of encephalomalacia due to the blast injury occurred 3 months later. The details were as follows:

Case report.—A 24-year-old white American private was admitted to Kennedy General Hospital, Memphis, Tenn., on 9 February 1943, after transfer through a chain of evacuating hospitals, with the transfer diagnosis of "Psychoneurosis; War Neurosis."

While in battle in Guadalcanal in November 1942, 3 months earlier, he was lying prone in a shallow trench when an enemy mortar shell exploded nearby, rendering him unconscious. He was taken to a hospital by litter and was told that he remained unconscious for 8 hours. According to his own statement, he remained dazed and amnesic for the next 2 weeks. The hospital records indicated that there was no sign of external injury to the head or elsewhere. During this period, he took nourishment and attended to his physical needs but was restless and complained frequently.

About 1 week after the trauma, he was removed to a base hospital, where he remained 6 weeks. He complained of headache, dizziness, tinnitus, and irritability to noises. He was described as being depressed, with a diminished affect and a tremor of his hands. Occasional unsteadiness of gait was noted, as well as past-pointing in the finger-to-nose test. Medication afforded him no significant relief from his headache, and it remained extremely severe for about 6 weeks. It was apparent from the hospital record that the patient's symptoms were interpreted as of functional origin.

His complaints remained essentially unchanged in quality upon admission to the neuropsychiatric service of the Kennedy General Hospital except that the headache was no longer as severe as it had been for the first several weeks. He appeared well nourished. He was somewhat restless and tense, but he mingled freely with the other patients and participated in various hospital recreational activities. Psychiatric examination did not reveal any personal determinants indicating a psychoneurosis. He continued to complain of headaches, but his tension subsided considerably.

On 12 February 1943, 3 days after admission, while straining at stool, he had a sudden, extremely severe pain in the temporal region (side not specified), which became gradually, progressively, and relentlessly worse. He had extremely severe nuchal pain and vomited several times. Neurologic examination revealed nuchal rigidity and Kernig's sign, apathy, and restless somnolence. The deep reflexes were slightly preponderant on the left side, with the biceps jerk spreading to the fingers, and a positive Hoffmann sign. Plantar flexion was normal on the left, but on the right, there was fanning of the toes. There was a lower facial droop on the left side.

Serologic studies of the blood, as well as the chemistry and cytology of the urine, were normal. The cerebrospinal fluid was grossly and uniformly bloody.

The patient's mental attitude and emotional state were normal except for his restlessness and emotional response to his pain, and the display of some annoyance when the neurosurgeon attempted to elicit a detailed history of the trauma and the subsequent course leading up to his recent acute complication. He was loathe to talk about his condition, his explanation being that he felt that he had not been believed when he had been interrogated at various times in the past. Asked to state his chief complaint in his own words he said:

"I don't 'complain,' sir. But only since you insist: I have had constant headache and dizziness since I recovered consciousness, and treatment has given me no relief. I have been told that I am psychoneurotic and that my complaints arise from my mind. I am not competent to discuss a medical diagnosis, but to me my complaints have been very real. The headache I have now is new and different from the one I had before. I don't care what the diagnosis is, as long as I get some relief."

This patient grew progressively and sporadically worse, with crises of slow pulse, syncope, and excruciating headache. He was seen in neurosurgical consultation because the

persistence of severe headache since the onset of his illness made it desirable to rule out chronic subdural hematoma, complicated by recent subarachnoid hemorrhage.

The possibility of chronic subdural hematoma seemed slight, but because of the urgency of his present condition, bilateral trephine explorations were carried out in the early morning of 19 February 1943. They revealed only some blood in the subarachnoid space, with increased cerebral tension and edema of the cortex.

The clinical course continued progressively bad. The patient became apathetic and febrile. Fixed, irregular, dilated pupils developed, the left being larger than the right He expired on 20 February 1943, after Cheyne-Stokes breathing, a rapid, thready pulse, and apnea had developed.

The clinical diagnosis was (1) ruptured congenital aneurysm in a case of cerebral blast concussion or (2) cerebral blast concussion with delayed posttraumatic hemorrhage (spät-apoplexie).[49]

The autopsy findings (Maj. Emmerich von Haam, MC) were as follows:

Gross pathology.—The skull and dura, aside from the bilateral posterior trephine openings, were normal. The leptomeninges were normal. The subarachnoid space, chiefly around the pons and brain stem, contained hemorrhage. The brain weighed 1,280 gm. The sulci were slightly flattened. There were no congenital aneurysms or varices. The basilar vessels showed no evidence of injury or arteriosclerosis. The cervical spinal cord removed with the brain showed no gross pathologic change.

Sectioning the brain after fixation revealed a massive hemorrhage involving and destroying the posterior part of the internal capsule, the caudate nucleus, and parts of the thalamus on the left side. The hemorrhage had perforated into the lateral ventricle. The tissue surrounding the hemorrhage was soft and grayish brown and contained numerous small hemorrhages. The point of the bleeding vessels could not be determined. The brain tissue of the right hemisphere was edematous.

Microscopic pathology.—Acute changes noted in the site of the lesion were massive hemorrhage with extensive destruction of the brain tissue and numerous secondary hemorrhages. Other sections of the brain showed diffuse edema and an occasional thrombosed vein which was markedly dilated.

Chronic changes in the tissue adjacent to the hemorrhage were characterized by severe myelin degeneration with beginning gliosis and some hemosiderin pigment. The ependymal lining was intact, but subependymal gliosis and edema were present. No pathologic changes were found in the cerebellum.

The cause of death was attributed to a severe intracerebral hemorrhage with increased intracranial pressure and extensive destruction of brain tissue. The age of the patient and the normal appearance of the entire arterial system, including the basilar artery and its branches, made arteriosclerotic, syphilitic, or mycotic arteritis rather improbable. No miliary aneurysms were found.

The pathologic diagnoses were severe intraventricular hemorrhage and extensive, subacute, traumatic encephalomalacia (due to blast concussion). The post mortem findings, with the clinical history, pointed to the relationship of this brain hemorrhage to the blast injury suffered in the Pacific war area.

CLINICAL FEATURES OF BLAST CONCUSSION

Atmospheric Blast Concussion

In a neuropsychiatric-neurologic-neurosurgical center of the Zone of Interior, Cramer, Paster, and Stephenson observed a large group of cases of the closed head injury syndrome in which blast concussion had been the responsible

[49] Bollinger, O.: Ueber traumatische Spät-apoplexie. Internat. Beitr. z. Wissensch. Med. 2: 459–470, 1891.

factor. Most of the patients had been evacuated from overseas as neuropsychiatric problems. About half had been given the primary diagnosis of psychoneurosis, further qualified by such diagnoses as war neurosis and conversion hysteria. In a few instances, the diagnosis of chronic subdural hematoma had been made early in the course of the illness, on the basis of persistent headache, abnormalities of the reflexes, inequalities of the pupils, and, very occasionally, a shift, demonstrated roentgenologically, of the pineal gland to one side of the midline. These patients had had surgical explorations to rule out hematoma.

It is of interest that this hospital did not receive any cases of blast injury in which subdural hematoma was found. This is in contrast to the observations of Abbott and his associates and Hamlin. The apparent absence of subdural hematomas does not rule out the possibility of the occurrence of small subdural hematomas or effusions such as one often sees in autopsies in cases of skull injury in which these particular lesions could not be diagnosed ante mortem because they were not acting as expanding lesions.

In a few other cases, in which there were objective neurologic changes, blast concussion was the primary diagnosis. In one case, acute cerebral hemorrhage due to blast injury was diagnosed immediately. For the most part, however, it was apparent that a psychiatric, interpretative approach was used during the early observation and diagnosis of most of these cases. It was more readily apparent in the Zone of Interior, on an average of 5 months after the injury, that not psychogenic factors but the residua of an organic trauma were producing the persistent symptoms.

The selection of cases for this study was made from patients in whom direct trauma, either by missile or blunt blow, or by gross acceleration and sudden deceleration of the head, could be ruled out with as much certainty as can be brought into a clinical problem of this type.

The causes of trauma were explosive missiles (mortar shells in about a quarter of the cases), artillery shells of both Allied and enemy origin, and aerial bombs. Most of the soldiers had had some sort of protection by trenches. Several of them, exposed to blasts from heavy shells, estimated that they had been within the lethal range of that type of shell, while some believed that they were within 50 to 150 feet of exploding 155-mm. shells. About a third of the patients were in the same hole or slit trench, or in the same immediate site, as other soldiers who were either killed outright by the blast or were injured by it, in contrast to injury by the fragments of the shell or debris from the explosion.

Early symptoms.—Early symptoms were as follows:

1. *Unconsciousness.*—This was usually immediate. In a few cases it was delayed for a matter of moments, or minutes, during which the individual had the opportunity to attempt to do something practical, such as run to the assistance of other wounded or seek better shelter. In these cases, the neurologic picture, both early and late, was significant of definite cerebral hemorrhage.

About 20 percent of the patients were unconscious for less than 30 minutes, 60 percent were unconscious for from 9 to 18 hours, and the remaining 20 percent were unconscious for from 2 to 5 days. It is, of course, impossible to try to estimate for what part of this period the soldier was in coma and for what part in stupor and apathy. Because of the amnesia which followed, the patients usually estimated the period of lost consciousness to be longer than that indicated on available detailed records. The records more frequently described the patients as being confused, dazed, and disoriented. A further complication arises from the fact that hypnotics were administered in effective doses early in the course of treatment.

2. *Headache.*—The first awareness after regaining consciousness was usually of an extremely severe bursting type of headache, which continued unabated, for a number of days or up to a few weeks, before it began to remit to any notable degree. This headache did not respond to analgesics, and time alone seemed to be the chief leaven.

3. *Tinnitus.*—This symptom occurred almost invariably but was not as intolerable as most of the other symptoms.

4. *Dizziness.*—This was frequently described in terms of true vertigo and had about the same frequency and course as the tinnitus.

5. *Tremors.*—These took various forms and occurred in about half of the cases. Usually, they were generalized and consisted of violent involuntary trembling of all four extremities and the body. In a few cases, the tremors were monolateral or were confined to one extremity, usually the arm; in these cases, the homolateral side of the face was also involved. Occasionally, the tremor was confined to the head. Other involuntary movements observed were seeming purposive movements. In one case a facial motor tic developed.

6. *"Nervous" symptoms.*—"Nervousness" was the term most patients used to describe their other symptoms. The most frequent complaint was the inability to tolerate loud or sudden noises or sudden movements, which often precipitated marked startle responses. Restlessness, anxiety, and fearfulness were frequent. Fear was either spontaneous or in response to stimuli, such as the explosion of a shell or the passage of aircraft overhead. Some patients were wildly disoriented as well as fearful during the early phases and required heavy sedation, which doubtless contributed to many of the reports of prolonged amnesia. Insomnia and battle dreams occurred frequently.

7. *Symptoms of neurogenic origin.*—Anesthesia and paralysis were usually very transient and were of various types, quadriplegic, hemiplegic, or monoplegic.

Late symptoms.—The late symptoms were as follows:

1. *Headache.*—In about 60 percent of the cases, the severe headache usually did not persist without surcease for longer than 1 to 4 weeks. In about 20 percent, it persisted longer, sometimes with spontaneous relief and at other times with relief following medication. No constant references either to changes in the quality or location of the headache could be elicited, although

altering the position of the head sometimes played a part in the production or severity of the symptom.

2. *"Nervous" symptoms.*—"Nervousness" was complained of by most of the patients. It was difficult for them to elucidate this symptom further. The inability to tolerate sudden or loud noises was the next most serious complaint. About a quarter of the patients stated that they could not adjust to the excitment of social gatherings and had been forced to forego this activity because of their loss of equanimity. The same was true when they found themselves in the presence of arguments and discussions. They became acutely uncomfortable and were forced to withdraw, whether the discussions were friendly, serious, or contentious.

3. *Dizziness.*—This partook of true vertigo in less than 10 percent of the cases. Most patients described only momentary periods of feeling dazed or light headed.

Physical examination.—Unfortunately, complete neurologic examinations of the early phases of these conditions were infrequent in the medical records, and, as already noted, they were more often interpretative than objective.

In the last phases, despite the anxiety and dejection which still colored the patients' emotional behavior, their bearing was nevertheless unusually cooperative and relatively dispassionate and objective. The attitude displayed by the patient whose case is described in detail in the section on pathologic changes (p. 234) serves as a prototype for the group.

Neurosurgical evaluation.—The group of patients who were selected by the neuropsychiatric service for neurosurgical evaluation were examined as critically as though they were suspected of harboring brain tumors. For the most part, gross neurologic discrepancies did not persist. Slightly more than a third of the patients still displayed some tremor, usually fine and rhythmic, occasionally of a coarser type, and most often involving the extremities on the side on which there was a slight hyperreflexia. The head and face were involved only infrequently. About 25 percent displayed slight but definite and persistent inequalities in the deep reflexes, one side having a relative hyperreflexia. Pupillary inequality, however, occurred more frequently than the inequality of the reflexes; it was observed as often on the right side as on the left. It was noted on repeated observation in slightly less than 50 percent of the cases that one pupil was consistently larger than the other. When both conditions were found in the same individual, they were more often contralateral than ipsilateral. This observation was one of the factors leading to the request for neurosurgical consultation, since this finding is well known in chronic subdural hematoma.

In the earlier course of the collaboration, pneumoencephalography was carried out in these particular patients. There was usually a visible and measurable enlargement of one of the lateral ventricles, although no evidence of subdural hematoma was found. The enlargement of the ventricle was

almost always contralateral to the hyperreflexia. It was interpreted as evidence of a slight cerebral atrophy resulting from the encephalopathy following the trauma.

In a comparable study, Aita and Kerman [50] reported significant findings in seven cases (20 percent of their series). Their findings consisted of pathologic reflexes, brain-stem cerebellar-tract signs, brain-stem cranial nerve disturbances, masked facies, tremor, and, in one case, unilateral optic nerve injury resulting in blindness. The group sustaining concussion by solidborne head injury showed significant changes in nine cases (30 percent), pathologic reflexes being the most outstanding finding. Aita and Kerman specifically avoided the inclusion of cases in which gross primary injury to the brain or skull had occurred and rigorously selected those cases in which the clinical histories indicated concussion to have been caused by blast alone. They thus chose to group the two categories of cerebral concussion and encephalopathy together under the closed head injury syndrome.

Reports from the literature.—The case of a man sustaining cerebral blast concussion was described by Tunbridge.[51] The accident was viewed by eye witnesses, as follows:

A soldier, 32 years of age, was in an open square with two friends when an enemy aircraft dived to attack a neighboring building. The other two men fled for cover, while the patient decided to lie flat in a small gulley. The bomb dropped within 5 yards of the gulley. He remembered nothing further of the incident and was unconscious until he awoke in an ambulance. On admission to the hospital, he was highly disoriented and irritable and had a complete amnesia for the incident. He remained disoriented for about 48 hours. He did not know that one of his friends had been killed by the explosion. The amnesia was not broken down by Pentothal Sodium (thiopental sodium).

Physical examination of this patient showed hemorrhage into, and congestion of, the eardrums. His face was pitted with small particles of dust. Roentgenograms showed no fracture of the skull, but lumbar puncture revealed uniformly bloodstained spinal fluid under 200 mm. H_2O pressure; this procedure gave relief from headache. This soldier eventually recovered and returned to duty.

Barrow and Rhoads [52] also described as one of the clinical characteristics of blast concussion in the patients whom they observed a pronounced decrease in psychomotor activity. Their patients were listless and apathetic and seemingly overcome by fatigue and lassitude. They were content to stay in one place, made no conversation, and had few requests. They did not complain or cry out with pain. They did not request water or sedation. They were men stunned and stupefied. Unconsciousness was of short duration. Tremor such as that sometimes associated with intense emotion was common at the

[50] See footnote 25, p. 240.

[51] Tunbridge, R. E.: Cause, Effect and Treatment of Air Blast Injuries. War Med. 7:3–6, January 1945.

[52] See footnote 21, p. 221.

time of hospital admission of conscious patients. Convulsions were observed in some of those who were fatally injured, and others had muscular twitchings. Memory was normal for the events leading up to the period of unconsciousness and was not impaired after recovery of consciousness.

The vital condition of these patients was characterized by hypotension, a slow pulse, slight fever, and shallow, sighing, slow respiration. When death occurred, it was almost instantaneous. A survival of as long as an hour was usually followed by recovery.

Similarly, Klemm,[53] selecting 36 patients out of 90 such admissions during one month, studied them 10 to 20 days after they had sustained blast concussion. The injuries resulted from a variety of missiles, which were estimated as having exploded from 2 to 15 feet away. Klemm found that the clinical history conformed to a uniform pattern, which was quite like that described. Of the patients considered to have had pure blast injury, 25 percent had hemoptysis, 25 percent had epistaxis, and 50 percent had vomiting. He did not report the details of neurologic examinations, which showed no remarkable changes. Two patients exhibited generalized myoclonus, which in one case persisted during sleep. Persistent apathy and fatigue were noted. Klemm had no reservations concerning the etiologic relationship of the blast factor or the diagnosis of a true concussion, but he was unable to determine to what extent the harrowing battle experiences had contributed to the persistence of the patients' apathy and fatigue.

Stewart, Russell, and Cone [54] also reported a small series of patients exposed to blast. Their patients showed no mark of external violence and had no focal neurologic signs of injury to the brain. Some of them, however, when examined some days later, had xanthochromic spinal fluid and evidence of damage to the anterior horn cells of the spinal cord. Their mental state was characteristically either apathetic and indifferent or depressed.

In common with others, Rogers [55] "* * * noted the apathy, lassitude and dejection of certain of the human victims of air raids." He thought that bloody or xanthochromic spinal fluid indicated small scattered cerebral hemorrhages in the central nervous system and believed that they occurred much more often than had been recognized.

In a naval hospital in the Zone of Interior, Bailey [56] found that 25 percent of the patients considered to have had blast concussion displayed organic symptoms of various degrees, while 75 percent were regarded as having purely neurotic symptoms. The latter symptoms were chiefly anxiety and occasional evidence of hysterical fragments, but no obsessive-compulsive states were observed. Electroencephalograms and details of the neurologic examinations were not reported.

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[53] Klemm, R. A.: Atmospheric Blast Concussion: Medical Aspects. U. S. Nav. M. Bull. 44:1228–1230, June 1945.

[54] See footnote 40, p. 230.

[55] See footnote 43, p. 231.

[56] Bailey, P.: Neuropsychiatric Casualties Resulting From Exposure to Atmospheric Blast. Arch. Neurol. & Psychiat. 55, 551–553, May 1946.

A battery of psychologic tests and psychiatric, neurologic, and electroencephalographic examinations were employed by Lynn[57] in the differentiation of organic states as opposed to the neurotic reactions in closed head injuries. In 2 of the 13 cases selected for this special study, the history and the evaluation were definitely corroborative of blast concussion as the primary cause of the persistent syndrome.

Immersion Blast Concussion

In a symposium on immersion blast injuries, Hamlin[58] presented the neurologic observations in 12 persons out of 35 who were injured by underwater explosion while swimming or floating after the loss of their ship. Five had been definitely rendered unconscious, and another was probably unconscious. Ten had red blood cells in the spinal fluid. Most had headaches. Other findings, in varying degrees, were amnesia, disorientation, somnolence, and personality changes. One patient, who exemplified the typical blast-concussion phenomena, was of particular interest because he had sustained an earlier air-blast concussion, as well as another immediately preceding his exposure to immersion blast.

This patient had bursting retro-orbital headache, tinnitus, sensitivity to noises, and nightmares of battle experience. He had spontaneous, involuntary flexion movements of the upper and lower extremities involving postural muscle groups; elevation and forward rotation of the shoulders; and occasional contractions of the muscles of the neck and trunk. The movements were paired and became exaggerated under observation and conscious effort to control them. They ceased with sleep. His speech was halting, emissive, and aphasic. When distracted, he had difficulty returning to his train of thought. His memory performance and calculation tests were poor. He had agnosia for right and left. Yet his personality made a good impression, and the findings were considered, by most observers, to be more organic than functional and suggestive of extrapyramidal as well as diffuse insult to the cerebral cortex.

Cerebral Contusion From Blast

A nonfatal case of blast injury with neurologic signs but without direct trauma to the head of "concussion" (unconsciousness) was described by Garai.[59] A 29-year-old soldier heard a bomb in the air and threw himself into a gutter. The bomb burst a short distance away, and, as he started to arise, a second bomb fell behind and to the left of him; the resulting blast blew him a few feet across the pavement. He did not strike any object and was not unconscious; he remembered seeing a man collapse some distance ahead. When the patient arose to walk, he experienced pain in the small of his back, blood came from

[57] Lynn, J. G., Levine, K. N., and Hewson, L. R.: Psychologic Tests for the Clinical Evaluation of Late "Diffuse Organic," "Neurotic," and "Normal" Reactions After Closed Head Injury. A. Research Nerv. & Ment. Dis., Proc. (1943) 24: 296-378, 1945.

[58] See footnote 45, p. 233.

[59] Garai, O.: Blast Injury: Non-Fatal Case With Neurological Signs. Lancet 1: 788-789, 17 June 1944.

his mouth, and he had bloodstained sputum and hematuria, all typical symptoms of blast injury.

On examination 36 hours later at Sutton Emergency Hospital, he was rational and composed. He had a slight icterus but no ecchymosis. Neurologic examination revealed the right pupil to be moderately dilated, without light reaction, and sluggish to accommodation; this finding still remained detectable a year later. The left plantar reflex was extensor in reponse. The cerebrospinal fluid was under 100 mm. H_2O pressure; it contained 20 mg. of protein, was clear, and contained no cells. The Wasserman, Kahn, and Lange tests were all normal. The general physical examination revealed bloodstained sputum, diminished excursion of the left side of the chest, patchy impairment of the percussion sound, and displacement of the trachea toward the left. There was impaired translucence of the left chest on roentgenologic examination, without evidence of tuberculosis. The pulse rate was 70 and the blood pressure 130/75. The abdomen was tender to palpation in the left loin. The urine, for 1 week, contained some blood and albumin.

Garai considered that—

> The right sided pupillary changes associated with signs of pyramidal involvement on the left suggest a lesion, possibly haemorrhagic, in the peri-aqueductal grey matter of the upper part of the midbrain. This may have been the result of contrecoup; or the effects of blast on the thorax and abdomen may have caused a sudden increase in venous pressure in the CNS with resulting haemorrhages.

The electroencephalographic records of this case in the acute and chronic stages, which are of great interest, are reported in the section of this chapter devoted to the electroencephalogram (p. 248).

In 26 out of 259 cases of injury from flying bombs, Bell [60] observed that there were 4 instances of cerebral concussion and 3 of cerebral contusion. Two of the three patients in the second group had blood in the cerebrospinal fluid; one had sudden paralysis of the left arm, and one, described under the section on pathologic changes (p. 240), had free blood cells in the brain tissue. Bell also described two cases of paralysis of the pupillary mechanism, and, although he attributed the paralyses to fear, it seems more proper to consider them as of organic rather than psychogenic origin, since it is highly improbable that even acute fear alone can produce a persistent paralysis of the pupillary light reflex. The condition of one of these patients was described as acute; his pupils were widely dilated and did not respond to light. The other patient was an intelligent woman, aged 24 years, who had widely dilated pupils; the right did not respond to light, and the left contracted only slightly. In a few days, the left eye improved, and in about 6 months she made a full recovery. It should be noted that there was also marked sweating in this case, and the inference is that both the pupillary reflex and the sweating mechanisms should properly be considered as evidences of lesions in a cerebral vegetative center resulting from the intrinsic changes precipitated by the blast injury.

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[60] See footnote 38, p. 229.

THE ELECTROENCEPHALOGRAM IN BLAST CONCUSSION

Three groups of workers have made encephalographic studies in relatively large numbers of cases of closed head injury due to blast. Von Storch [61] obtained electroencephalograms during the first hours and days after exposure to blast injury in 33 patients. Aita and Kerman [62] studied 34 patients in later stages. Stephenson [63] obtained routine electroencephalograms in 441 cases in which the diagnosis of blast concussion had been made, based on the clinical criteria already enumerated. In Stephenson's group, unconsciousness had occurred in 329 cases. Positive evidence of blast damage as demonstrated by bleeding from the ears or nose, rupture of the eardrum, or conjunctival hemorrhages was found in 77 cases. Five patients who had never had convulsions before the injury gave histories of convulsive seizures following exposure to blast. The history of exposure to blast was doubtful in 2 patients included in the series, and 3 were believed by the referring medical officer to be suffering from psychoneuroses, with questionable exposure to blast injury.

Whereas many of the patients with milder blast concussion encountered by Fabing and by Von Storch were returned to duty, the patients on whom Stephenson's study was based represented those who had received more serious concussion and had been returned to a hospital in the Zone of Interior. These studies, therefore, represent only the late, residual changes. The minimum time which elapsed between the injury and the electroencephalogram ranged from 3 weeks in 1 case to at least 80 weeks in 6 cases, the mean being 24.4 weeks. The fact that changes in the electroencephalogram persisted so long after trauma emphasizes the probability of real, irreversible traumatic changes in the brains of these individuals.

The Results of Electroencephalography

The electroencephalograms obtained from the individuals studied by Stephenson ranged from entirely normal to pathognomonically abnormal, as judged by standard works on the subject.[64]

In figures 47 and 48, the distribution of normal, borderline, and abnormal electroencephalograms in eight clinical groups of soldier patients is compared with normal controls. The group suffering postconcussional symptoms after blast injury had a larger percentage of normal readings than the group with skull defects, epilepsies, focal injuries, and miscellaneous organic conditions, but fewer normal readings than the group with migraine, other headaches, and miscellaneous psychoses. The percentage distribution is represented in table 9, in which it will be seen that a control series of 140 cases of normal individuals

[61] See footnote 22, p. 222.

[62] See footnote 25, p. 224.

[63] See footnote 26, p. 224.

[64] (1) Davis, P. A.: Technique and Evaluation of the Electroencephalogram. J. Neurophysiol. 4: 92-114, January 1941. (2) Gibbs, F. A., Gibbs, E. L., and Lennox, W. G.: Electroencephalographic Classification of Epileptic Patients and Control Subjects. Arch. Neurol. & Psychiat. 50: 111-128, August 1943. (3) War Department Technical Bulletin (TB MED) 74, 27 July 1944.

(civilians) contained only 9.2 percent of abnormal tracings, whereas a series of blast concussion cases contained 15.9 percent of abnormal records.

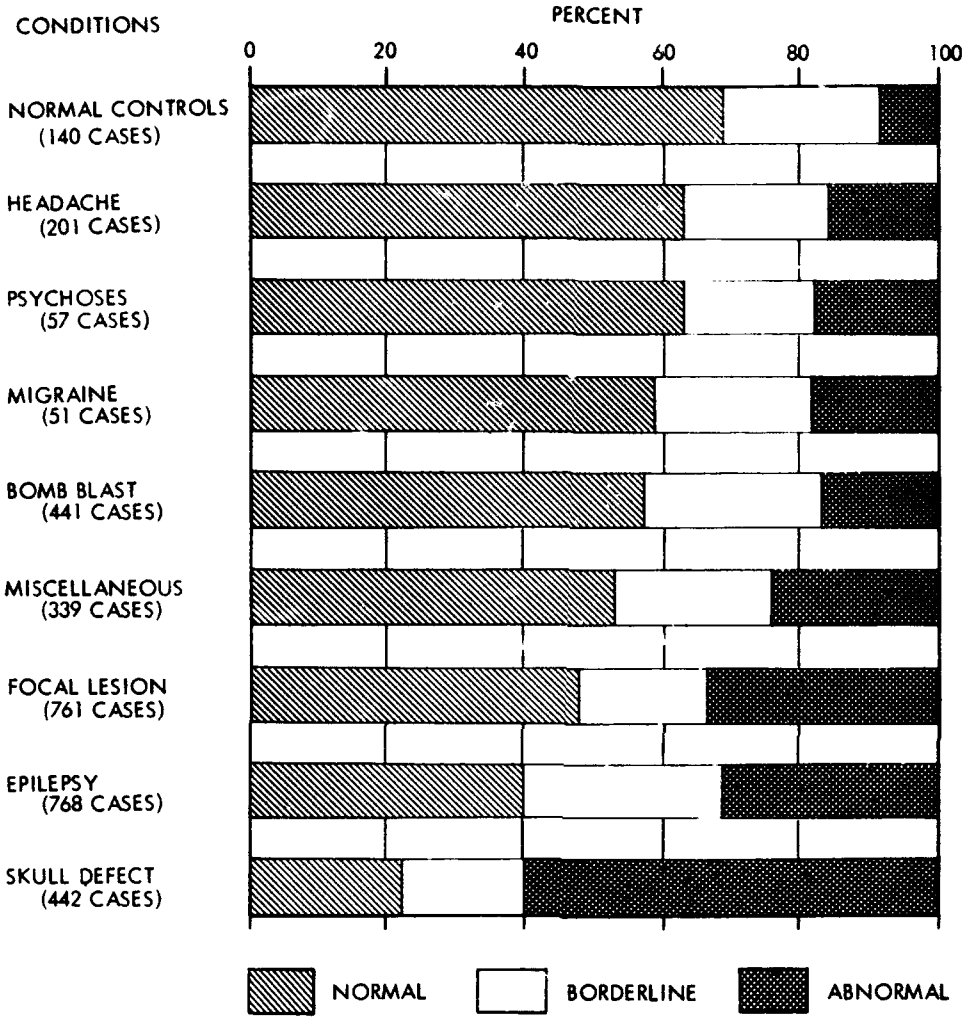


FIGURE 47.—Distribution of normal, borderline, and abnormal electroencephalograms in eight clinical groups of symptoms and signs compared with normal controls.

TABLE 9.—*Percentage distribution of normal, borderline, and abnormal electroencephalograms in two series of late cases of blast concussion*

Series	Normal	Borderline	Abnormal
	Percent	*Percent*	*Percent*
Controls [1]	68. 6	22. 2	9. 2
Blast concussion [1]	56. 5	27. 6	15. 9
Blast concussion [2]	57. 0	36. 0	7. 0

[1] Cramer, F., Paster, S., and Stephenson, C.: Cerebral Injuries Due to Explosion Waves—"Cerebral Blast Concussion." A Pathologic, Clinical and Electroencephalographic Study. Arch. Neurol. & Psychiat. 61: 1–20, January 1949.

[2] Aita, J. A., and Kerman, W. Z.: The Closed Head Injury Syndrome Due to Blast. Bull. U. S. Army M. Dept. 6: 411–427, October 1946.

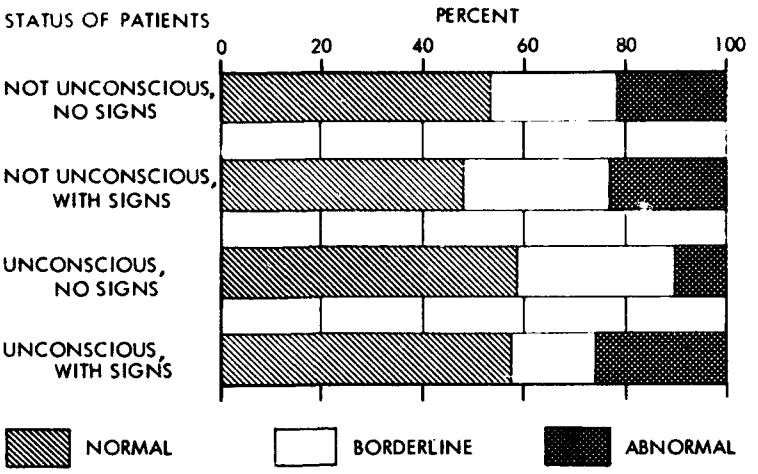


FIGURE 48.—Distribution of normal, borderline, and abnormal electroencephalograms after bomb-blast concussion with and without unconsciousness and with and without physical signs of blast injury.

Stephenson's figures in table 9, when subjected to the chi square test (a statistical test which measures the degrees of difference between the actual frequencies) show definite, statistically significant deviation from the normal. The incidence of borderline and abnormal records obtained from patients exposed to blast injury falls well outside the limits of probable chance. In the group of patients who had signs of neurologic disturbance, the recordings show a greater incidence of abnormal electroencephalographic findings than in those without abnormal neurologic signs.

Aita and Kerman included a greater number of cases under the borderline heading. From the point of view of statistical probability, it would seem that more of their cases might have fallen under the abnormal classification. This point is raised because the incidences of normal findings in both series are within 0.5 percent of each other, whereas the incidence of abnormal findings in Aita and Kerman's study is identical for solidborne concussion, blast concussion, and psychoneurosis (7.0 percent). Heppenstall and Hill [65] also found a 57-percent frequency of abnormal encephalograms in the postconcussion syndrome.

In the group of acute cases studied by Von Storch, the electroencephalogram, in patients injured by blast, with questionable evidence of unconsciousness, tended more toward abnormality than it did in those patients who were definitely rendered unconscious by the blast injury. Stephenson's group also noted a greater aberration toward borderline and abnormal electroencephalograms in the patients who had not suffered a loss of consciousness than in those who had; his examinations were made in the later stages. In the later stages, however, the patients who had positive physical signs of blast trauma had more definite abnormality in the electroencephalograms than did those without physical signs. By way of comparison, Stephenson noted that the patients in

[65] Heppenstall, M. E., and Hill, D.: Electroencephalography in Chronic Post-Traumatic Syndromes. Lancet 1: 261-263, 27 Feb. 1943.

both series who had been injured by a solid blow to the head had more electroencephalographic abnormality than those injured by blast concussion. This was true both in the study by Aita and Kerman and in that by Stephenson. Both studies were comparable in respect to the time intervals between the acute concussion and the electroencephalogram under consideration, as well as in the proportions of cases which were normal and not normal.

The types of electroencephalographic abnormalities described by Stephenson included the following:

1. Increase in the percentage incidence of intermediate activity [66] of 13 to 18 cycles appears as follows:

Condition:	*Percentage*
Blast concussion	27. 7
Closed head injury	26. 6
Open head injury	23. 1
Normals	20. 0
All others	23. 3

2. Moderate increase in the incidence of disruption of the alpha activity into double its frequency, giving rise to the occurrence of double alpha.[67]

3. Suppression of alpha activity (table 10).

Although these figures do not appear statistically significant, they approach very closely to the borderline and appear to indicate a trend. A larger group of control cases with the same relative incidence would render the findings definitely significant. The close agreement between the incidence of intermediate activity in blast-concussion and closed head injuries confirms the impression gained in the laboratory that the blast-concussion group is, at least

TABLE 10.—*Comparative frequency of double alpha and suppression of double alpha in blast concussion and other conditions* [1]

Group classification	Number	Frequency	
		Double alpha	Suppression of alpha
Bomb blast	441	9. 1	6. 6
Controls	140	6. 4	2. 1
Epilepsy	768	4. 5	3. 9
Focal lesions	761	6. 7	10. 7
Migraine	51	5. 9	2. 0
Headache, nonspecific	201	4. 5	2. 0
Skull defects	442	5. 7	24. 7
Psychotic states	57	5. 3	5. 3
Miscellaneous	339	4. 7	2. 6

[1] Cramer, F., Paster, S., and Stephenson, C.: Cerebral Injuries Due to Explosion Waves—"Cerebral Blast Concussion." A Pathologic, Clinical and Electroencephalographic Study. Arch. Neurol. & Psychiat. 61: 1-20, January 1949.

[66] Brazier, M. A. B., and Finesinger, J. E.: Characteristics of the Normal Electroencephalogram. J. Clin. Investigation 23: 303-311, May 1944.

[67] Williams, D., and Reynell, J.: Abnormal Suppression of Cortical Frequencies. Brain 68: 123-161. June 1945.

electroencephalographically, only a subdivision of the larger group of closed head injuries, including concussion from any source.

There also occurred a moderate increase in the incidence of disruption of the alpha activity into double its frequency, giving rise to the double alpha. The increase in this characteristic is not sufficient to warrant the assumption of any definite conclusions, but, again, it represents a trend.

Suppression of alpha activity occurred significantly more often in the electroencephalograms of patients with blast concussion than in normal controls. It was found more often in cases of closed head injury and most often in cases with the very severe skull defects. Williams and Reynell considered this suppression of alpha activity to be "always abnormal * * * [and occurring] around penetrating gunshot wounds of the brain, and also in the cortex underlying nonpenetrating gunshot wounds, and depressed fractures, and also in other circumstances in which brain is violently percussed."

When one analyzes the incidence of double alpha and of suppression of alpha activity, as shown in table 10, the evidence in Stephenson's series again points toward definite brain damage.

Von Storch also observed that the electroencephalograph revealed evidence of cortical dysrhythmias which were more severe and persisted longer in the patients with concussion caused by a solid object. Blast concussion produced changes which were less severe, less prolonged, and more variable in degree. Although electroencephalographic changes often persisted longer than neurologic or psychometric changes, all three tended to run a parallel course with respect to the duration of the changes. Since Von Storch considered unconsciousness to be essential to the diagnosis of concussion, those cases in which it had not been definite were not diagnosed as instances of concussion. Cortical dysrhythmias, however, appeared in a large number of the patients who were exposed to blast but in whom there was no definite evidence of unconsciousness. There were 17 patients in this group, 10 of whom manifested neurotic or psychopathic tendencies, while 7 were otherwise normal individuals. About a third of these patients manifested no abnormalities detectable by craniofacial, neurologic, electroencephalographic, or psychologic examination, but over two-thirds (70 percent) had positive evidence of injury as judged by these four types of examination. Of 16 patients who had been rendered unconscious by blast injury, only 9 percent showed no evidence of abnormality in the examination, while 91 percent had positive findings.

In Garai's case (p. 243), a man who sustained a blast injury to the lungs and other organs had clinical evidence of a cerebral lesion affecting the pupillary innervation of the right eye and the pyramidal tract to the left side. Electroencephalography 48 hours after trauma showed a 9-cycle dominant rhythm, 40 microvolts, and equal inhibition in right and left hemispheres on opening the eyes. The parietal areas had a 4- to 6-cycle frequency of 40 microvolts, greater on the right side. This finding remained about the same 30 days after trauma. At this time, "* * * 6-cycle waves in small bursts of significant voltage [were] seen occasionally from all areas." Five days after trauma the

same had been true for all areas, with less evidence of a significant focus. One year later, the general character of the dominant rhythm was unchanged. There was generalized irregularity, with tendencies to slow frequencies still obvious. There was a 6-cycle rhythm of significant voltage from all areas, greatest in the right parietal area.

Garai, bearing in mind that 10 percent of the normal population have abnormal rhythms, considered the following factors in favor of an acquired dysrhythmia in this case:

1. There was an asymmetry of the rhythm in the two hemispheres.
2. There was a change in the rhythms in the course of time.

Von Storch noted that the various groups of cases tended to show electroencephalographic changes which, if not characteristic for individual cases, were at least typical of the groups, as shown in table 11. In general, electroencephalographic, neurologic, and psychiatric abnormalities tended to follow a parallel course with respect to duration and degree of change. In some instances, cerebral dysrhythmias persisted longer than other evidence of encephalopathy.

The higher incidence of borderline and abnormal cases which Stephenson found in blast concussion was significantly greater than in his control group, as was the suppression of alpha activity. The additional increase in intermediate activity and the double alpha were not statistically meaningful, but the summation of these characteristics indicated a reasonable probability that there was residual cerebral damage in these cases. The persistence of findings for as long as 2 years after injury, the mean being 3 months, also lent emphasis

TABLE 11.—*Comparative electroencephalographic trends in cases of concussion due to solid blows and blast, and cases with significant psychogenic complications* [1]

Observations	Solid blows	Blast	Significant psychogenic complications
Voltage	Normal or low [2]	Normal	Low.
Frequency	Normal [3] or mild to moderate random frequency.	Normal with slight random activity.	Mild random activity.
Bursts of abnormal activity.	Mild to marked high voltage, slow activity.	Few if any	Occasional, slow.
Response to hyperventilation.	Little or none	None	Considerable. Rhythms suggested psychomotor epilepsy and probably represented pretraumatic abnormalities.

[1] Von Storch, T.: A Study of Cerebral Concussion. *In* Annual Report, 33d General Hospital, 1945.
[2] 25 to 50 microvolts.
[3] 10 seconds.

to the probability of relatively severe damage in those patients whose condition became chronic and who were evacuated.

CEREBRAL CONCUSSION AND BLAST CONCUSSION

Theories of the Causes of Concussion

The elucidation of two principal mechanisms by which cerebral concussion is currently believed to be produced may be simplified as follows:

1. In the acceleration-deceleration theory, the phenomena are the result of a differential of momentum between the brain and skull, when the latter has been caused to move at the rate of about 28 feet per second. Denny-Brown [68] attempted to relate all concussion to this mechanism.

2. The compression theory, for which Scott [69] and Webster and Gurdjian [70] found experimental proof, depends on the belief that increase in the intracranial pressure, whether by the transcranial or paracranial route, will produce typical cerebral concussion. It is this etiologic category that most of those who have been interested in explaining the phenomenon of blast concussion have found the most suitable, since it is by the paracranial route that the blast forces appear most likely to pass to the brain. It would appear that the experimental work of recent years contains the cogent facts on which definitive formulations regarding the basic cerebral alterations can be made.

Structural alterations in concussion.—Groat, Magoun, Dey, and Windle [71] proved that acceleration concussion and compression concussion were alike in their clinical manifestations. Windle, Groat, and Fox [72] also proved that these conditions are typified by the same histologic changes and, furthermore, that these histologic changes occur immediately after the blow is delivered.

In controlled experiments, these observers delivered concussive and subconcussive blows to guinea pigs, the animals being prepared in such a manner that it was certain that formalin reached the brain through its blood vessels within 8 to 30 seconds, or sooner, after wounding. Histologic studies showed that in the animals which were sacrificed immediately, there were significant alterations in arrangement, size, and shape of the Nissl bodies of many neurons of the basal ganglia, chiefly the small-celled ganglia of sensory function, such as Deiters' nucleus and the sensory nuclei of the trigeminus. These were the same nuclei in which Groat and his associates observed chromatolysis in other animals in their series which were sacrificed a day or more after concussion. Some of the Nissl bodies were fragmented and disarranged by the blow; in other cells, their particles gave the appearance of agglutination. Similar changes in the Nissl bodies were observed in two animals which were killed by strong blows.

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[68] See footnote 12, p. 218.
[69] See footnote 3 (1), p. 215.
[70] See footnote 3 (3), p. 215.
[71] See footnote 4 (1), p. 216.
[72] See footnote 4 (3), p. 216.

Many of the changes observed in animals sacrificed 14 to 24 hours after concussion resembled those seen when death occurred immediately after the blows.

Of particular note was the observation that the intracellular changes found in animals which had received two subconcussive blows were equivalent to those in animals in which one single, stronger blow had been sufficient to produce the concussion. Windle and Groat [73] stated that from the cytological point of view it is inconceivable that extreme degenerative changes involving ballooning of the cell and complete loss of the Nissl substance can be reversible.

They further observed that the chromatolysis, as studied in successive stages of its progression in a series of animals in the postconcussional period, led to the eventual disappearance of as many as half the cells in certain nuclear masses in the brain stem. This change occurred in relative proportion to the severity of the concussion.

Experimental Blast Concussion

Studies comparable to those just cited have not been carried out by observers who have performed the experimental work in blast concussion, and the number and scope of investigations have been very limited. Krohn, Whitteridge, and Zuckerman [74] proved that (atmospheric) blast produces its effects through the body surfaces and not by way of the trachea. Their experiments bore out the general clinical observations that the greatest effect is upon the aircontaining viscera. They did not attempt an analysis of the finer structural changes in the brain-stem nuclei, by which the effects of blast concussion upon the brain could be compared with those of direct cerebral concussion. In rabbits whose bodies were protected while their heads were exposed to blast pressures of 200 pounds per square inch, they found no thoracic or abdominal lesions, but there were subpial hemorrhages over the olfactory bulbs and bruising of the occipital lobes.

Corey [75] also observed that epidural and subdural hemorrhage occurs when the head of an animal is immersed under water coincidentally with a detonation. This phase of the experiments cannot be compared to any clinical counterpart but suggests that if the body be amply protected blast concussion is less apt to occur.

When the animals in the experiments by Krohn and his associates had the heads protected and the blast was transmitted through the body walls, typical damage to the lungs and other viscera was found. Some hemorrhage into the ventricles of the brain was also noted in rabbits but not in other species. The cause of death in those animals which succumbed to the blast was either an undefinable, so-called primary shock when death was immediate or complications of pulmonary damage when it was delayed.

Electrocorticograms were made in 2 cats and 2 monkeys. In the first cat, there was a small depression of cortical activity from 14 to 17 seconds

[73] See footnote 4 (2), p. 216.

[74] Krohn, P. L., Whitteridge, D., and Zuckerman, S.: Physiological Effects of Blast. Lancet 1:252-258, 28 Feb. 1942.

[75] See footnote 17, p. 219.

after the explosion, followed by a temporary restoration of the previous activity. The animal, however, died in 14 minutes. In the second cat, the record was not readable for 5 seconds; 5 seconds later, electrical activity almost disappeared; after a further 12 seconds, it returned; and it became apparently normal after another 11 seconds.

In one monkey, the record was not readable for 20 seconds, and no abnormality was observed when it again became readable. In the other monkey, before the explosion, there was a well-developed discharge of alpha waves of 9 to 11 cycles per second, as well as occasional large waves of 2 per second, attributed to Nembutal (pentobarbital sodium). Twenty-three seconds after the explosion, there was a small burst of alpha waves of 9 per second, then a period of slow waves, larger and more frequent, which disappeared, the record becoming normal again in 30 minutes. The animal died of asphyxial convulsions in 1 hour and 40 minutes. The authors compared these results with those of Denny-Brown,[76] in which, after direct blows to the head in experimental animals, all cortical activity was abolished immediately, returning after 3 minutes with slow regular waves reminiscent of concussion in the human.

The data from these two groups of experiments are not satisfactory for comparison, however, particularly since two of the records in the cases of Krohn and his group were simply reported as not readable during the first several seconds and there is no knowledge of what they may have shown during that period.

Corey reported experiments to test the gross effects of blast. He observed that guinea pigs and rabbits outside a radius of 80 feet from exploding charges of 1,750 pounds of TNT survived the explosion, but he gave no details of later examination. Guinea pigs and rabbits placed behind a tree 32 feet from an explosion and behind a parapet 50 feet away were killed. Likewise, goats 32 and 50 feet away, in trenches, whether protected by kapok or unprotected, were killed. A guinea pig 50 feet away, placed in the long axis of the charge, and a rabbit 75 feet away, behind a tree, survived. Others 50 to 75 feet away died in 24 hours. Corey stated that injuries did not occur when the animals were outside the flash area but were constant when they were within it. Microscopic changes, the nature of which he did not specify, were found in the central nervous system in 2 out of 7 animals studied, and transient depression of the central nervous system, which was not qualified further, also occurred.

Mechanism of Blast Concussion

The acceleration theory.—The concept that cerebral concussion may occur without direct action of the traumatic force upon the head is difficult for many to accept.

Denny-Brown,[77] who has written authoritatively on some phases of cerebral concussion, asserted: "Concussion, or indeed any similar physical damage

[76] Denny-Brown, D., and Russell, W. R.: Experimental Cerebral Concussion. Brain 64: 93–164, September 1941.
[77] See footnote 2, p. 215.

to nervous tissue resulting from the force of an explosion transmitted by air or by water * * * remains an unproven condition."

There are, no doubt, many who, for various reasons, share this opinion.

On practical grounds, however, this statement must be considered ready for revision, in the light of the numerous published facts which have appeared since Denny-Brown's observations (1945) and many of which are reported in this chapter. Denny-Brown himself has not attempted to explain the phenomenon in a positive way but implies that secondary injuries have occurred to the skull or that acceleration-deceleration has always occurred in some manner.

Fulton [78] has suggested that Denny-Brown's acceleration-deceleration theory may be applied to this problem as follows: There are two components to be considered in blast. The first is a primary increase in pressure to 100 pounds or more per square inch, and the second is the rate of movement. He continues as follows: "A wave of moving pressure that gives acceleration of the body by virtue of its impact on the body wall—and hence imparts an acceleration to the body as a whole, including the head—reproduces in a very precise manner the type of acceleration or deacceleration known to be essential for the traumatic-concussion syndrome."

The compression theory.—Others [79] have applied to the problem the concept that the compression wave travels to the cerebrum through the body mediums, chiefly by way of the blood stream. This general viewpoint was not new to World War II, but it would appear that it has been a more or less original conception of various observers.

The following statements furnish a synthesis of this concept:

1. It is characteristic of the shock wave that its rate of speed varies with the compressibility of the mediums through which it passes. Its speed depends upon many factors, including the distance from the explosion and the reflectance of the wave by solid objects and surfaces in its path.

2. In air, the speed of the shock wave corresponds to that of sound, which is about 1,100 feet (335 meters) per second. While the wave is traveling through water, the speed is about 4,800 feet (about 1,460 meters) per second. These relationships also hold while the shock wave is passing through a living body. Thus, when the rapidly moving compression wave strikes the body, it is transmitted through the various components of its anatomic structure at variable rates of speed and with variable results. Upon striking the body walls, it is transmitted faster through the blood stream than it is through compressible tissues and the gas-containing viscera. It is here, therefore, that the greatest damage occurs, also because of another characteristic of the shock wave; namely, that, at the junction or surface between fluid and gas, a shock wave moving through the fluid produces a disruption of the surface. This is exemplified, outside the body, in the case of an underwater explosion, by the so-called doming of the surface of the water which occurs as the shock wave reaches the

[78] See footnote 27, p. 225.

[79] See footnotes 26, p. 224; 39, p. 229; 40, p. 230; and 45, p. 233.

surface, as distinguished from the later pluming, or geyserlike eruption, which occurs when the gases from the explosion, following after the shock wave, have reached the surface. This same characteristic of the wave holds true for its effects within the body. Thus, the most typical injury due to blast is a rupture of the walls of the pulmonary alveoli and of the gut, with attendant hemorrhage.

3. The shock wave is also transmitted into the cranium and into the spinal canal by the venous system and probably also by the arterial system. Several possible factors are at once brought into play. There is a sudden, violent increase in the intravascular tension of the cerebrum, with immediate alteration of the physiology of the cerebral circulation and the possible rupture of vessels. That there will be instantaneous vascular reflex alterations with secondary stasis, edema, and diapedesis is inescapable. That there may be hemorrhages of various sizes and degrees and locations, as there are in closed head injuries in general, is also a foregone conclusion. At the same time, however, there will be a concussion of the brain (and at times of the spinal cord) independently of the vascular alterations themselves. The concussive wave will be applied directly to the brain tissue as a shock wave not only by way of the vascular system but also by way of the spinal fluid.

At this point, it is highly important to differentiate clearly in one's mind between two qualities of the blood stream. First, as a circulating body of fluid, the blood stream has the faculty, on the venous side of its circuit, of reversing the direction of flow during phases of negative pressure. Second, irrespective of its circulatory function, the blood stream represents merely a large body of fluid, and in this respect, it is capable of transmitting a shock wave in essentially the same manner as any body of fluid. It is this aspect which is important in the explanation of blast concussion as a phenomenon of cerebral compression.

A discussion of the physics of the blast wave by Sutherland [80] clarifies this problem. The point must be emphasized that, as the shock wave itself moves onward through any substance, there is not a movement or displacement of the matter itself with the exception of the displacement at surfaces of unequal compressibility. There is merely an oscillation back and forth about equilibrium positions of the particles of the matter through which the wave is passing.

An understanding of the facts just stated throws light upon the problem and should correct certain misconceptions. For example, in the context of Denny-Brown's [81] objections to the idea of blast concussion, it is presumed that a violent retrograde flow of blood into the cranium would have to occur in order to produce cerebral trauma. On the contrary, according to the physical principles of blast, the shock wave alone would traverse the blood stream. It would pass at a rate enormously faster than the blood could possibly flow. Its effects in the brain and various other parts of the body must be considered

[80] See footnote 18, p. 219.

[81] See footnotes 2, p. 215, and 12, p. 218.

to occur within about a ten-thousandth of a second after the blast wave has struck the body wall. This view of the mechanism of transmission of the shock wave to the cerebrum corresponds to that brought out in the experiments by Scott and by Webster and Gurdjian in their work with compression concussion, and in the dynamics proposed by Young.[83]

The Origin of Symptoms

There have been three principal approaches to the explanation of the blast concussion syndrome, as follows:

The psychiatric approach.—The most manifest aspect of the problem in its acute phase, after recovery from the initial unconsciousness, is the psychic disturbance. Thus, a psychiatric approach has been the most frequent one, except when there were frank complications of a surgical nature. In their preliminary report on the war neuroses of North Africa, Grinker and Spiegel laid stress on the admixture of true postconcussional symptoms with those which were considered to be psychogenic. Many of their reports of cases in which an explosion (frequently close enough to kill others in the same immediate site) precipitated a psychiatric disorder were so typical of blast concussion that they should normally fall into that category. The need for differentiating the cases of psychogenic origin from those of concussional origin was recognized, so that patients with concussions could be treated in one way and those with psychiatric conditions in another.

The difficulties encountered in making these differentiations on the basis of the symptomatology alone is patent, for essentially identical somatic and vegetative disturbances can be seen in individuals who have suffered organic injury to the brain and in those in whom intense psychic stimuli have liberated the symptoms. Therefore, it is obvious that anxiety states, regressions, and conversions do occur. This would be expected in the light of the preoccupation which many soldiers in combat have with thoughts of their loved ones at home; the safety of their own persons and those of their buddies and fellow soldiers, for whose welfare they have a highly developed sense of responsibility; and the regard for admitted fear which everyone has who is in or about to engage in combat. On the other hand, it is equally obvious that a purely interpretative approach, as contrasted with a systematic study employing all the modalities of neurologic analysis, will, in general, lay undue emphasis on the psychogenicity of the symptoms. It largely depends upon the clinical background and personal point of view of the examiner whether he will describe and treat the presenting symptoms as psychogenic, or as neurogenic (in the sense of a physiologic functional disturbance), or as manifestations of a true organic disturbance due to injuries of the central nervous system.

Functional neurogenic interpretation.—The point of view maintained by Fabing is that the syndrome is of functional neurologic origin; that is, a true neurosis. The symptoms are considered to arise from alterations in the physi-

[83] Young, M. W.: Mechanics of Blast Injuries. War Med. 8: 73-81, August 1945.

ology of the brain in its various levels of functioning, as a result of the intense stimulus of the blast force, but irrespective of any organic injuries.

In this point of view, it is proposed that the symptoms arise from cerebral mechanisms as conceived by Pavlov and explained in his terms. For example, all of the patients in Fabing's series had amnesia for the sound of the explosion, although some may have remembered seeing its flash. Even under narcosynthesis, in which most other experiences during the postconcussional amnesia were recalled, the actual sound of the explosion remained a hiatus in the unconscious memory. According to the Pavlovian theory employed, this would be due to the fact that the intense sound wave, to which the blast wave is equivalent, acts as an ultramaximal stimulus. In passing through the auditory nerve, the wave does not produce normal excitation, in the sense of hearing. Instead, it produces in the temporal lobe an ultraparadoxical state of activity, which inhibits the realization of the hearing of the sound. Consequently, even under Pentothal Sodium (thiopental sodium) narcosynthesis, the sound of the explosion is never recalled. According to this theory, it is entirely outside the memory because it has never been registered as an experience. Fabing has also explained the other symptoms of the syndrome by the use of these same conceptions and Pavlovian terminology. Unfortunately, this method of interpretation is somewhat too verbose and weighty to allow further epitomization here.

Fabing's further premise is that organic injuries, including hemorrhages, do not produce the symptoms of this syndrome, but are merely epiphenomena to the physiologic disturbance.

Organic neurologic explanation.—The consideration of symptoms as specific evidence of structural disorganization of various specialized areas of the brain has been proposed by Cramer, Paster, and Stephenson. Their interest in this phase was stimulated by experiences with cases which were typical of the cerebral blast syndrome and in which evidence of encephalopathy was displayed. These patients had persistent organic neurologic findings, incontrovertible trends from the normal pattern in the electroencephalograms, and, in one case, a delayed posttraumatic cerebral hemorrhage in the neighborhood of the thalamus from areas of softening produced by a previous blast concussion and contusion.

Opposed to the attitude that either a purely physiopathologic or psychopathologic disturbance produces this syndrome is the estimate of Aita and Kerman and of Cramer, Paster, and Stephenson, that there is truly organic substratum in a substantial number of selected cases. This estimate was based on the persistence of symptoms and physical signs in their cases, and the trends away from the normal encephalograms, in contrast with the relatively normal psychometric and psychiatric studies. It appeared to Cramer and his coworkers that evidence of disturbance of the brain-stem centers was found in the unconsciousness, the tremors, the vegetative disturbances, and the hyperacusis which accompanied the startle reaction; the confusion and the emotional lability; and the physical signs, particularly the inequality of the pupils.

The persistent dysrhythmias in the electroencephalogram may well be of the same origin, in the light of the experiments of Kennard and Nims.[83] They found, in *Macaca mulatta*, that damaging the cortex alone produced little or no change in the pattern, whereas added damage to the basal ganglia produced profound and permanent rearrangements. The electroencephalogram was proved to be intimately related to corticogangliar function and not a summation of cortical potentials alone. The most pronounced changes in their experiment was "hypersynchrony" ("cusping" or "saw-toothing"). Stephenson found 9.1 percent of blast concussion cases had double alpha.

Unconsciousness, from this point of view, is attributed to disturbance in the centers controlling sleeping and waking in the hypothalamus, midbrain, and hindbrain. Davison and Demuth [84] have reported their findings in various conditions in which lesions at the diencephalic and mesencephalic-metencephalic levels were associated with disturbances of sleeping and consciousness. Jefferson [85] discussed this theory in relation to cerebral concussion. From this point of view, the unconsciousness is seen as being proportional only to the type and degree of involvement of these centers. Unconsciousness is not an indication of the severity of the injury to the brain at large; it is the focal symptom of altered function of specific areas in the brain, just as aphasia, apraxia, and paralysis are similar symptoms.

That this alteration in function may also be produced by psychogenic stimuli, just as many other conditions, including peptic ulcer, may be produced, is recognized. Unconsciousness, or any other single symptom or sign, cannot be taken alone as a substantiation of concussion, but each must be evaluated with the whole. Unconsciousness was a first symptom in many soldiers suffering psychogenic ailments in battle. As reported by Von Storch, however, it was the general opinion of all concerned in his study of concussion that the soldiers examined in forward echelons tended to underestimate, rather than emphasize, the aspect of unconsciousness. Direct observation was possible in many patients admitted to the hospital unconscious after blast injuries, and in others, eyewitnesses were available to prove the unconsciousness which was used as a criterion of concussion. Yet concussion with unconsciousness due to blast could not be differentiated by any single method of study from the manifestations of injury due to blast without loss of consciousness. In electroencephalograms in the latter cases, there was more tendency toward abnormality than in those of patients who had been rendered unconscious. This tendency occurred to a lesser extent in patients hit by solid objects who did not become unconscious. From these observations, therefore, one could infer either that

[83] Kennard, M. A., and Nims, L. F.: Effect on Electroencephalogram of Lesions of Cerebral Cortex and Basal Ganglia in Macaca Mulatta. J. Neurophysiol. 5:335-348, September 1942.

[84] (1) Davison, C., and Demuth, E. L.: Disturbances in Sleep Mechanism: A Clinicopathologic Study: III. Lesions at the Diencephalic Level (Hypothalamus). Arch. Neurol. & Psychiat. 55:111-125, February 1946. (2) Davison, C., and Demuth, E. L.: Disturbances in Sleep Mechanism: A Clinicopathologic Study: IV. Lesions at the Mesencephalometencephalic Level. Arch. Neurol. & Psychiat. 55:126-138, February 1946. (3) Davison, C., and Demuth, E. L.: Disturbances in Sleep Mechanism: A Clinicopathologic Study: V. Anatomic and Neurophysiologic Considerations. Arch. Neurol. & Psychiat. 55:364-381, April 1946.

[85] (1) Jefferson, G.: Neurological Interpretation of Acute Head Injury. Tr. Am. Neurol. A. 69:13-16, 1943. (2) Jefferson, G.: Nature of Concussion. Brit. M. J. 1:1-5, January 1944.

the changes indicated actual cerebral concussion on a subclinical basis (or, rather, concussion without unconsciousness) or that the changes in electrical activity of the cerebrum were due to preexisting abnormalities.

Against the latter interpretation are Stephenson's observations, which were made in the same type of patient but at a much later period. He also found that the group subjected to blast who were not rendered unconscious but who developed the postconcussional syndrome and in whom psychometric and psychiatric studies favored an organic type of disturbance had more electroencephalographic disturbances than those who had been unconscious. Contrasting these findings, however, with his figures for normal controls and for other types of disturbance, he considered them as definitely following the trend for blast concussion.

Tinnitus and true vertigo, two relatively short-lived symptoms, are most likely due to direct concussion of the auditory and vestibular mechanisms in the inner ear. On the other hand, the hyperirritability to noises and sounds and the sensations of floating, as well as a part of the persistent nonvertiginous dizziness, are considered by Cramer and his associates to be due to basic lesions in the auditory and vestibular nuclei and their representations in the thalamus. This is the point of view which writers in the German language, as exemplified by Stier,[86] have for many years been elucidating in respect to the postconcussional syndrome in general. It is also in conformity with the knowledge regarding the lesions which occur in concussion, as cited earlier in this chapter (p. 228).

Headache, which is the chief symptom immediately upon regaining consciousness, is as difficult to explain on any single factor in this syndrome as it is in concussion due to solid blows and in other conditions in which it occurs. It may be as generic in origin as are the effects of the all-pervading shock wave, which may be conceived of as affecting not only the brain and its vessels but all parts of the integument of the brain as well. As is evident in the reports of autopsies, all of the meninges may be involved by hemorrhage, which also may be subpial and intracerebral. Cerebral edema has been reported. The trigeminal pathways, which mediate most of the headaches of dural origin, are involved at times. The thalamus, which is the accepted penultimate center for the elaboration of the sense of pain, may also be the site of lesions. Posttraumatic headache, as emphasized by Brenner, Friedman, Merritt, and Denny-Brown [87] is of organic origin, irrespective of its greater incidence in cases in which there are bad psychic, social, and economic states.

It is proposed, therefore, by those whose occupation and preoccupation is in the field of neurologic surgery and organic neurology, that there is sufficient evidence for the somatic origin of this typically recurring train of symptoms to permit explaining them on this basis without invoking purely psychic mechanisms for their production. It is true that no single fact or

[86] Stier, E.: Uber Zwischenhirnschadigung durch Schädeltrauma. Arch. f. orthop. u. Unfall-Chir. 37: 446–448, 1937.

[87] (1) Brenner, C., Friedman, A. P., Merritt, H. H., and Denny-Brown, D. E.: Post Traumatic Headache. J. Neurosurg. 1: 379–391, November 1944. (2) See footnote 2, p. 215.

mode of diagnosis suffices alone to establish the diagnosis. Yet each method of study reveals definite trends from the normal, and, when the results of systematic study are combined, there is little room for doubt that blast concussion not only is an entity but also results in an organic disturbance of the nervous system. It may well be, as Grünthal, Hiller, and Marburg [88] and those whom they quote have suggested, that there are not only somatic predetermining factors in the causation of posttraumatic symptoms but that there many also be factors in the personality which actually predispose to the incurrence of head injuries. Certainly, it is common experience that the individual who has already suffered one concussion is greatly predisposed to develop inordinate symptoms after successive or subsequent head trauma. This clinical fact has an organic basis, as pointed out in the experimental work on concussion quoted earlier (p. 251).

Mental symptoms are not necessarily influenced by the pretraumatic personality, as Adler [89] and Kozol [90] have observed, each in a study of 200 cases. Pretraumatic psychoneurosis does not necessarily determine the presence or absence of posttraumatic neurosis, and, conversely, a normal pretraumatic personality is no guarantee of the absence of psychoneurosis after head trauma. Various nontraumatic factors, such as physical disability, the family status, and economic factors, may influence the posttraumatic sequelae; yet it is the injury itself which primarily determines the nature of the symptoms, be they somatic or psychic.

This point of view militates against any formulation which endeavors to explain the phenomena of cerebral blast injury or concussion upon the basis of a purely psychogenic or functional "higher" physiologic basis. In order to apply that formulation, one would perforce also have to explain the many other phenomena, including instantaneous death, on the same basis. Two or more men in combat, let us say, are very close together, in one trench or emplacement or vehicle. They are exposed to the blast of a close-by exploding shell, whose fragments do not strike them. They do not move from their primary positions. When some of them die outright and others become unconscious, it is indeed difficult to concede that the one who lives to manifest the symptoms of a postconcussional neurosis does so because of a predisposition to neurosis or a subconscious desire for gain, any more than one can concede that the others had a predisposition toward dying.

THE TREATMENT OF BLAST CONCUSSION

The end in view in differentiating the true concussions from the anxiety states is that eventual treatment should be directed along appropriate lines.

[88] Grünthal, E., Hiller, Fr., and Marburg, O.: Traumatische, Präsenile und Senile Erkrankungen Zirkulationsstörungen. *In* Handbuch der Neurologie, edited by O. Bumke and O. Foerster. Berlin: Julius Springer, 1936, vol. XI, pp. 1–100.

[89] Adler, A.: Mental Symptoms Following Head Injury: A Statistical Analysis of 200 Cases. Arch. Neurol. & Psychiat. 53: 34–43, January 1945.

[90] Kozol, H. L.: Pretraumatic Personality and Psychiatric Sequelae of Head Injury. I. Categorical Pretraumatic Personality Status Correlated With General Psychiatric Reaction to Head Injury Based on Analysis of 200 Cases. Arch. Neurol. & Psychiat. 53: 358–364, May 1945.

It has been recognized that the patients whose symptoms are chiefly of organic or postconcussional origin may require relatively long-term treatment, including evacuation from the battle zones, when the symptoms are persistent. Rest and broad psychotherapeutic treatment may lead to early rehabilitation of the lesser concussions, but only time will heal the more severely injured brain. On the other hand, the neurotic states require concerted psychotherapeutic measures, and, when combat exhaustion is a large contributing factor, these casualties may be rehabilitated within a relatively short time by appropriate physical and mental relaxation and diversion.

It is a fortunate circumstance that the use of Pentothal Sodium narcosis and narcosynthesis not only produced marked therapeutic results in both the neuroses of battle and the cerebral injuries but also served as a means of differential diagnosis to some degree. When there were frankly psychogenic moieties in the blast concussion syndrome, patients were very greatly benefited by this measure. The symptoms of frankly organic type, that is, headache, dizziness, and hyperacusis, were often greatly modified by this treatment, but, in many instances, they persisted, irrespective of all forms of therapy, and disappeared only very gradually, with the passage of time. Evaluation of the neurologic, psychometric, psychiatric, and electroencephalographic and pneumoencephalographic findings during this period indicated that there were continuing subacute and chronic intranuclear degeneration and cerebral changes, probably in the nature of glial cicatrization in the areas of encephalopathy.

CHAPTER XII

Cranioplasty

David L. Reeves, M. D.

HISTORICAL NOTE

Except for the repair of occasional defects caused by accidents, cranioplasty in civilian neurosurgical practice before World War II had been chiefly directed to the correction of deformities of the skull resulting from osteomyelitis, from the removal of infected bone flaps, and from the excision of hyperostosing tumors. There was rather general agreement that, whenever possible, autogenous bone was the material of choice for the grafts necessary in the operation.

The literature in the field of cranioplasty was exhaustively reviewed by Grant and Norcross [1] in 1939 and again, from the standpoint of the use of metals, by Venable and Stuck [2] in 1943. Both reviews showed the good results that could be obtained by the use of other types of materials.

The cranial defects which followed battle injuries stimulated an interest in the subject in World War II, as in all previous wars. Although other materials were used, cranioplasty with tantalum or the acrylic resins best fulfilled the needs of the military neurosurgeons who were called upon to handle the large numbers of casualties with head injuries. The experimental work reported by Burke [3] in 1940, Carney [4] in 1942, and Pudenz [5] in 1942 and 1943 had shown that tantalum was a most satisfactory alloplastic material for the repair of cranial defects. Experiences with its use in the repair of defects caused by battle-incurred wounds were reported by a number of military surgeons, including Fulcher [6] in 1943; Reeves [7] and Robertson [8] in 1944; Woodhall and

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[1] Grant, F. C., and Norcross, N. C.: Repair of Cranial Defects by Cranioplasty. Ann. Surg. 110: 488–512, October 1939.

[2] Venable, C. S., and Stuck, W. G.: A General Consideration of Metals for Buried Appliances in Surgery. Surg., Gynec. & Obst. 76: 297–304, April 1943.

[3] Burke, G. L.: The Corrosion of Metals in Tissues; and an Introduction to Tantalum. Canad. M. A. J. 43: 125–128, August 1940.

[4] Carney, H. M.: An Experimental Study With Tantalum. Proc. Soc. Exper. Biol. & Med. 51: 147–148, October 1942.

[5] (1) Pudenz, R. H.: The Use of Tantalum Clips for Hemostasis in Neurosurgery. Surgery 12: 791–797, November 1942. (2) Pudenz, R. H.: The Repair of Cranial Defects With Tantalum. J. A. M. A. 121: 478–482, 13 Feb. 1943.

[6] Fulcher, O. H.: Tantalum as a Metallic Implant To Repair Cranial Defects. J. A. M. A. 121: 931–933, 20 Mar. 1943.

[7] Reeves, D. L.: The Repair of Traumatic Cranial Defects. Bull. Los Angeles Neurol. Soc. 9: 112–119, March–June 1944.

[8] Robertson, R. C. L.: Repair of Cranial Defects With Tantalum. J. Neurosurg. 1: 227–236, July 1944.

Spurling,[9] Mayfield and Levich,[10] Schnitker and McCarthy,[11] and Woolf and Walker [12] in 1945; and Bradford and Livingston [13] and Turner [14] in 1946.

Whether the plate used for cranioplasty was formed by preoperative molding in a die and counterdie or by rough molding at the operating table with a hammer and rounded form, the inherent malleability of tantalum allowed adjustment by cutting or contour changing at the time of operation, and thus eliminated the need for multiple operative procedures. In addition to its malleability, tantalum has the further desirable qualities of noncorrosiveness, inertness in tissue, nonabsorbability, and absence of toxic ingredients.

As a result of the progress in the field of plastics, as well as the wartime stimulation to the study of the problem of cranioplasty, it was only natural that the acrylic resins should also be employed for the repair of skull defects. In 1943, Gurdjian, Webster, and Brown[15] described their method of repairing large skull defects with acrylic and reported that one patient was in excellent condition with no evidence of deleterious reactions, at the end of 15 months. In 1943, Kerr [16] reported seven such cases.

In 1946, Elkins and Cameron[17] reviewed their experiences in 70 cases of skull defect repaired with acrylic plates between June 1943 and January 1945. They concluded that methyl methacrylate was a satisfactory material for the repair of cranial defects, that it was sufficiently strong to assure permanency of repair, that it produced a minimum of foreign-body reaction in the tissues, and that it gave excellent cosmetic results. They perfected a technique by which the plate could be inserted in a one-stage operative procedure.

Acrylic plates possess certain advantages in that they are not subject to physical effects incident to changes in temperature or to the effects of electric phenomena, and that they do not interfere with the taking of pneumoencephalographic films. Their preparation, however, is more difficult than the preparation of tantalum plates, and it is questionable whether the acrylic resins will ever prove as satisfactory for the repair of large defects as tantalum.

PREPARATION OF TANTALUM PLATE

During World War II, methods used for forming tantalum plates were similar to those used for other alloplastic preformed materials. Adequate

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[9] Woodhall, B., and Spurling, R. G.: Tantalum Cranioplasty for War Wounds of the Skull. Ann. Surg. 121: 649–671, May 1945.

[10] Mayfield, F. H., and Levich, L. A.: Repair of Cranial Defects With Tantalum. Am. J. Surg. 67: 319–332, February 1945.

[11] Schnitker, M. T., and McCarthy, W. D.: Osteomyelitis of the Skull—Its Treatment With Penicillin and Repair of the Defect With Tantalum. Surgery 18: 94–115, July 1945.

[12] Woolf, J. I., and Walker, A. E.: Cranioplasty, Collective Review. Surg., Gynec. & Obst. 81: 1–23, July 1945.

[13] Bradford, F. K., and Livingston, K. E.: Failure in Early Secondary Repair of Skull Defects With Tantalum. J. Neurosurg. 3: 318–328, July 1946.

[14] Turner, O. A.: Repair of Defects of the Skull. Arch. Surg. 53: 312–326, September 1946.

[15] Gurdjian, E. S., Webster, J. E., and Brown, J. C.: Impression Technique for Reconstruction of Large Skull Defects. Surgery 14: 876–881, December 1943.

[16] Kerr, A. S.: The Use of Acrylic Resin Plates for Repair of Skull Defects. J. Neurol., Neurosurg. & Psychiat. 6 (N. S.): 158, July–October 1943.

[17] Elkins, C. W., and Cameron, J. E.: Cranioplasty With Acrylic Plates. J. Neurosurg. 3: 199–205, May 1946.

descriptions have been published by Schnitker and McCarthy; Mayfield and Levich; Woodhall and Spurling; Hemberger, Whitcomb, and Woodhall;[18] and Woodhall.[19] Impressions of the deformity were obtained either by the indirect (primary) method or by the direct (secondary) procedure. The one-stage method was more commonly used than the two-stage procedure. In either case, the technique for swaging the plate was the same.

Preparation of Mold

After the patient's scalp had been shaved, the defect was outlined, with an allowance of an overlap of a centimeter, by means of an indelible pencil, markings by which persisted on the cast. The material used for the impression was a dental impression compound, such as Kerr's red compound for the indirect method and the green compound for the direct method, modeling clay, or a hydrocolloid, such as Truloid.

After an application of mineral oil, the defect was filled in with the dental impression compound, which had been softened in warm water and worked manually until pliable. For the purpose of retaining the dental stone or plaster of paris, the compound was applied considerably beyond the area of the defect. It was hardened by the application of cold wet towels. When the hardened compound was removed, the outline and depth of the skull defect were revealed as an elevated mass in this primary impression. The sides of the dental compound were built up with wax strips or boxing wax to contain the dental stone or plaster, which was poured in to form a model. A crossmark was scraped into the depressed area of the dental stone or plaster of paris, to represent the depth and extent of the bony defect. This area was filled in with tinfoil and then with dental wax, which was contoured with a heated spatula to correspond to the surrounding scalp outline. The crossmark scraped in the dental stone kept the wax restoration from moving. The convexity of the wax fill was overemphasized, to compensate for the subsequent inlay of the plate.

A mold, including the wax restoration and an ample mass of the adjacent skull contour, was made with some one of the molding sands and a die was poured with zinc, or, in the case of larger defects, with Hydromite.[20] After the die had cooled, its surface was painted with a solution of alcohol and talc. A soft clay, such as Moldine, was formed about the circumference of the zinc die to act as a mold for the counterdie of poured lead. A similar procedure was used when Hydromite was employed.

Because the hydrocolloid impression material gave a more accurate reproduction of the detail of the surrounding skull or scalp contour, it was preferred by most neurosurgeons for multicontoured and large frontal defects. The material was heated in a double boiler until it was of a smooth consistency and

[18] Hemberger, A. J., Whitcomb, B. B., and Woodhall, B.: Technic of Tantalum Plating of Skull Defects. J. Neurosurg. 2: 21–25, January 1945.

[19] Woodhall, B.: Cranioplasty. *In* Surgical Treatment of the Nervous System, edited by Frederic W. Bancroft and Cobb Pilcher. Philadelphia: J. B. Lippincott Co., 1946, pp. 63–74.

[20] Hydromite (United States Gypsum Company) is a mixture of stone and plastic.

was then cooled to 120° F. for impression over the scalp. To limit the flow of the hydrocolloid and to facilitate removal of the impression, a cardboard base, fitting like the brim of a hat without a crown, was placed over the calvarium. A wire measurement of the circumference of the head was taken with heavy copper wire to obtain the proper hat-brim size. The impression material was painted over the defect and the surrounding contour with a soft brush to a thickness of about 1 inch. Wire mesh might be added to strengthen it, and, after it had been cooled with towels soaked in ice water, it could be reinforced further with a layer of plaster of paris. After the mold had set, it was removed with the cardboard, the outline of the defect was traced with a knife blade over the marked outline, and the impression was poured in dental stone. The remainder of the preparation did not differ from that already described for the dental compound.

Preparation of Plate

The 0.015-inch tantalum plate was then cut to approximate contour, with an increased diameter of approximately 1 cm. as an allowance for the varying convexity of the plate and for the possible enlargement of the bony defect at operation. The plate was swaged between the die and counterdie by means of a handpress or hydraulic press. Pressure had to be applied gradually and evenly, to allow stretching of the metal. After molding had begun, the plate was removed and inspected for marginal wrinkles in the metal. The edges of the excess metal could be trimmed off and any furrows or wrinkles obliterated with a ball-peen hammer. Kinking or furrowing was more apt to occur in the swaging of large and complicated plates. For purposes of drainage, a single hole was punched in the proposed dependent portion of the plate or in its central area, and the plate was reswaged. Some surgeons preferred plates preformed with numerous small perforations. Before the plate was used, it was washed in carbontetrachloride, rinsed in water, treated for 10 to 15 minutes in a bath of warm dilute (5 percent) nitric acid, rinsed thoroughly in water again, and finally sterilized in the autoclave.

A method of using the female die and a heavy rubber pad in the pressing cylinder gave excellent results and eliminated a time-consuming procedure. A modification of this method was described by Baker [21] (fig. 49). Using a skin-marking pencil, he outlined the shape of the plate which would cover the defect, allowing for an overlap of about 1 cm. He then filled in the skull defect directly with ordinary modeling clay until the clay encroached on the line marked on the skin and its surface was symmetrical with that of the opposite side of the skull. The surface of the clay dried within a few minutes. It was then covered with clear fingernail polish to prevent its being incorporated into the dental stone used to make the mold. The dental stone was placed on the calvarium to form an identical mold; when it had hardened, it could easily be removed. The impression made in the stone represented the shape and size of the tantalum plate

[21] Baker, G. S.: Cranioplasty With Tantalum Plate: A New Method of Forming the Plate Prior to Operation. Proc. Staff Meet., Mayo Clin. 21:177-182, 1 May 1946.

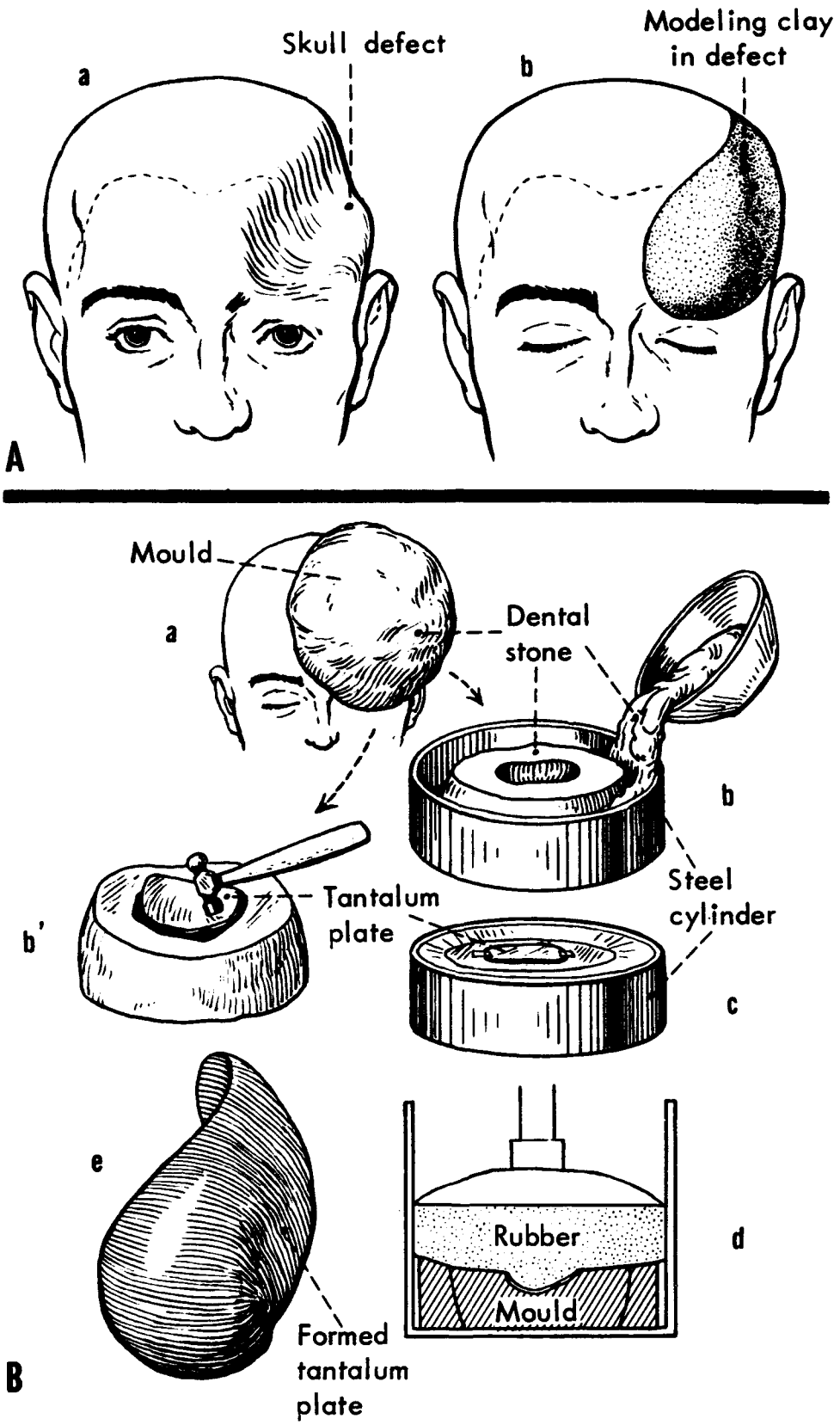


FIGURE 49.—Baker technique for construction of tantalum plate. A. Filling of skull defect with modeling clay. B. Methods of forming mold and plate.

required. The plate could then be hammered out on the mold until it had been shaped.

An alternative method used a power press. The mold was placed in a steel cylinder and surrounded by more dental stone until it was held firmly in position; the plate could be swaged into the desired contour by the pressing method, using a rubber pad. It was Baker's experience that smaller plates within the hairline could be hammered out satisfactorily on the mold but that larger plates were best swaged out in a power press.

If secondary repair of a skull defect was necessary after the debridement of bone in acute injuries of the skull, the two-stage procedure necessitated the preparation of a bone ledge around the defect and a direct impression of the mortise joint and surrounding bone, with subsequent restoration of the defect with a tantalum plate. A direct impression of the defect could be obtained at the time of operation by use of sterilized dental compound, usually Ker green compound, which becomes softened more readily and at a lower temperature than the red compound. The hydrocolloid impression material could be similarly used after it had been autoclaved and cooled to 98.6° F.

Simpler methods of forming the tantalum plates were also used with excellent results, particularly in defects of less complexity. These methods included the preforming of plates about a basic model of the skull, the hammering of plates with the use of preformed dental-cement molds or a basic metallic model, and the similar preparation of the plate at the operating table by hammering from a basic model. It was surprising what satisfactory results could be obtained even with seemingly crude methods. The surgeon could readily determine the result of his efforts by fitting the hammered plate against the defect until it appeared correct.

OPERATIVE FIXATION OF TANTALUM PLATE

Cranioplasties with tantalum could be performed under local anesthesia with the exception of those for frontal defects, which required general anesthesia. Scars on the scalp for the most part were of the linear variety, which allowed approach to the skull defect through the original scar. Coronal incisions were desirable in frontal defects. The usual craniotomy incision, making use of sagittally directed scars and circumscribed vertical scars, was desirable in defects of the temporal bone because the mass of muscle in this region and the increased vascularity after injury made direct exposure of the defect difficult. Cranioplastic incisions about a horizontal sc r might result in necrosis at the incision line. Plastic repair of a broad, thin scalp scar adherent to underlying tissue was done before cranioplasty and might even require grafting.

Two methods of applying the plate to the calvarium were used, the inlay method and the onlay method. Many neurosurgeons found the onlay method quite satisfactory for defects over the calvarium. The inlay method, in

Reeves' opinion, required unnecessary prolongation of the operative procedure without sufficient justification for its use.

Inlay Technique

After the skull defect was exposed, the pericranium was incised about 1 cm. about the border of the defect. This portion was resected, along with any excess connective tissue over the defect. Bone resection, removal of skull fragments, resection of cerebral scar, dural repair, and other measures were carried out as indicated. The border of the proposed ledge in the outer table was outlined on the bone with an instrument, such as the ordinary dental scaler, and the border was cut circumferentially with the lineator, an instrument designed for this procedure by Hemberger and his associates.

A bone ledge was cut out along this circumferential outline to a depth of approximately 2 mm. with a Stout No. 3 dental chisel. The preformed tantalum plate was adapted to fit this ledge, the necessary adjustments being made with heavy scissors or contour pliers, or by hammering on a basic model of metal. Fixation of the plate was secured by tantalum wedges, which were triangular trimmings of the tantalum sheet used to immobilize the plate in the manner in which glazier points are used to immobilize a glass windowpane. Seats for the tantalum points were made in the outer border of the bony ledge with a triangular pointed instrument (perforator). The wedges were tapped into position with a wedge director and were turned inward over the edge of the plate. Ordinarily, four to six wedges were sufficient for a plate. If tantalum screws were used, it was found best to have perforations made along the edge of the plate to accommodate them. Tantalum wires through the outer table of the calvarium and the edge of the plate proved satisfactory, but the tantalum wedges or screws were mechanically superior. Closure of the scalp was carried out in the usual manner, and a firm dressing, with pressure distributed evenly, was applied.

Onlay Technique

This method needs no additional description. Tantalum screws were very helpful in the fixation of the tantalum plate in such a procedure.

Often a combination of the inlay and onlay methods was helpful: the inlay method was carried out at a location at which an overlying plate might be noticeable, and the onlay method was carried out over the calvarium, where the plate would not be seen. In large defects over the calvarium, the onlay method with the use of tantalum screws allowed a very rapid and satisfactory fixation of the plate, while the inlay method might be very tedious and time consuming, without any particular benefit and with possibly a detrimental effect on the patient.

CRANIOPLASTY WITH ACRYLIC RESINS

The direct method was usually employed in the preparation of acrylic plates. Gurdjian, Webster, and Brown used the indirect or primary method, the preparation being carried out in much the same manner used for the tantalum plate. The desired amount of acrylic was cut with a bandsaw and then gently heated with a dry flame from a bunsen burner until it was soft and pliable. It was adapted to the outer surface of the mold, and, in the process, the edges of the glass were beveled to produce an accurate fitting. Holes were drilled into the edges for the purpose of fixing the graft into position by means of such suture material as tantalum. Only a small amount of readjusting was necessary at the time of the operative repair of the defect. The acrylic was exposed to dry heat on a bunsen burner outside the operating room or in the operating room if local anesthesia was being used for the procedure.

For the direct method of preparing acrylic plates, Elkins and Cameron placed in the wound a sterilized piece of dental base plate which had previously been heated in a pan of hot water until it was pliable. After it had been molded on the skull to the desired contour, it was removed from the wound and placed in cold sterile water for a few seconds. The base plate had to be larger than the defect, since it was used as an impression tray and since it determined the outer contour of the completed acrylic plate. Two retention holes were punched in the center of the tray by means of a heated blunt instrument. A layer of impression material was spread on the inner surface of the impression tray, of sufficient thickness to permit an accurate measurement of the depth of the bone edges. The material was pressed firmly into the inner surface of the tray, some being allowed to extrude through the retention holes. An impression of the bony defect was taken by reinserting the tray with its inner coating of wax into the wound and pressing it down firmly. The tray was removed and the impression was inspected for defects. If the operator was satisfied with the impression, it was placed in a pan of cold water for hardening and was then sent to the dental laboratory. The retractors were removed from the incision, and a sterile dressing was applied to the wound without disturbing the drapes.

The material Elkins and Cameron found most advantageous was a previously prepared mixture of two-thirds beeswax and one-third petrolatum product; this was either boiled or autoclaved to insure sterility. The impression material was firm when cool and had to be softened by molding in the hands.

In the dental laboratory, the tray and its impression were embedded in ordinary dental stone in the lower half of a Pryor injector flask. After it had hardened, the stone was coated with silex. The upper half of the flask was filled with dental stone, and the halves were placed together. The lid of the flask was placed in position, and the pressure plunger was adjusted to the estimated top of the tray. After 5 minutes, the pressure plunger was removed and the halves of the flask were separated. The wax impression material was boiled out and the tray was removed, after which the impression matrix was lined with light tinfoil and packed with acrylic resin in sufficient quantity for

packing pressure and for an allowance of about 20-percent shrinkage. The top of the injector flask and the pressure plunger were reinserted, and the plunger screw was adjusted until the indicator was one-fourth inch above the housing. The flask was placed in boiling water for about 35 minutes for curing; during this process, the plunger screw required several readjustments.

After the heating, the flask was allowed to cool for 10 minutes. Then the parts of the flask were separated, and the acrylic plate was removed for polishing. Several small drillholes were placed around the edges of the plate for wiring the plate into place in the defect, and two larger perforations were made in its center for drainage. Sterilization was accomplished by leaving the plate in Bard-Parker solution for 15 minutes. Elkins and Cameron estimated that the entire procedure for completion of the plate required on an average 1½ hours.

Small holes, corresponding as closely as possible to their counterparts in the acrylic plate, were drilled in the edge of the bone defect. The plate was fitted in position in the usual way and fastened in place by means of tantalum on chromium steel wire threaded through the holes in the bone and plate. Elkins and Cameron reported that the time for the entire procedure was between 2½ and 3 hours.

Either inlay plates the full thickness of the skull or onlay plates 2 to 3 mm. in thickness could be prepared by variations in the impression technique. For fixation of an onlay plate, metal screws were preferable.

INDICATIONS FOR CRANIOPLASTY

The indications for cranioplasty for war wounds of the skull were the same as for wounds of the skull seen in civilian life. As Grant and Norcross summarized them in their classical paper, they included the following: (1) Severe headache and other symptoms of the so-called syndrome of the trephined, such as dizziness, vague discomfort at the site of the defect, a feeling of apprehension and insecurity, mental depression, and intolerance to vibration; (2) the danger of trauma at the site of the defect; (3) pulsating or painful defects; (4) deforming and unsightly defects; and (5) the assumed relief of an associated convulsive state.

Woodhall and Spurling reported that the majority of their patients with battle-incurred wounds of the head noticed localized tenderness upon palpation in the area of the defect and complained of localized pain, which had a tendency toward spontaneous resolution. Cranioplasty benefited these subjective complaints. Vertigo and generalized headache, which were less common complaints, seemed unaffected by cranioplasty; in all probability, they represented sequelae of the cerebral injury. The revision of unsightly defects produced uniformly gratifying results.

Woodhall and Spurling, in an investigation of the effect of cranioplasty upon the electrical activity of the brain, studied 26 cases before and after cranioplasty. They found no postoperative change in the character of the

cortical electrical activity in 12. In 10, there was evidence of improvement, and in 4 the abnormal activity became even more manifest with the development of epileptogenic foci. Bradford and Livingston did not think that the cranial defect was the cause of convulsive seizures or that its repair would significantly alter the incidence of convulsions. Reeves' experience was in accord with theirs.

One of the complications influencing reparative surgery is the presence of infection. Bradford and Livingston pointed out that secondary intervention for tantalum plating in the presence of infection, or immediately following wound healing, could not be justified on the basis of scattered successes. It was their belief that secondary operation to cover a cranial defect should be delayed for a minimum period of 3 months after primary healing and for a period of 8 months after healing if frank infection had been present. They likewise concluded that, when a patient had a cerebral hernia, treatment should be directed toward the underlying pathologic processes and not toward mechanical block of the escaping cerebral mass. They also pointed out that complete coverage with good scalp is essential for successful cranioplasty with tantalum, and that, if the cranioplasty with tantalum is not successful, the plate must be removed promptly.

The Posttraumatic Convulsive State

Ascroft,[22] in studying the occurrence of epilepsy after gunshot wounds of the head, examined the records of 317 patients at the British Ministry of Pensions whose wounds had been incurred in World War I. Cases of blunt injuries and of wounds of the cerebellum were excluded. The followup period varied from 7 to 20 years. The series represented a fair sample of head injuries severe enough to warrant award of a pension but not to cause death within 4 years.

Epilepsy had developed in 34 percent of these 317 cases. The incidence of convulsions was twice as great in cases in which the dura had been penetrated (45 percent) as in cases of scalp and skull wounds in which the dura was intact (23 percent). Epilepsy naturally was found to be more common after wounds affecting the sensorimotor cortex than after those of the polar regions of the cerebral hemisphere.

In a series of 76 penetrating injuries of the brain observed over a period of 1 year or less, Woodhall and Spurling [23] found that convulsions had developed in 15 patients within 4 months of the injury, while, in another 15 (a total of 40 percent), there was electrical cortical activity highly suggestive of an epileptogenic focus. Later, Woodhall reported that in wounds of dural-cortical penetration, the statistics revealed an incidence of posttraumatic epilepsy of from 35 to 50 percent and that the principal time for its onset was within the first year, between the 4th and 12th months, after injury. In other cases, the onset might occur within the following 5 years, or even later. Wood-

[22] Ascroft, P. B.: Traumatic Epilepsy After Gunshot Wounds of the Head. Brit. M. J. 1:739-744, 17 May 1941.
[23] See footnote 9, p. 262.

hall also stated that in spite of adequate early debridement and more improved methods of combating infection, the incidence of posttraumatic epilepsy in a series of war wounds in World War II appeared quite similar to the incidence in previous reports and that, during the early period of observation, it had remained at a level of 40 percent.

STATISTICAL DATA

A personal study, in 1946, of admissions reported from 16 Army neurosurgical centers in the United States revealed 4,322 patients with skull defects from craniocerebral wounds. When one considers the cases operated on in naval centers and in overseas installations, it seems not unreasonable to assume that at least 5,500 cranioplasties were performed during World War II. Obviously, if this large series of cases were properly studied over a sufficient period of time, a valuable contribution to this subject could be made.

In 1945, Woodhall and Spurling [24] analyzed 79 cases of tantalum cranioplasty. The majority of the skull defects were caused by shell fragments, a fact which suggests failure to survive the impact of the higher velocity missiles of rifle and machinegun. Numerous other cases are on record in the literature.

The distribution of the 194 cranioplasties upon which the experience recorded in this chapter is based is significant. No operations were performed in 1941 or 1942, and only three were performed in 1943. In 1944, 36 skull defects were repaired, and in 1945, the number rose to 155, or 80 percent of the whole series.

Information is available concerning the cause of the defect in only 121 of the 194 cases; in 71 cases the defects were caused by shell fragments. There were no neurologic defects in 24 of the 125 cases in which sufficient data were available to determine this point. All of the 24 cases of convulsive disorders followed dural-cortical penetration, and most of them had become evident within the first year following the injury. Undoubtedly, many other cases of epilepsy occurred within this group but were not adequately recorded. If these patients could have been followed carefully for a period of 5 years, there seems little doubt that the incidence of convulsive disorders would have been found to be 35 to 50 percent. Unfortunately, adequate records of electroencephalographic findings are not available.

In 4 of the 194 cases, removal of the plate was necessary because of infection. In two of these, the cranioplasty had been completed in spite of the presence of a low-grade infection. In the early cases, it was not appreciated that pressure necrosis of atrophic scalp overlying the plate was likely to occur when plating was carried out under scalp wounds that had healed leaving thin wide scars; two plates were removed for this reason. Revision of the plate had to be performed in two orbital defects because the plate overlapped the orbital ridge and prevented adequate opening of the upper lid. When

[24] See footnote 9, p. 262.

the plates were inlaid only to the edge of the orbit, this difficulty was corrected. Revision was also necessary in a third case.

The preoperative and postoperative use of penicillin became fairly routine procedure in these cranioplasties. No operative or postoperative deaths occurred in this series, and the morbidity rate of all tantalum repairs was surprisingly low.

CASE REPORTS

The following case reports represent special experiences with cranioplasty at the neurosurgical centers at Hoff General Hospital, Santa Barbara, Calif., Birmingham General Hospital, Van Nuys, Calif., and Hammond General Hospital, Modesto, Calif.

Nontraumatic Skull Defects

Case 1.—A soldier with a history of intermittent attacks of upper respiratory infection and sinusitis since April 1944 was admitted to Letterman General Hospital, San Francisco, Calif. In New Caledonia, a diagnosis had been made of possible osteomyelitis of the left frontal bone secondary to sinusitis. A subperiosteal abscess in this region was incised on 23 September 1944, and a sequestrectomy of the left frontal bone was carried out on 7 October 1944.

The patient was subsequently evacuated to Hammond General Hospital, where, in January 1945, physical and roentgenologic examination revealed a large circular defect involving the major part of the left frontal bone in its lateral superior portions. The edges of the defect were irregular, but there was no evidence of a spreading bone infection.

A tantalum cranioplasty was performed on 6 April 1945. The inlay technique was used over the frontal area and the onlay technique elsewhere, with fixation by means of tantalum wires. The plate was hammered out on a basic dental cement model. The postoperative course was uneventful, and the cosmetic result was satisfactory. The patient was given a disability discharge in August 1945.

Traumatic Skull Defects

Case 2.—An officer in the Army Air Forces was admitted to Hoff General Hospital because of a large skull defect in the left frontotemporal region, resulting from the removal of fragments from a compound comminuted, depressed fracture and of foreign bodies in that area. He had been struck in the left naso-orbital region by a shell fragment from an antiaircraft battery during a bombing mission on 13 June 1943 and had become unconscious immediately. On his return to the base, depressed bone fragments, metallic fragments, and embedded pieces of his oxygen mask were removed from the wound. His condition was critical during the first 24 hours after operation. Thereafter, he recovered uneventfully and was later returned to the United States for further observation and treatment.

When he was admitted to the Hoff General Hospital on 21 September 1943, his overseas operative report described a large laceration of the dura in the left frontotemporal region with clots of blood and herniation of cerebral tissue. Beneath the skull fragments was a large, rectangular metallic foreign body, about 1.5 cm. by 5 cm., and there were several smaller foreign bodies, about 0.5 cm. by 2 cm., resembling pieces of rubber tubing. There were multiple lacerations of the nose on the left side, and a large laceration of the left nasofrontal region of the skull medial to the left eye was filled with clots of blood and cerebral tissue.

Examination at Hoff General Hospital revealed a large frontotemporal cranial defect (fig. 50) measuring 7.6 cm. by 12.7 cm., with destruction of 3.8 cm. of the left orbital bone.

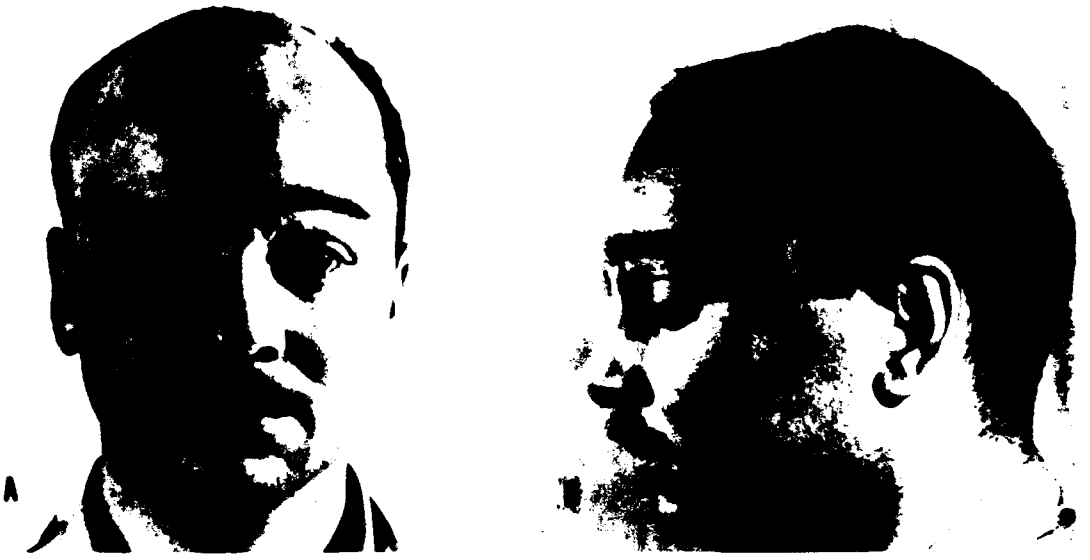


FIGURE 50. A. Frontal view 7 months after operation. B. Lateral view 7 months after operation.

Redundant granulation tissue and slight drainage were noted in the left naso-orbital area of the incision as well as at its posterior margin in the left temporal area. There were healed scars along the left side of the nose. The patient stated that the drainage areas often closed and then re-opened. He also mentioned that he tired easily, but he had observed no right-sided weakness, *nor had he been troubled with headaches.* He had little ambition or drive, and he forgot names easily. Diplopia occurred when he looked upward and to the right, but the left eye seemed so weak that this did not bother him. He had realized an impairment in the sense of smell. There was no evidence of speech defect and no obvious aphasia.

Neurologic examination disclosed the patient's inability to identify camphor in either nostril; a noticeable atrophy of the left optic disk; diplopia on gazing upward and to the right, with an evident weakness of the left internal and inferior rectus muscles; vision of 20/50 corrected to 20/40 in the left eye; and restriction of the temporal field and enlargement of the blind spot.

Roentgenograms of the skull showed a large area in which bone was absent. A single small metallic fragment, 3 by 3 by 2 mm., was seen in the soft tissues overlying the midportion of this area. Near the orbital bone there were also a few calcified areas which appeared to represent small, depressed fragments of bone.

Because of the redundant granulation tissue and continued drainage at the ends of the old craniotomy incision, as well as the evidence of retained foreign bodies, the old incision was reopened on 15 October 1943, with the patient under Avertin with amylene hydrate and ether anesthesia. The dense scar tissue overlying the skull defect was excised, the anterior portion of the left frontal lobe was explored, and 5 small pieces of bone and 1 small piece of rubber, which was a fragment of the patient's oxygen mask, were removed. The small metallic fragment and portions of devitalized and lacerated cerebral tissue in the anterior portion of the left frontal lobe were also removed. A pattern outline of the skull defect was taken at the time of operation on a piece of sterile cellophane. The interrupted subcuticular silk sutures were also removed, and the scalp flap was resutured exteriorly with interrupted sutures of medium black silk. The patient was given a transfusion of 500 cc. of citrated blood at the conclusion of the procedure.

His postoperative course was uneventful, and the wound healed without further drainage. Roentgenograms on 31 October 1943 showed that the previously visualized fragments of bone and the metallic fragment had been removed.

On 23 February 1944, a repair of the cranial defect was carried out, with the patient under Avertin with amylene hydrate and ether anesthesia. It was believed that sufficient time had elapsed to preclude the possibility of further infection. An accurate tantalum plate of the defect was prepared by means of impressions, molds, and models. The previous scalp incision was reopened and carefully dissected laterally, exposing the skull defect. The scar tissue and dura had regenerated over the brain and were surprisingly normal in appearance. After adjustments had been made in the plate, drillholes were perforated around the edge of the bony defect with an electric drill, and the plate was wired firmly in place with tantalum wire. The scalp flap was then replaced, and the subcutaneous tissues were approximated with interrupted sutures of medium black silk. The skin was closed with interrupted end-on mattress sutures of fine black silk. Before closure, a small amount of powdered sulfathiazole was sprinkled in the wound. At the conclusion of the procedure, the patient was given a transfusion of 500 cc. of citrated blood. He was taken from the operating room in good condition.

The postoperative convalescence was uneventful and the cosmetic result was good (fig. 50A and B). Roentgenograms of the skull on 6 March 1944 showed the previous defect in the left frontal and parietal region covered by a perforated metal plate held in position by several wire loops.

Jacksonian convulsions had developed before the operation in the Zone of Interior, and after operation the patient continued to have convulsions about every 2 months. They were never fully observed. This was one of the cases handled before adequate appreciation of the technique of the direct or secondary method of cranioplasty had been developed.

Case 3.—A 34-year-old officer was struck by machinegun bullets on 16 April 1945, in Germany. He sustained a severe lacerating wound involving the scalp, skull, dura, and brain, with an associated left hemiparesis, and a compound comminuted fracture of the skull which led to a large deformity of the right parietal region.

Primary debridement was done on the day of injury. A piece of fascia lata from the left thigh was sutured over the dural defect, and the scalp wound was closed in layers. When the patient regained consciousness, he was found to have an almost complete left-sided paralysis. The cranial wounds healed without difficulty, but a large skull defect was left. A gradual improvement in the paralysis ensued.

Roentgenograms of the skull disclosed a defect of the right parietal region measuring 11 by 5 by 4 cm., with evidence of old fracture lines extending into the lateral border of the right orbit. On 16 October 1945, a tantalum cranioplasty was performed, the inlay technique being used along the upper portion of the plate and the onlay method elsewhere. Tantalum wedges were used for fixation of the plate.

The patient made an uneventful recovery, and the cosmetic result was satisfactory. After operation, he had one major convulsive seizure, an adequate description of which was not obtained. When he was last seen, in November 1946, he looked quite well; he had put on weight; the hemiparesis had greatly improved, and the cranioplasty had caused him no trouble.

Case 4.—This soldier sustained penetrating, bifrontal wounds of the head as the result of machinegun fire on 30 November 1944, near Metz. He had a compound comminuted fracture of the skull and a laceration of the brain in the area involved. Debridement was carried out at an evacuation hospital, and he reached Hammond General Hospital on 14 January 1945.

Roentgenograms of the skull on 22 January 1945 revealed a large bony defect affecting both frontal bones. In addition to the skull defect, the patient had loss of memory and an incomplete type of motor aphasia.

Tantalum cranioplasty was carried out on 4 April 1945 by the inlay technique, with fixation by means of tantalum wires. The plate was prepared by hammering it out on a dental cement mold. The postoperative course was uneventful and the cosmetic result good. The patient was given a disability discharge on 22 August 1945.

Case 5.—A soldier sustained a rifle-bullet wound of the right eye and frontal lobe of the

brain, with an associated comminuted fracture of the supraorbital ridge, 30 July 1943, on Munda. Debridement and enucleation of the right eye were performed on 31 July at a station hospital in New Caledonia. He was evacuated to Hammond General Hospital, 2 September 1943. Petit mal seizures became evident in February 1944, and there was a grand mal episode on 23 July 1944. These major seizures continued until they were controlled by phenobarbital medication.

General physical examination disclosed nothing unusual other than the defect of the right frontal region and right anophthalmos. Roentgenograms of the skull revealed a large bony defect involving the right anterofrontal bone and extending inferiorly from the coronal suture to the right orbit.

A tantalum cranioplasty was performed on 31 October 1944 by the onlay technique, with fixation by means of tantalum wire. The plate was preformed by the indirect method and was hammered out on the dental mold.

The postoperative course was uneventful, and the patient was transferred to Dibble General Hospital, Menlo Park, Calif., on 14 December 1944, for fitting of an artificial eye and any further plastic procedures necessary. Photographs 17 days after operation revealed a satisfactory cosmetic result.

Case 6.—A soldier received a penetrating injury to the right supraorbital region from a shell fragment in October 1944, in Italy. The injury resulted in a compound comminuted fracture of the affected area, as well as a laceration of the right frontal lobe of the brain. Craniotomy, with removal of the comminuted bone fragments and devitalized cerebral tissue, was carried out overseas.

Except for the skull defect in the right frontal region (fig. 51A), examination at Hammond General Hospital in December 1944 disclosed nothing unusual. A tantalum cranioplasty was performed on 12 February 1945. The plate was prepared by the indirect method and was hammered out on the dental mold. The onlay technique was used, and fixation was carried out by means of tantalum wire.

A good cosmetic result was obtained, and the patient was given a disability discharge on 30 May 1945. Anteroposterior and lateral roentgenograms of the skull before and after cranioplasty with tantalum are shown in figure 51B and C, and the postoperative appearance of the patient is shown in figure 51D.

Case 7.—A soldier who was injured by a bomb fragment on 24 September 1943, in New Guinea, sustained a compound comminuted fracture of the skull and an underlying laceration of the parasagittal region of the brain. Debridement was performed overseas. A tantalum cranioplasty with the inlay technique and fixation by means of tantalum wires was carried out in the Zone of Interior on 22 May 1944. The scalp overlying the defect was rather thin in two areas, but, at this stage of the experience with tantalum cranioplasty, the likelihood of subsequent pressure atrophy was not sufficiently appreciated. The plate, however, had to be removed on 19 July 1944 for this reason. Granulation tissue had filled in the dead space beneath the tantalum plate and also had filtered through the perforations in it, thus holding it firmly in position.

A second attempt at repair was carried out on 3 October 1944. Although it had been believed that skin grafting would be required, the condition of the scalp appeared sufficiently satisfactory to make it appear that a plastic procedure of this sort might be unnecessary. The patient made an uneventful recovery, and the procedure seemed to be successful.

Some time before cranioplasty, convulsive seizures had developed. The patient was given a disability discharge on 18 January 1945. About a month later, an area of necrosis in one of the thin areas of the scalp necessitated his return to a Veterans' Administration hospital for further treatment.

Case 8.—A 33-year-old soldier received a severe pentrating wound of the right frontoparietal region of the skull and brain from an enemy shell fragment on 14 November 1944, in Germany. An associated compound comminuted fracture resulted in a large residual skull defect (fig. 52A). With the patient under local anesthesia, debridement of the wound, with removal of bony fragments, was carried out overseas on 14 November 1944.

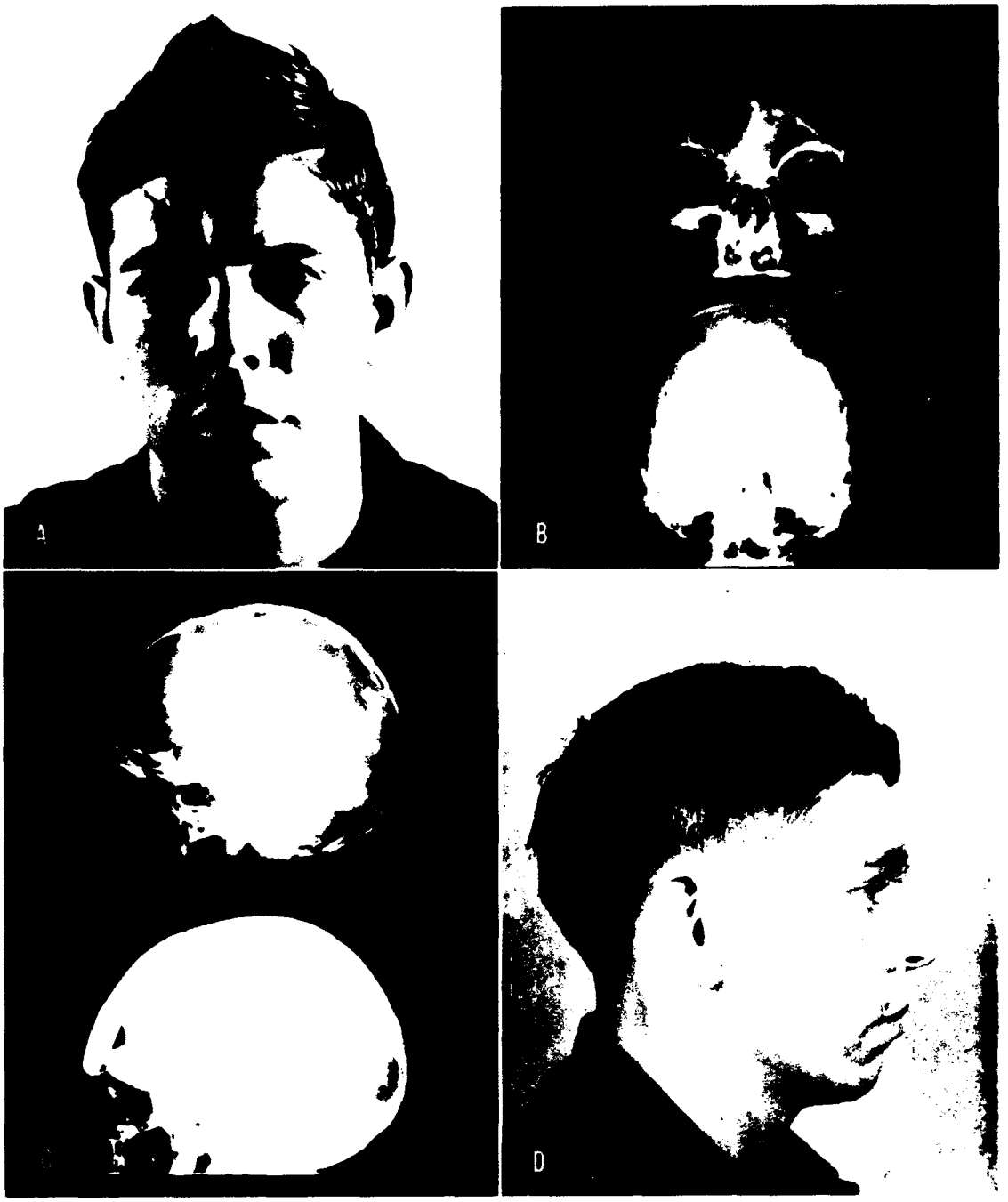


FIGURE 51. A. Frontal view of patient with large disfiguring deformity of right supraorbital region. B. Anteroposterior roentgenograms of skull before and after tantalum cranioplasty. C. Lateral roentgenograms before and after tantalum cranioplasty. D. Lateral view 3 months after operation.

The patient reached Hammond General Hospital on 29 January 1945. Paralysis of the left arm had been observed at the 25th Evacuation Hospital, and there was a similar involvement of the leg after operation. Improvement gradually occurred in both areas, more notably in the leg than in the arm.

Roentgenograms in the Zone of Interior revealed a skull defect in the right parietal area near the midline measuring 7 by 8 cm. (fig. 52B and C). While at home on furlough,

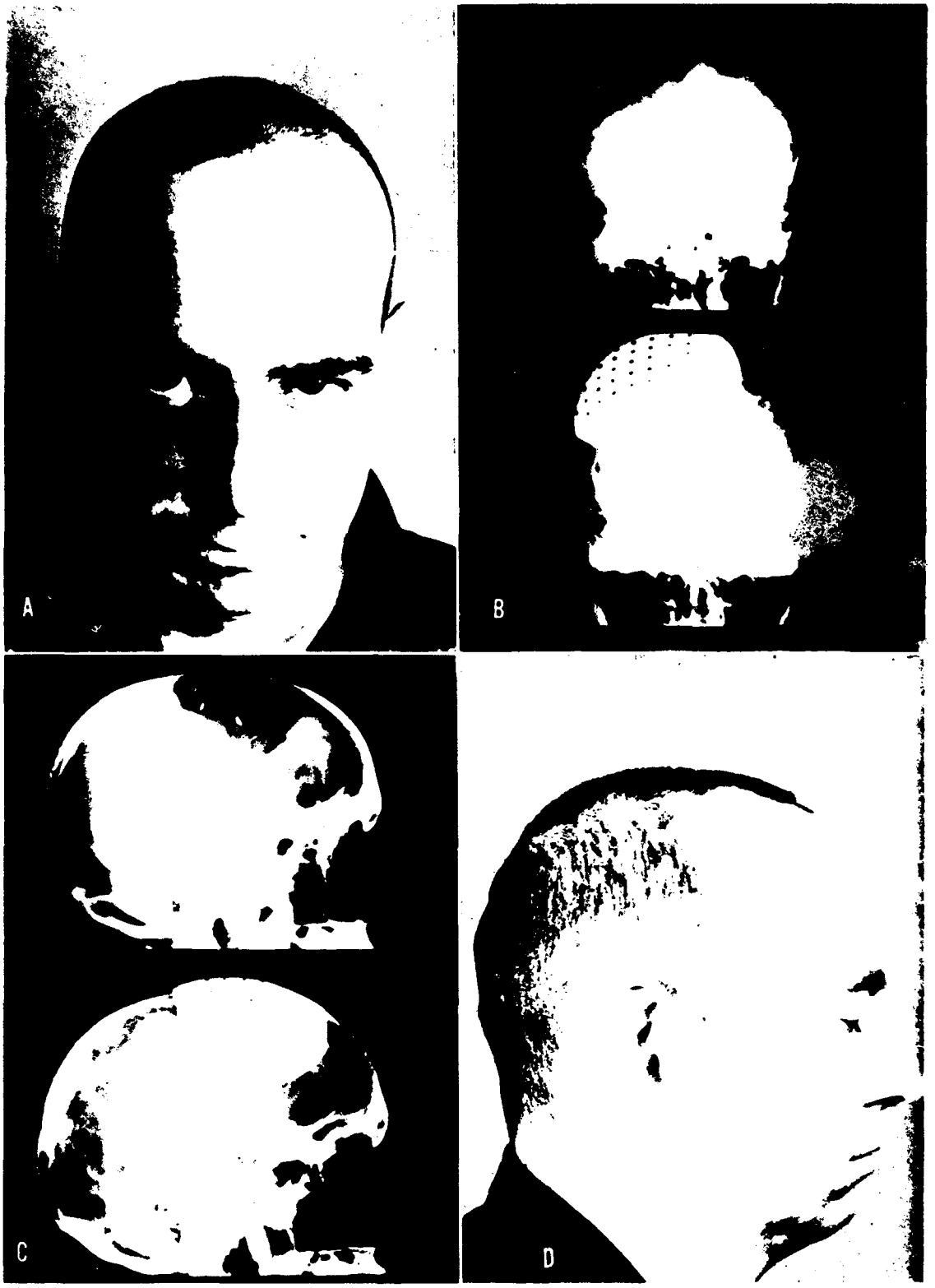


FIGURE 52. A. Frontal view of patient with large residual defect of right frontoparietal region. B. Anteroposterior roentgenograms before and after tantalum cranioplasty. C. Lateral roentgenograms before and after tantalum cranioplasty. D. Lateral view 17 days after tantalum cranioplasty.

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the patient had a convulsive episode. A tantalum cranioplasty was performed on 5 April by the onlay technique, with fixation by means of tantalum wire. The plate was preformed by the indirect method and was hammered out on the dental cement mold rather than by means of a pressing cylinder.

The postoperative course was uneventful, and the cosmetic result was satisfactory (fig. 52D). The patient was discharged to a Veterans' Administration hospital for separation from the service on 7 September 1945. A letter dated 1 May 1947 indicated that he did not even realize that the plate was in his skull, since his head "felt fine all of the time." He was apparently continuing to have occasional convulsive episodes.

SUMMARY AND CONCLUSIONS

In World War II, the experience with Vitallium, Ticonium, and tantalum for cranioplasty left little justification for the employment of other metallic materials, and, at the end of the war, most of those familar with the subject considered tantalum the metal of choice for the repair of the larger defects. While acrylic plates may have certain advantages, in that they lack temperature changes and are inert to electric phenomena, their preparation is more difficult, and it is doubtful that they are as satisfactory in large defects as tantalum.

Autogenous bone or cartilage continues to offer an adequate means for the repair of the smaller skull defects, particularly those involving the frontal sinuses and the region of the glabella. Final selection will probably remain somewhat the personal choice of the surgeon who is more familar with, and more enthusiastic about, one technique than another.

It is generally agreed that indications for cranioplasty include the following. (1) The repair of deforming and unsightly defects; (2) the danger of trauma at the site of the defect; (3) pulsating and painful defects; and (4) headaches and other symptoms of the syndrome of the trephined, including discomfort at the site of the defect. The relief of an associated convulsive state has also been considered as an indication, but it is not believed that the cranial defect is the cause of convulsive seizures or that its repair alters their incidence appreciably.

Secondary intervention for tantalum plating in the presence of infection or immediately following wound healing cannot be justified on the basis of scattered successes. It is believed wise to consider a minimum delay of 3 months after primary healing before a secondary procedure to cover a cranial defect is performed, and to allow 8 months after healing if frank infection has been present. Complete coverage with good scalp is essential to successful tantalum cranioplasty; if the cranioplasty is not successful, the tantalum plate should be removed promptly.

CHAPTER XIII

Posttraumatic Epilepsy

A. Earl Walker, M. D.

ADMINISTRATIVE CONSIDERATIONS

In November 1945, Cushing General Hospital, Framingham, Mass., was designated as a center for the treatment of posttraumatic epilepsy, and arrangements were made for the transfer to it of all patients with this disease then under treatment in Army general hospitals. Convoys began to arrive early in December, and patients continued to be received at irregular intervals for the next 6 months.

In all, 246 patients were treated for posttraumatic epilepsy at this center. A few others who were admitted with this diagnosis were found, on examination, not to be suffering from the disease.

To process so large a number of cases within a short time would have been impossible without the organization developed in the course of the war by Lt. Col. Frederic H. Lewey, MC, and Lt. Col. W. P. Van Wagenen, MC, which provided functionally united, although administratively distinct, sections of neurology and neurosurgery (p. 21). The existence of this organization made it feasible to utilize at once all the aptitudes and special skills available in the hospital and to begin the study of patients immediately without waiting for specially trained personnel. The plan may not have been ideal, but without it the posttraumatic epilepsy program would have encountered grave difficulties.

All patients were admitted to the neurologic service, on which they were worked up by the staffs of the neurologic and neurosurgical sections, under the supervision of Maj. F. A. Quadfasel, MC. Clinical and electroencephalographic studies were made, autonomic functions were investigated, psychologic examinations were conducted, and vocational counseling was provided. The American Red Cross assisted in the securing of social histories and in the rehabilitation of the patients.

CLINICAL INVESTIGATION

Anamnesis and General Physical Examination

Because the histories which accompanied the patients on their transfer to Cushing General Hospital were of varying degrees of completeness, it was found necessary to adopt a standard form for the clinical history. This form was prepared after a careful investigation of the epilepsy history outlines in current use at the neurologic centers in Montreal, Boston, New York, and

Chicago. The form (appendix C, p. 411) was designed as a guide for ward officers to insure that all pertinent data would be elicited and recorded in definite order. It also made it possible to analyze the clinical history with comparative ease.

Each patient was given a complete physical examination in conformity with Army general hospital procedure.

Neurologic Examination

Because almost all of these patients had neurologic deficits, the majority of which were quite severe, it was considered advisable to have the neurologic examination follow a more flexible form than the Army neurologic examination sheet (55–E–10–A), which was set up for patients with few abnormalities. Accordingly, a special outline (appendix C, p. 414) was formulated for the recording of the neurologic examination.

Officers who had had considerable experience in neurology obtained the detailed neurologic history and carried out the examination. Both history and examination were doubly checked for accuracy and completeness by the neurologist and the neurosurgeon.

Other Special Studies

Roentgenologic examination.—Routine roentgenograms of the head were made at the time of admission. All previous films of the skull were reviewed. Pneumoencephalograms were reread and a note concerning them was incorporated in the history.

Examination of spinal fluid.—Unless the patient had had a recent, complete examination of the spinal fluid, a lumbar puncture was performed. Careful observations were made of the spinal fluid pressure under normal conditions and following jugular and abdominal compression. Cytologic, serologic, bacteriologic, and chemical studies were made on the spinal fluid.

Ophthalmologic examination.—An ophthalmologic consultation was requested on each patient, with particular emphasis on visual field disturbances.

Otolaryngologic examination.—The otolaryngologic consultant was asked to emphasize hearing and vestibular disturbances.

Aphasic examination.—Each patient was given an examination designed to reveal possible disturbances of speech (appendix C, p. 417). In normal individuals, this examination served as a rough test of the individual's intelligence and could be completed in a few minutes. In aphasic individuals, it indicated the type and severity of the impairment and could be used for comparative purposes at a later time.

Psychologic examination.—A number of tests were used as indicators of psychologic performance, as follows:

1. The Wechsler-Bellevue test was considered to give a general rating of the individual's intellectual ability.

2. The revised Binet vocabulary test was somewhat more dependent upon a special form of learning.

3. The Wechsler memory scale was also a test of somewhat special aptitudes.

4. The Bender Visual Motor Gestalt test was used as a possible indicator of brain damage.

5. The critical fusion frequency test was included as a criterion of focal brain damage.

6. The Goodenough test was included for evidence of brain damage.

It was hoped that these tests, each serving a special function, would constitute a battery which might differentiate a patient with cerebral injury from a normal individual and might indicate whether a return to normal or a progression of the cerebral process could be expected.

Autonomic function.—Since vasomotor disturbances were often present in individuals suffering from posttraumatic epilepsy, the autonomic examinations included the following: (1) Sweat tests, (2) plethysmograms, (3) electrocardiograms of oculocardiac reflex, (4) electrocardiographic alterations, and (5) oscillometry.

Electroencephalograms.—Routine electroencephalograms were made of each patient after he had had no medication of any kind for 3 days. Standard hyperventilation tests were carried out at the end of the routine run. If an abnormal focus of activity was present, extra electrodes were placed and an accurate localization was made.

Social history.—With the assistance of the American Red Cross, the family of each patient was interviewed in order to obtain more specific and accurate information of his childhood. Red Cross workers were given a suggested outline of the social history desired (appendix C, p. 419).

Evaluation of Findings

These studies constituted the basic workup of each patient admitted to the hospital. When complicating factors were present, they were taken care of by appropriate means. Not a few patients were suffering from osteomyelitis or fractures of bones and required orthopedic treatment. Some required plastic reconstruction of the face. Others presented special otolaryngologic problems. As the result of their cerebral wounds, a few were paraplegic and needed special care.

When the case had been fully worked up, it was presented to a combined neurologic-neurosurgical conference in which the problems were reviewed and medical and surgical therapy was decided upon and initiated.

ANALYSIS OF CLINICAL FINDINGS

The records of the patients suffering from posttraumatic epilepsy and treated at Cushing General Hospital have been extensively analyzed in the hope of identifying factors which influence the development of the convulsive state.

Age distribution.—The age incidence of this series corresponds well with the age distribution in the wartime Army. There were 96 patients between 19 and 25 years of age, 83 between 26 and 30, 54 between 31 and 35, and 13 were over 35.

Type of injury.—Of the 246 patients in this series, 236 had open wounds and 10 had closed injuries of the head. Bilateral or multiple wounds were present in 40 cases. The open injuries were variously caused by shell fragments (154), bullets (58), shell fragments and bullets (2), blunt objects (15), a knife (1), and unknown agents (6).

Location of wound.—The sites of cranial and cerebral wounds in this series were scattered over the entire head, with a predominance of lesions in the parietal region of the skull. In a number of cases, perforating wounds were present with extensive damage to more than one lobe of the brain and in some cases to both hemispheres (table 12).

History of unconsciousness.—The period of impaired consciousness varied greatly (table 13) and did not seem to be directly proportional to the extent of the apparent cerebral wound. Shell fragments appeared to cause a greater and more prolonged impairment of consciousness than bullet wounds of the brain, but the difference is probably not very significant. It is noteworthy that almost a quarter (23.4 percent) of the patients with craniocerebral wounds for whom this information is available had no period of unconsciousness.

Debridement.—The patients who comprise this group were more severely wounded than the patients who would comprise a random sample of head injuries (fig. 53).[1] The figures are not biased, however, by the factor of late

TABLE 12.—*Location of head injury in 240 patients with posttraumatic epilepsy*

Location of injury	Lobes involved			
	Right side	Left side	Side unknown	Total
Frontal lobe	13	20	3	36
Frontoparietal lobe	9	14	4	27
Frontotemporal lobe	2	3	1	6
Parietal lobe	27	38	4	69
Parietotemporal lobe	9	8	2	19
Temporal lobe	4	6	0	10
Parieto-occipital lobe	7	16	2	25
Occipital lobe	1	5	2	8
Both frontal lobes				3
Both parietal lobes				4
Both temporal lobes				1
Vertex				4
Other combinations				28
Total	72	110	18	240

[1] Maltby, G. L.: Penetrating Craniocerebral Injuries: Evaluation of the Late Results in a Group of 200 Consecutive Penetrating Cranial Wounds. J. Neurosurg. 3: 239–249, May 1946.

TABLE 13.—*Duration of impairment of consciousness after head injuries in 287 patients with posttraumatic epilepsy*

Period	Unconsciousness	Confusion
None	46	23
1 day	51	38
More than 1 day	99	151
Unknown	41	25

debridement, for, in the 168 cases in which the time of initial wound surgery was known, the operation was done within 24 hours of wounding in 132 cases, between 2 and 4 days afterward in 28 cases, and after 5 days in only 8 cases.

The severity of the injury may be judged by the extent of debridement. In at least 154 of the 178 cases in which the extent of the debridement was known, dural penetration had occurred. Debridement of the skull and scalp was necessary in 4 cases and debridement of the dura in 12. Surgery was limited to the scalp in only eight cases.

Infection.—In spite of the early treatment which most of these patients received, infection occurred in 82 of the 186 cases in which the character of wound healing was known. This is an unusually large proportion. Age may have played some part in the rate of infection, for secondary wound healing occurred in 30.5 percent of the patients over 25 years of age against only 15 percent in patients under 25 years. The type of chemotherapeutic or anti-

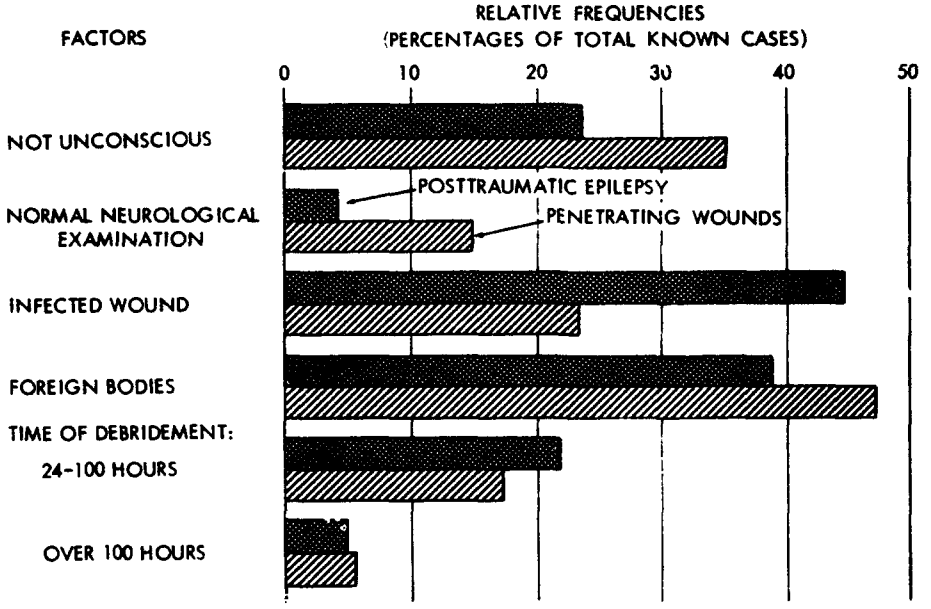


FIGURE 53.—Relative frequencies of various factors in selected series of posttraumatic epileptics and in a nonselected series of patients suffering from penetrating wounds of head.

biotic therapy did not seem to influence the rate of infection. About the same numbers of infected and noninfected patients received sulfonamide drugs and penicillin.

Intracranial foreign bodies.—This study supplies no evidence that the presence of intracranial foreign bodies is a factor in the production of epilepsy. The majority of the patients (141) had no demonstrable foreign bodies within the brain.

Neurologic defects.—The severity of the cerebral wounds in these cases is evident from the fact that, at the time of their admission to Cushing General Hospital, all but 14 of the 246 patients presented one or more neurologic abnormalities.

Electroencephalographic abnormalities.—A preliminary survey of the electroencephalographic records in this series indicated that the tracings were normal in only 31 cases. A generalized dysrhythmia, such as is seen in the idiopathic forms of epilepsy, was present in 25 cases. Localized or focal abnormalities occurred in 180 cases. The great majority of the records showed slow wave foci (one-half to 6 per second) about the site of the cranial defect and brain wound (fig. 54). A much smaller number showed spiky waves or spikes confined to the defect.

For comparison, an analysis was undertaken of the records of a control series of nonepileptic patients admitted to Cushing General Hospital for the treatment of craniocerebral injuries.[2] This series was composed of patients with somewhat less severe wounds of the head than the group with posttraumatic epilepsy. It is not surprising, therefore, that a slightly smaller percentage of focal abnormalities was found in the control group than in the epileptic group. The generalized dysrhythmias were of about equal frequency in both series. The incidence of localized spiky waves or spikes, which are usually considered indicative of epileptic activity, was the same in both. Serial electroencephalographic examinations were made in a number of cases in both groups, but it was not possible to predict from these examinations an epileptic diathesis or to tell if the patient had had convulsive seizures. From this investigation, it was concluded that the electroencephalogram was not an adequate indicator or prognosticator of the convulsive state in cases of penetrating brain wounds.

Posttraumatic syndrome.—Although the posttraumatic syndrome is usually thought to be associated chiefly with mild head injuries, both headaches and dizziness were complained of by 54 patients in this series.

Epileptogenic Factors

The statistical analyses suggest that a number of factors predispose to posttraumatic epilepsy, as follows:

1. The severity of the local injury seems to be important, as may be

[2] Kaufman, I. C., and Walker, A. E: The Electroencephalogram After Head Injury. J. Nerv. & Ment. Dis. 109: 383-395, May 1949.

assumed from the high incidence of dural penetration (87 percent), electroencephalographic changes (86.8 percent), and neurologic damage (94.3 percent) in this series.

2. Sepsis in the wound may be a predisposing factor in epilepsy. It was present much more frequently, according to Maltby, in this series than in an unselected series of penetrating head wounds.

3. The location of the wound seems to play a role in the production of posttraumatic epilepsy. If the incidence of craniocerebral wounds in the different parts of the head was the same in World War II as in the First World War, the distribution of wounds in any unselected series would be 27 percent frontal, 25 percent parietal, 17 percent temporal, and 15.5 percent occipital.[3] In this series, the majority of injuries were in the parietal region of the head; approximately 18 percent were in the frontal area and about 5 percent in each of the temporal and the occipital regions. Posttraumatic epilepsy thus seems to occur much more frequently with lesions in the central region than with lesions elsewhere. On the other hand, the frequency of attacks, once the attacks were established, did not seem to be related to the site of the wound or to the presence of foreign bodies.

4. It was the clinical impression that convulsions were precipitated by a cranial operation, such as cranioplasty. The impression was borne out by the fact that 45 patients had convulsions within a week after this operation.

This analysis does not support some of the previous hypotheses concerning the causation of posttraumatic epilepsy, as the following data show:

1. Heredity did not appear to be an important factor in its development. In this series, only 4.5 percent of the patients' relatives had convulsions, as compared to an incidence of 3.4 percent in normal families and an incidence of 17 percent in epileptic families.[4]

2. Intracranial foreign bodies did not seem to predispose to epilepsy. The majority of the patients in this series had no demonstrable foreign bodies within the brain, and the incidence of such objects was no higher in this series, according to Maltby, than in an unselected series.

3. Cerebral dysrhythmias did not appear to be an important consideration in posttraumatic epilepsy. The percentage of cases exhibiting these phenomena was not appreciably greater in this series than in the normal population.

Time of onset of epilepsy.—The time after injury at which the epilepsy occurred varied greatly. In 27 percent of the cases, the seizures came on within 3 months and, in another 30.7 percent, between 3 and 6 months after injury. The delayed onset of attacks did not seem to have prognostic importance nor was it related to the anatomic site of the injury.

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[3] Kerr, H.: A Statistical Analysis of Gunshot Wounds of the Head. *In* The Medical Department of the United States Army in the World War. Washington: Government Printing Office, 1927, vol. XI, pt. 1, p. 842.

[4] Lennox, W. G.: Personal communication.

THE EPILEPTIC ATTACK

Auras

The type of seizure in this series did not, in general, differ from the type seen in idiopathic epilepsies. It is true that focal manifestations (present in 75.8 percent of the cases) were more common than in an unselected group of patients suffering from convulsive seizures. The most common localizing sign was an aura—motor, sensory, or visual—which ushered in the attack. The types of aura in 239 patients with posttraumatic epilepsy are shown in the following tabulation:

Type of Aura:	*Number of patients*
None	63
Motor	64
Somatosensory	54
Visual	23
Complex	17
Vertiginous	8
Auditory	5
Epigastric	4
Diplopic	1
Total	239

The occurrence of an epileptic warning seemed to be related to the location of the injury. In the patients who had no aura, there was a high incidence of frontal wounds; in those with motor aura, there was a high incidence of frontal and parietal injuries; and in the group with sensory aura there was a high incidence of wounds in the parietal region.

Motor aura.—The motor phenomena preceding a fit were usually present in the upper extremity, less commonly in the face, and only occasionally in the leg. An entire half of the body was occasionally involved. The predominance of auras in the arm is probably only a correlate of the extensive cortical representation of the arm and hand. Both inhibitory and excitatory phenomena occurred as auras. A weakness or heaviness of the arm was occasionally encountered, and an inability to speak was not infrequently stated to be the first manifestation of an attack. Excitatory phenomena were more common; they consisted of twitching of the face, eye, or fingers; jerking of the arm or leg; or turning of the head and eyes away from the side of the cranial wound.

The motor manifestations persisted either for a very short period or for many minutes. They might or might not be followed by a generalized seizure. Some patients found that massage of the twitching part stopped the progress of the attack. In a few cases, the aura started in one member and spread to another, but a true Jacksonian march was seldom encountered. In only a single case did the patient describe a motor aura followed by a sensory hallucination, although the reverse was common.

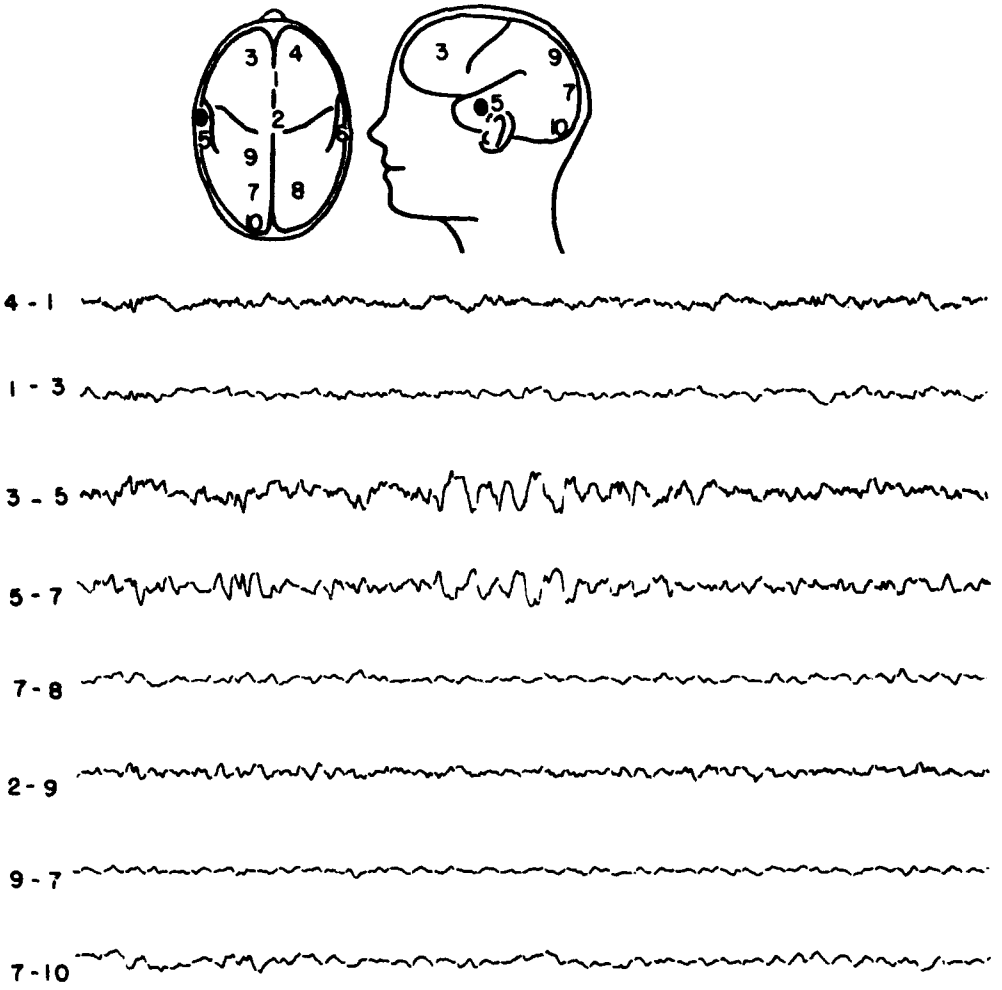


FIGURE 54.—Electroencephalogram of patient with penetrating wound of left temporal region 23 months before this record was made. The slow wave out of phase activity in the third and fourth tracings is due to a focus under point 5.

Somatosensory aura.—Just as with motor phenomena, sensory auras were referred to the face, hand, or arm, and, less often, to the trunk and leg. Occasionally, the neck, axilla, abdomen, or spine was the site of the paresthesia. The individual parts of the foot were not singly involved as were the thumb and fingers.

The usual type of paresthesia was a sensation of numbness. It was sometimes described as a "funny feeling," "as if the hand were off," "a tickling sensation," "a tingling," or "a pins and needles" feeling. One patient had a kinesthetic aura as if the right arm were swinging in a circle, and another a feeling as if the arm and leg were about to move. "Burning" was seldom used to describe the sensation; only one patient complained of the hallucination of coldness. Pain as an aura was encountered in only two cases. One patient stated that his warning consisted of the "going to sleep" of the right hand and then the right leg, followed by painful abdominal cramps and spasms of the

right hand and leg. Frequently, the sensory aura was followed by motor phenomena such as twitching or jerking of the paresthetic extremity. Occasionally, olfactory, auditory, and visual hallucinations rounded out the aural cycle.

Visual aura.—The common visual aura consisted of a blurring of vision in one or both visual fields. The impairment of sight was frequently described as dots, a black ball, a circle, or a curtain moving across the visual field in an undulating or pulsating fashion. Frequently, the scotoma began in the periphery of a hemianoptic sector and proceeded across the vertical meridian to involve the normal field, causing a complete loss of vision for a few minutes. With the onset of blindness, a generalized attack sometimes developed.

The visual aura often consisted of white or colored "lights" in the affected visual fields. Occasionally, the patient described his warning as a pyrotechnic display with all the colors of the rainbow. The hallucination might develop in an hemianoptic field. Occasionally, a blinding light preceded a grand mal attack. One patient described his aura as "little yellow stars followed within a minute by blindness," either lasting 2½ hours or followed within a few seconds by a generalized seizure. Another patient maintained that he saw blue flashes of light in his right lower quadrantanoptic visual field and then "bedpans, ducks, books, trains, and magazines" moving to the left. When lateralized, the hallucination occurred in the visual field contralateral to the cerebral wound. It might be followed by numbness of the face, arm, or hand on the side of the aura, and, less commonly, by twitching. One patient complained of his "eyes going out of focus" and another of seeing double at the onset of the attack.

Complex aura.—These are bizarre auras. One patient spoke of his attacks beginning with a confused feeling, a sizzling noise, and then a cry as the tonic phase started. "Sweating and blackouts" were the words used by another patient to describe his aura. Dreamy states occurred but were unassociated with gustatory and olfactory hallucinations. Although the patient was always asked about it, a full-blown uncinate attack was not encountered. A patient with a bilateral inferior frontal lobe injury was warned of his attack by "a funny feeling in the stomach, smell of burnt almonds, and a droning noise like an aeroplane." A number of patients had auras composed of auditory, visual, and motor components. A ringing of bells, flashes of light, and movement of the right arm ushered in the attacks of a patient who had a wound in the left occipital region. The onset of the epilepsy in another patient was characterized by ringing in the left ear, followed by a feeling of coldness of the left side of the lip and face, and burning of the fingers of the left hand. Still another noted that, before the attack, sounds became fainter, the left side of his face, lips, teeth, and tongue burned, and his jaw muscles tightened, so that he could not talk. These are but a few examples of the varied complex auras encountered in this series of patients with posttraumatic epilepsy.

Vertiginous aura.—A number of patients complained of dizziness just before the generalized attack. It is usually difficult to assess this sensation,

but in some cases there appeared to be a true vertiginous element in the dizziness. One patient stated that objects spun around and he became nauseated.

Auditory aura.—Uncomplicated auditory auras were encountered infrequently in this series. Usually, a sonic warning was accompanied by a visual, motor, or sensory hallucination. One patient "heard voices, saw bright lights," and then became unconscious. Humming in both ears ushered in the attack in one case. A sound of "motors running followed by a stinging sensation in the right hand" constituted another aura. Usually, the aura was not lateralized, but one patient maintained his auditory premonition was in the left ear.

Epigastric aura.—The epigastric auras consisted of a feeling of uneasiness in the pit of the stomach, nausea, a choking sensation, cramping abdominal discomfort, and, occasionally, vomiting. Such visceral auras were infrequent. Occasionally, the attack was initiated by other manifestations such as sweating.

Although in some cases the attacks consisted only of focal manifestations, generalized seizures developed in 87 percent of the cases in this series. Other variants of the epileptic diathesis were less commonly seen. Fifteen patients had petit mal attacks, and two had psychomotor episodes. In none of these cases was there an electroencephalographic spike and dome pattern characteristic of petit mal or psychomotor epilepsy.

MEDICAL MANAGEMENT

As a general rule, all patients were treated by nonsurgical measures before surgical procedures were considered with the exception of those suffering from such conditions as uncontrollable status epilepticus, infections of the scalp, or cranial defects which required repair.

Anticonvulsant Medication

It had been hoped that medical therapy could be regulated by determinations of the blood and spinal fluid levels of the anticonvulsant agent used, but this did not prove practical. Without this information such therapy was clearly empiric, but it was nonetheless employed and gave good results.

In the first cases encountered, treatment was begun with small doses of phenobarbital (0.1 gm.) at night, the dose being increased if an attack occurred. When this regimen proved inadequate, phenobarbital was given in 0.1-gm. doses twice daily and Dilantin Sodium (diphenylhydantoin sodium) was given in the same dosage 3 to 6 times daily.

In 16 of the 20 cases in which this regimen was tested, daily doses of Dilantin Sodium (5 capsules or more) proved toxic. Ataxia and dizziness were the most frequent side effects, but in three instances extreme ataxia, vomiting, weakness, and nystagmus developed after this treatment had been carried out for several weeks. Complete investigation of the neurologic status of the affected patients, including pneumoencephalographic studies, disclosed no

other cause for the symptoms. The patients slowly improved when Dilantin Sodium therapy was discontinued, but, in one case, it was more than 3 months before the nystagmus disappeared.

If epileptic seizures were not controlled by a regimen of phenobarbital in doses of 0.1 gm. 2 or 3 times a day (the administration being regulated according to the degree of drowsiness it produced), combined with Dilantin Sodium in doses of 0.1 gm. 4 times a day, medical management was regarded as unsuccessful and surgical measures were considered.

Results of Anticonvulsant Therapy

The results of medical management, in spite of the difficulties just related, were on the whole quite good. One evidence of its results is the fact that at the beginning of the program practically all of the hospital personnel observed one or more seizures, while after a few months nurses were sometimes on the ward for months before they witnessed an epileptic attack.

Of 238 patients submitted to this regimen, seizures were controlled by it in 110 patients for periods of at least 6 months. In 64 of these 110 cases, the results were achieved by phenobarbital alone, usually in doses of 0.1 gm. daily. In 26 cases, Dilantin Sodium, usually in doses of 0.3 gm. daily, combined with phenobarbital in doses of 0.1 gm. daily, proved sufficient. In nine cases, Dilantin Sodium was used alone in doses of 0.3 gm. daily. The remaining patients were treated by various combinations of these drugs. It is quite possible that in many of this group Dilantin Sodium alone would have proved adequate, but the routine required did not permit testing.

In 22 cases, attacks were controlled from 4 to 6 months by the measures described. At the end of these periods, the patients were either discharged or were transferred to other institutions. In 40 cases, the patients were transferred to other Army hospitals or to Veterans' Administration hospitals before they had been treated long enough to warrant conclusions.

In the remaining 66 patients, the attacks were not controlled by anticonvulsant medication. Forty of the patients later underwent cortical resection. The other 26 patients were not operated on chiefly because of neurologic defects, infected wounds, or unwillingness to undergo surgery; no patient was operated on if he did not wish to be.

As this analysis indicates, the chief reliance in the medical management of these patients was upon phenobarbital and Dilantin Sodium. Tridione (trimethadione) was used in a number of cases but had no apparent effect on psychomotor seizures.[5]

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[5] In all, 130 patients were discharged from Cushing General Hospital with their epileptic attacks controlled either for 6 months or more (110) or for 4 to 6 months (20). At the end of 12 to 18 months after their discharge, 51 had had no convulsions, 19 had had only 1 seizure, and 22 had had several seizures. Few of them had continued taking the medication as prescribed and about 10 percent had discontinued it entirely. Under the circumstances, complete control of seizures in almost half of the 130 patients was regarded as distinctly encouraging. Social and economic results were, however, distinctly less promising. (Walker, A. E. and Quadfasel, F. A.: Followup Report on a Series of Posttraumatic Epilepties. Am. J. Psychiat. 104: 781–782, June 1948.)

General Measures

An extremely important phase of the treatment of these patients had to do with counseling them about their condition. Many had been unduly alarmed by gossip and rumors. Their morale was invariably much improved when their status was discussed with them tactfully but frankly. They were told that there was no ground for the stigmata so often attached by the laity to the epileptic state; that the seizures were simply an indication of the brain injury; that they could be controlled to a large extent if not entirely eliminated; that epilepsy does not lead to insanity, does not necessarily prevent marriage, and should not be kept secret from fiance, wife, or family; that unless attacks were very frequent, they were not incapacitating; and that the drugs used for the control of epileptic seizures are not habit forming. These explanations and their frank discussion did much to restore self-confidence and improve morale.

Patients were also advised about their general activities and way of life, the following facts being emphasized:

1. An epileptic must take certain precautions, exactly as do patients suffering from many other medical diseases.
2. Because of the danger of injuring himself in an attack, he should not bathe in deep water unattended or work around moving machinery into which he might fall in a fit.
3. Driving an automobile is dangerous not *only* to himself but to the public.
4. If the patient had frequent attacks, he was advised to carry with him a padded tongue depressor to place between his teeth when he felt an attack beginning.

General hygiene.—Experience has shown that epileptics are less likely to have seizures if their general physical condition is good and if they are mentally and physically active. A regular routine of exercise, wholesome meals at the usual hours, and adequate sleep each night was therefore provided in the hospital. A special diet was not used, but excesses were discouraged. Because seizures are frequently associated with constipation, the patients were advised to cultivate regular evacuation habits. Moderate smoking was allowed, but all alcoholic beverages, which tend to precipitate attacks, were forbidden.

Management of Seizures

It was found that the relatives of a patient and even hospital attendants did not have a healthy attitude toward patients subject to convulsions. The whole situation was discussed with them, and they were instructed in the proper care of a patient having a seizure. If the patient is prevented from injuring himself, they were told, the attack, while terrifying to behold, does not endanger life. Because the seizure is usually self-limited, the observer can be of more assistance by following a few simple directions than by rushing

to telephone a doctor. The clothing about the patient's neck should be loosened. To prevent tongue biting, a padded tongue depressor can usually be inserted between the molar teeth without using force. During the clonic phase of the attack, the patient should not be restrained, but he should be protected from injury by removing him from the proximity of radiators and furniture and by placing a pillow or other soft object under his head to prevent bruises and lacerations. During this clonic stage, the attendant should not leave the patient. It lasts only a minute or two following which he relaxes. Then, to maintain a patent airway, he may be turned on his side so that the saliva may drool from the corner of his mouth. He may now be moved to a bed, preferably one with sideboards.

Since an observant nurse or relative can give valuable information about the mode of onset and progress of the patient's attack, it was worthwhile instructing these persons in the points to note during and following an attack. These included the mode of onset of motor phenomena; the spread of the attack; the type of movement; involvement of one or both sides; the state of consciousness; tongue biting; incontinence of urine or feces; cyanosis; injury; duration of the attack; and, following the clonic phase, the period of confusion or excitement, weakness of an arm or leg, disturbance of speech, or any other unusual occurrence.

SURGICAL MANAGEMENT

Surgical treatment was considered entirely as an adjuvant to medical therapy for posttraumatic epilepsy. It was not regarded as a substitute for it, and most patients were not operated upon until intensive medical treatment had failed to control the attacks.

Indications

The most frequent indications for surgical intervention for epilepsy were the following: (1) Sizable bone fragments or other foreign bodies in the brain, especially if they were superficial and near the motor cortex; (2) a cranial defect larger than 2 cm. in diameter; and (3) convulsions not controlled by nontoxic amounts of anticonvulsant medication.

Intracerebral foreign bodies.—At the primary debridement of a wound of the head, all foreign bodies, bone fragments, and necrotic material were usually removed. If, however, a foreign body had been overlooked and was discovered months or years after an uneventful convalescence, it was difficult to decide whether it should be removed. The role of intracerebral bone fragments and foreign bodies in posttraumatic epilepsy has still not been clearly demonstrated, but there is no doubt that their presence in the brain is usually associated with greater pathologic reaction than occurs after surgical wounds.

If, therefore, convulsive seizures had developed and if foreign bodies present were superficial and readily accessible without damaging normal brain excessively, it was the practice at Cushing General Hospital to remove them,

with adjacent diseased tissue. If, however, the particles were small and if the patient's attacks could be controlled by anticonvulsant medication, they were usually left in situ.

Cranial defects.—Defects in the skull smaller than 2 cm. in diameter, unless they were situated in the frontal region and were disfiguring, usually required no surgical treatment. Such defects, especially in young individuals, gradually decrease in size because of bony proliferation at the margin of the defect, and in the course of a year or two they may be completely filled in by a thin layer of new bone. If there is no bony regeneration, a thick, firm, fibrous membrane covers the opening so that even the pulsations of the brain may not be palpable. These defects were therefore not usually repaired.

Defects larger than 2 cm. in diameter were usually repaired, since cranioplasty by protecting the underlying brain and eliminating the constant pulsations of the cerebral tissue may decrease or even eliminate convulsive seizures.

Persistent seizures.—Inability to control convulsive seizures by medical treatment constituted the most frequent indication for surgical therapy for posttraumatic epilepsy. If convulsions persisted in spite of therapy with Dilantin Sodium and phenobarbital in doses just below the toxic level, surgical intervention was considered justifiable.

Contraindications

A surgical procedure on the brain must be considered as carrying a certain risk. This danger must be weighed against the possible advantages of any operative procedure. Because epilepsy is not usually a lethal disease, no method of treatment should be employed that is attended by a heavy risk to life, especially if the results of the procedure are not well established. There were, therefore, certain contraindications to the surgical treatment of epilepsy at Cushing General Hospital.

Extensive neurologic disturbances.—It appeared unwise to subject an individual who was hemiplegic, paraplegic, or quadriplegic to a serious operation for the relief of convulsive seizures if the seizures played a very minor role in his disability. The condition of severely aphasic patients was also carefully evaluated before operation. Only if it seemed likely that the elimination of their attacks would enable them to lead a relatively normal existence were they subjected to operation. If they would still be seriously handicapped, even though they would no longer have attacks, the danger of aggravating the aphasia and the risk of operation were considered to overweigh the possible gain from the procedure.

Mental disturbances.—If the patient was mentally disturbed, the advisability of operation was always doubtful. Psychoneurotic tendencies leading to alcoholic excesses constituted, for instance, a poor background for surgery. Mental impairment to a degree which rendered the patient incapable of following medical advice and treatment was likewise regarded as a contraindication to surgery.

Chronic infections of scalp or cranium.—Infection as a contraindication to exploration of the cerebral cortex is self-evident. Appropriate medical and surgical measures were instituted in all such cases to clear up the infection.

General debility.—Because a cortical resection is a major surgical procedure, and one of considerable magnitude, surgical intervention was delayed in any instance of debility until the patient's general condition was improved.

Timing of Surgery

If foreign bodies were present in the brain or if a cranial defect existed in the skull at the time of the patient's first convulsive seizure, the optimum time for surgical intervention depended upon the time which had elapsed since the original brain injury. If less than 6 months had elapsed, immediate removal of the foreign bodies, with cranioplasty, was considered advisable because of the excellent chance that the epilepsy could be controlled medically. If, however, the attacks had begun more than 6 months after the injury or if the patient was first seen at a later time, it was thought wiser to await the results of a test of medical therapy before carrying out any surgical procedures. If the attacks persisted, as previous studies indicate they usually do, the cortex could be more extensively explored at the time the foreign bodies were removed and the skull defect was repaired.

If the patient's primary wound had healed by first intention, the cortical exploration was carried out whenever it was decided that medical management had failed. If the primary wound was infected, a cortical resection was not considered to be advisable until the wound had been completely healed and free of drainage for at least 6 months and preferably for a year.

Special Preoperative Examinations

When cortical excision was to be undertaken, it was considered advisable for additional psychologic examinations to be made before operation so that any alterations following operation might be more readily evaluated. Certain of the tests previously made were repeated, and a Rorschach examination was always carried out.

Accurate localization of the area of cerebral cortex giving rise to the convulsive manifestation in posttraumatic epilepsy was necessary for surgical treatment of the condition. Since it seemed possible that an epileptogenous focus might be more susceptible than normal cerebral cortex to a convulsant agent, an attempt was made to stimulate the focus selectively by altering the chemistry of the blood. An attempt was made to locate the focus without inducing a generalized seizure by using electroencephalographic recordings to determine the activation before clinical manifestations appeared.

Activated electroencephalography.—Electroencephalograms were made with electrodes placed conventionally upon the scalp, with additional electrodes about the site of the healed wound or cranial defect, and with at least one

electrode on a comparable position on the opposite side of the head. For monopolar recording, an electrode attached to the ipsilateral ear was used as a common lead. All anticonvulsant medication was eliminated for 3 days before the electroencephalographic recordings were made.

Hyperventilation.—It is well known that alterations of the acid-base equilibrium of the blood will accentuate or diminish the likelihood of convulsive seizures. The usual hyperventilation technique induces a buildup in 20 to 30 percent of epileptic patients. The attempt to measure the respiratory exchange was found to be too exacting for epileptics with neurologic disorders. In a group of seven patients, all of whom seemed willing to cooperate, an attempt was made to obtain standard hyperventilation at a rate of 15 cycles per minute, each cycle representing an exchange of 20 cc. of air per pound of body weight. Kymographic recording of the respiratory exchange showed that the individual patients achieved from 40 to 112 percent of the standard. In one patient (breathing 14.2 cc. of air per pound of body weight 15 times a minute), a Jacksonian convulsion developed during the hyperventilation. The other six did not have significant alterations in the electroencephalograms. Hyperventilation, therefore, did not seem to be a consistent, satisfactory method for activating an epileptic focus.

Forced hydration.—While clinical convulsions may be induced by forced hydration in 25 to 40 percent of individuals subject to epilepsy, it is a difficult procedure to control quantitatively. In 8 patients, 4 of whom had posttraumatic epilepsy and 4 posttraumatic encephalopathy without seizures, the hydration test described by Penfield and Erickson [6] was carried out after anticonvulsant drugs had been withheld for a few days. Under this regimen, such severe nausea and vomiting developed within 24 hours in all but one of the patients in the epileptic group that the test had to be discontinued. Gains in weight from 1.5 to 5.5 pounds were noted. None of the patients showed electroencephalographic changes, although one had a generalized seizure 2 hours after the test. Gastrointestinal disturbances did not occur in the nonconvulsive group, and none of the patients showed electroencephalographic alterations.

Alcohol.—Clinical experience shows that seizures are prone to occur a day or two after an epileptic has indulged in alcoholic beverages. In view of this fact, electroencephalograms were made on a group of 16 patients, 12 of whom were epileptic, after the rapid (within 2 to 3 minutes) intravenous administration of 40 to 150 cc. of 10 percent alcohol. The studies were made during administration, immediately after, and at intervals up to 72 hours after the alcohol had been injected. In the four instances in which blood alcohol levels were determined, they ranged between 100 and 150 mg. percent. In the nonepileptic group, the alpha rhythm became more prominent, and the amplitude of the previous abnormal activity decreased. All the patients

[6] Penfield, Wilder, and Erickson, Theodore C.: Epilepsy and Cerebral Localization. Springfield: Charles C Thomas, 1941.

showed clinical evidences of intoxication, although none were unable to walk. No clinical or electroencephalographic manifestations of epilepsy were observed.

Tridione (trimethadione).—Because grand mal had developed in some patients under treatment with Tridione for petit mal, 12 patients with posttraumatic epilepsy were tested with this drug; the doses, which ranged from 50 to 500 mg. each, were given variously from 1 to 3 times (a total of 26 trials). In no instance was there any appreciable effect on the electroencephalogram, but one patient, about 30 minutes after receiving the drug and after the electroencephalographic recording had been completed, had a focal epileptic attack which did not become generalized.

Electric shock.—Because electric shock has proved to be such a simple and relatively innocuous convulsant, it was used in a group of 11 patients with posttraumatic epilepsy. Seven patients were given 200 milliamperes for 0.05 to 0.15 second with a 60-cycle sine wave current. They had only startle reactions or mild tonic spasms at the instant of application of the stimulus. Four patients who received 2 to 5 milliamperes of square wave pulsating direct current for 2 to 3 seconds felt only a sensation of warmth. The electroencephalograph was turned off just before the shock and was turned on again immediately afterward. In no instance was there significant alteration in the electroencephalogram 5 seconds after the stimulus.

Penicillin.—Since penicillin had been shown to have a convulsive effect when applied to the cerebral cortex, 100,000 units were given intravenously to 5 patients, 4 of whom had organic brain disease. The electroencephalographic record was followed for 35 to 50 minutes. In no instance was any change noted.

Sodium cyanide.—Because the cytochrome-oxidase system is impaired in advanced paretics in whom convulsive manifestations are common and because cyanide is a specific poison for these enzymes, it was thought advisable to determine its effect upon patients with posttraumatic epilepsy. A 2-percent solution of sodium cyanide was therefore administered intravenously to 17 patients, 14 of whom had seizures. Three patients were given 0.3 mg. per kilogram of body weight, and the remainder were given 0.4 mg. per kilogram of body weight. One patient in the first group had no clinical or electroencephalographic reaction. In all the other patients, a respiratory gasp occurred 8 to 20 seconds after the injection, and there appeared a progressive slowing of the predominant activity of the electroencephalogram. In one patient, for example, the gasp occurred 20 seconds after the injection in the antecubital vein. The predominant rhythm in all leads 5 seconds later, was 8 per second; in another 5 seconds, it was 6 per second, and 8 seconds later it was 2 per second. Fifty seconds after the injection, widespread muscular twitching occurred throughout the body. Within 70 seconds after inhalation of two ampules of amyl nitrate, the slow waves had disappeared. In some cases, the head and eyes turned to one side without constant relationship to the site of injury. Occasionally, opisthotonos developed. The reaction to the cyanide was the same in patients with epilepsy and in those without it.

Acetylcholine.—Since it had been suggested that acetylcholine may be related to epileptic states, 18 patients, 15 of whom had epilepsy, were given 100 to 300 mg. of this drug at rates of injection varying from 20 to 60 mg. per minute. No change in the electroencephalogram was noted in any case except that in one instance, there was a decrease in amplitude of the waves for 2 minutes after the injection. The patients experienced a feeling of warmth and occasionally coughed, but they had no other clinical manifestation related to the injection.

Metrazol (pentylenetetrazol).—It had been known for some time that clinical seizures could be induced in patients subject to epileptic attacks by a smaller dose of Metrazol than that needed to cause convulsions in normal individuals. For activation of the epileptogenous focus, Metrazol, usually in 2-cc. doses (200 mg.), was injected as rapidly as possible into the antecubital vein while an electroencephalogram was recorded (fig. 55). The needle was left in place, and as soon as alterations were seen in the tracing, which was usually within 30 to 60 seconds, phenobarbital sodium (0.26 gm.) was administered intravenously in an attempt to prevent the development of clinical convulsions. In one group of patients, the Metrazol was given by intramuscular administration, 0.6 mg. per kilogram of body weight being injected into the deltoid muscle. Phenobarbital sodium was given intravenously or intramuscularly as soon as changes were seen in the electroencephalogram, which was usually 3 to 4 minutes after the drug had been given.

In the majority of cases, the changes occurred predominantly or solely in the tracings from one region (localized electroencephalographic alterations). The results of Metrazol activation in tests of 97 patients with posttraumatic epilepsy are shown in the following tabulation:

Findings	*Patients showing these findings*[1]
Generalized electroencephalographic alterations	7
Localized electroencephalographic alterations	58
Localized and generalized electroencephalographic alterations	10
Generalized slow waves	5
Localized slow waves	33
Generalized spikes	6
Localized spikes	31
Generalized electroencephalographic seizures	6
Localized electroencephalographic seizures	18
Clinical generalized convulsion	14
Clinical focal convulsion	10

[1] Although a number of patients were activated more than once, in this table a positive result in any category is stated only once for each patient. Some patients had positive results on repetitions of the tests, which were sometimes carried out 4 times.

These alterations took the form of slow waves or humps with a frequency less than the alpha rhythm, or of single or multiple spikes, at times occurring rhythmically and giving the pattern of a localized electroencephalographic seizure. Abnormalities present before activation were usually aggravated by the Metra-

zol. Such localized electroencephalographic alterations occurred in 60 percent of 97 patients tested (fig. 55).

In a smaller number of cases, generalized electroencephalographic alterations consisting of single or multiple slow waves or spikes were present simultaneously in tracings made from several parts of both sides of the head. In

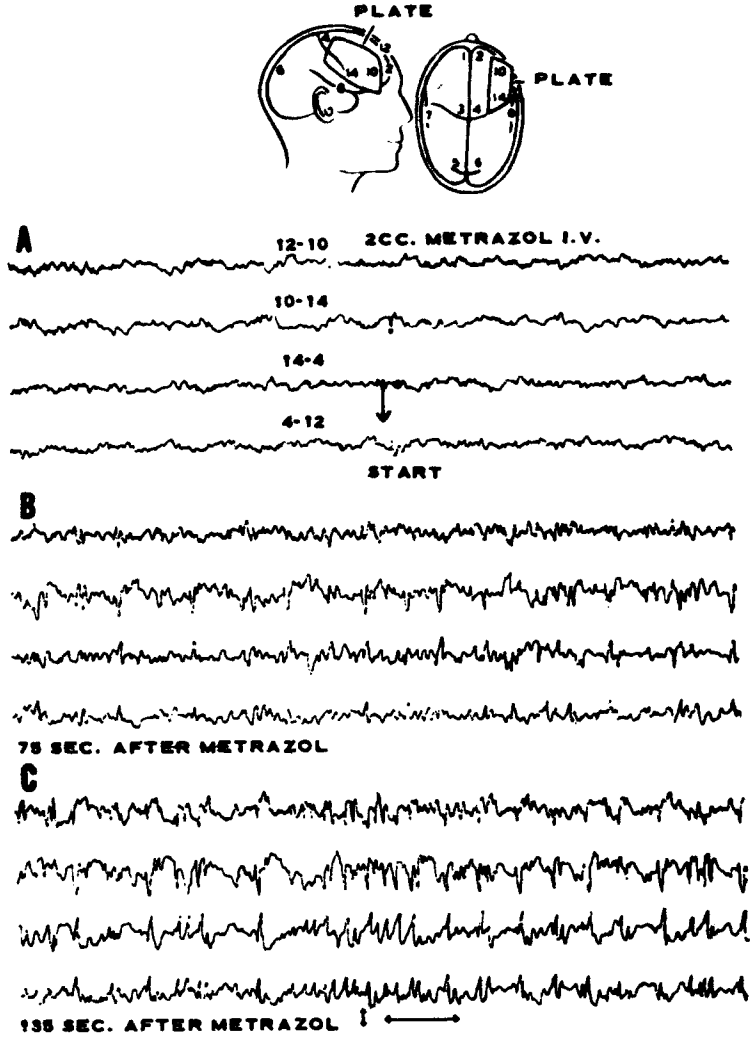


FIGURE 55.—Electroencephalogram taken during Metrazol activation. The electrodes were placed on the scalp about a plate in the right frontal region. A. Record taken during intravenous administration of 2 cc. of Metrazol (200 mg.). B. Spiking seen in all records 75 seconds after injection, most pronounced and out of phase in tracings from electrode 14. C. Spikes of greater amplitude 135 seconds after administration of Metrazol.

some instances, the rhythmic waves and spikes occurred simultaneously in all parts of the head recorded, giving the pattern of a generalized electroencephalo graphic seizure.

In a small number of cases, the patients experienced sensory or motor phenomena such as they usually had at the onset of their spontaneous seizures.

In approximately half of these cases, the attack consisted only of this aura. In the remainder, the attack began as, or developed into, a generalized seizure involving all parts of the body and associated with loss of consciousness.

The seizures induced by Metrazol had the characteristics of the patient's ordinary attacks. The fit was ushered in by the same aura, progressed in the usual order, and was associated with identical motor phenomena. The severity of the seizure was comparable to that of the spontaneous convulsion. There was, however, no relation between the frequency and number of attacks which the patient had had and their occurrence on Metrazol activation.

Because a single Metrazol activation gave positive localizing electroencephalographic findings in less than half of the cases studied, examinations were repeated on a number of patients whose primary examination had been negative. About a third of this group had localized electroencephalographic alterations on the second test. Reexamination was also done on a number of patients who had had focal electroencephalographic alterations on the first test. Although the majority again had focal abnormalities, some showed no electroencephalographic alterations on the second test. It was therefore concluded that focal activation was not consistently found in the same patient on repeated examinations, although, when it was present, the focus appeared to be at the same locus.

If the activation was carried out while the patient was taking anticonvulsant medication, the incidence of activation was lower. In a series of 40 such activations, 3 patients (7.5 percent) showed generalized electroencephalographic changes, and 15 patients (37.5 percent) showed localized alteration, but none of the patients in this group had clinical convulsions.

In a group of 11 patients, 6 mg. of Metrazol per kilogram of body weight were administered intramuscularly. Only 1 patient had a clinical generalized seizure, and only 2 showed localizing electroencephalographic changes. After 9 of the 11 patients in this group were given 2 cc. of Metrazol intravenously, all showed focal electroencephalographic alterations. It would seem, then, that, though the intramuscular method of administration of Metrazol was less apt to produce clinical seizures, it was much less efficient in activating an epileptogenous focus.

On the whole, activated electroencephalography by Metrazol was concluded to be a useful diagnostic procedure and of real value in localizing an epileptogenous focus.

Pneumoencephalography.—In order to ascertain the amount of brain substance lost and the degree of ventricular distortion, pneumoencephalograms were made routinely before a cortical exploration (table 14). One hundred and nine patients were thus studied from 3 months to 3 years after the head injury. A few patients had more than one pneumoencephalographic study. In the entire group, only 14 pneumoencephalograms were reported as normal. Four of these patients had closed injuries of the head. In only three patients did the ventricular system fail to fill. In 9 patients, 3 of whom had closed injuries of the head, the ventricles were symmetrically dilated. The pneumoencephalo-

TABLE 14.—*Pneumoencephalographic observations in 109 patients with penetrating wounds of the head*

Series	Normal findings	Nonfilling of ventricles	Unilateral ventricular dilatation	Bilateral ventricular dilatation	Bilateral ventricular dilatation with outpouching
	Percent	*Percent*	*Percent*	*Percent*	*Percent*
Posttraumatic epilepsy series	12. 8	2. 8	18. 3	8. 2	57. 9
Nonselected series [1]	27. 2	5. 8	29. 0	16. 5	29. 0

[1] Troland, G. E., Baxter, D. H., and Schatzki, K.: Observations on Encephalographic Findings in Cerebral Trauma. J. Neurosurg. 3: 390–398, September 1946.

grams of all patients suffering from closed injuries of the head were either normal or showed symmetrical ventricular dilatation.

The most common finding in cases of penetrating head injuries was a bilateral ventricular enlargement with an outpouching at the site of the skull defect (fig. 56). This finding was present in 63 patients. In 12 patients, unilateral ventricular enlargement with an outpouching was present on the side of the lesion, and in 8 patients, a simple unilateral ventricular dilatation was present on the side of the lesion (fig. 57).

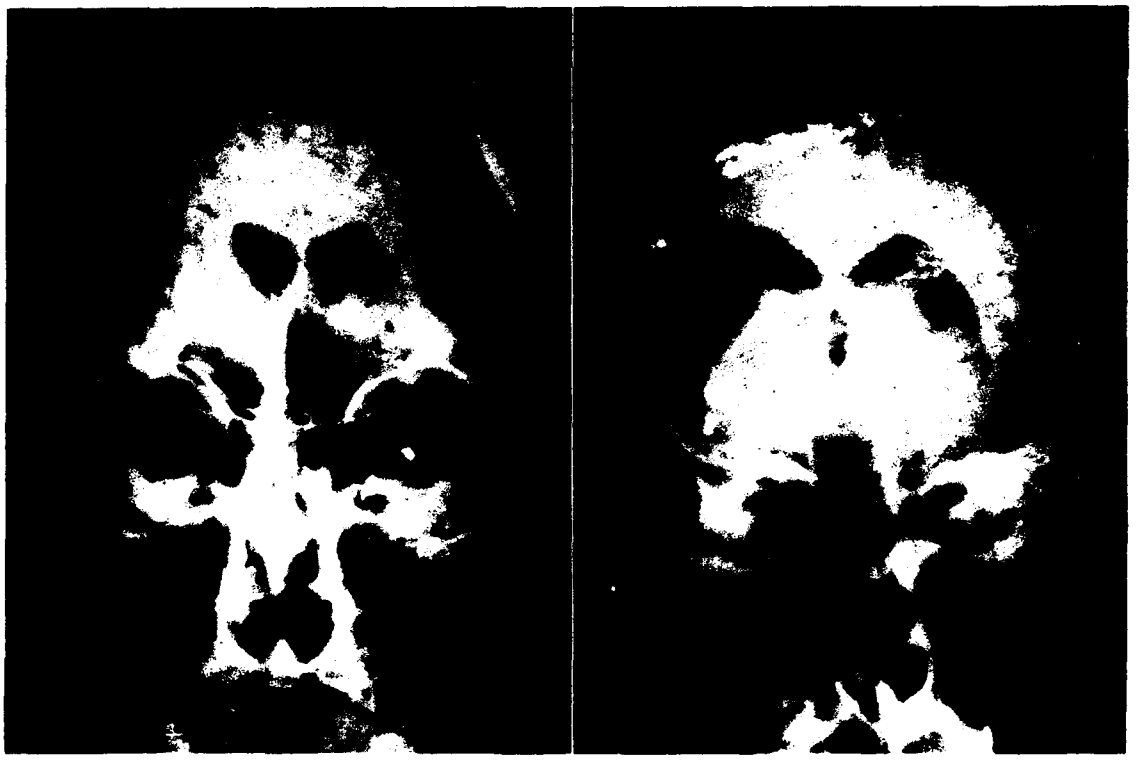


FIGURE 56.—Pneumoencephalograms of patient with posttraumatic epilepsy, showing mild generalized ventricular dilatation, somewhat more pronounced on left side, and retraction of entire system to left. The enlargement of the third ventricle is well demonstrated. A. Anteroposterior view. B. Posteroanterior view.

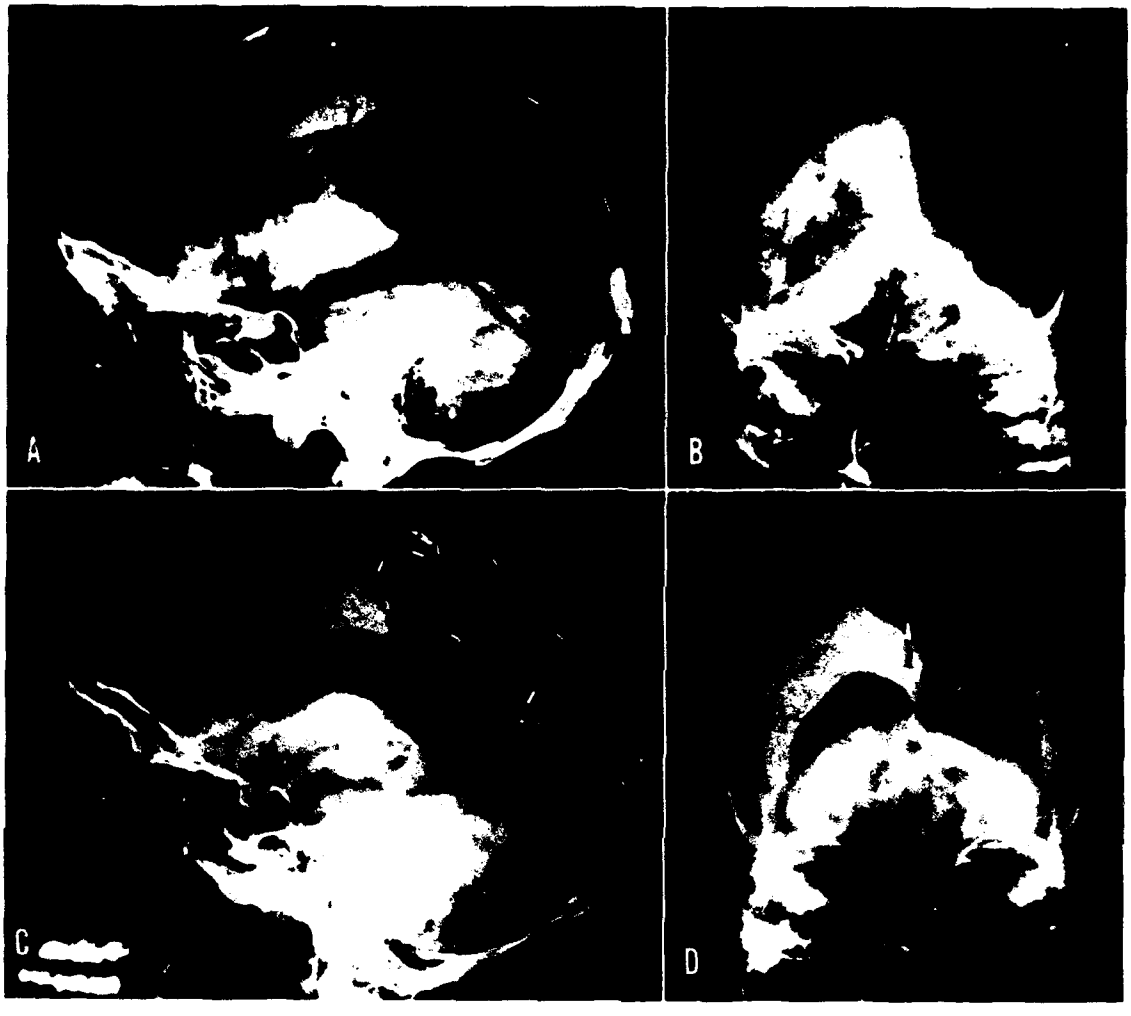


FIGURE 57. Lateral (A, C) and posteroanterior (B, D) pneumoencephalograms of patient suffering from posttraumatic epilepsy, showing enormous right trigonal outpouching at site of cranial defect and appearance of ventricular system after resection of scar to ventricle (C and D).

These ventriculographic changes are not peculiar to patients with posttraumatic epilepsy. Practically the same findings were present, in fact, in a nonselected group of patients with head injuries reported by Troland and his associates.[7] The incidence of severe ventricular distortion was, however, higher in this series than in the control series studied at Cushing General Hospital.

The subarachnoid space was frequently poorly demonstrated, especially on the side of the lesion, but cysts or dilated sulci were occasionally visualized. An asymmetrical filling of the subarachnoid spaces was considered an indication of arachnoidal proliferation.

In many patients, especially those with considerable internal hydrocephalus, the air remained in the ventricular system for as long as 2 to 3 weeks before absorption. This observation was demonstrated in roentgenograms made at

[7] Troland, C. E., Baxter, D. H., and Schatzki, R.: Observations on Encephalographic Findings in Cerebral Trauma, J. Neurosurg. 3:367-388, September 1946.

intervals after pneumoencephalography and by clinical auscultation of the shaken head; the splash of the water was quite audible until the air was absorbed.

Dye (phenolsulfonphthalein) was injected into the lateral ventricles of eight patients, and the rate of excretion in the urine was determined. Almost all seemed to have slow rates, but, since normal patients vary so greatly, it cannot be stated with certainty that this phenomenon indicated an impairment of the absorbing mechanism.

Preoperative Medication

Because the majority of patients with posttraumatic epilepsy had had infected wounds of the scalp and brain, prophylactic chemotherapy was given before secondary craniotomy. Sulfadiazine (2 gm. every 4 hours) was given for 24 hours before operation, and penicillin (30,000 units every 3 hours) was given for 12 hours. This regimen was continued for 5 days after operation.

ANESTHESIA

Intravenous Pentothal Sodium (thiopental sodium) was used in the first craniotomies for posttraumatic epilepsy, but the cortical activity was observed to be markedly depressed. Afterward, therefore, local anesthesia was secured by infiltration of the scalp with 1 percent procaine. Morphine (0.015 gm.), given as preoperative medication, did not appear to affect the cortical activity. It is a tribute to the determination and stamina of these patients that under these circumstances, only one lost his courage during the course of the operation, although the procedure occasionally lasted 5 hours.

TECHNIQUE OF OPERATION

Incision

The site of the epileptogenous focus, the size and shape of the skull defect, and the extent and position of scalp scars were important considerations in planning the incision. Frontal foci were exposed adequately by a bifrontal incision behind the hairline, which left a perfect cosmetic result. For central, parietal, temporal, or occipital exposures, a horseshoe-shaped incision was used unless a previous incision prevented it.

A skin flap was not reflected across a previous scar for fear of ischemic necrosis of the distal scalp. When a scar passed transversely in the temporal region, it was necessary to use an S-shaped scalp incision, and to reflect two flaps, in order to gain sufficient exposure. If a previous horseshoe-shaped incision had been made but was too small to provide adequate exposure, the anterior or posterior limb was carried farther out, leaving at least 3 cm. of scalp between the two incisions to prevent ischemic necrosis. If a tripod scar was present, it was converted into a modified horseshoe incision.

Incisions circumscribing cranial plates were preferred to those passing over the plate. It was thought that slight tension on the suture line due to previous loss of scalp substance might cause the incision to disrupt and expose the plate. If the incision skirted the plate, granulation tissue from the periosteum would soon fill in the gap, and there would be less danger of losing the plate.

If the skull defect was small, the bone flap was planned to incorporate the entire defect. If the hole was large, an appropriate flap was made anterior, posterior, superior, or inferior to the defect, to expose the previously determined epileptogenous focus. The flap was hinged, if possible, by temporal muscle.

Two-Stage Surgery

Usually, the craniotomy and cortical exploration were done in one stage. If, however, the epileptogenous focus lay near the midline, it was considered wisest to expose the cortex in two stages. In one patient with a focus near the midline in the frontal region, status epilepticus developed during a cortical exploration. Bleeding from openings into the longitudinal sinus due to avulsion of pacchionian granulations assumed alarming proportions and could not be controlled during the seizure by fibrin foam or manual compression.

A two-stage operation was therefore devised to minimize the future development of such a serious complication. At the first procedure, the bone flap was turned down and all bleeding from the longitudinal sinus was controlled. Tantalum foil or plate was laid over the sinus, so that a secondary craniotomy could easily be done without causing further hemorrhage. The two-stage procedure had the added advantages that the first stage could be performed under general anesthesia and that the discomfort of the patient at the second operation was greatly shortened.

Exposure of Field

If a plate was present in the cranial defect, it was inspected for evidence of infection as soon as the scalp flap was reflected. In many cases, a small amount of clear fluid was present between the scalp, the dura mater, and the plate. The thin membrane over the plate was usually white, smooth, and glistening, but if infection was present the membrane appeared yellow and granular. Yellow fluid about the plate suggested infection. If there was any question, a piece of the membrane was removed immediately for bacteriologic study. At times, organisms were found on smears, although cultures remained sterile probably because of the inhibitory action of the preoperative chemotherapy.

If a plate was not present, the dura mater was separated from the margin of the defect and from the scalp over the defect to prevent lacerations of the brain when the bone flap was lifted.

After reflection of the bone flap and control of all bleeding, the cortex

was exposed by incising the dura mater and carefully elevating it from the underlying arachnoid to avoid tearing fine adhesions and causing subpial bleeding. In many cases, a subdural membrane was found firmly adherent to the dura mater. If the meningocerebral scar was small, the dura mater was separated from it by sharp dissection. If the scar was extensive, the dura mater was cut only from the margin adjoining the suspected epileptogenous focus. The exposed brain tissue was carefully inspected and photographed so that, in case subsequent operative procedures had to be undertaken, the area would be objectively recorded. A rough sketch of the exposed area was found useful for orienting points of stimulation.

Appearance of brain.—In many cases, when the dura mater was opened, a subdural membrane firmly adherent to the dura mater was encountered (fig. 58). In some cases, the membrane was 2 to 10 mm. in thickness and quite extensive, overlying as much as half of a hemisphere. In other cases, the membrane was of filamentous thinness. The meningocerebral scar, whether small or large, was firm and tough and had to be cut by sharp dissection. The adjacent brain appeared normal in some cases, but frequently it was yellow

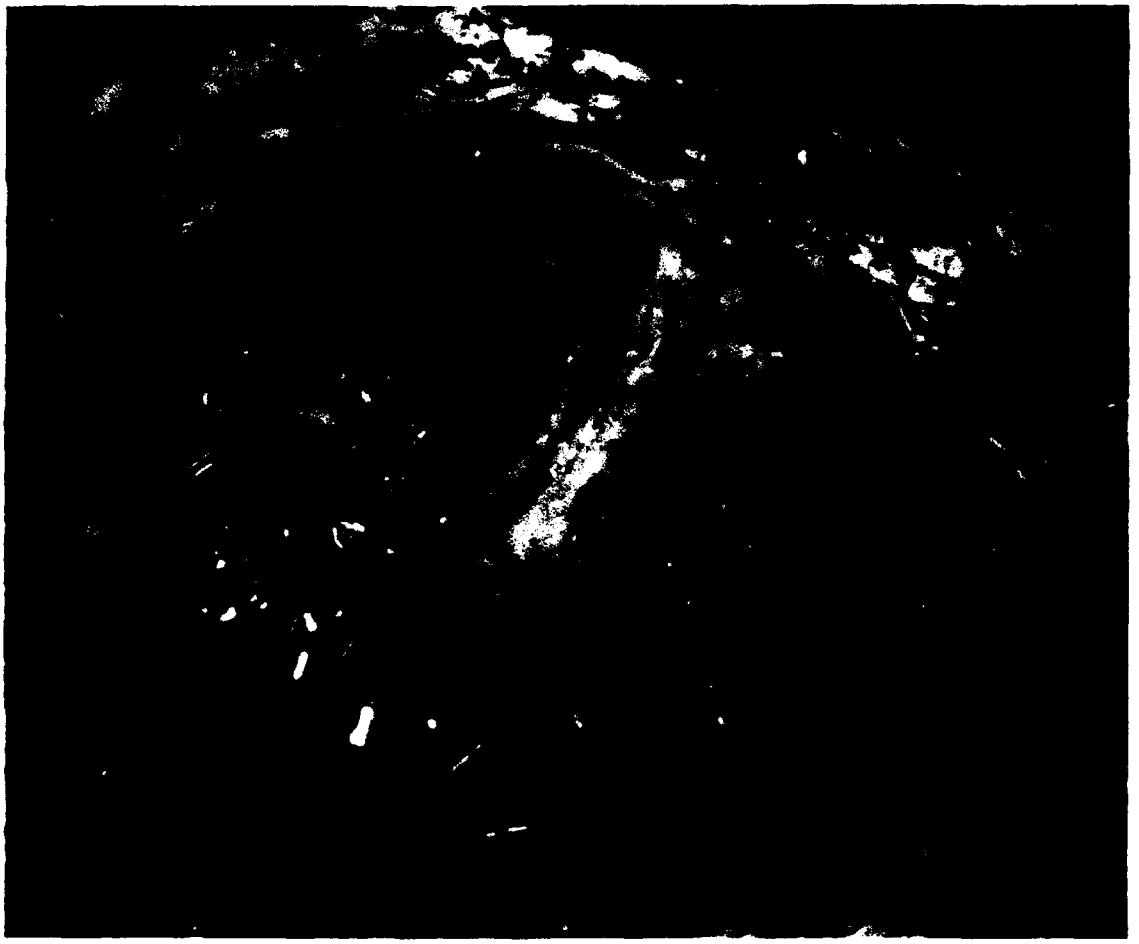


FIGURE 58.—Exposed cortex showing severe arachnoidal proliferation adjoining meningocortical scar.

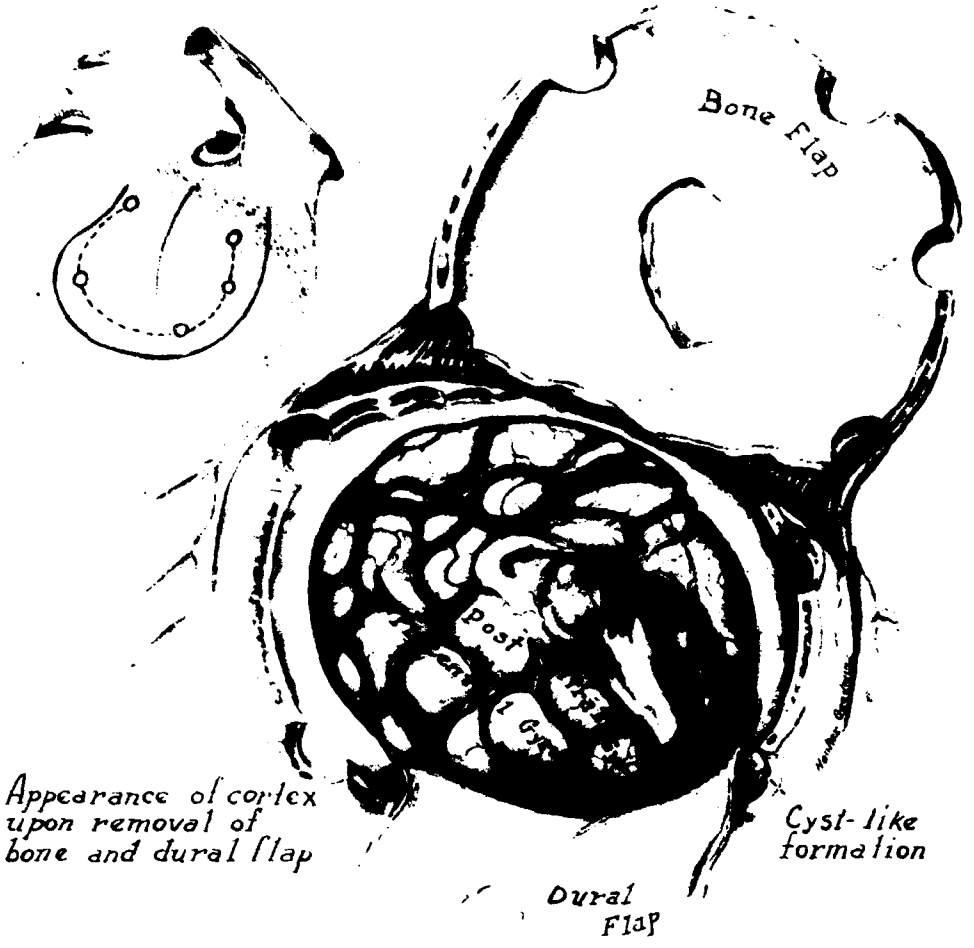


FIGURE 59.—Exposed cortex showing arachnoidal proliferation with cystlike formation at site of meningocortical scar.

or brown, softened, and cystic. The arachnoid was often thickened and translucent and occasionally contained white calcified plaques. Because of atrophic gyri, arteries and veins, which are usually concealed in sulci, appeared on the surface of the brain (fig. 59). Traction of the scar produced distortion of superficial channels (fig. 60).

Studies on Cortex

An electrode holder, devised by Capt. Curtis Marshall, MC, was used to place leads on any portion of the exposed cortex and to group as many as 10 leads about a small focus (fig. 61). For routine examinations, the leads were placed at regular intervals over the entire cortex. The cortical activity was usually tested, 8 points at a time being led into the 4 channels of the electroencephalograph. If an area of abnormal activity was found, this focus was more closely analyzed by grouping the electrodes about the abnormal point and using serial recording (fig. 62).

Electrocorticograms.—Slow wave foci were present in practically all cases, but they were not considered as of localizing value so far as the epilepsy was concerned. Spontaneous focal electrical alterations such as isolated spikes, bursts of spikes, or spiky waves (epileptic manifestations) were present in 14 of 39 cases thus examined. These foci were adjacent to, but not in, the cerebral scar. They were usually confined to an area of cortex not more than 3 to 4 sq. cm. and were often confined to one convolution.

Stimulation of cerebral cortex and electrocorticographic activation.—Three types of electric stimulators were tried. The ordinary sine wave 60-cycle



FIGURE 60.—Cerebral cortex adjacent to large scar which has caused traction on sylvian vessels.

stimulator with an output voltage from 0.1 to 20 volts proved quite satisfactory. A stimulator having a variable saw-tooth form (kindly loaned by Dr. Percival Bailey, Illinois Neuropsychiatric Institute, Chicago, Ill.) gave adequate results. Because the electroencephalograph was used immediately after the stimulation, a direct pulsating current stimulator, such as the square wave stimulator of Pollock, which caused polarization of the tissues blocking the amplifiers, was not satisfactory.

After the spontaneous activity of the cerebral cortex was tested, an attempt was made to induce the focus to fire artificially. Because of previous experiences (p. 297), Metrazol was tried as an activator, but the fear of inducing a

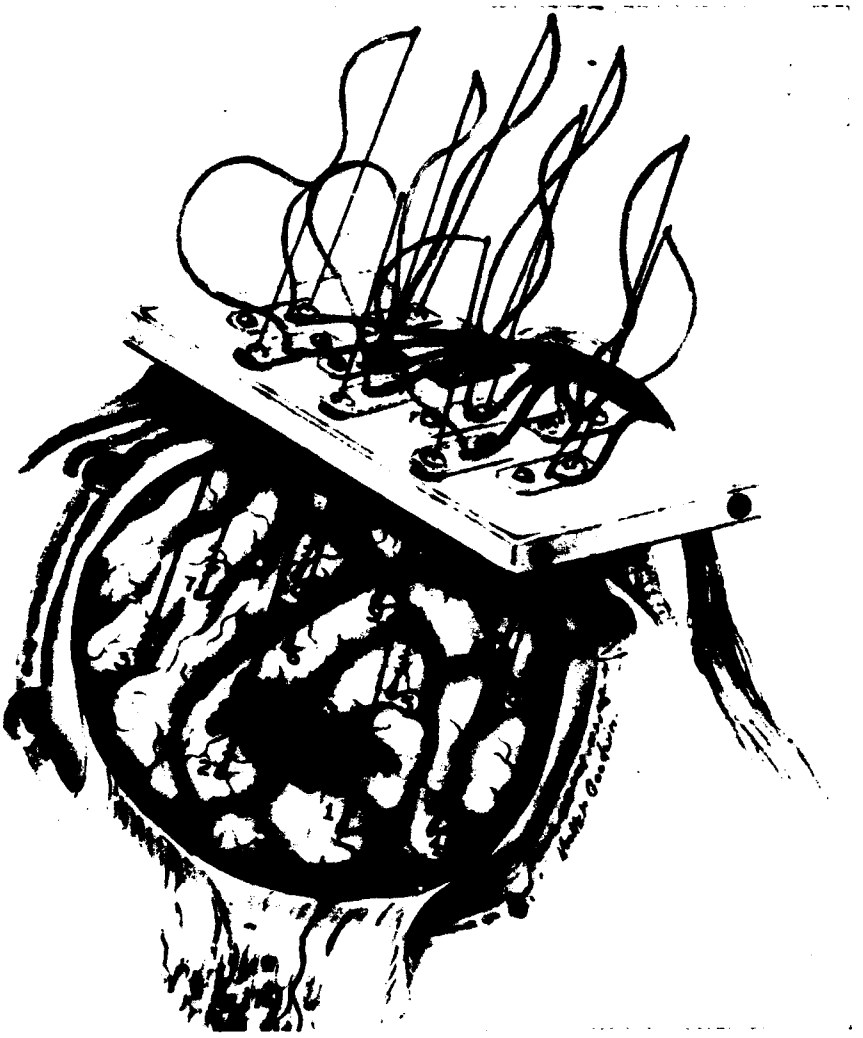


FIGURE 61.—Electrode holder clamped to margin of calvarium with electrodes placed upon cortex for recording.

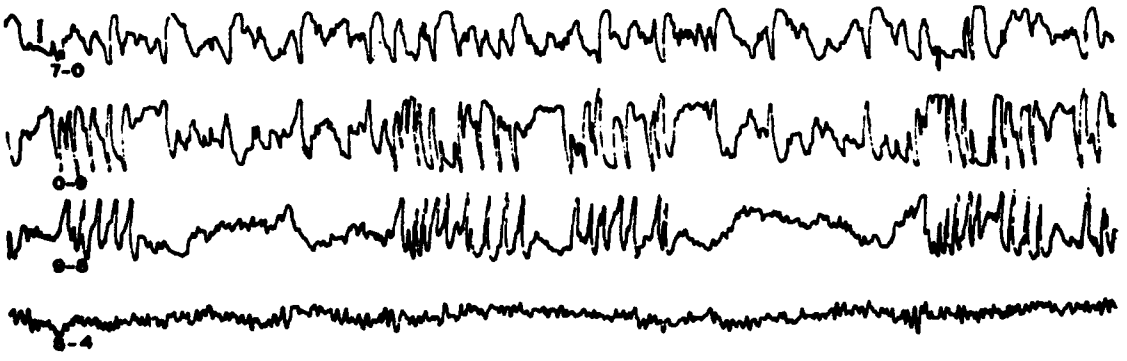


FIGURE 62.—Spontaneous electrocorticograms of epileptogenous foci in right inferior frontal region. The record shows out-of-phase spiking in the first three leads, indicating two foci, one at point 0 and one at point 9.

generalized seizure, as well as the ease of activating the cortex by electrical stimulation, led to abandonment of the method.

The usual method of electroactivation consisted of serial stimulation of representative points of the cerebral cortex by means of a sine-wave current from 1.5 to 2.5 volts for 5 seconds, followed by observation of the electrical activity of the cerebral cortex, the motor responses, and questioning of the patient regarding induced sensory phenomena. If no change was observed in the electrocorticogram after 1 minute, another point was stimulated, and so on until the entire cortex had been mapped (fig. 63A). An afterdischarge

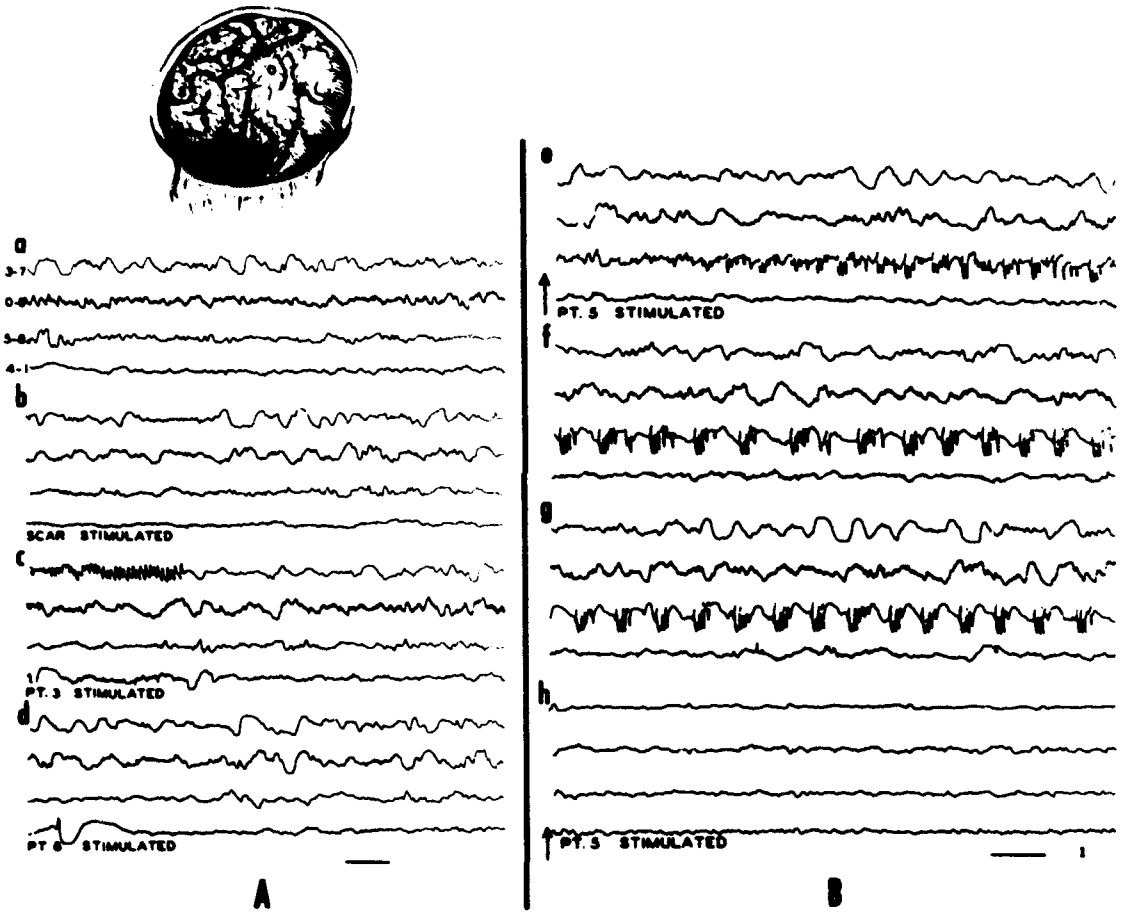


FIGURE 63.—A. Electrocorticograms in case of psychomotor and grand mal epilepsy: a. Spontaneous electrocorticogram from points indicated; b. record taken after stimulation of scar (2 volts sine wave current for 5 seconds) (electrocorticogram has remained normal); c. record taken after stimulation of point 3 (note slight afterdischarge in the first tracing); d. record taken just after stimulation of point 8. B. Record taken after stimulation of point 5: e. Afterdischarge in third tracing; f. record of same area 1 minute later; g. record of same area 2 minutes later (the patient, who was operated on under local anesthesia, felt quite normal. The triangular gyrus was removed by subpial dissection); h. record after removal of triangular gyrus and stimulation of same area with 2 volts for 5 seconds. There is no afterdischarge, nor could any be obtained by stimulation of the adjacent cortex. The horizontal line at the base indicates an interval of 1 second, the vertical line a calibration of 50 microvolts.

would occasionally occur for a few seconds at the point stimulated, but this phenomenon was considered within physiologic limits. Generally, however, an area of cortex adjacent to the scar could be found which, on stimulation, caused a long-lasting spiky afterdischarge (fig. 63B). This epileptic discharge might persist for as long as 30 minutes. In many cases, the sensory or motor aura which preceded the patient's spontaneous attacks was associated with this afterdischarge. Occasionally, the electrocorticographic attack would progress to a clinical seizure, but in some cases the patient did not have any motor or sensory concomitants during the electrocorticographic discharge. Of 29 patients in whom such activation was used, 11 had no clinical phenomena associated with the afterdischarge. The remainder had sensory or motor focal manifestations which, in seven patients, progressed to a generalized seizure. Most of the 11 patients who had no motor or sensory phenomena associated with the epileptic afterdischarge had had no aura with their spontaneous attacks.

Epileptogenous foci localized in this manner were adjacent to the cerebral scar in 18 cases and were in a gyrus slightly removed from, but parallel to, the scar in 4 instances. In eight cases, the foci were separated from the scar by one or two nonepileptogenous convolutions. The extent of the focus could be determined by triangulation or push-pull arrangement of electrodes. The foci activated by electrical stimulation were located at approximately the site predicted by the electroencephalogram recorded after Metrazol activation. In those cases in which spontaneous epileptic electrocorticographic alterations were present in one area of the cortex, electroactivation brought out a long afterdischarge from that cortical focus.

Multiple epileptogenic foci were present in very few cases, but occasionally a focus migrated from one cortical area to another within a period of minutes. On one occasion, the epileptogenous area wandered around a scar through an arc of 180° within a period of 28 minutes.

The threshold of excitability appeared to be fairly constant, but it was elevated for 1 to 4 minutes after a long afterdischarge. The activation usually occurred at the site of stimulation, but, in 15 percent of the cases, epileptic activity was initiated in adjoining gyri, which were sometimes removed as much as 2 to 3 cm. from the point of stimulation.

These observations seemed to indicate that certain cortical areas adjacent to cerebral scars could be excited by the production by normally subliminal stimuli of a local state of hypersynchrony characterized by epileptic electrocorticographic manifestations. Although these phenomena had many of the characteristics of cortical afterdischarge, their lower threshold, more localized activity, and longer duration suggested that they were different at least in degree That areas capable of such activity were potentially epileptogenous seemed highly probable. That they were the foci from which spontaneous epileptic attacks originated lacked demonstration, though this seemed a reasonable inference in those instances in which the electrocorticographic attack was associated with an aura identical to that in the spontaneous attacks. If the focus was

some distance from the scar, the suspicion was entertained that another, and perhaps primary, focus was located nearer the injury.

By the use of this technique, it was possible in many cases to locate both the motor cortex and an epileptogenous focus without inducing a generalized seizure. Without the aid of the electrocorticogram repeated stimulations might have precipitated a seizure as the result of facilitation of repetitive excitations. If a generalized seizure occurred during exploration of the cortex, it was usually difficult to locate an epileptogenic focus because of the marked depression of cortical activity which persisted for hours after a particularly severe seizure.

It was often difficult to find the precentral cortex by stimulation techniques in patients suffering from posttraumatic epilepsy. Many had a hemiparesis due to a subcortical lesion, in which event little response could be expected from cortical stimulation. As cortical reorganization and recovery proceeded, the excitability of the cortex probably increased. The longer the time after injury, therefore, the better were the responses induced by electrical stimulation.

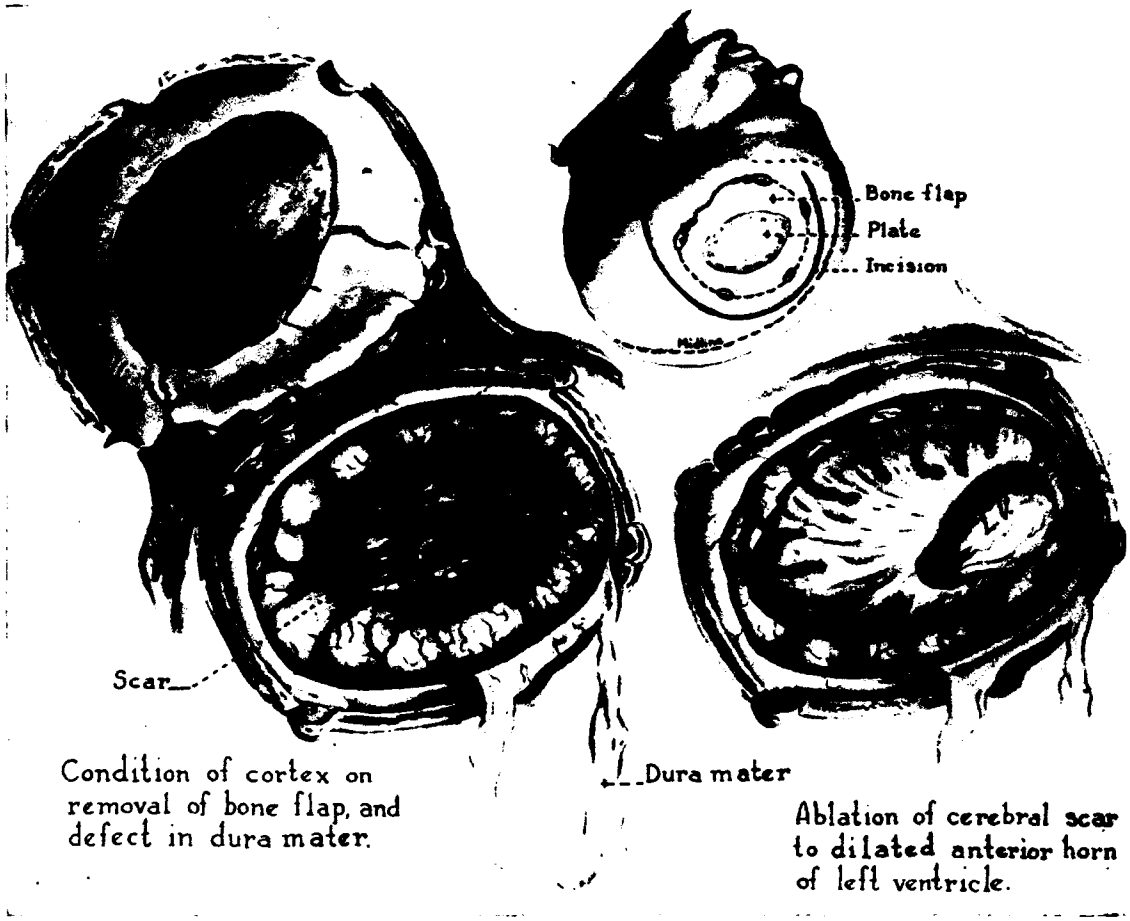


FIGURE 64.—Surgical field showing extensive left frontal cerebral scar containing bone fragments. The scar was removed to the ventricle.

In those cases in which the clinical aura and electrocorticographic manifestations of an attack could be simultaneously induced, it seemed probable that the stimulated focus was responsible for the patient's spontaneous attacks. In those cases in which only the electrocorticographic epileptic manifestations could be elicited, the evidence that the focus stimulated was responsible for the attacks was not conclusive. There was no doubt, however, that such areas were functioning abnormally and were potentially, if not actually, epileptogenous.

Surgical Removal of Scar and Focus

After the epileptic focus had been located and its extent determined by a secondary application of the electrodes about it, it was removed by one of several techniques, as follows:

1. The focus and the scars were excised down to the ventricle (fig. 64).
2. The focus and the scar were removed down to white matter (fig. 65).
3. The focus was ablated by subpial dissection (fig. 66).

Several considerations determined which of these techniques would be employed. If there were foreign bodies in the scar, the scar and the focus

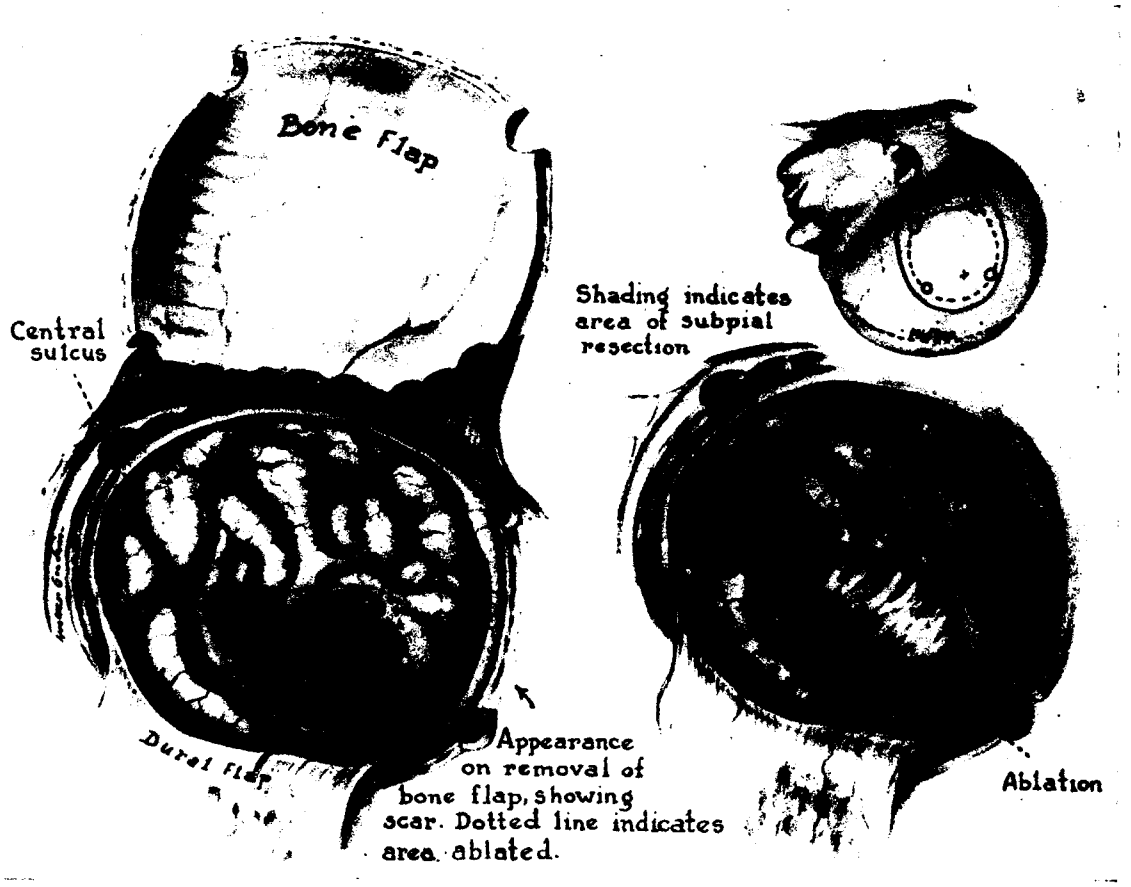


FIGURE 65.—Surgical field before and after removal of scar and epileptogenous focus. The wound was a bullet "crease" of the parietal bone, without fracture of the bone. In spite of the absence of penetration, a large scar was present in the parietal region.

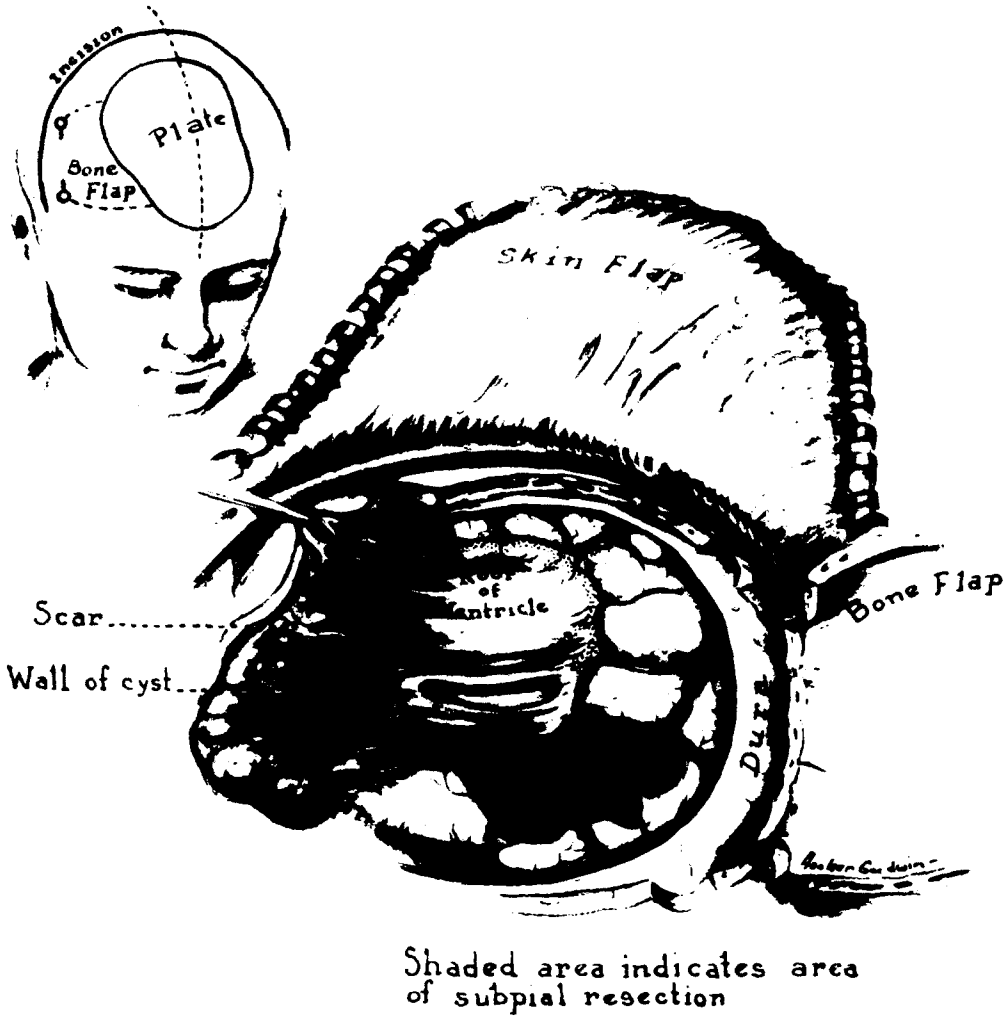


FIGURE 66. Surgical field showing removal of large scar containing cystic areas near midline of right hemisphere. An epileptogenous focus removed by subpial dissection is shown by dark shading.

were excised. The danger of complications was lessened if the ventricle was not opened. If the scar was large and adjacent to the motor area, extensive resections were avoided because of the fear of exacerbating a paralysis or an aphasia. Irrespective of the procedure carried out, the blood supply to the remaining cerebral cortex was disturbed as little as possible.

A pia-arachnoid incision was made in the avascular portion next to the scar, and the involved gyrus was removed completely from trough to trough (fig. 67). After removal of the focus, the electrodes were placed upon the adjacent cortex to check the completeness of the extirpation by stimulation with the same strength of current used previously. In some cases, it was found that the cortex adjacent to the focus gave rise to a convulsive pattern. In some cases, before all convulsive activity could be eliminated, a rather extensive subpial dissection of several gyri running into the scar had to be

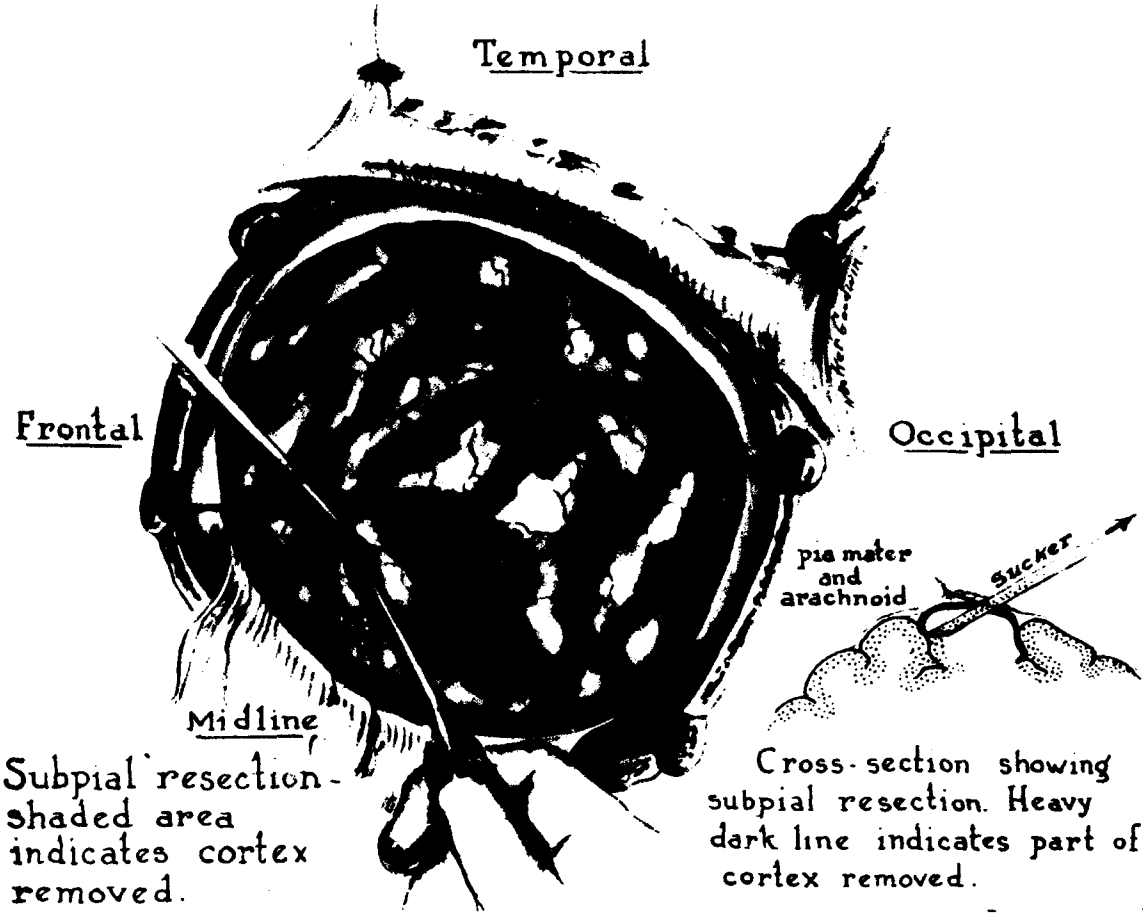


FIGURE 67.—Technique of subpial resection. The cortical ablation is carried to the depth of the sulcus.

carried out. During the ablation, speech and motor power were checked frequently. A defect in the dura mater was repaired by temporal fascia or pericranium.

After closure of the dura mater, the bone flap was replaced. If a tantalum plate formerly covered the defect, it was replaced by a Lucite plate to make subsequent roentgenograms of the skull more revealing (fig. 68).

PATHOLOGIC EXAMINATION OF EXCISED TISSUE

The microscopic examination of the tissues removed (fig. 69) showed that the cerebral scars consisted of connective and glial tissue with numerous capillaries and had a varying amount of inflammatory cellular reaction. Neurones were not present in the center of the scar. The scar did not seem to possess any histologic characteristics different from those of scars in nonepileptic individuals.

The removed epileptic foci, even when they appeared grossly normal, were in all instances composed of abnormal cortical tissue. The cytoarchitecture

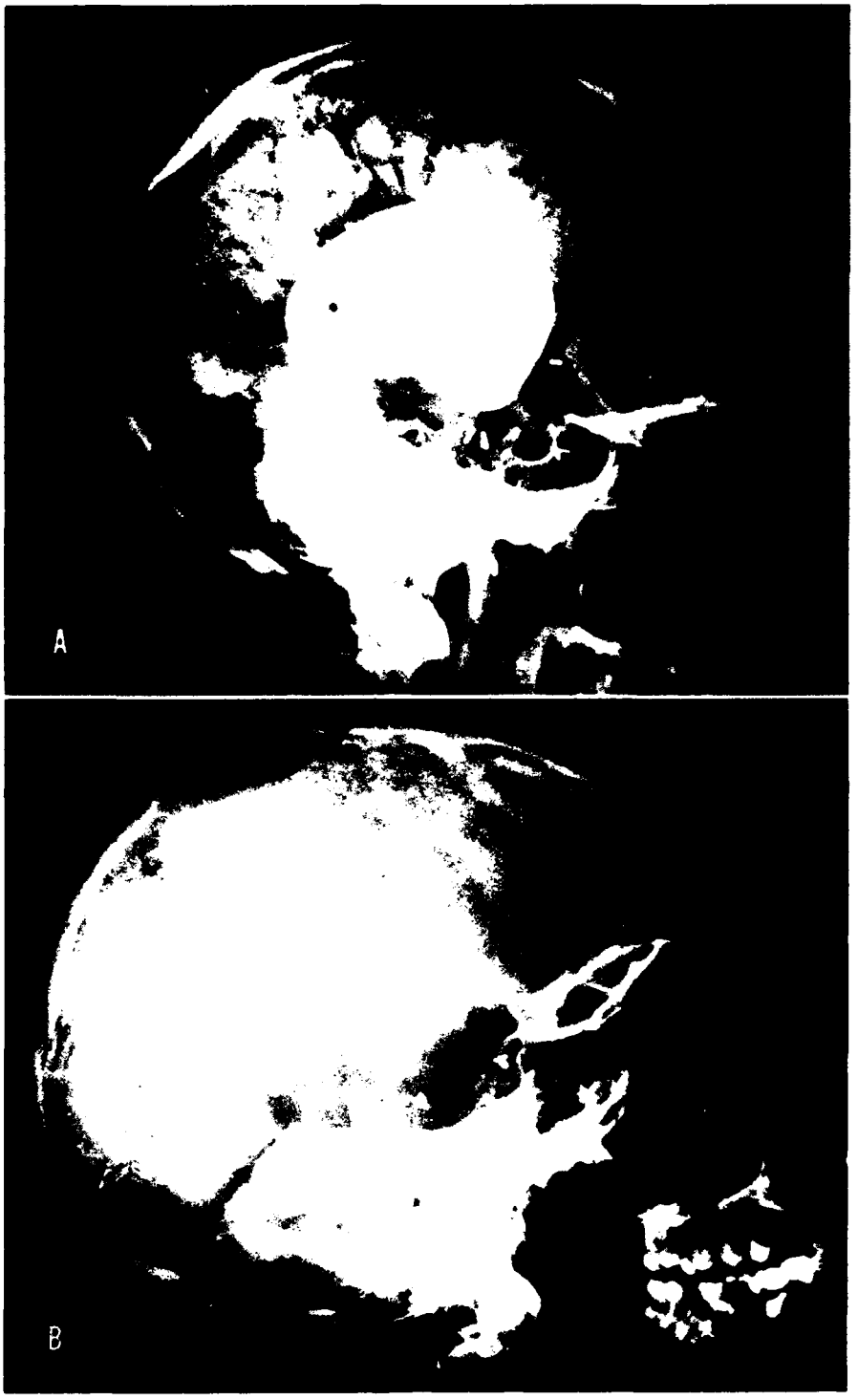


FIGURE 68. Roentgenograms of skull after cranioplasty. A. Obscuration of ventriculogram by tantalum plate. B. Transparency to X-rays of Lucite plate which replaced tantalum plate.



FIGURE 69.—Photomicrograph of cortex adjacent to scar showing invasion of cortex by reticular tissue. The arachnoid is markedly thickened over this area. (Perdrau preparation X.)

of the cortex was disturbed, and the neurones were decreased in number. Those present were dark staining, and the nucleus, nucleolus, and cytoplasm were distinguished with difficulty. Glial tissue was increased, and in some cases the connective tissue was also increased, particularly about capillaries. The arachnoid usually was thickened and occasionally contained chronic inflammatory cells.

POSTOPERATIVE CARE

The patient usually withstood the operative procedure well. In some cases, plasma and blood were given during the operation. If not, a transfusion was always started when he was brought back to the ward.

In some instances, the patient was anesthetized with intravenous Pentothal Sodium during the closure of the wound. As soon as he began to wake up, sodium phenobarbital (0.2 gm.) was given by hypodermic injection. Phenobartital (0.1 gm.) was then prescribed 3 times a day. On a few occasions, when orders for such postoperative medication were omitted, convulsions occurred within the first few days after operation. Apparently, although there was greater likelihood of attacks at this time, their occurrence was not an indication of therapeutic failure; after these few days, the patients might have no further attacks.

Fluids were limited to approximately 1,500 cc. per day for the first 3 or 4 days to prevent postoperative nausea and vomiting. The patient was allowed up as soon as he desired, which was usually by the third or fourth day.

Ordinarily, there were few problems following the craniotomy.

CRANIOPLASTY

Those patients who had a cranial defect but whose attacks were controlled by anticonvulsant medication required only a cranioplasty. Although sheet tantalum proved to be easily shaped, its radiopacity was undesirable, since epileptic patients may require repeated roentgenographic examinations. Acrylic plates, although more troublesome to form, were preferable to tantalum for this reason. The surgical technique of cranioplasty (p. 261) is so well standardized that no comment on it is required.

After operation, the patient was given phenobarbital, 0.1 gm. 3 times a day, to prevent the attacks otherwise likely to occur within a few days after operation. In a week, the medication was reduced to the preoperative level.

POSTOPERATIVE EXAMINATIONS

At the end of 1 month, and again at the end of 3 months after craniotomy, the patient had complete neurologic, psychologic, and electroencephalographic examinations. In general, his neurologic status was not appreciably altered by the operative procedure. Several patients had an increase in hemiparesis or aphasia lasting for a few weeks, but in most instances these disturbances returned to their previous level within 3 months. All patients were advised to continue anticonvulsant medication for 3 to 5 years.

It is recognized that the results of any procedure for the relief of convulsive seizures are usually good or excellent for the first few months, or even for the first few years, after operation. Immediate results suggest that surgical measures may have a limited place in the management of posttraumatic epilepsy which has not responded to medical measures (tables 15 and 16).

OCCUPATIONAL THERAPY AND REHABILITATION

A patient who was under treatment for posttraumatic epilepsy at Cushing General Hospital was rehabilitated as quickly as possible, in order to improve

his mental outlook. To this end, he was seen by a vocational counseling group, whose responsibility was to assess his individual problems and potentialities. A form was used for this purpose (appendix C, p. 420). Advice concerning future activities was predicated upon the results of this investigation, the Army Classification Test score, and psychologic examinations.

TABLE 15.—*Early results of cortical excision in 39 patients with posttraumatic epilepsy*

Results	Scar resection	Scar resection to ventricle	Excision of epileptogenous focus	Excision of subdural membrane	Total
No attacks for 3 months after operation	9	3	9	2	23
Attacks only during first 3 days after operation	0	2	2	0	4
Aura only	1	0	3	0	4
More attacks, much as before	0	2	6	0	8
Total	10	7	20	2	39

TABLE 16.—*Later results of cortical excision in 36 patients with posttraumatic epilepsy* [1]

Results	Scar resection	Scar resection to ventricle	Excision of epileptogenous focus	Excision of subdural membrane	Total	Control group [2]
No attacks	2	3	7	0	12	1
Aura only or 1 attack	1	1	4	1	7	2
More than 1 major attack	6	3	7	1	17	17
Total	9	7	18	2	36	20

[1] All investigations were carried out approximately a year after operation.

[2] This group consisted of patients who were not controlled by medical management and who, because of neurologic incapacity, infection, or personal prejudice, were not considered suitable for operation.

In some cases, training for a job was begun in the hospital, or high school credits were obtained for schooling carried out in the hospital while the patient was under treatment. In this way, his rehabilitation was started before he left the hospital. After discharge, he was assisted in obtaining suitable work. Many commercial firms had a sympathetic comprehension of the difficulties of these patients and were quite willing to employ them in certain positions. The social service organizations in the community, the American League Against Epilepsy, and the American Red Cross all assisted in occupational and social adjustments. Thus, for some patients, at least, the ultimate goal in the treatment of posttraumatic epilepsy—a return to a normal social and economic life—was successfully reached.

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CHAPTER XIV

Speech Disorders Resulting From Gunshot Wounds of the Head and Neck

William G. Peacher, M. D.

HISTORICAL NOTE

Rehabilitation of Army personnel with speech defects was carried out in World War I at a single center, General Hospital No. 11, located at Cape May, N. J.[1] Speech reeducation was given at this center to a total of 54 patients; the defects of 37 of them were subsequently classified in the following categories: Aphasia, 14; imperfect phonation, 13; stammering, 3; aphonia, 2; stuttering, 2; aphasia and stuttering, stammering and stuttering, and multiple neuritis, 1 each. When this installation was closed on 7 July 1919, facilities for therapy and the 20 patients still in the hospital were transferred to General Hospital No. 41 at Staten Island, N. Y. When the last report was made on the subject, 1 November 1919, 14 soldiers were still hospitalized with speech defects, but it was anticipated that their treatment would be completed by the end of the year.

Not a great deal of information exists concerning the incidence of speech disorders following craniocerebral injuries in World War I or concerning the method of treatment of these defects. Frazier and Ingham[2] reported in the official history of the Medical Department that among 200 patients with craniocerebral injuries observed at General Hospital No. 11, 16 had residual speech defects 6 months after injury. Ten of these defects were of the motor or dysarthric type, and three were of the sensory type, with alexia as the most prominent symptom. The remainder were of the mixed type. Improvement was noted in all, following speech reeducation. In 1919, Ingersoll,[3] in a paper concerning the management of wounds of the nose and throat on the otolaryngologic section at Base Hospital No. 11, stated that vocal therapy was beneficial in injuries of the jaw, pharynx, and larynx, but he gave no statistics.

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[1] The Medical Department of the United States Army in the World War. Washington: U. S. Government Printing Office, 1927, vol. XIII, pt. I, pp. 181–188.

[2] Frazier, C. H., and Ingham, S. D.: Neurological Aspects of the Effects of Gunshot Wounds of the Head. *In* The Medical Department of the United States Army in the World War. Washington: Government Printing Office, 1927, vol. XI, pt. 1, p. 801.

[3] Ingersoll, J. M.: Injuries of the Nose and Throat Due to Bullet and Shell Wounds Laryngoscope 29: 624–632, November 1919.

CONCEPTS OF SPEECH DIFFICULTIES EARLY IN WORLD WAR II

Between the World Wars, although considerable progress was made in the treatment of speech defects, the main emphasis was on the dysphasias. The study of all speech disorders was accelerated by the magnitude of the fighting early in World War II, as well as the large numbers of head and neck wounds, but considerable time passed before the full importance of the problem was appreciated and adequate methods of examination and treatment were evolved.

Until April 1943, no comprehensive program for the management of speech disorders existed in the United States Army. Several unofficial attempts, however, were made earlier. Medical officers interested in the subject treated functional disorders, such as stuttering, articulatory disorders due to poor habits, and speech defects due to foreign dialects. Darley and Spriestersbach,[4] for instance, reported improvement after only 3 weeks of individual and group therapy in 22 soldiers treated in the spring of 1942 at Fort Riley, Kans. A similar brief program was conducted at Fort Storey, Va., as part of the rehabilitation program, but detailed results are not available.

ESTABLISHMENT OF THE ARMY PROGRAM FOR THE CORRECTION OF SPEECH DEFECTS

Complete diagnostic and therapeutic facilities for soldiers with both organic and functional speech disorders were first set up under Army auspices on 7 May 1943 at Brooke General Hospital, Fort Sam Houston, Tex. It is fortunate that they were provided at that time, for casualties were just beginning to be received from the fighting in North Africa and in the Solomons. It is also fortunate that the speech clinic was attached to the neurosurgical service, since the majority of patients evacuated from overseas with speech defects were admitted to this section for cranioplasty and for treatment of various neurologic residuals. This clinic was operated until 22 February 1945, when the necessary facilities, including personnel and patients, were transferred to McGuire General Hospital, Richmond, Va.

In contrast to the centers later designated for speech training, the speech clinic at Brooke General Hospital remained attached to the neurosurgical service as long as it was in operation and was never made an adjunct of the neurologic service.

In April 1945, The Surgeon General took official cognizance of the need for a program of speech training for military casualties and delegated the responsibility to Army hospitals with specialized neurologic and neurosurgical centers. Speech therapy was made the responsibility of neurologic sections, following definitive surgery, and the patients were to be retained in hospital until maximum benefits had been achieved.[5] Speech clinics were developed in all but two

[4] Darley, F., and Spriestersbach, C.: War Notes. J. Speech Disorders 8: 189–190, June 1943.

[5] War Department Technical Bulletin (TB MED) 155, 25 Apr. 1945.

of the designated installations; patients assigned to those two hospitals were transferred to other installations in the same service command when they were ready for speech training.

MANAGEMENT OF SPEECH DISORDERS IN OVERSEAS THEATERS

The exigencies of modern warfare permitted no formal speech therapy in combat zones, and treatment was limited to the causative injury. Thorough debridement of all craniocerebral wounds and the institution of supportive therapy not only saved lives and decreased morbidity but also prevented complications which might subsequently have influenced the speech mechanism. Treatment of the speech defect at this time would not have been desirable because of the presence of an acute pathologic reaction, which usually took several weeks to subside.

No organized program for the correction of speech defects was developed in communications zones because of the necessity for early evacuation of patients for further specialized reconstructive therapy, including repair of scalp and skull defects and, as necessary, resection of cerebrodural and cortical scars. Facilities for detailed neurologic, neurosurgical, and psychologic investigation were frequently available; however, the pressure of hostilities, the ensuing casualty loads, and the need of definitive surgical treatment for such urgent conditions as hematomas, meningitis, brain abscess, and retained bone and metallic fragments, as well as the frequent necessity for redebridement, secondary closure of wounds, and skin grafting, made it impossible to undertake the prolonged studies and treatment necessary in speech defects.

In spite of the circumstances, however, interested fellow patients and Red Cross workers were able, in some cases, to begin the work of reeducation and psychotherapy by teaching the patients such familiar and useful words and sentences as "Hello," "Good morning," and "I am hungry, cold, tired." Some patients were also taught the alphabet and were taught to count before they reached the Zone of Interior.

ADMINISTRATIVE CONSIDERATIONS OF SPEECH CLINICS IN ZONE OF INTERIOR

Assignment to Hospitals

Patients evacuated to the Zone of Interior with speech defects were returned on the basis of their wounds rather than on the basis of residual speech disorders. In the Zone of Interior, they were assigned to hospitals on the basis of the major medical or surgical defect present, the speech disorder again being regarded as incidental. The assignment was, in general, as follows:

Neurosurgical centers received patients with (1) gunshot wounds of the head and neck which had resulted in cranial defects and such neurologic

residuals as hemiplegia, parietal lobe signs, convulsions, and visual field disturbances, and (2) cranial nerve injuries. Gunshot wounds of the head and neck were sometimes accompanied by dysphasia, dysarthrophonia, or both. Injuries of the facial (peripheral) nerve could be accompanied by dysarthria, injuries of the vagus (recurrent laryngeal) nerve by dysphonia, and injuries of the hypoglossal nerve by dysarthria.

Plastic surgery centers received patients with (1) maxillofacial wounds, including involvement of the tongue, lips, and floor of the mouth, which were sometimes accompanied by dyslalia; (2) gunshot wounds of the larynx, which were sometimes accompanied by dysphonia; and (3) palatal defects, which were likely to be associated with rhinophonolalia.

Neuropsychiatric centers received patients suffering from (1) anxiety states, sometimes accompanied by stuttering; (2) conversion hysteria, sometimes accompanied by aphonia and dysphonia; and (3) other neuroses and psychoses, sometimes accompanied by dyslogia.

Aural rehabilitation centers received patients suffering from deafness resulting from chronic middle-ear infection, heavy gunfire, and skull trauma, in which dysaudia was the usual speech defect.

At Brooke General Hospital, where the first speech clinic was established in World War II, the most common speech disturbances encountered as a result of gunshot wounds of the head and neck were dysphasia, dysarthria, dyslalia, and dysphonia. Some instances of aphonia and stuttering were on a functional basis and had resulted from exposure to enemy action. Remedial speech training, which was deferred until the completion of specific neurosurgical procedures, was carried out by speech pathologists and psychologists under medical supervision. All speech defects encountered were treated except such common functional defects as articulatory and vocal disturbances arising from poor habits, low educational training, and use of foreign dialects. Trained personnel were insufficient, and all facilities were devoted to patients whose speech disorders were on an organic rather than on a functional basis.

War Department Technical Bulletin entitled "Aphasic Language Disorders" (TB MED 155), published on 25 April 1945, recognized the need for therapy in the field of speech but contained recommendations for the management, study, and classification of only the dysphasias. The program in each designated center of treatment was made the direct responsibility of the medical officer locally in charge.

Function of Speech Clinics

The primary function of Army speech clinics was the diagnosis of speech disorders in military casualties and their reeducation, with the object of returning them to useful military duty or to civilian life after maximum benefits had been received from hospitalization for other disqualifying neurologic or physical sequelae of injuries or both. Every effort was made to restore the soldier to an adequate social, mental, and economic status in his community before he was

discharged from the hospital. A secondary purpose of the speech clinic was to disseminate information derived from experience by ward rounds, conferences, lectures, and publications, for the benefit of other centers and other patients.

Personnel of Speech Clinics

In each designated center for speech training, a psychologist, preferably with a background of training in speech, was assigned to the neurologic service to assist in evaluation of patients as well as in their reeducation. Except at Brooke General Hospital where a neurosurgeon was in charge, the director of each clinic was a neurologist whose responsibility it was to organize the program, maintain a close liaison with all personnel participating in it, and evaluate and interpret the results of all examinations so that correct treatment could be outlined. Teachers of speech were in short supply throughout the war, and no technical school for their training was ever developed. It was therefore often necessary for the neurologist in charge of a speech clinic to train the workers in the installation himself. These workers consisted of occupational therapists, American Red Cross workers, members of the Women's Army Corps, and similar personnel who were selected for the positions because of their previous background and their interest in the problem. Students and instructors in speech from adjacent colleges often volunteered to assist in the program, during the school year as well as in vacation periods.

DIAGNOSTIC AND THERAPEUTIC REGIMEN

Diagnostic Regimen

When a patient with a speech defect was admitted to one of the designated general hospitals in the Zone of Interior, he was examined by the director of the speech clinic (a neurologist), who supervised all subsequent medical and surgical therapy and directed the program devised for speech rehabilitation. When all definitive treatment had been concluded, the patient was interviewed by the speech therapist, who, after conferring with the neurologist and neurosurgeon, outlined the specific program for his care and reeducation. It was the rule to discuss the situation intimately and at an early date with the patient's relatives also in an effort to secure an early psychologic adjustment, to allay tensions and anxiety, and to present as encouraging a picture as was justified of the results to be expected from treatment.

The routine of diagnostic study began with the examinations usually carried out on any neurologic patient. In addition to the history and physical and neurologic examinations, routine laboratory tests were made, consultations were requested, and other studies were carried out as indicated, including roentgenologic examination, special studies of the blood and spinal fluid, laryngoscopy, audiometry, psychometry, electroencephalography, and similar measures. In the occasional case in which cranioplasty or craniotomy was

necessary and the dura was entered, inspection, palpation, biopsy, and stimulation of the cortex added to the completeness of the study. In some instances, these manipulations actually produced improvement in speech by the release of cerebrodural scars and improvement in the cerebrospinal fluid circulation.

Diagnostic studies always included those listed in TB MED 155, in addition to those preferred by the particular neurologist in charge of the speech clinic. All analyses were based on the principles previously established by Jackson, Head, Goldstein, and Weisenburg and McBride. In general, the dysphasic patient was subjected to the following battery of tests in addition to the basic examinations already listed:

1. *Psychometric studies.*—Wechsler-Bellevue; Kent EGY; Rorschach; Bender Visual Motor Gestalt; thematic apperception test; Minnesota multiphasic test; Shipley-Hartford test; measurement of abstract behavior, including the Goldstein-Scheerer cube and stick tests; Weigl-Goldstein-Scheerer color form sorting test; and mechanical aptitude and vocational preference studies.

2. *Language studies.*—Head, Weisenburg and McBride, Chesher, and Halstead and his associates. Specific testing of the cognitive functions, such as reading, writing, arithmetic, and spelling, was included in this group. Because of the overlap of various speech disorders, investigations for dysarthria and dysphonia most commonly associated with the dysphasias were not overlooked.

3. *Nonlanguage studies.*—Various form boards (Seguin, two-figure, casuist, etc.); Pintner nonlanguage tests, drawing tests, and Knox cube tests, among others.

Therapeutic Regimen

Patients with speech defects could be centralized on special wards, with adjacent classrooms, at four of the special centers (Bushnell General Hospital, Brigham City, Utah, Kennedy General Hospital, Memphis, Tenn., O'Reilly General Hospital, Springfield, Mo., and McGuire General Hospital). Similar arrangements were made at Percy Jones General Hospital, Battle Creek, Mich., at the convalescent center (Fort Custer Annex). Officers lived in separate quarters but attended classes with enlisted men.

The individualized program outlined for each patient took into consideration his previous experience, education and environment, the results of diagnostic studies, the opinions of the various consultants, and the need for a possible change in his occupation because of residual disability. Training further took into account residual function and was directed along lines of substitution through other association pathways. An active reconditioning program was a part of every individual program. In some instances, with due regard to the condition of the individual patient, arrangements were made for credit in advanced subjects with local high schools and colleges. United States Armed Forces Institute courses were also extensively utilized.

Equipment used during the rehabilitation of dysphasic patients included all material supplied by occupational therapy departments in the arts and

crafts selected for the individual; magazines and comic books for picture and word stimulation; flash cards; textbooks on spelling, arithmetic, reading, writing, and other subjects; stimulus letters, words, and numbers made of cardboard, wood, or metal; playing cards (numbers), crossword and jigsaw puzzles; miniature objects; coins; calendars; ward telephones, to improve conversation and aid psychologic adjustment; clocks; blackboards; phonograph recordings; tachistoscopes; and a variety of other items, which, with those already listed, were adapted to the needs of the individual patient. Recording machines, of the recorder-playback type, which were available at DeWitt General Hospital, Auburn, Calif., Thomas M. England General Hospital, Atlantic City, N. J., Percy Jones General Hospital, McGuire General Hospital, and Wakeman General Hospital, Camp Atterbury, Ind., proved most useful in permitting comparative studies of the patient's progress as well as providing opportunity for him to recognize and correct his own errors.

The time devoted specifically to speech therapy depended upon the characteristics and degree of the speech disorder in the individual case. At first, half an hour twice daily was as much as the usual patient could tolerate. Later, as improvement occurred, practice sessions could be lengthened. Although individual training was emphasized throughout, group therapy proved valuable in such fields as writing, reading, arithmetic, and spelling. Frequent examinations were given, and records were maintained to determine progress. Conferences and seminars were held to discuss common problems, disseminate information, and adopt new suggestions.

The usual individual schedule was maintained on an 8-hour basis but was entirely flexible. A typical program follows:

0800 to 0830: Ward work detail.
0900 to 1000: Physiotherapy; classes in writing, reading, and arithmetic.
1000 to 1100: Orientation and education program (moving pictures, concerts, lectures, etc.).
1100 to 1200: Speech therapy.
1300 to 1400: Physical training.
1400 to 1600: Occupational therapy; speech therapy; diversional and recreational activity; educational activities, including classes in reading, writing, arithmetic, and spelling.
1900 to 2100: Local night courses for credit.

Furloughs.—All patients with speech defects, if their physical condition permitted, were given furloughs immediately after completion of the initial studies made on their entrance into the centers. Periodic leaves were also given at 2- to 3-month intervals. Instructions were given to the patient himself and to members of his family, so that lessons could be continued while he was away from the hospital. The psychologic values of these leaves more than balanced the loss in training time which resulted.

CLASSIFICATION AND MANAGEMENT OF SPEECH DISORDERS

The classification of speech disorders adopted in Army treatment centers was based on categories already well known to physicians interested in them. Additions were made as military necessity required. The defect was listed according to the major disorder present, with full recognition, of course, that more than one defect often existed in the same patient. Dysphasia and dysarthria, for instance, often coexisted. Patients with palatal perforations presented impairment of both phonation and articulation, while those with maxillofacial wounds not infrequently showed concomitant structural and neurologic involvement of the peripheral speech apparatus.

Dysphasia

Patients in this category were classified into 1 of 5 possible groups, depending upon the predominating defect revealed by examination. These defects included the following: (1) Motor or expressive dysphasia; (2) sensory or receptive dysphasia; (3) amnesic dysphasia; (4) mixed or expressive-receptive dysphasia; and (5) aphasia, which might be total or complete.

The term "dysphasia" to denote impairment of language was first used by Head [6] and was in general use by British speech pathologists. It was preferred in Army speech centers as being more characteristic and more accurately descriptive than the former term "aphasia," which was reserved for complete loss of speech.

Since, owing to delays in evacuation, Army patients usually were not observed until 4 to 8 weeks after injury, subclassification into such groups as transcortical and subcortical dysphasia was not possible. For the same reason, when the patients were first seen, usually only one major defect was present, such as impairment in comprehension of speech and writing (sensory or receptive dysphasia); difficulty in expression of language and writing (motor or expressive dysphasia); or inability to name objects, qualities, and conditions (amnesic dysphasia). Sometimes the defects were mixed. It was believed that soon after injury the majority of speech defects could be regarded as mixed (expressive-receptive), all language processes being involved. During the same period, complete loss of speech (aphasia) might be present, but it was unlikely to be permanent unless severe, irreparable cerebral damage existed. Lesions such as those described by Wernicke, Gerstmann, and others were occasionally observed.

Dysarthria

The term "dysarthria" was used to denote a disorder of articulation on the basis of a lesion of the central or peripheral nervous system. It could occur (1) with injuries of the central nervous system, including the pyramidal tracts

[6] Head, Henry: Aphasia and Kindred Disorders of Speech. New York: Macmillan Co., 1926.

(corticobulbar and corticospinal), extrapyramidal tracts, frontopontocerebellar tracts, and cerebellar pathways; or (2) with injuries of the peripheral nervous system, including the facial nerve, the palatal branches of the vagus nerve, and the hypoglossal nerve. Although articulatory defects assumed the major role in such cases, phonatory problems were associated with most central nervous system lesions. For this reason, the term "dysarthrophonia" is now being suggested as a more descriptive term than dysarthria in this type of case.

Since dysarthria was usually the result of a craniocerebral injury, patients suffering from it were admitted to the neurosurgical sections of designated general hospitals. It was usually associated with such other neurologic residuals as dysphasia, hemiplegia, and cerebellar manifestations. Only occasionally, in spite of the large numbers of gunshot wounds of the head which occurred in World War II, was it associated with central nervous system damage. The majority of cases were the result of lesions of the cranial nerves, chiefly of the facial nerve and the hypoglossal nerve. Lesions of the facial nerve caused impairment of accurate production of bilabial sounds. Lesions of the hypoglossal nerve interfered with the production of linguadental and linguarugal sounds. Lesions of the vagal and recurrent laryngeal nerve, although theoretically incorporated with this group, were classified with dysphonias.

Disorders of articulation due to central nervous system lesions were less frequent in the Army than in civilian life. When they occurred, they were usually associated with cerebellar injury alone or were present in conjunction with pathologic changes productive of dysphasia. Defects which occurred under the latter circumstances were grouped with the dysphasias. In a supratentorial lesion, therefore, dysarthria was commonly found with other evidence of central nervous system involvement. The infrequency of dysarthria in the presence of extensive damage to the frontal, parietal, and temporal lobes is interesting, especially in view of the high incidence of articulatory disturbances in the pyramidal and extrapyramidal disorders in childhood. The explanation is undoubtedly the site of the lesion: dysarthria is usually manifest in bilateral involvement and is much less common on a unilateral basis. It should be emphasized that these conclusions are derived from examinations made 1 or 2 months after injury. It is quite conceivable that minor defects were initially present and were overlooked by both examiner and patient.

In examinations for dysarthria and dyslalia, although long test phrases and sentences (such as Methodist Episcopal, Third Riding Artillery Brigade, round the rock the ragged rascal ran) are commonly mentioned in the neurologic literature, their use was discouraged, as they occasion difficulties even in normal persons. Standardized words, phrases, and sentences weighted with the individual sounds to be tested were found far more useful and practical.

Another important fact learned in the speech clinics concerned the previous recording of a patient's speech defect according to the examiner's in

terpretation of his response to questions. Individual differences were found to make this system lacking in uniformity and therefore very unsatisfactory. An accurate phonetic analysis of the defects present by the use of the international phonetic alphabet largely compensated for this lack of uniformity.

Speech defects due to lesions of the peripheral facial and hypoglossal nerves only occasionally required specific therapy. Adequate compensation by substitution had often been achieved by the time the patients were seen in Zone of Interior hospitals. Central nervous system dysarthria, however, almost always required active treatment. Accurate production of the various sounds and their strengthening was an essential part of the training. Exploration of the involved nerves, which was sometimes necessary, often ameliorated the speech defects. The treatment of associated speech disorders was part of therapy.

Dysphonia

Under the heading of dysphonia were included cases of voice impairment due to involvement of the laryngeal mechanism. The causes might be organic, such as tumors, traumas, inflammatory processes, and congenital abnormalities, or functional, such as poor vocal habits and aphonia on the basis of conversion hysteria.

Only occasional cases of dysphonia of traumatic origin were observed at the speech clinics in the Zone of Interior. Patients with gunshot wounds of the neck often died. If they survived, there were multiple symptoms because of the many closely related vital structures in this region.

Dysphonia resulted from the following causes: (1) Direct trauma to the larynx; (2) paralysis of the vagus nerve or of its recurrent laryngeal branch; and (3) indirect trauma to the larynx or the vagus nerve or its recurrent laryngeal branch, with resulting temporary edema, hemorrhage, and similar manifestations. Late extrinsic contracture of scars occasionally caused dysphonia by distortion of the laryngeal mechanism.

Laryngeal paralysis of central nervous system origin was observed only infrequently. Cerebellar lesions usually resulted in errors of both articulation and phonation (dysarthrophonia), with the former predominating. The laryngeal syndromes of Jackson, Collet-Sicard, Vernet, and Tapia were also infrequent. A few cases were observed following chronic inflammatory processes, chemical and thermal trauma, injury at thyroidectomy, bilateral cord involvement, paralysis of the superior laryngeal nerve, and excision of tumors from the vocal cords. Patients with functional dysphonia on the basis of conversion hysteria were treated at neuropsychiatric, not neurosurgical, centers.

In addition to routine studies, the complete examination of a patient with dysphonia included laryngoscopy, vocal recording, and the investigation of associated speech defects. Specific questions to the patient concerning pitch, intensity, quality, and rate were found essential for decisive conclusions. Formerly, when reliance had been placed on the replies to such general ques-

tions as whether hoarseness or voice changes had been noticed, negative answers were often received, even in the presence of obvious changes in the quality of the voice.

Vocal exercises resulted in considerable improvement in most cases of neurologic or structural damage to the laryngeal mechanism. Esophageal speech was preferred to an artificial larynx in avulsion injuries and after laryngectomy for tumors. The resection of intrinsic and extrinsic scars distorting the larynx occasionally resulted in improvement, and a patient was occasionally benefited by exploration of the vagus or recurrent laryngeal nerves. Physiotherapy, in the form of heat, massage, and electrical stimulation over the course of the involved nerve, often expedited recovery in traumatic speech defects. Medical and surgical measures were used as necessary. Patients with aphonia on a hysterical basis were placed under the care of the neuropsychiatrist.

Dyslalia

In the category of dyslalia were included disorders of articulation due to actual structural involvement of the organs of articulation (the lips, tongue, and palate). These disorders might be due to congenital defects, such as cleft lip and palate, or might be acquired, such as tumors of the tongue and palate, palatal perforations, and gunshot wounds of the tongue with loss of substance and late cicatricial contracture between the tongue and the floor of the mouth. Functional causes of dyslalia, such as low educational standards, sigmatism (lisping), foreign dialects, and the regional differences apparent in American speech were obviously not of wartime importance and were excluded from this category. Dyslalia and dysarthria often coexisted in the same patient as the result of the anatomic location of maxillofacial wounds.

Because lesions of the palate are associated with impairment of both articulation and phonation, the term "rhinophonolalia" has been suggested as a substitute for rhinophonia, which is in use by the British school, and for rhinolalia, which is advocated by the American Speech Correction Association.

Patients with dyslalia, which was usually the result of structural damage to the articulatory mechanism following maxillofacial injuries, were ordinarily admitted first to plastic surgery centers. Because of anatomic relationships, dysarthria due to injuries of the seventh and twelfth cranial nerve in their peripheral courses was often associated with dyslalia.

Dyslalia was observed after all of the following injuries and conditions, in some of which, as noted, other speech disturbances also occurred: (1) Injuries to soft tissues, particularly the lips and cheeks; (2) loss of various portions of the tongue; (3) wounds of the floor of the mouth with late cicatricial contracture, usually involving the tongue; (4) maxillofacial fractures with concomitant soft-tissue injury which, when occurring in tracheotomized patients, were also at times associated with dysphonia; (5) mandibular fractures with deformity accompanied in some instances by vocal changes related to intensity and rate but with pitch and quality usually not affected unless laryngeal damage was

also present; (6) loss of teeth, particularly the incisors; (7) trismus; (8) cranial nerve injuries, with injuries of the peripheral portion of the facial and hypoglossal nerves at times accompanied by dysarthria and injuries of the facial nerve and its recurrent laryngeal branch by dysphonia; (9) perforations of the hard and soft palates, which were associated with rhinophonolalia; and (10) structural damage to the larynx, with dysphonia present if the damage was incomplete and with aphonia if the damage was complete.

Facilities for correcting speech defects were not available at plastic surgery centers unless these centers had also been designated as special centers for neurology and neurosurgery. If they had not been, either patients needing speech training were transferred to a speech center at the conclusion of reconstructive surgery or local arrangements were made for instruction by civilians.

Methods of examination were similar to those carried out for dysarthria, both dyslalia and dysarthria being disorders of articulation. Because dyslalia was so often associated with maxillofacial wounds, specific speech therapy sometimes had to be postponed for as long as 12 to 24 months. Satisfactory compensation was achieved in many cases of fracture with soft-tissue damage by replacement of missing portions of the lips, cheeks, and other tissues with local and distant flaps. Later speech reeducation was sometimes necessary, particularly in the following cases: (1) Cicatricial contractures between the tongue and the floor of the mouth in which increased lingual mobility achieved by surgical release of adhesions often facilitated the solution of the subsequent speech problem, (2) loss of considerable portions of the tongue, and (3) structural and neurologic damage or both to the laryngeal mechanism.

Therapy usually consisted in strengthening the various involved components of the articulatory apparatus by appropriate exercises, with the substitution of compensating movements to achieve the desired sounds when there was tissue loss or replacement. Imitation, mechanical direction, mirrorwork, ear training, chewing methods, pushing exercises, kinesthetic stimulation, and similar devices were used as necessary. Once the correct sound was produced, it was strengthened by appropriate exercises. Esophageal speech was taught when avulsion wounds of the larynx were associated with maxillofacial injuries. Occasionally, after corrective surgery of palatal perforations, residual nasality necessitated additional therapy.

Dysphemia

The category of dysphemia or stuttering included instances of obvious hesitation and repetition in the speech pattern with occasional secondary manifestations. The cause was usually psychologic, but dysphemia was also noted following craniocerebral injuries.

Dysphemia was classified according to whether it had existed before induction or had developed in service. A fairly large number of patients in this category were observed at speech clinics during treatment of other medical and surgical conditions. The usual cause was some mild emotional conflict,

personality disorder, or reaction to adverse environmental circumstances. These patients were not considered to have severe enough disabilities to be classified as psychoneurotic. In general, they performed their routine duties faithfully and competently. Many of them saw action and adjusted as well as any other randomly selected group of nonstutterers. Stuttering did not prevent their promotion in the enlisted ranks, and some of them received commissions.

Postinduction stuttering was usually the result of some intense emotional experience, such as exposure to repeated enemy bombing, prolonged campaigns, and severe wounds. The etiology was listed as combat fatigue when the symptom disappeared within a few weeks following rest, psychotherapy, and specific medical and surgical measures. When the anxiety state continued to recur, even after therapy had been continued over a long period of time, the patient was classified as psychoneurotic and was transferred to a neuropsychiatric center. In most cases, insufficient neurologic evidence was present to suggest that an organic factor was responsible. Some patients with dysphasia exhibited hesitation in their speech, as well as occasional secondary manifestations such as facial grimacing, but true stuttering could not be said to be present.

Large numbers of stutterers in the Army were not treated at all. Patients who were admitted to the hospital for investigation, however, were studied intensively and were given individual and group therapy. A detailed history was taken, and the personality structure was observed, particularly in relation to the patient's reaction to his symptoms and environment. Psychometric studies included the Wechsler-Bellevue intelligence scale, projection techniques such as the Rorschach method, and thematic apperception and Bender Visual Motor Gestalt tests. Therapy was directed toward the underlying emotional disorder. Specific treatment of the stuttering complex was not attempted. Principles of general semantics and mental hygiene were stressed. Individual and group psychotherapy was practiced daily and was coordinated with active reconditioning. The patient was encouraged to verbalize his fears and anxieties and to objectify them in relation to his early childhood experiences. Projection techniques, with the reenacting of common problems, were employed. Narcosynthesis was helpful in the occasional carefully selected case. Negative practice was also sometimes helpful; by this method, the patient is taught to stimulate his blocks with later introduction of the new pattern to normal, everyday situations.

Dyslogia

The term "dyslogia" was used to include disorders of articulation accompanying various neuroses, with the exception of aphonia on the basis of conversion hysteria and psychoses.

Patients with dyslogia were usually under the supervision of the neuropsychiatrist at a neuropsychiatric center; psychiatric rather than articulatory principles of training were followed, though analyses applicable to the dysarthrias and dyslalias could sometimes be applied. Methods of examination

and treatment, however, were usually directed toward the various neuroses and psychoses with which the symptom was associated and were not directed primarily toward the articulatory defect.

Dysaudia

The term "dysaudia" was used for cases of impairment of articulation due to defective hearing, whether it was congenital or followed craniocerebral injuries, acoustic trauma, aural infections, and similar conditions.

Patients with dysaudia were treated at the Army aural rehabilitation centers for the deafened and hard of hearing, where therapy was directed chiefly toward improvement of hearing and prevention of speech deterioration. This was accomplished by the provision of a hearing aid, speech reading, phonetic stimulation, and psychotherapeutic techniques. Methods of examination and other principles of therapy used in dysarthria and dyslalia were also applicable to dysaudia.

DISPOSITION

The length of hospitalization of patients with speech defects depended upon the severity of the disorder, the adaptability of the individual to treatment, and the associated disabilities, which were often of far greater consequence than the speech difficulty. Dysphasia was usually associated with other neurologic residuals, such as cranial nerve injuries, hemiplegia, convulsions, hemianopsia, astereognosis, and varying degrees of intellectual impairment, including confusion, loss of recent memory, disorientation, impairment in learning capacity, defects in cognitive abilities, loss of visual-motor coordination, personality disorders, and delirium.

From the standpoint of the speech difficulty only, patients with minor defects were usually ready for disposition within 2 or 3 months after admission to the hospital. Those with more serious defects sometimes required hospitalization for a year or more before any improvement was noted. Occasionally, even though spontaneous speech appeared adequate, further reeducation was necessary in such subjects as reading, arithmetic, and writing. In such cases, arrangements were made for additional training through the Veterans' Administration. In January 1947, 2 centers were designated for this purpose, 1 at Van Nuys, Calif., the former Birmingham General Hospital, and 1 at Framingham, Mass., the former Cushing General Hospital. It was also made possible for veterans to obtain speech therapy on a local contract basis if the arrangements were approved by the Veterans' Administration and did not conflict with the organization of existing centers.

As a rule, the presence of a speech defect, if there were no other injuries and disabilities, did not disqualify a man for some form of useful military duty. Spontaneous speech and comprehension of speech adequate for ordinary conversational purposes were interpreted as indicating maximum hospitalization benefits in patients with dysphasia. On this basis, had the necessity continued,

they could have been returned to duty. As already noted, however, most of them were separated from service on the basis of associated disabilities and neurologic residuals.

STATISTICAL DATA

Surveys made at intervals during the war and in October 1945 (tables 17 and 18) give some idea of the patient load and of the distribution and management of cases at the speech centers. The majority of the centers found dysphasia the most common defect.

Final figures from Percy Jones General Hospital for the entire period during which it operated as a speech center (November 1944 to November 1946) show that during this time the hopsital received 107 patients with dysphasia, 28 with dysphemia, 6 with dysarthria, 2 with dysphonia, and 4 with emotional difficulties associated with poor articulation and foreign accent.

TABLE 17.—*Summarized data on Army program for speech disorders at Zone of Interior general hospitals*

General hospital	Date of organization of clinic	Personnel in charge	Average caseload
Bushnell	1 June 1945	Neurologist	8
Cushing	16 Nov. 1944	Psychologist	30
DeWitt	21 Jan. 1945	Speech therapist	40
Halloran	3 Apr. 1945	do	6–7
Hammond	28 Aug. 1945	do	5–6
Kennedy	December 1944	Neurologist	20–25
McCloskey	1 Mar. 1945	Neuropsychiatrist	15
McGuire [1]	7 May 1943	Neurosurgeon	20–30
Northington	24 July 1945	Neuropsychiatrist	25
O'Reilly	June 1944	Neurologist	15–25
Percy Jones	6 Nov. 1944	do	15–25
Thomas M. England	1 June 1945	Psychologist	20
Wakeman	February 1945	Neurologist	6

[1] Formerly at Brooke General Hospital.

TABLE 18.—*Distribution of speech disorders at Brooke and McGuire General Hospital*

Period	Cases treated	Wounded in action	Speech disorder				
			Dysphasia	Dysarthria	Dysphonia	Dyslalia	Dysphemia
7 May 1943–31 Aug. 1944	120	16	32	47	12	5	24
1 Sept. 1944–31 Dec. 1944	97	25	16	26	16	14	25
1 Jan. 1945–30 Sept. 1945	243	205	81	42	37	27	56
1 Oct. 1945–4 Oct. 1946	263	205	32	40	16	164	11
Total	723	451	161	155	81	210	116

In January 1946, the results of a survey of dysphasic patients still in Army hospitals, which was carried out by the Office of the Surgeon General, constituted a central register for patients with this disorder. In November 1946, 11 patients with dysphasia were still receiving speech reeducation at Halloran General Hospital, Staten Island, N. Y., but they were expected to be ready for final disposition when the installation was closed on 31 December 1946.

CHAPTER XV

Clinicopathologic Aspects of Fatal Missile-Caused Craniocerebral Injuries

(An Analysis of 24 Cases)

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The clinicopathologic study of fatal missile-caused injuries to the head upon which this chapter is based was undertaken for two reasons. The primary purpose was to establish criteria that would serve to indicate to the clinical neurosurgeon which of these wounds is apt to be lethal and to which, to express it very bluntly, he should not devote the time and effort which could be expended, with a better chance of success, upon wounds that are potentially less lethal. The secondary purpose was to set up a collection of carefully documented pathologic materials on this type of head injury for permanent storage and use in the Army Institute of Pathology (now the Armed Forces Institute of Pathology), Washington, D. C.

As with many another research project, the secondary of these two objectives proved to be the easier of fulfillment, as well as, perhaps, the more important. Not a great deal that was conclusive was established about the lethality of head injuries as a result of this study. On the other hand, the protocols of each of the 24 cases investigated, together with the gross and microscopic specimens, all carefully photographed, were deposited with the curator of the Army Institute of Pathology early in 1947. They constitute a truly invaluable collection, and, as with all other specimens entrusted to the Institute, the materials are available at all times to students of pathology and to other physicians interested in the particular field of head trauma.

MATERIALS AND METHODS

This study was based upon 24 extensive craniocerebral injuries, all of which were caused by missiles and all of which terminated fatally. Ten patients died without operation; the other fourteen were treated surgically. The materials were obtained during the last phase of the Italian campaign, through the cooperative efforts of the 8th, 13th, 16th, 28th, 94th, 170th, and 171st Evacuation Hospitals.

In all instances, autopsies were performed at these forward hospitals, either by a pathologist or by a neurosurgeon. All possible clinical material

was collected, and all details of the gross injuries of the scalp, the skull, and the brain and its coverings were carefully recorded.

The brains were fixed in formalin for a period of 10 days and then were forwarded to the 2d Medical Laboratory. Here the gross specimens were photographed, to show the extent of the surface injuries, before the brains were sectioned coronally. Photographs were made of the most important levels, and representative blocks of tissue were cut for histologic study. The microscopic sections were cut, stained, and studied at the Army Institute of Pathology. Clinical and autopsy data were forwarded with the specimens.

In 10 of the 24 cases, the cranial and cerebral injuries were associated with injuries of other parts of the body. In at least 3 of these 10 cases, the other injuries were severe and may have played a contributory part in the fatal outcome. In every instance, however, the cerebral injury was thought to be the primary cause of death.

The anatomic location of the destructive lesions in each case, together with the survival time after wounding, is summarized in table 19. Space does not permit the publication of the protocols of all 24 cases in this chapter, and repeated references will therefore be made to this summarized table throughout the ensuing discussion. The listing is according to the hours of survival and not according to the chronologic order in which the cases were handled. Certain case reports are also reproduced in detail, to illustrate certain of the fundamental surgical implications of the study.

Survival time in these 24 cases ranged from 30 minutes at one extreme to slightly over 10 days at the other. In nine instances in which the time of wounding could not be ascertained with certainty, the period of survival was estimated on the basis of the interval between the time of tagging by the medical aidmen and the recorded time of death. In four instances in which the information concerning the survival time was particularly vague, the approximate period could be deduced from other sources which were regarded as reliable.

This study includes wounded men who survived long enough to reach a forward hospital. Battlefield deaths were excluded, since the time factor in such deaths is necessarily unknown. It is of interest, however, that in 1,000 autopsies performed by Capt. William W. Tribby, MC, on casualties found dead in the field between the Salerno landings and the crossing of the Arno, craniocerebral injuries alone accounted for 133 deaths. In another 268 cases, in which there were injuries of other parts of the body in addition to the head injuries, the craniocerebral wounds were of such severity as to have been a possible, if not probable, cause of the fatality. In other words, in 40.1 percent of 1,000 battlefield deaths, head injuries were the actual or the probable cause of the fatal outcome.

CRANIAL LESIONS

Five of the twenty-four head injuries which make up this series represented perforating (gutter) wounds of the cranium. The remainder were penetrating wounds. The characteristics of the wound of entrance, as well as of the wound

TABLE 19.—*General topography of destructive lesions in 24 penetrating wounds of the brain*

Case No.	Frontal lobe	Parietal lobe	Temporal lobe	Insula	Occipital lobe	Corpus striatum	Septum pellucidum	Corpus callosum	Lateral ventricle	Thalamus	Hypothalamus	Third ventricle	Mesencephalon	Cerebellum	Period of survival[1]
															Hours
1	x		x	x		x	x	x	x						0.5
2	x		x	x		x	x		x						5
3		x	x	x	x										13
4	x	x	x		x										13
5		x	x						x					x	16
6	x	x			x			x	x						18
7	x	x			x	x	x		x	x	x				25
8	x					x	x	x	x		x				26
9		x			x				x					x	31
10	x	x			x	x		x	x	x	x	x	x	x	31
11	x	x	x			x			x						63
12	x	x	x		x				x	x					65
13			x		x				x						60
14	x		x						x						60
15	x	x													72
16			x		x				x					x	79
17			x						x						85
18	x	x	x	x	x										94
19	x		x												96
20	x					x				x					134
21	x	x	x		x				x	x				x	118
22	x							x							150
23			x		x			x	x					x	156
24		x	x		x	x		x	x	x	x				240
Total	16	13	16	4	13	8	4	7	17	6	4	1	1	6	

[1] Estimated for cases 6, 8, 10, 13, 14, 15, 19, 20, and 23. Survival time in cases 13 and 14 (German prisoners of war) was known to be between 48 and 72 hours, and in case 23 between 144 and 168 hours.

of exit (if there was a wound of exit), were essentially the same in all cases. Bone fragments of varying size, depending upon the size of the missile, were carried to various depths below the inner table of the skull. In certain through-and-through wounds, bone fragments were found throughout the path of the missile. In one instance of penetrating injury, an everted fracture was observed at the point of intracranial ricochet of the missile. The linear fractures which radiated in a variety of patterns from the wounds of entrance seemed to have no particular clinical significance.

One type of fracture, however, the so-called discontinuous fracture, seemed of quite special significance. This term implies the existence of one or more fracture lines in the base of the skull which do not in any way connect with the primary fracture. These fractures were usually multiple, and they were most often seen in the region of the cribriform plates, the orbital roofs, and, to a lesser extent, in the sphenoidal ridges and tegmental plates of the petrous bones. Fractures of this type were present in 9 cases; 3 were instances of perforating, and 6 were instances of penetrating, wounds of the skull.

The exact mechanism of discontinuous fractures is not yet clearly understood. It has been suggested that they result from great internal pressure suddenly produced within the cranial cavity by the effects of a high-velocity missile. On the other hand, the wavelike deformation spreading through a cranium struck by a missile may be a significant factor. These shock waves were studied in 1945 by Harvey, Butler, McMillen, and Puckett.[1]

Regardless of the mechanism of their formation, it was necessary to bear in mind the possibility of discontinuous fractures as an important feature of all penetrating or perforating missile wounds of the cranium. They could produce hemorrhage and cerebral damage at a considerable distance from the missile track, and they also could permit the entry of air or infection or both. There was definite evidence of injury to that part of the brain which corresponded to the site of the discontinuous fracture in 4 of the 9 cases in which this type of fracture was present (cases 1, 4, 10, and 21) and less conclusive evidence of injury of the brain in a fifth case (case 2). The base of the frontal lobe, including the region of the olfactory bulb and tract, was invariably the area involved.

In two cases, the dura was torn at the site of the discontinuous fracture, and it may also have been torn in a third case. In one instance, and possibly in another, the brain was also damaged at the site of the discontinuous fracture. The findings suggest that in this type of injury, the brain and the dura may be involved together or independently.

PATHOLOGIC CONSIDERATIONS

Gross Damage

In 18 of the 24 head injuries in this series, the ventricular system was directly involved by a laceration of the ventricular wall. In 17 of these 18

[1] Harvey, E. N., Butler, E. G., McMillen, J. H., and Puckett, W. O.: Mechanism of Wounding. War Med. 8: 91–104, August 1945.

cases the lateral ventricle was affected. The third ventricle, however, was affected in only one instance (case 10). Intraventricular hemorrhage was present in every instance in which the ventricles were wounded, the degree varying from a small amount of liquid blood to a complete cast of blood which filled the entire system. In one case, in which there was no apparent gross lesion of the ventricular system, a solid blood clot was found blocking the sylvian aqueduct; presumably it resulted from rupture of a subependymal vessel.

The lateral ventricle was damaged 17 times, the frontal and temporal lobes 16 times each, and the occipital and parietal lobes 13 times each (table 19). No other structures were damaged with such high degrees of frequency. The insula was injured in four cases.

Of the other telencephalic structures which suffered direct injury by the missiles, the corpus striatum was damaged in 8 cases, the corpus callosum in 7, and the septum pellucidum in 4. The diencephalon was directly involved in 7 cases, including 3 cases with damage to the thalamus alone (cases 12, 20, and 21), 3 cases with damage to the thalamus and hypothalamus (cases 7, 10, and 24), and 1 case with damage confined to the hypothalamus (case 8). In one instance, the lesion of the thalamus and hypothalamus was combined with damage to the mesencephalon in the diencephalomesencephalic border zone. This was the only case in which a gross lesion of the mesencephalon was evident. Gross damage to the cerebellum was noted in six cases. There was no evidence of a gross lesion in the region of the fourth ventricle, including the pons and the medulla oblongata, in any case in this series. The assumption seems reasonable that wounds of these structures resulted in death on the battlefield and that they therefore would not be represented in a series which excludes battlefield deaths.

Multiple injuries were the rule in all cases, and in only 1 instance (case 15) was gross damage confined to as few as 2 structures.

Histopathology

Since the survial periods in the 24 cases of severe brain injury in this series ranged from 30 minutes to approximately 10 days, the material was well suited for a study of early tissue changes and reactions in the central nervous system. The survival periods, however, did not extend to the stages in which Wallerian degeneration or retrograde cell reaction can best be demonstrated. They also did not extend to the stages at which reparative processes were in full development.

Microscopic examination of the brain lesions always revealed a central zone of hemorrhage and necrosis in the path of the missile, with immediate and complete destruction of tissue elements. Adjoining this zone was a zone of hemorrhage and edema, in which there were severe alterations of the ischemic type in the nerve cells, with less apparent damage to glial elements. In some instances, the elements of the mesodermal vascular structures in this zone showed few cellular alterations, but occasional necrosis of the walls was observed,

with exudation of fibrin or of a coagulating protein precipitate. These findings accord with previously recorded observations by Baggenstoss, Kernohan, and Drapiewski [2] and Rand and Courville.[3]

In all 24 cases, the definite zone of edema and hemorrhage extended along the missile track for distances varying from 3 mm. to 1 cm. or more. Hemorrhage and other vascular damage, however, also occurred in foci of apparently irregular and unpredictable patterns at varying, and often considerable, distances from the missile track, and diffuse edema not contiguous with the primary wound was noted in many instances. It is necessary to bear in mind, therefore, that in penetrating wounds of the brain, in addition to the local damage along the missile track and in its immediate vicinity, there are other and more distant lesions of the type found in cerebral contusion. It is furthermore possible, as Miller [4] pointed out in 1927, that cerebral concussion alone, without microscopic changes demonstrable by present technical methods, might give rise to various symptoms. Damage caused by laceration and contusion may lead to the series of traumatic reactions described by Bennett and Hunt [5] in 1933 and designated as traumatic encephalitis. Concussion, however, is believed to give rise to changes which are of a more rapidly transient nature.

Another point to be emphasized is the possibility advanced by Scheinker and Evans,[6] that in head injuries alterations in the parenchyma may be secondary to vascular disturbances. Central vasoparalysis, according to these authors, is of special importance, and vascular alterations are interpreted as due to local retardation or immobilization of the blood flow, associated with an increase in the permeability of the vessel walls for serous fluid and red blood cells. In the cases in this series, it was difficult to evaluate accurately the relative importance of primary mechanical impact, and the possibility existed that secondary ischemia might be due to mechanical vascular lesions, such as rupture, as well as to functional vasoparalysis.

Edema.—In brain injuries, as Hassin [7] pointed out in 1943, the cerebral parenchyma may assume a sievelike appearance, in which vacuolated acerolae are separated by thin trabeculae. Edematous fluid fills the adventitial spaces of Virchow-Robin and may, in addition, cause rupture of the glial pedicles attached to the membrana limitans perivascularis, which remains firmly adherent to the adventitial tissue. A second space filled with fluid is thus found external to the perivascular limiting membrane and within the cerebral parenchyma; this space, which represents the perivascular spaces of His and

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[2] Baggenstoss, A. H., Kernohan, J. W., and Drapiewski, J. F.: The Healing Process in Wounds of the Brain. Am. J. Clin. Path. 13:333–348, July 1943.

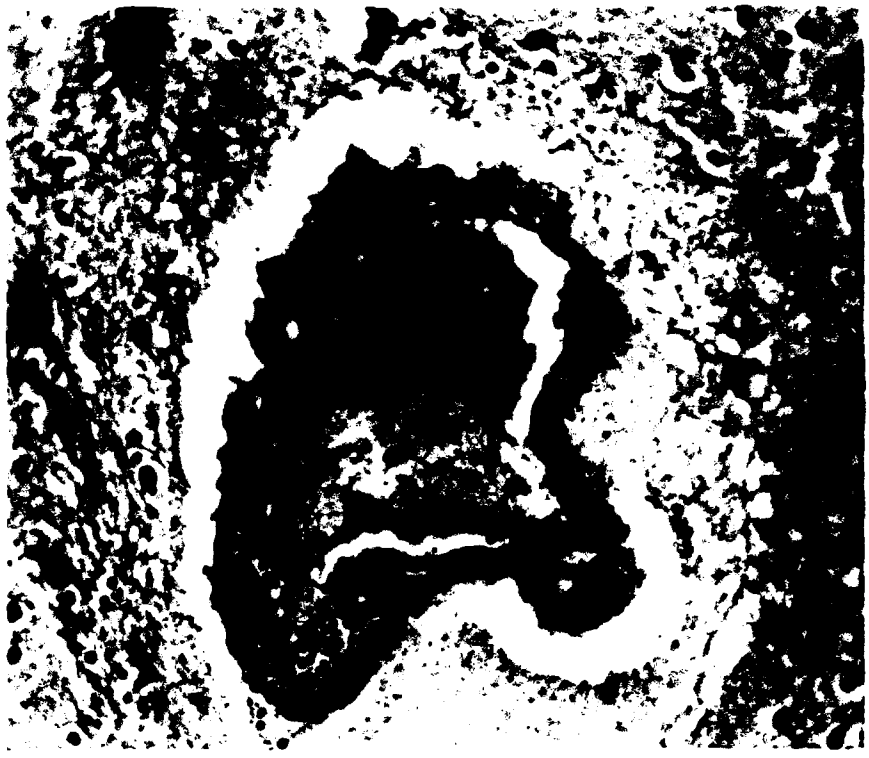
[3] Rand, C. W., and Courville, C. B.: Histologic Studies of the Brain in Cases of Fatal Injury to the Head. VI. Cytoarchitectonic Alterations. Arch. Neurol. & Psychiat. 36:1277–1293, December 1936.

[4] Miller, G. G.: Cerebral Concussion. Arch. Surg. 14:891–916, April 1927.

[5] Bennett, A. E., and Hunt, H. B.: Traumatic Encephalitis: Case Reports of So-Called Cerebral Concussion With Encephalographic Findings. Arch. Surg. 26:397–406, March 1933.

[6] Scheinker, I. M., and Evans, J. P.: Histologic Studies of the Brain Following Head Trauma. V. Alterations in the Vessels of the Central Nervous System Following Trauma. A. Research Nerv. & Ment. Dis., Proc. 24:98–130, 1945.

[7] Hassin, G. B.: General Pathological Considerations in Brain Injury. *In* Injuries of the Skull, Brain and Spinal Cord, edited by Samuel Brock. Baltimore: Williams & Wilkins Co., 1943. pp. 14–40.



AFIP 100004

FIGURE 70.—Arteriole in head of caudate nucleus containing coagulum and with predominantly mononuclear round-cell infiltration of Virchow-Robin space. There is pronounced perivascular edema. A perivascular space of His and Held has been created. Hematoxylin and eosin stain. (× 210)

Held, may be traversed by broken or elongated glial processes (figs. 70, 71, and 72). These spaces are artificial and can result from tissue shrinkage but may also be caused in cerebral trauma by the pressure of edema in a locus of lesser resistance. Edematous fluid also surrounded the ganglion cells in the central parenchyma, giving rise to the periganglionic spaces of Obersteiner (figs. 73 and 74), which are not present in normal tissue. Damage to the synaptic structures surrounding the nerve cells, plus interference with their function, must be considered a likely result of the formation of these perigang-lionic spaces.

Although edema, which represented one of the characteristic zonal features of cerebral injury, was usually most pronounced in the immediate vicinity of the injury, it could also be found at a considerable distance from the site of injury and often involved the leptomeninges. There was no apparent regularity in its extent and distribution, these considerations seeming to depend upon a number of variable factors, among which the central vasoparalysis emphasized by Scheinker and Evans may be included. Thus, edema was present, but was not conspicuous, in a patient whose survival time was 30 minutes. It was pronounced in a second patient with a survival period of 5 hours, and it was still present in a third with a survival period of about 10 days. The wide



AFIP 100000

FIGURE 71. Infiltration of Virchow-Robin space of an artery in the internal capsule. The infiltrating cells are polymorphonuclear leukocytes and mononuclear cells. The lumen contains a fibrin clot. There is a high degree of edema in the surrounding white substance and a perivascular space of His and Held is apparent. Hematoxylin and eosin stain. (× 210)

distribution of cerebral and leptomeningeal edema has previously been emphasized by Hassin, and also by Osnato and Giliberti,[8] Greenfield,[9] and Brock,[10] among others.

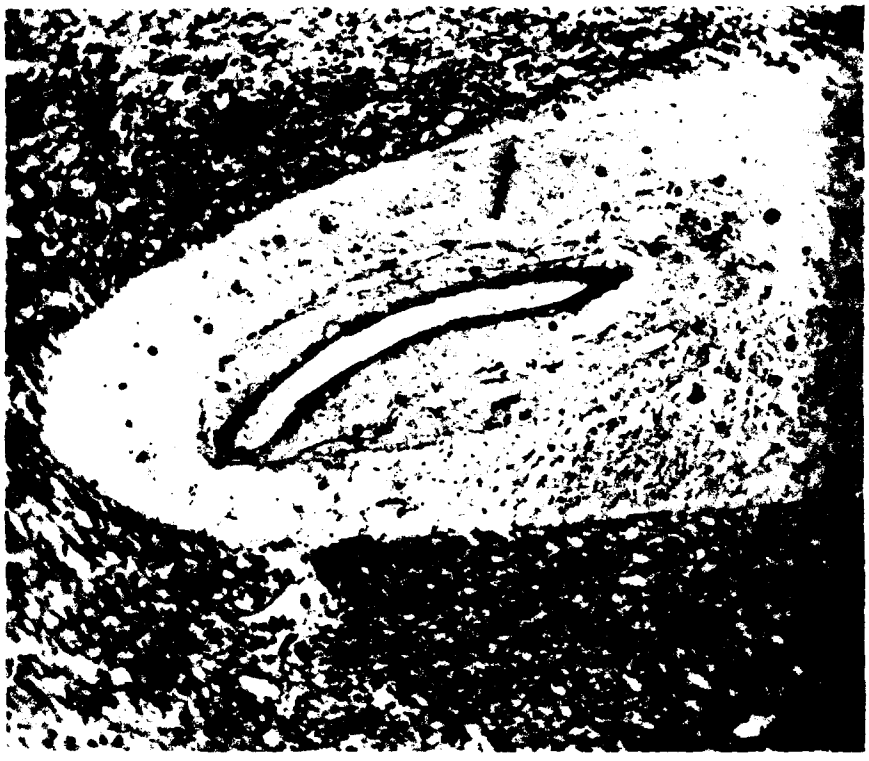
A vascular change occasionally noted in edematous areas consisted of collapsed capillary loops which appeared as solid, hyalinized bands (fig. 75).

Hemorrhage. —Hemorrhagic lesions were of various but interrelated types. Massive hemorrhage which infiltrated necrotic tissue and leptomeninges was associated with actual lacerations. Petechial hemorrhages of the ring type and so-called ball hemorrhages were observed especially in the immediate vicinity of the zone of complete destruction. Scattered petechial hemorrhages and more minute hemorrhages were found at considerable distances from the missile track and probably resulted either from actual laceration of the vessels in the missile track or from contrecoup. Other hemorrhages were presumably due to rupture of vessels subjected to mechanical stress at the time of impact. The stress might take the form of a pull or a shearing force or even sudden

[8] Osnato, M., and Giliberti, V.: Postconcussion Neurosis; Traumatic Encephalitis; A Conception of Postconcussion Phenomena. Arch. Neurol. & Psychiat. 18:181-214, August 1927.

[9] Greenfield, J. G.: Some Observations on Cerebral Injuries. Proc. Roy. Soc. Med. 32:43-52, November 1938.

[10] Brock, S.: General Considerations in Injuries of the Skull, Brain and Spinal Cord. *In* Injuries of the Skull, Brain and Spinal Cord, edited by Samuel Brock., Baltimore: Williams & Wilkins Co., 1943. pp. 1-13.



AFIP 100128

FIGURE 72.—Perivascular edema in medullary center of hemisphere. *Transudate with protein precipitate is filling both the Virchow-Robin* space and the space of His and Held. Arrow shows outer lining of Virchow-Robin space. Hematoxylin and eosin stain. (× 160)



AFIP 100013

FIGURE 73.—Perivascular and interstitial hemorrhage, moderate edema, and ischemic nerve cell changes in cortex adjacent to wound. Hematoxylin and eosin stain. (× 70)

overfilling of the tissues by cerebrospinal fluid which distended the Virchow-Robin spaces and thus disrupted the tissues.



AFIP 100231

FIGURE 74. Edematous cortex in vicinity of laceration with satellitosis around damaged large pyramidal cell. This picture suggests neuronophagia. Hematoxylin and eosin stain. (× 515)



AFIP 99087

FIGURE 75. Distorted cytoarchitectural pattern, perivascular hemorrhage and edema in cortex adjacent to missile wound. The pyramidal cells are oriented at various angles. Collapsed capillary loops appear as hyalinized bands. Hematoxylin and eosin stain. (× 210)

Petechial hemorrhages caused by fat embolism have been emphasized by a number of authors, including Hassin and Martland and Beling.[11] There was little evidence of fat embolism in this series, however, except in a single instance (case 11), and even in it the evidence was not conclusive. There were numerous fat droplets in vessels in regions in which no hemorrhages were

[11] Martland, H. S., and Beling, C. C.: Traumatic Cerebral Hemorrhage. Arch. Neurol. & Phychiat. 22:1001-1023, November 1929.

present, while the hemorrhagic areas did not differ from similar areas in other cases in which there was no definite evidence of fat embolism.

The findings in this series of cases indicate the importance of distinguishing between hemorrhages caused by direct trauma and those caused by concussion at a distance from the trauma.

Changes in nerve cells.—In this series, the nerve cells in the zone of complete destruction were entirely crushed and disintegrated. Except for poorly staining granular detritus, there were no traces left of [illegible] structures. In the zone of edema and hemorrhage, the changes [illegible]ose frequently described as due to ischemic cell disease. The cells were shrunken and stained darkly with both hematoxylin-eosin and Nissl stains. The nuclei were pyknotic and the cell processes, especially the apical dendrites of pyramidal cells, were wavy or tortuous (figs. 76, 73, 77, and 74). The changes described ranged in



AFIP 99993

FIGURE 76.—Nerve cell changes of ischemic cell disease type in cortex adjacent to missile wound. Shrinkage, homogeneous staining, pyknosis of nucleus, and tortuous dendrites are characteristic. Slight pericellular edema has caused the so-called spaces of Obersteiner. A few collapsed capillary loops can be recognized. Hematoxylin and eosin stain. (× 235)

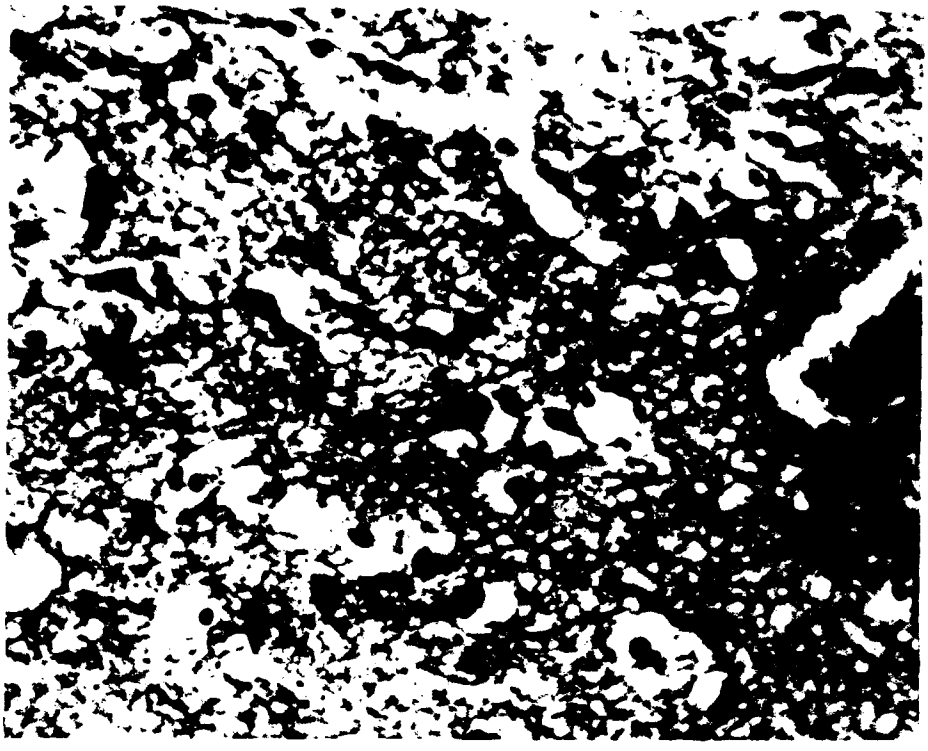


AFIP 9887

FIGURE 77.—Edema of subependymal cell plate and desquamation of ependymal cells of head of caudate nucleus. Sievelike edema of gray substance of caudate nucleus with ischemic nerve cell changes. Hematoxylin and eosin stain. (× 160)

severity from slight elongation and narrowing of the cells, with some increase in diffuse staining reaction, to extreme shrinkage (figs. 78 and 79). Large, edematous pericellular spaces could be seen around the shrunken cells. Vacuolization of the cells, with loss of affinity for stains, was sometimes observed; so-called ghost cells were present when this had occurred. The typical retrograde cell or axon reaction characterized by swelling of the cell, peripheral displacement of the nucleus, and tigrolysis, was not found to be well developed in this series. On the basis of the lesions observed in it, this is not a conspicuous cell change in the early stages of acute brain trauma.

Pronounced cell changes of the ischemic type were found following a survival period of only 30 minutes. On the whole, in this series, they were rather definitely confined to the immediate vicinity of the traumatized region, though they were occasionally observed in minor degree in regions at a considerable distance from the actual trauma. On the other hand, it is of interest that in a number of instances in which direct trauma was confined to the telencephalon, cell damage of fairly conspicuous degree was found in the inferior olivary nucleus of the medulla oblongata (fig. 80). Complete disappearance and disintegration of Purkinje cells were noted in the immediate vicinity of the lesion whenever the cerebellar cortex was lacerated or contused (figs. 81 and 82), which seems to indicate (1) that these cells represent elements especially sensitive to trauma and (2) that the cells of the inferior olivary nucleus are apt to show evidence of damage in brain injuries even if they are not apparently directly involved.



AFIP 100119

FIGURE 78.—Edematous necrosis of cortex in parieto-occipital region *with highly shrunk nerve cells.* There are a few infiltrating polymorphonuclear leukocytes and mononuclear cells in addition to glia nuclei and collapsed capillaries. Hematoxylin and eosin stain. (× 400)

In addition to damage to individual cells, cytoarchitectural changes resulting from laceration of the cortex were recorded, due to the immediate effect of the traumatizing agent. They included distortions of the laminar pattern and irregularities in the arrangement of the displaced cortical cells, which appeared oriented at various divergent angles. These changes, which had previously been reported by Rand and Courville in 1936, were designated as primary or early, in contradistinction to the secondary or late cytoarchitectural changes resulting from the pull of cortical scars. The cases reported in this chapter presented, of course, only primary changes because the longest period of survival was in the neighborhood of 10 days.

Changes in nerve fibers.—In areas of complete destruction, the axons had lost their property of being stained by silver impregnation and appeared as fine granular detritus, indicating that immediate necrosis had occurred. In the surrounding zone, damage to nerve fibers was indicated by swelling and the formation of varicosities (figs. 83, 84). Swollen fibers occasionally showed a reticular network of neurofibrillae with vacuolated, elongated meshes, and the swollen myelin sheaths had acquired a certain affinity for diffuse impregnation with silver salts (fig. 85A and B). Swollen axons were also fairly distinctly outlined by the use of hematoxylin-eosin and Nissl stains. The axons assumed a sinuous course, with many loops, and appeared fragmented, though



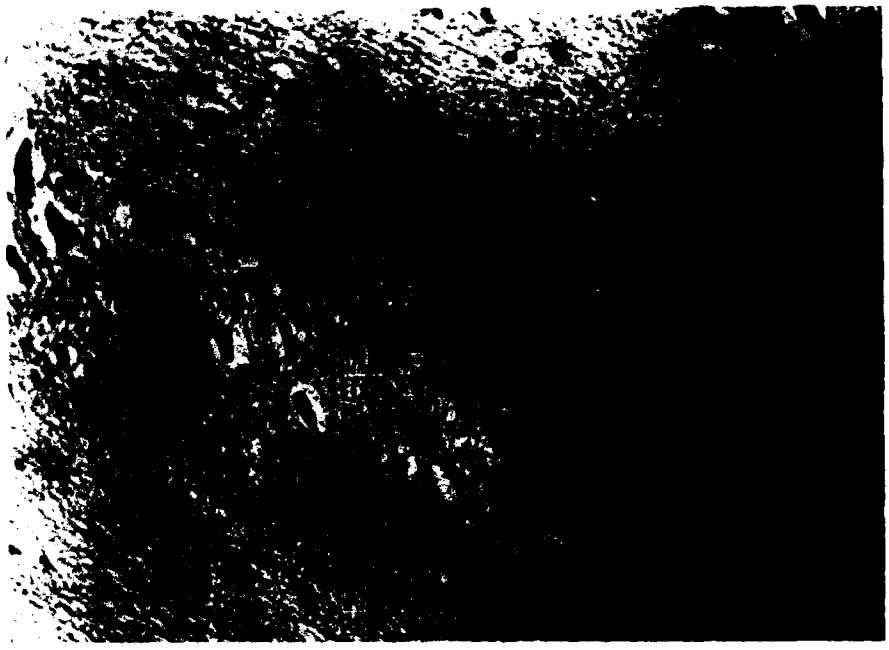
AFIP 100202

FIGURE 79. Hyalinized ghost nerve cells in necrotic area of cortex adjacent to parietal lobe wound. Hematoxylin and eosin stain. (× 400)

it is difficult to say whether this picture represented early actual fragmentation or whether the interruptions were due to the extension of the tortuous loops beyond the planes of the sections (fig. 86A and B). End bulbs of globular, fusiform, and clublike appearance (figs. 87, 88) and often of reticulated texture resembled those described by Ramón y Cajal [12] and by Rand and Courville.[13] The general findings of these authors, as well as the conclusions which they drew, could be confirmed in this material, but in most instances it was not possible to ascertain whether a structure of end-bulb type was a true end bulb or was merely an artificial break resulting because the plane of the section had cut off a varicosity in a still continuous axon. There was no evidence of regenerative processes, but it must be kept in mind that the periods of survival in this series were comparatively short, none exceeding 10 days. Further studies on fiber

[12] Ramón y Cajal, Santiago: Degeneration and Regeneration of the Nervous System, translated by Raoul M. May. New York: Oxford University Press, 1928.

[13] Rand, C. W. and Courville, C. B.: Histologic Changes in the Brain in Cases of Fatal Injury to the Head. V. Changes in the Nerve Fibers. Arch. Neurol. & Psychiat. 31: 527-555, March 1934.



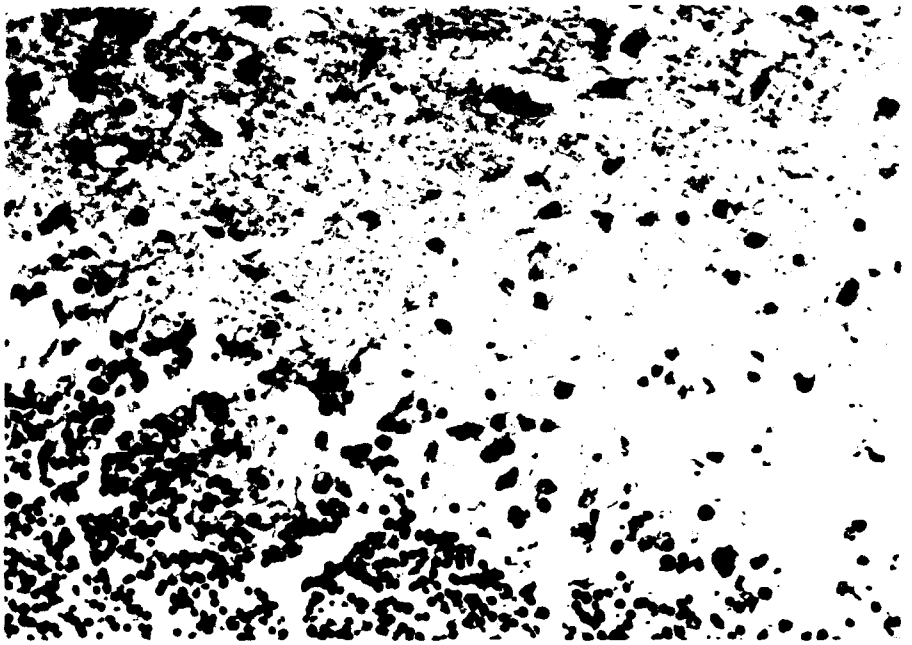
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FIGURE 80. Nerve cell changes of ischemic type in inferior olivary nucleus. Nissl's stain. (× 210)



AFIP 100228

FIGURE 81. Cerebellar cortex adjacent to lacerated area. Infiltration of molecular layer with polymorphonuclear leukocytes. Most Purkinje's cells have disappeared, but two sclerosed cells in the end stage of ischemic cell damage are indicated by arrows. Hematoxylin and eosin stain. (× 160)



AFIP 100230

FIGURE 82. Complete disappearance of Purkinje's cells in cerebellar cortex adjacent to cerebellar wound. Hematoxylin and eosin stain. (× 190)



AFIP 100610

FIGURE 83. Swelling, tortuosity, and possible fragmentation of swollen axons in white substance of damaged parietal lobe. Bodian silver impregnation. (× 315)



AFIP 100598

FIGURE 84.—Varicosities, possible end bulbs, and edema in white substance of damaged parietal lobe. Bodian silver impregnation. (× 315)

degeneration and possible later abortive regenerative processes were thus precluded.

Changes in the ependyma and choroid plexuses.—The changes described by Rand [14] and by Rand and Courville [15] as occurring in the choroid plexus almost immediately after injury to the brain could be clearly recognized in the cases in this series. They consisted of edema of the stroma, hydropic swelling of the choroid epithelium, and increased vacuolization of these epithelial cells (fig. 89A and B). Although hyalin bodies were frequently found in the stroma of the villi, it is doubtful that their presence represented a change due to trauma, since such degenerative changes are occasionally seen in otherwise normal material. These structures may be regarded as precursors of psammoma bodies.

Severe subependymal edema was frequently seen. Sometimes it resulted in distortion of ependymal cells (fig. 90). Numerous pyknotic nuclei were seen within the cells of the ependymal lining (fig. 91). The changes in both the ependyma and the choroid plexus frequently occurred at a considerable distance from the site of direct lesions. In many instances in which the wound was in the telencephalon, they were found in the fourth ventricle.

Changes affecting glial tissue.—The reaction of the astrocytes was rather inconspicuous in early stages in the cases in this series. These cells were found disintegrated in the zone of complete destruction, but in the surrounding zone swelling of astrocytes was noticed after approximately 18 hours (fig. 92). This

[14] Rand, C. W.: Histologic Studies of the Brain in Cases of Fatal Injury to the Head. I. Preliminary Report. Arch. Surg. 22:738–753, May 1931.

[15] Rand, C. W., and Courville, C. B.: Histologic Studies of the Brain in Cases of Fatal Injury to the Head. II. Changes in the Choroid Plexus and Ependyma. Arch. Surg. 23: 357–425, September 1931.

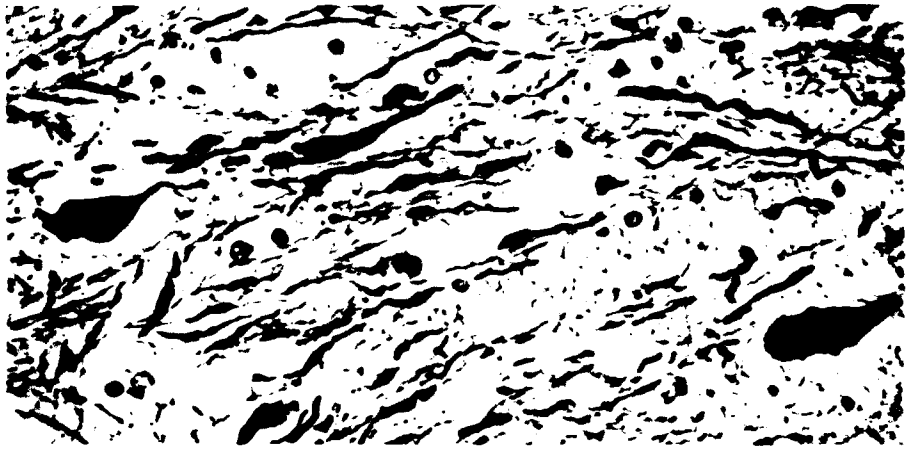


FIGURE 85. A and B. Swelling and possible fragmentation of axis cylinders near damaged area of internal capsule. Bodian silver impregnation. A, ×315; B, ×800.



AFIP 100609, 100605

FIGURE 86.—A and B. Swelling, tortuosity, and possible fragmentation of axons in white substances of damaged parietal lobe. There is also evidence of edema. Bodian silver impregnation. (× 275)



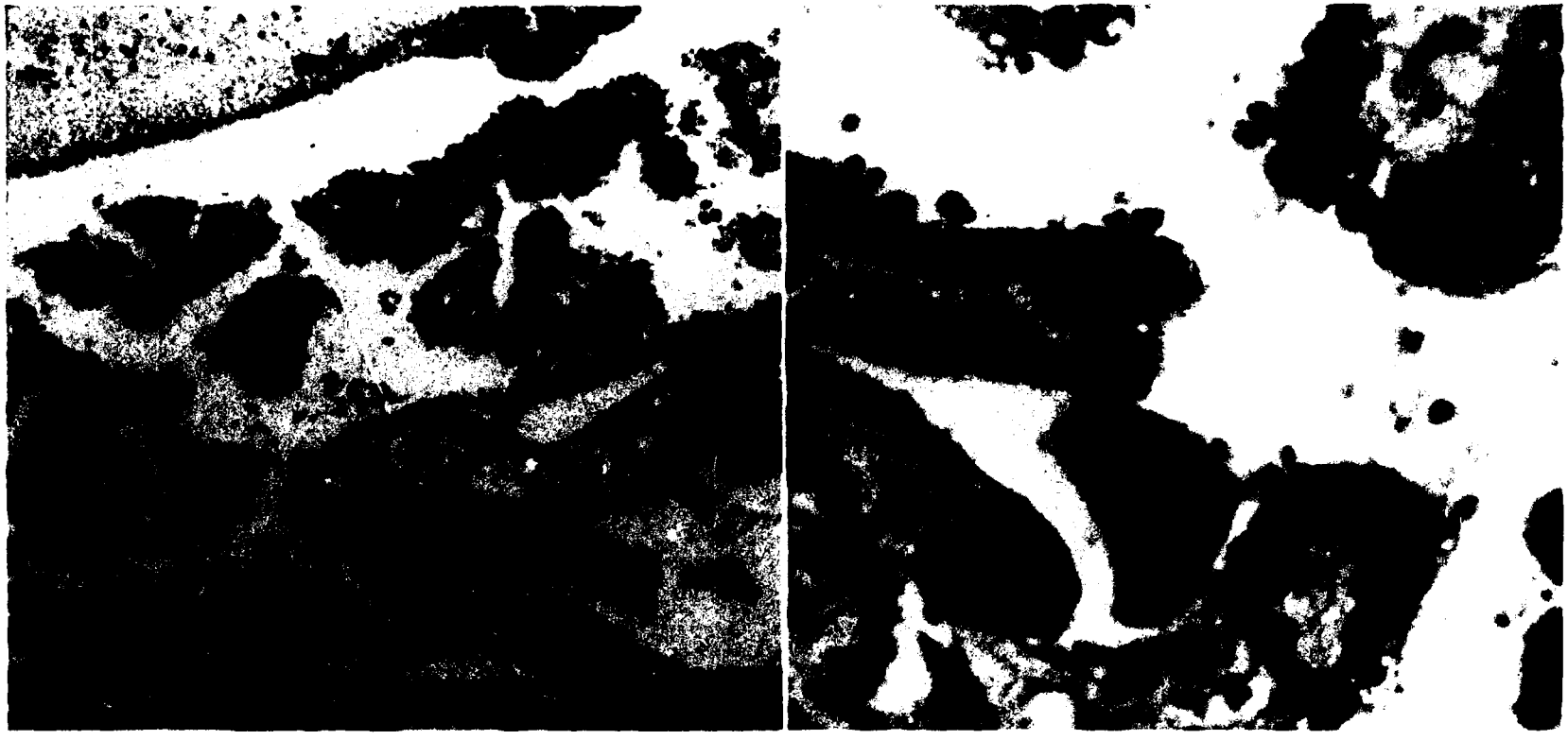
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FIGURE 87.—Swelling, tortuous course, and varicosities of axis cylinders near damaged area of internal capsule. The two large varicosities are possibly so-called end bulbs. Bodian silver impregnation. (× 315)



AFIP 100597

FIGURE 88.—Varicosities and possible end bulbs of axons in medullary center of damaged parietal lobe. Bodian silver impregnation. (× 450)



AFIP 100007, 100008

FIGURE 89.—A and B. Early changes in choroid plexus of fourth ventricle. Edema of stroma, swelling and vacuolization of choroid epithelium. Hyalin bodies in stroma are presumably not related to traumatic changes. Blood cells can be seen in the lumen of the fourth ventricle. Hematoxylin and eosin stain. A. (× 140) B. (× 500)



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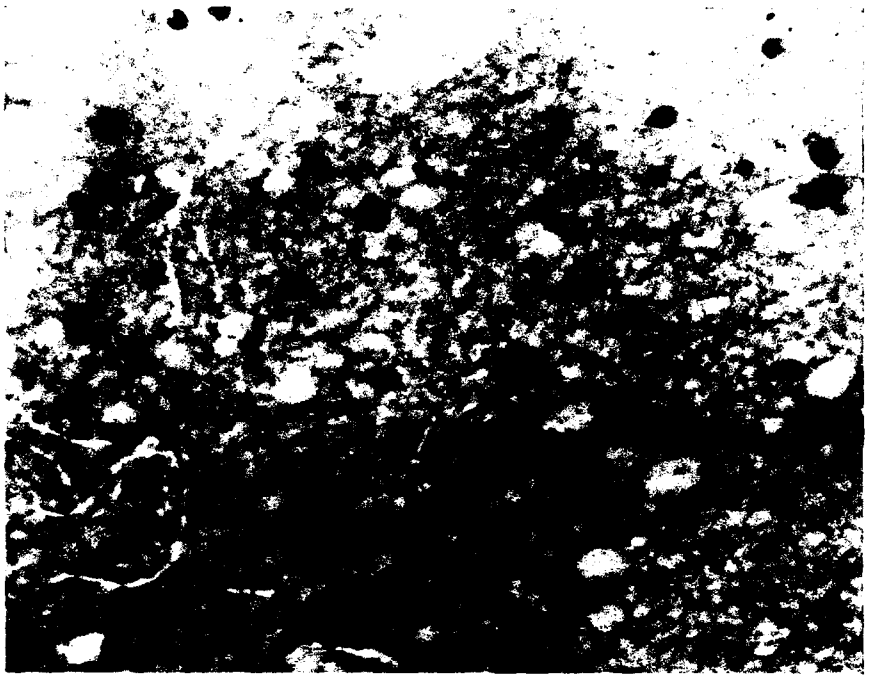
FIGURE 90.—Edema and distortion of ependyma lining fourth ventricle. Red and white blood cells can be seen in the ventricular lumen. Hematoxylin and eosin stain. (× 545)



AFIP 100121

FIGURE 91.—Pyknotic nuclei within the ependymal lining of the lateral ventricle and pronounced subependymal edema. Hematoxylin and eosin stain. (× 545)

finding was interpreted as representing the onset of so-called ameboid changes. Further accentuation of the regressive process was noted in cases in which the survival period was longer. In addition, the presence of constriction of nuclei and double nuclei indicated a mild degree of amitotic cell division, but conspicuous proliferation was not observed. These observations were in accord with the findings of Baggenstoss, Kernohan, and Drapiewski, who stated that astrocytes play a minor role in the reparative process in brain injuries. Oligodendroglia cells showed a strong reaction, indicated by swelling (fig. 93), occasional pyknosis, amitotic division (fig. 94), and a granular or vacuolated ap-

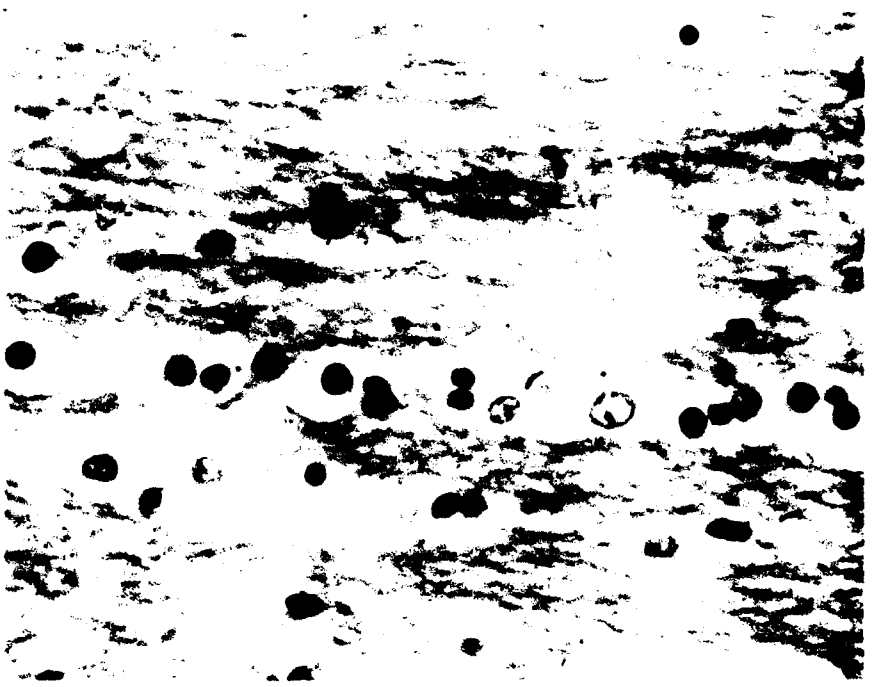


AFIP 100109

FIGURE 92.—Edema in white substance of damaged parietal lobe. The cytoplasm of astrocytes has become homogeneous and their nuclei are pkynotic. This swelling of perinuclear cytoplasm is believed to represent an early stage of so-called ameboid transformation. Hematoxylin and eosin stain. (× 545)

pearance. In several cases, definite evidence of accumulation of oligodendroglia cells around damaged nerve cells suggested neuronophagia or at least increased satellitosis (figs. 74, 95). The changes of oligodendroglia were thus both regressive and progressive in character.

Microglia (mesoglia) cells showed swelling and transformation into rod cells (fig. 96). Evidence of amitotic cell division could also be found. The changes in these cells were essentially progressive. Their activity during the stages studied in this series was, however, rather inconspicuous, and the findings mentioned are in accord with Hassin's observations.



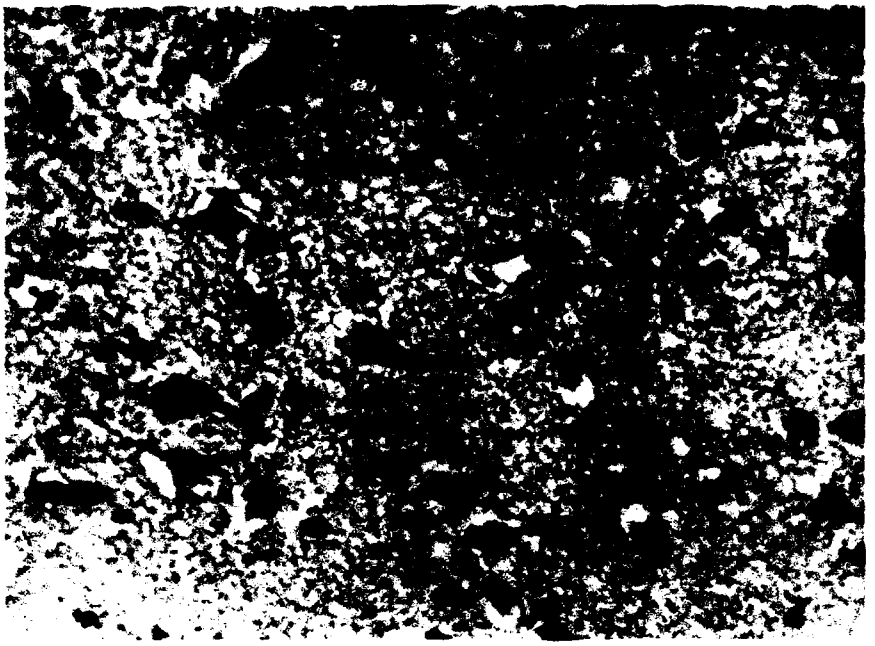
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FIGURE 93. Conspicuous [illegible] of oligodendroglia cells in white substance of damaged frontal lobe. Hematoxylin and eosin stain. (× 545)



AFIP 100001

FIGURE 94. Hemorrhage, mild edema, and pronounced ischemic nerve cell changes in cortex adjacent to missile wound. The paired and closely adjacent glia nuclei (arrow) are suggestive of a certain degree of amitotic cell division. Hematoxylin and eosin stain. (× 310)



AFIP 100336

FIGURE 95.—Ischemic nerve cell changes in frontal cortex near wound area. There is some suggestion of neuronophagia. Hematoxylin and eosin stain. (× 315)

Marginal gliosis, as described by Winkelman and Eckel,[16] was not a conspicuous feature in this series of cases, although in two instances there was observed a very mild indication of increase in glia cells in the first cortical layer. In these cases, marginal gliosis consisted of an increase of all three forms of glia cells and was purely a local phenomenon (figs. 97, 98). In a few cases in which the survival period was several days, there were, in addition to mitotic figures of proliferating endothelial cells, some mitotic figures in the brain substance which could not be definitely identified in respect to cell type but which might have been dividing glia cells (figs. 99, 100, and 101). It is thus not impossible that glial elements may undergo mitotic as well as amitotic cell division in the early stages of reaction following trauma.

Changes in the histogenous and hematogenous elements of the vascular apparatus.—Other important vascular changes found in this series, in addition to rupture of blood vessels, hemorrhage, and edema, which already have been discussed, included severe congestion of the veins and capillaries. Extreme distention was associated with stasis. Fibrin thrombi, and necrosis and hyalinization of the vascular walls, were also noted. These changes occasionally occurred not only in the vicinity of traumatic lesions but also at a considerable distance from them.

Scheinker and Evans,[17] who studied vascular changes in cerebral trauma, described vasoparalysis, general and local, associated with increased permeabil-

[16] Winkelman, N. W., and Eckel, J. L.: Brain Trauma; Histopathology During the Early Stages. Arch. Neurol. & Psychiat. 31: 956–986, May 1934.

[17] See footnote 6, p. 340.



AFIP 100352, 100355

FIGURE 96. A and B. Proliferation of capillaries and fibroblasts in damaged occipital cortex. Rod cells are also present. Hematoxylin and eosin stain. A. (× 160) B. (× 435)

ity, degeneration, and necrosis of the vessel wall, and chronic vascular changes. All the actual histologic alterations observed by Scheinker and Evans were also observed in the material in this series except for chronic vascular changes, which could not have developed within the survival periods of these casualties.

Perivascular infiltration, which was found to be present a few hours after injury, represented the most conspicuous tissue reaction to trauma in this series (figs. 102, 71, and 103). The reacting cells were polymorphonuclear leukocytes, small mononuclear cells (presumably lymphocytes), and large mononuclear cells



AFIP 100218

FIGURE 97.—Cortical area of left parietal lobe suggestive of marginal gliosis. Hematoxylin and eosin stain. (× 70)

(monocytes or histiocytes). Polymorphonuclear cells frequently predominated, which seemed to indicate that a large part, at least, of the reacting cells were of hematogenous origin. On the other hand, it appeared likely that histogenous elements present in adventitial tissue took part in the reaction. These various cells, originating both from the blood stream and the walls of the vascular apparatus, infiltrated into the borders of the zone of necrosis and throughout the neighboring zone of edema and perivascular hemorrhages (figs. 104 and 105). Macrophages with blood pigment were seen in the leptomeninges when the survival period was more than 3 days and in the brain substance when it was about 5 or 6 days.

Compound granular corpuscles were found when the survival period was 3 days or more (figs. 106, 107, and 108), which is in accord with the observations of Baggenstoss, Kernohan, and Drapiewski.[18] As these authors pointed out, most of these corpuscles appeared to arise from adventitial cells and from large mononuclear cells, which possibly were of hematogenous origin. There was little evidence that microglia cells contributed significantly to the formation of compound granular corpuscles at these stages, although some gitter cells might have been formed by them.



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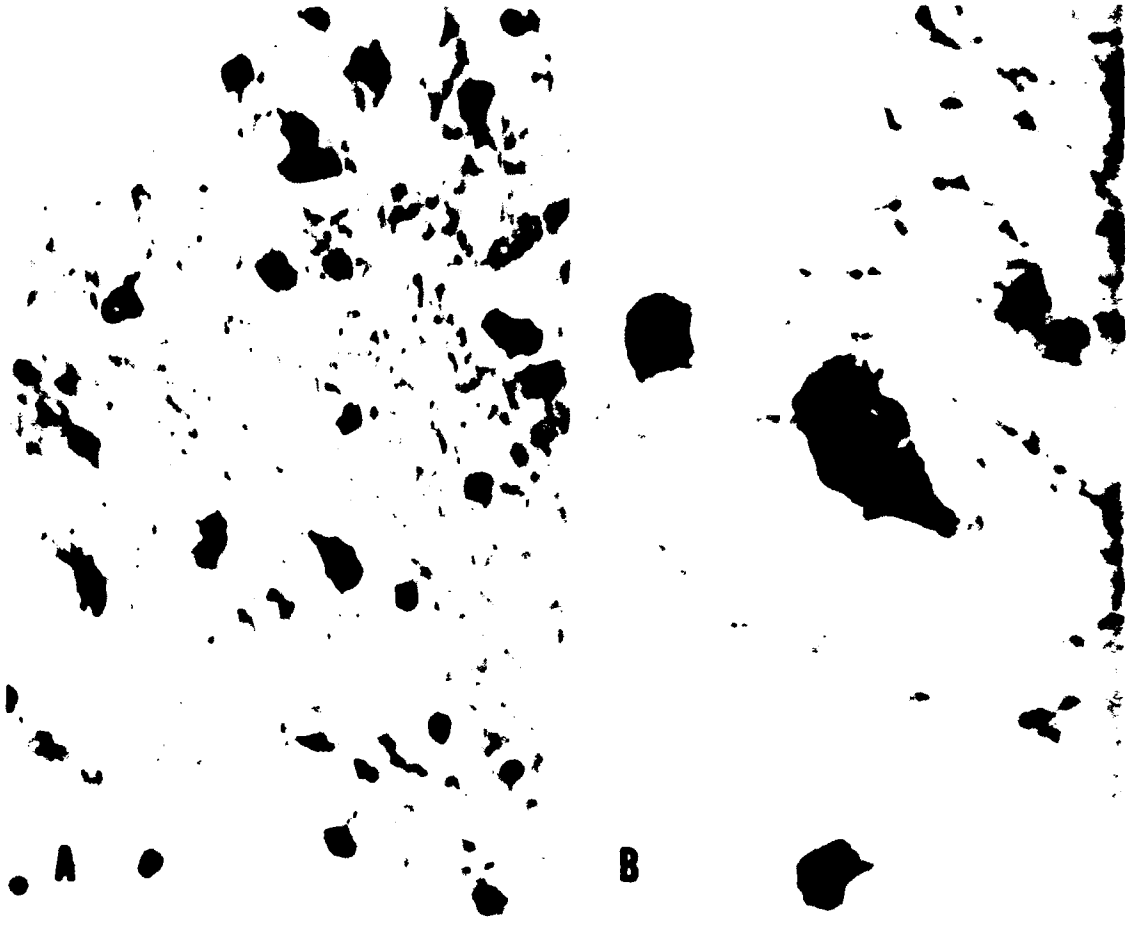
FIGURE 98.—Slight marginal gliosis in molecular layer of frontal cortex in immediate vicinity of wound area. Hematoxylin and eosin stain. (× 90)

Proliferation of capillaries in damaged areas of the brain became noticeable after a survival period of more than 5 days (figs. 109A and B, 110, and 111). Obviously, only the early stages of this process could be followed in this series. In these cases, the endothelial cells were hypertrophied and formed one-layered, but thick, endothelial tubes or solid endothelial sprouts. Mitotic figures were frequent (fig. 112A and B). Fairly dense networks of newly grown capillaries were seen when the survival time was about 10 days. Proliferation of capillaries occurred in damaged areas of both white and gray substance but was more pronounced in the latter. Fibroblastic proliferation also occurred but was less predominant in these early stages.

[18] See footnote 2, p. 340.

AFIP 100223, 100222

FIGURE 99.—A and B. Mitosis indicating proliferation of reactive cell, presumably oligodendroglia or microglia, in damaged and highly edematous cortical area of parietal lobe. Hematoxylin and eosin stain. A. (× 160) B. (× 1600)



AFIP 100226, 100208

FIGURE 100.—A and B. Mitosis of reacting glial cell, possibly oligodendroglia or microglia, in edematous molecular layer of damaged cerebellar cortex. Hematoxylin and eosin stain. A. (× 250). B. (× 1600).



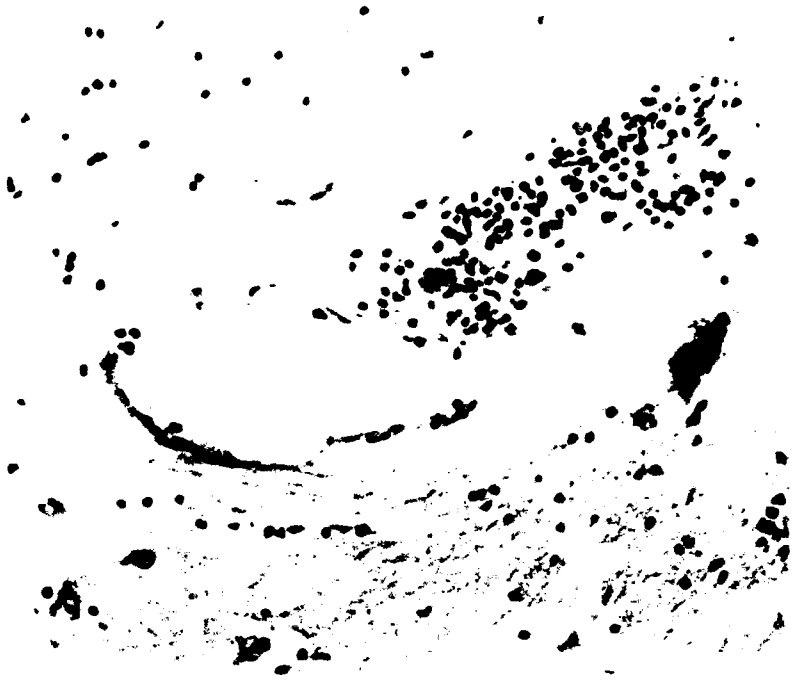
AFIP 100346

FIGURE 101.—Mitosis (equatorial plate) of reacting glious element in damaged frontal cortex. Hematoxylin and eosin stain. (× 1600)



AFIP 100002

FIGURE 102.—Small artery in region of diagonal band of Broca, with necrotic wall and perivascular infiltration. Infiltrating cells are polymorphonuclear leukocytes and small as well as large mononuclear cells. Hematoxylin and eosin stain. (× 210)



AFIP 60115

FIGURE 103. Vemule of nucleus amygdalae. Perivascular infiltration with mononuclear and polymorphonuclear cells. Hematoxylin and eosin stain. (× 235)



AFIP 60113

FIGURE 104. Infiltration with polymorphonuclear leukocytes and mononuclear cells in area of hemorrhagic necrosis of corpus striatum. Hematoxylin and eosin stain. (× 460)



AFIP 100201

FIGURE 105.—Marked infiltration with polymorphonuclear leukocytes in necrotic area of cortex adjacent to parietal lobe wound. Hematoxylin and eosin stain. (× 500)

Leptomeningeal changes.—The most striking alteration in the leptomeninges was hemorrhage followed by a cellular reaction. The erythrocytes in the meshes of the subarachnoid space were intermingled with polymorphonuclear leukocytes, histiocytes, large mononuclear cells, and lymphocytes. Proliferating fibroblasts could also be recognized, and red blood cells were occasionally seen enclosed within macrophages. The findings in this series corresponded to the changes in the leptomeninges described by Hassin [19] as the result of brain injury.

Infection.—A meningeal reaction of varying degree was observed in most cases in this series, but in only two cases (cases 14 and 24) was there definite evidence of pronounced purulent meningitis. In one of these cases (case 14), the ventricular lining also showed evidence of purulent ependymitis (fig. 113). In only one other instance could the unusually marked infiltration of the brain substance with polymorphonuclear leukocytes possibly be interpreted as an indication of infection. A certain amount of infection of the brain substance

[19] See footnote 7, p. 340.

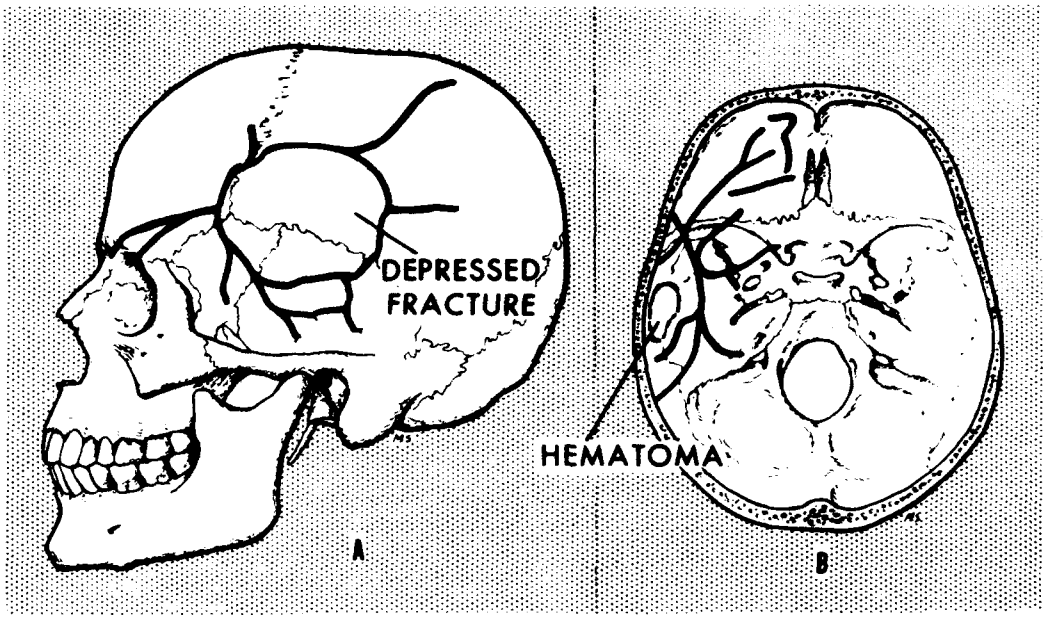
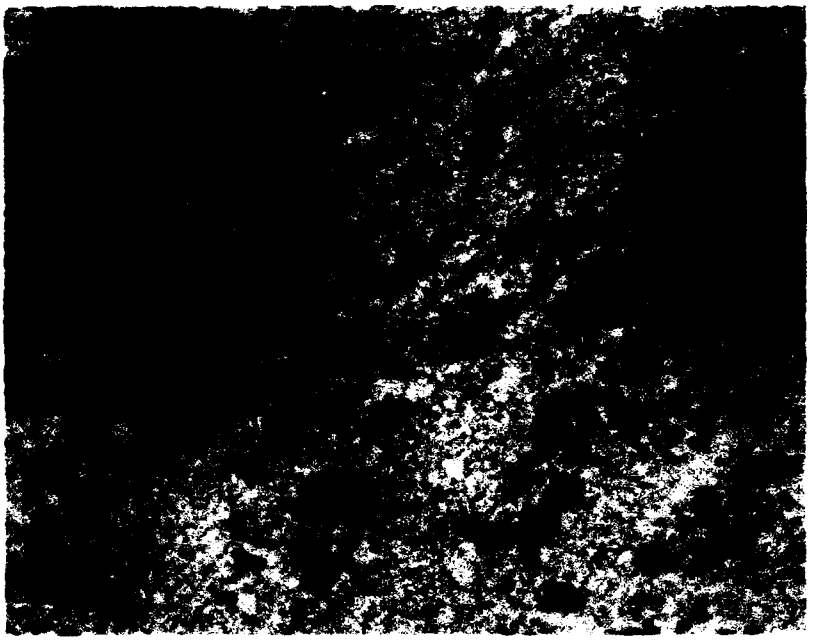
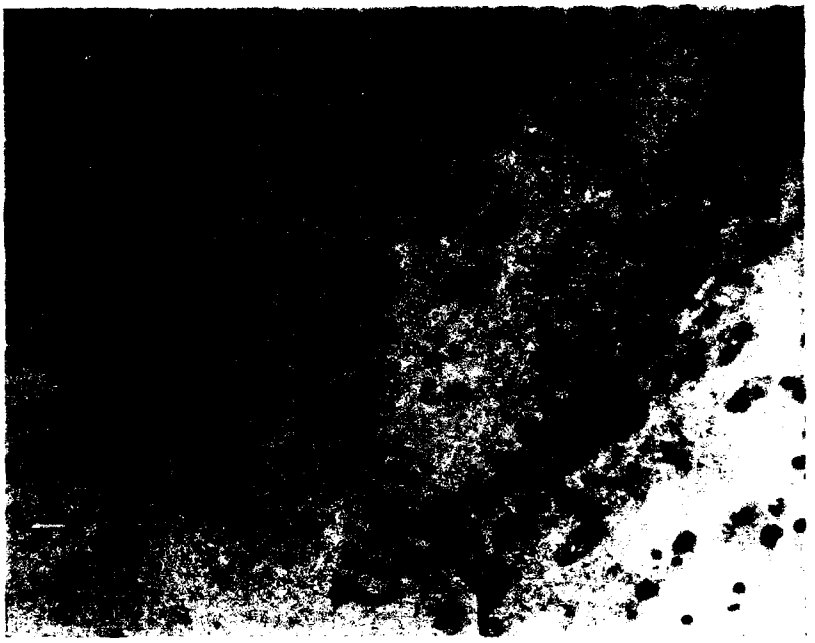


FIGURE 106.—A and B. Approximate pattern of skull fracture.



AFIP 100335

FIGURE 107.—Compound granular corpuscles in vicinity of sulcus terminalis near anterior end of damaged thalamus. Hematoxylin and eosin stain. (× 315)



AFIP 100342

FIGURE 108.—Compound granular cells in white matter of damaged occipital lobe. The grouping of such elements along a proliferating vessel is conspicuous. There is also some perivascular infiltration with mononuclear cells. Nissl's stain. (× 250)

was probably present in several other cases also. Although the material in this series differed in this respect from the practically aseptic cases studied by Baggenstoss, Kernohan, and Drapiewski, it is remarkable that the findings in both series were so similar, especially in respect to the predominance of polymorphonuclear leukocytes.

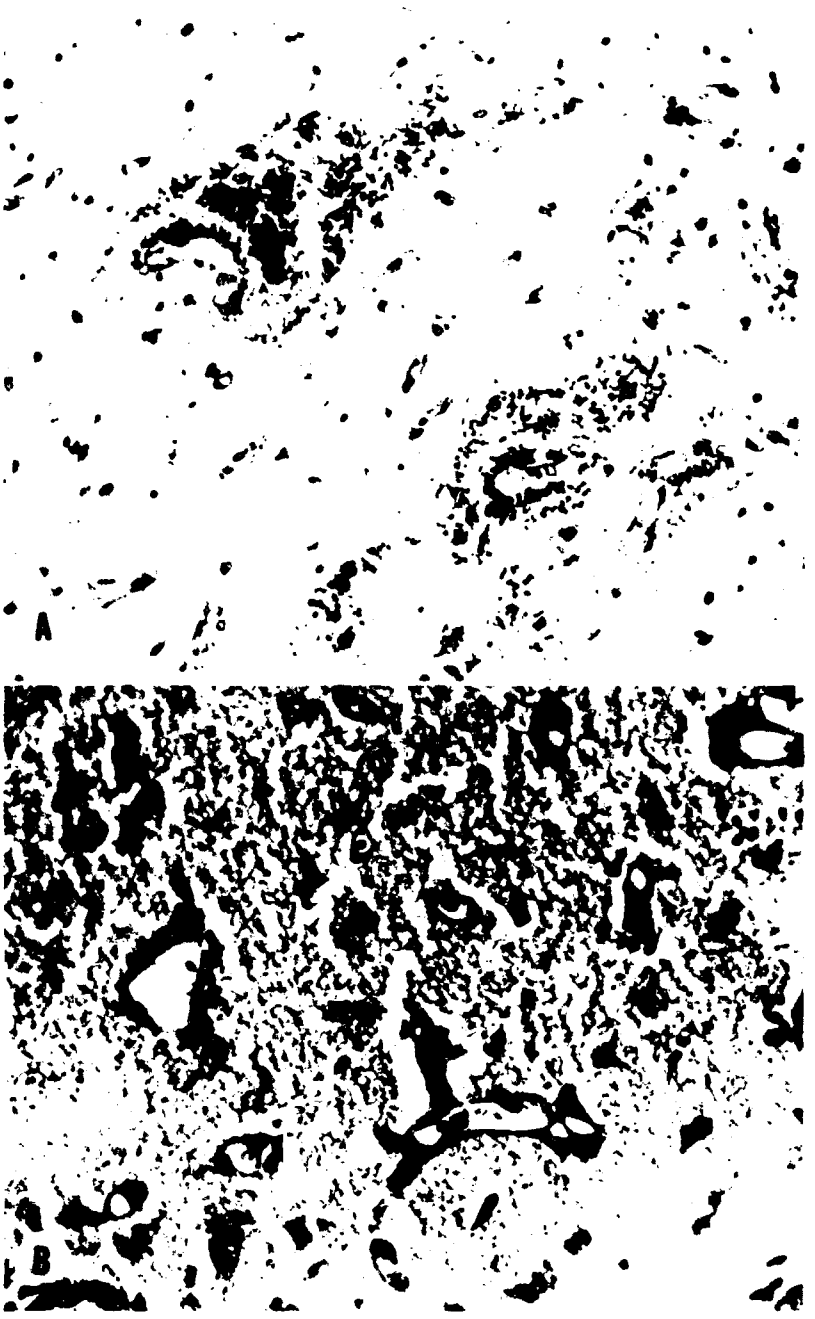
Sequence of reactive and other changes.—Generally speaking, the reaction of the brain tissue to trauma is far more sluggish than the reaction of tissues in other organs. The reactive processes during the early stages following injury, as they appeared in this series, were essentially carried out by the mesodermal components. The ectodermal elements showed only limited activity.

Summary of Pathologic Findings

On the basis of the observations made in the 24 mortal brain wounds reported in this chapter, the sequence of reaction and other changes in the cerebral tissue after trauma may be summarized as follows:

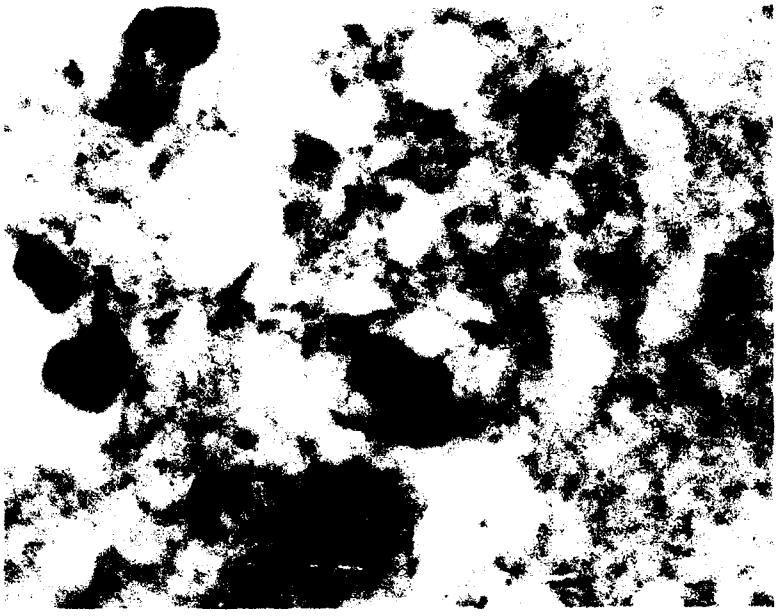
1. The early traumatic stage was characterized by disintegration of tissue in the path of the missile, hemorrhage and edema in the zone adjacent to the path of the missile, changes of the ischemic type in the nerve cells, and changes in the choroid plexus.

2. Within a few hours, perivascular infiltration set in, with a conspicuous participation of polymorphonuclear leukocytes. Thrombosis and necrosis of blood vessels proceeded as additional circulatory changes resulted from spread-



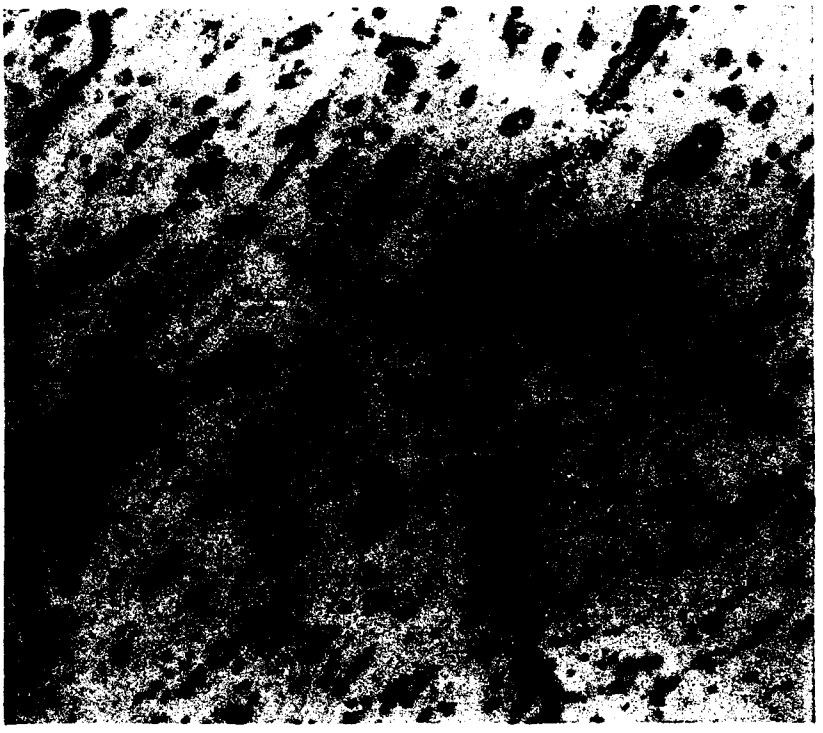
AFIP 100211, 100212

FIGURE 109.—A and B. Proliferation of endothelial cells, capillaries, and solid endothelial sprouts in damaged cortex of frontal lobe. Hematoxylin and eosin stain. A. (× 160) B. (× 275)



AFIP 100345

FIGURE 110.—Mitosis (equatorial plate) in early stage of compound granular cell. Edematous cortex adjoining medullary center of damaged occipital lobe. Hematoxylin and eosin stain. (× 1400)



AFIP 100341

FIGURE 111.—Conspicuous capillary proliferation in damaged occipital cortex. Hematoxylin and eosin stain. (× 185)



AFIP 100344, 100343

FIGURE 112.—A and B. Mitosis of endothelial cell of proliferating capillary in damaged cortex adjacent to wound. Hematoxylin and eosin stain. A. (× 800) B. (× 1900)



AFIP 100539

FIGURE 113.—Ependymitis in right lateral ventricle. A venule with perivascular infiltration is seen in an ependymal granulation. A fibropurulent exudate adheres to the ventricular wall. Hematoxylin and eosin stain. (× 210)

ing edema and hemorrhage. In damaged areas, axons began to swell, appeared sinuous, and showed varicosities.

3. Increased infiltration of hematogenous and histogenous elements took place into the cerebral parenchyma within the following period. Affected axons sometimes underwent fragmentation, and bulbs appeared.

4. After about 18 hours, swelling of oligodendroglia, microglia, and early ameboid transformation of astrocytes became noticeable, and pyknotic nuclei were observed within the ependymal lining. Glial elements of all three types displayed signs of occasional but extremely limited amitotic division.

5. After the second day, patchy areas of early demyelinization were observed in the regions of white matter affected by edema and hemorrhage.

6. After the third day, compound granular corpuscles made their appearance, and there was also, at this time, some inconspicuous evidence of neuronophagia.

7. After the fourth day, definite proliferation of capillaries set in.

SURGICAL CONSIDERATIONS

The implications of the preceding presentation, although it is entirely pathologic, are far more than academic. Most of the neurosurgeons in the Medical Corps at the beginning of World War II had had little, if any, first-hand experience with cerebral injuries caused by high-velocity missiles. Many of the younger men, in fact, had been trained in an atmosphere in which most

neurosurgery was ' ·tive and in which patients with severe head trauma were seldom seen.

As a result, these neurosurgeons were placed in forward hospitals, they had to learn the hard way, by trial and error, the practical details of military surgery. One of the most difficult of their problems was the correct selection of patients for operation. There were few rules to guide them, and no authoritative publications on the subject.

The fact that patients with severe damage to large parts of the brain, including the ventricular system, may survive for relatively long periods of time raises the question how one can distinguish, on the basis of the location of the anatomic lesion, between a mortal and a nonmortal brain injury. From a purely theoretical standpoint, it might seem that any injury of the brain would be directly mortal only insofar as it affected the function of the centers of the medulla oblongata which control respiration and cardiac action. On the other hand, extensive damage to higher parts of the brain, such as the cerebral hemispheres, diencephalon, mesencephalon, and cerebellum, can weaken the activity of the vital centers in a number of ways. Mechanical factors include the disruption of synaptic structures in the rhombencephalon through the pressure wave in the cerebrospinal fluid of the ventricles, through ventricular hemorrhage, and also through ge l cerebral edema. Hemorrhage and edema, combined with vasopar ay lead to anoxia of the vital centers. The toxic effects of devitalized ti and of infectious processes, even if they are mild, are additional factors which may endanger the function of these centers and may contribute to the formation of a vicious circle. These are all variables, many of which may escape observation, and the distinction between a mortal and a nonmortal brain wound therefore cannot be based exclusively on the extent of damage that is grossly, let alone cli ally, apparent.

The whole situation was confusing. All too frequently, a number of soldiers with head injuries would be received in the preoperative wards at the same time. The neurosurgeons, whose numbers were limited and whose time and strength were also limited, would be forced to decide which of these patients could most profitably be submitted to operation. Many times, craniotomies which were performed to be certain that wounded men be given every possible chance for life were found, at autopsy, to have been useless from the start because the cerebral injury had been lethal and beyond surgical relief from the moment if was sustained. Meantime, other patients with head injuries, less seriously wounded and with better prospects for survival, might have had their chances of life jeopardized by the delay in treatment.

By the time this particular study was undertaken, almost all of the neurosurgeons who participated in it were no longer inexperienced. All of them associated with the evacuation hospitals from which this material was derived had been through two or more active campaigns and had acquired the sound clinical judgment which, for most surgeons, can be acquired only by extensive experience. They were considered competent by this time to handle any type of trauma involving the nervous system. That competence probably accounts

for the fact that in these 24 cases, there was only one instance in which lesions were found at autopsy which might possibly have been corrected successfully by a more skillful surgeon.

The patients who made up the material for this investigation are logically divided into two groups. In the first group are the 10 patients who were recognized as hopeless surgical problems from the beginning and in whom no surgery was attempted. In the second group are the 14 patients for whom surgery was attempted and for whom, although their state seemed hopeless, there was a sufficiently reasonable doubt of the lethality of their wounds to warrant the attempt at operation.

GROUP I (NONSURGICAL)

In every one of the 10 cases in this group, autopsy studies revealed the soundness of the surgical decision to withhold operative intervention. The two cases presented here in detail represent a fair sample of the entire group. The first patient survived 30 minutes. The second survived 31 hours.

Case 1

Clinical History

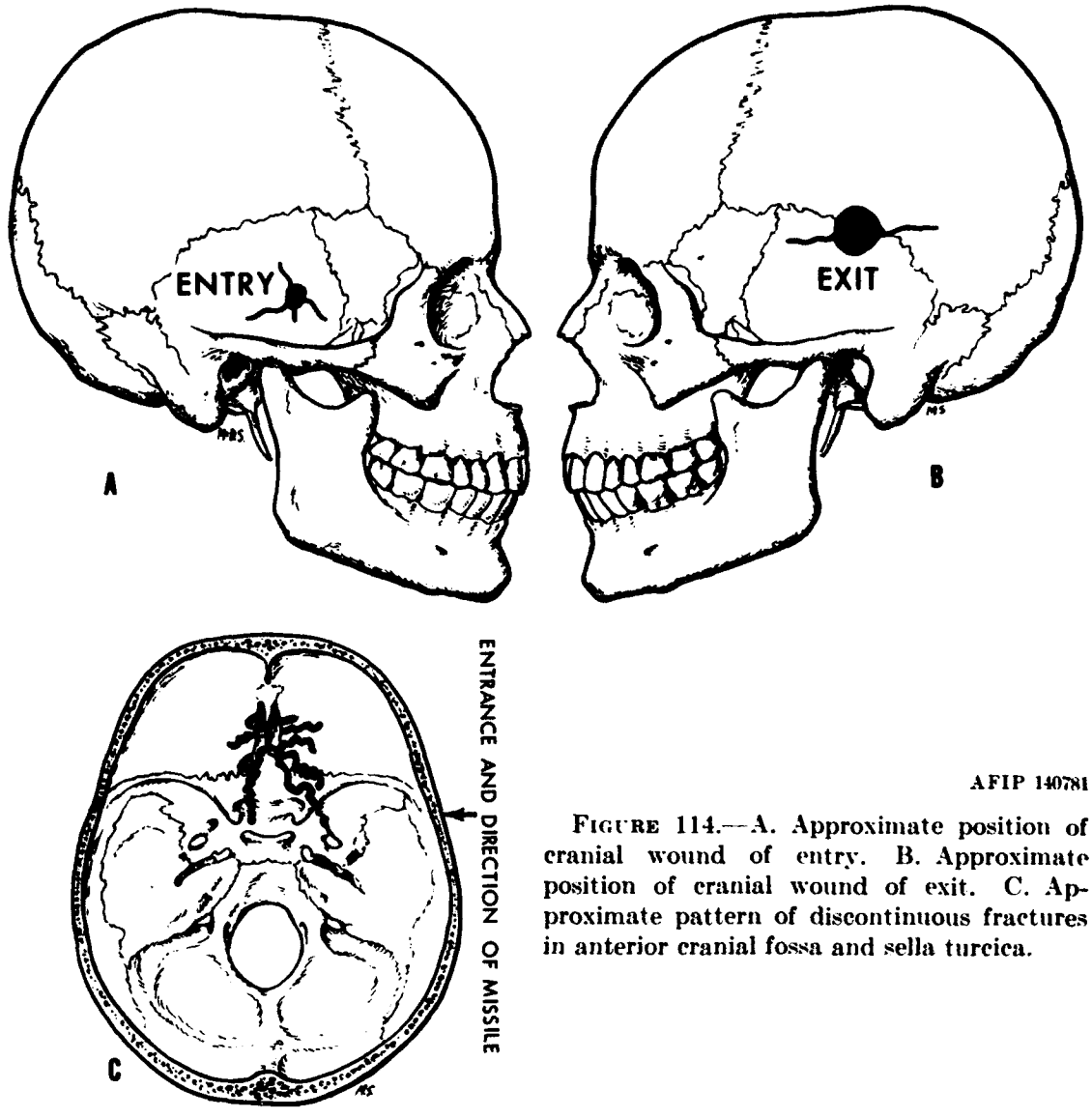
A soldier who became unconscious immediately after being struck by a bullet from a pistol fired at close range presented a wound of entry in the right temple and a wound of exit in the left parietal region. Cerebral herniation was present in both. Vigorous arterial bleeding in the right temporal wound had been partly controlled by a pressure bandage. The blood pressure was unobtainable. The pupils were dilated and fixed. No movements of any of the limbs were observed, and no reflexes of any type were obtained. Breathing was stertorous, and pulmonary edema was progressive.

Death occurred 30 minutes after wounding. Autopsy was performed 2 hours later.

Protocol

GROSS ANATOMY

Skull.—An oval opening about 7–9 cm. in diameter, with sharply cut edges, was present in the right temporal region, about 3.5 cm. above the external auditory meatus and 5 cm. posterior to the lateral corner of the right eye. Bloody fluid oozed from the wound. The skin and the scalp around the opening were free from burns, and no foreign particles (gunpowder) were observed. Another opening, between 12 and 15 mm. in various diameters, was present in the left temporoparietal region, about 9 cm. above the left external auditory meatus and 15 cm. from the lateral corner of the eye. The shape of this opening was stellar, due to the irregularly torn scalp. A small blood coagulum was seen in this wound, mixed with small particles of brain substance and of bone.



AFIP 140781

FIGURE 114.—A. Approximate position of cranial wound of entry. B. Approximate position of cranial wound of exit. C. Approximate pattern of discontinuous fractures in anterior cranial fossa and sella turcica.

Openings on both sides of the skull (fig. 114A and B) corresponded to the openings in the scalp. The opening on the right, which was located in the anterior third of the temporal squama, was sharply cut, oval, and of the same size as the opening in the scalp. Several fracture lines radiated from it for a distance of approximately 3–5 cm. The opening on the left, which passed through the suture between the temporal squama and the parietal bone, was also oval and sharply cut and was about twice as wide as the opening on the right. Through this opening, a fracture line ran in a nearly horizontal plane for about 4–5 cm. both anteriorly and posteriorly.

The anterior portion of the sella turcica and the entire lamina cribrosa, although these structures were not in the direct path of the projectile, were shattered into small fragments (fig. 114C), but no cuts were detected in the optic nerve.

Brain.—The point of entry of the projectile into the brain substance was in the right superior temporal gyrus, near the pole of the temporal lobe and approximately basal to the junction of the trunk and the posterior ramus of the sylvian fissure. The wound, an irregular tear, slightly over 1.0 cm. in diameter, was roughly funnel shaped, with the apex directly inward. It was filled with small, intimately intermingled fragments of brain tissue and coagulated blood.

The point of exit of the projectile from the brain was situated in the posterior part of the left inferior frontal gyrus, this location corresponding to the triangular and opercular portion of Broca's convolution, just above the dorsal end of the trunk of the left sylvian fissure. The wound was a nearly circular tear, somewhat over 2.0 cm. in diameter.

The leptomeninges showed widespread hemorrhage, particularly in the regions of both cerebral wounds. There was also some coagulated blood in the subdural space on the left. The base of the brain was contused and lacerated in the region of the medial orbital convolutions and of the gyrus rectus of both frontal lobes, this area corresponding to the area of the discontinuous fracture.

Coronal sections of the brain showed the course of the missile to be at a level slightly rostral to the anterior commissure. The track was rather clear cut, gross destruction of the brain substance being limited almost entirely to the path of the projectile, in a zone between 1 and 2 cm. wide (fig. 115). Severe,



AFIP 100825

FIGURE 115.—Coronal section through brain showing missile track.

grossly recognizable hemorrhagic lesions extended from the level of the rostrum of the corpus callosum to the level of the massa intermedia of the thalamus. There was also considerable diffuse hemorrhage along the fibers of the right internal capsule in the pes pedunculi, as far caudad as the most caudal levels of the thalamus.

In addition to the cortical areas already mentioned, the brain structures located in the path of the projectile included, from right to left, the anterior portion of the right insula, the right lentiform nucleus, the right internal capsule (anterior limb), the base of the head of the right caudate nucleus, the septum pellucidum, the base of the head of the left caudate nucleus, the left internal capsule (anterior limb), the left lentiform nucleus, the claustrum, and the insula. Both anterior horns of the lateral ventricle were perforated, and the entire ventricular system was filled with blood.



AFIP 100010

FIGURE 116.—Petechial hemorrhages in cerebral cortex adjacent to missile wound. The hemorrhages are perivascular and many are of the "ring" type. Hematoxylin and eosin stain. (× 70)

MICROSCOPIC ANATOMY

The cortex surrounding the site of both the entry and the exit of the projectile showed lacerations and numerous petechial hemorrhages of the perivascular, ball hemorrhage, and ring hemorrhage type (fig. 116). Although the patient survived for only about 30 minutes after wounding, the cortical nerve cells displayed definite alterations. The cell bodies appeared elongated, narrower than normal, densely stained, and homogeneous. The processes, especially the apical dendrites, were tortuous and corkscrew shaped; the nuclei were pyknotic (fig. 76). These changes have been described both as chronic cell alterations and as ischemic cell disease. In this case, they must be interpreted as rapidly occurring acute changes, possibly due to mechanical trauma as well as to ischemia. On the other hand, there was no evidence of any glious or mesodermal reaction. A slight degree of edema was present.

The lacerated cortical areas at the base of both frontal lobes presented identical histologic pictures. There was also considerable subarachnoid hemorrhage in the neighborhood of entry and exit of the missile and in the area of the laceration of the basal part of the frontal lobes.

In the areas of the basal ganglia traversed by the missile, the hemorrhages and cellular changes, which were similar to those in the cortex (fig. 117), in-



AFIP 100009

FIGURE 117.—Nerve cell changes of ischemic cell disease type in head of caudate nucleus adjacent to missile track. Slight edema of subependymal cell plate. Perivascular hemorrhage and perivascular edema along venule of nucleus caudatus. Hematoxylin and eosin stain. (× 160)

cluded subependymal hemorrhages in the vicinity of the projectile track and perivascular hemorrhages in the white substance of the region of the internal capsule. Although the ventricular system was filled with blood, there was no conspicuous histologic alteration of the ependymal lining. The choroid plexus, however, showed some evidence of early changes, corresponding to those described by Rand [20] after 2 to 4 hours (fig. 89A and B). The stroma was moderately edematous and the villi were ballooned, so that in some places the fibers of the stroma had become separated from the blood vessels. In addition, hyalin or fibrinoid bodies were seen in the stroma, though such findings are occasionally observed in normal cases. The choroid epithelium appeared swollen and hydropic, with irregular margins and increased numbers of vacuoles.

Case 10

Clinical History

The time of wounding was not known in this case. The patient had sustained a penetrating wound in the right parieto-occipital region, with moderate cerebral herniation and was in coma when he was admitted to an evacuation hospital 3 hours after tagging.

He was slightly restless and moved all limbs on painful stimulation. The temperature was 102° F., the pulse rate 100, and the respiration 28. Breathing was stertorous. The pupils were somewhat constricted and fixed. The deep reflexes were all more active on the left, and the Babinski and Hoffman reflexes were positive on that side. Superficial reflexes were absent. Roentgenologic examination revealed a severe comminuted fracture in the right parieto-occipital region; a large metallic foreign body lay near the sella turcica.

During the period of observation in hospital, the blood pressure rose to 180/50, the pulse to 130, and the respiration to 40 to 60. The temperature remained approximately 102° F. until death. No operation was undertaken.

Death occurred 31 hours after tagging, and autopsy was performed 10 hours later.

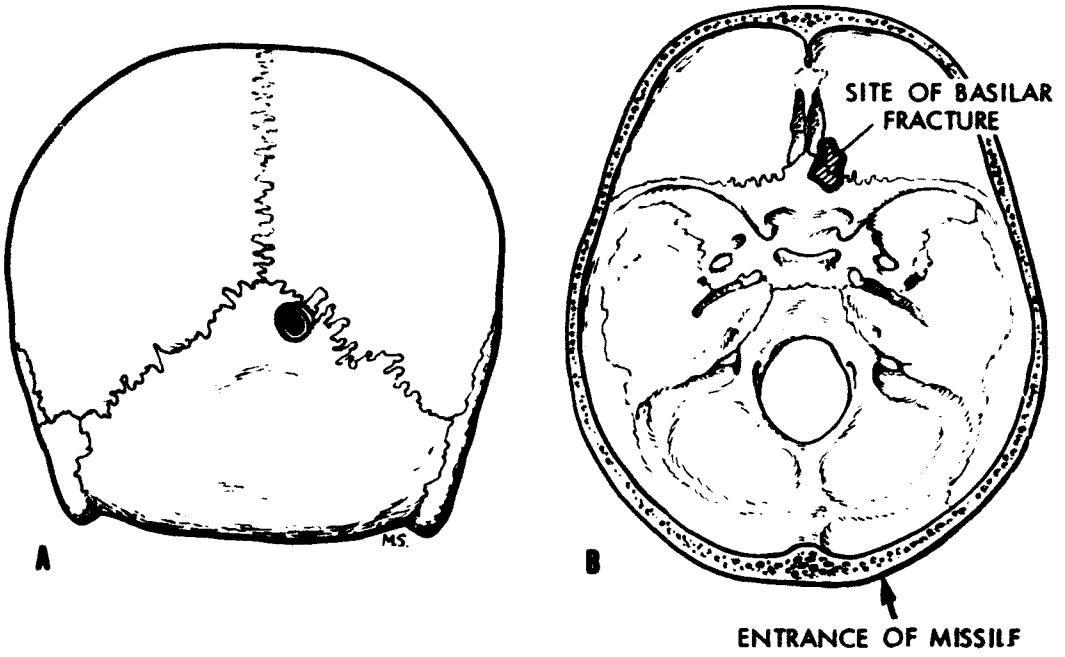
Protocol

GROSS ANATOMY

Skull.—There was a round opening, about 1.2 cm. in diameter, in the upper part of the right parieto-occipital region, about 1.5 cm. from the midline (fig. 118A). The defect was mainly in the occipital squama but also involved the right leg of the lambdoid suture. There was a discontinuous fracture in the medial portion of the right lesser sphenoid wing and the right cribriform plate (fig. 118B). There was some coagulated blood in the epidural as well as in the subdural space.

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[20] See footnote 14, p. 351.



IGURE 118.—A. Approximate location of wound of entry in skull. B. Approximate site of discontinuous fracture of cranial basis.

Brain.—An irregularly torn wound of the right hemisphere in the region f the junction of the occipital and parietal lobes, which involved the lobulus arietalis superior and the supramarginal gyrus (fig. 119), corresponded to the ole in the skull. The wound track led deep into the right hemisphere and made s exit in the region of the substantia perforata anterior. A thin, curved, regularly shaped shell fragment, with sharp edges, measuring about 1 by 1.2 n. in diameter, was found in the vicinity of the substantia perforata anterior. ilaterally, there was an area of laceration and contusion on the orbital surface f the frontal lobe, corresponding to the gyrus rectus, the olfactory bulb and act, and the adjacent orbital gyri. This lesion measured about 3 by 4 cm. nd was presumably related to the area of the discontinuous fracture.

Sections of the brain revealed extensive destruction of the right cerebral emisphere with damage to the ventricular system and the corpus callosum igs. 120 and 121). The structures involved were the third ventricle, the right alamus, including the diencephalomesencephalic border zone, the anterior art of the right hypothalamus, and a large part of the right striate body. Iassive intraventricular hemorrhage had occurred, and a cast of clotted blood lled the entire ventricular system. Widely scattered petechial hemorrhages ere seen throughout the entire brain substance, and there were small lacerations the right cerebellar hemisphere.

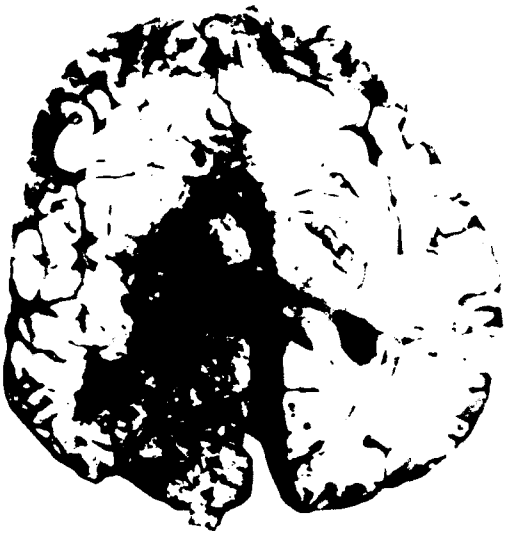
MICROSCOPIC ANATOMY

In the area of laceration at the base of the frontal lobes was a very marked ifiltration with polymorphonuclear cells throughout the edematous cortex,



AFIP 100824

FIGURE 119. Dorsal view of brain showing wound in right parieto-occipital region.



AFIP 100826

FIGURE 120. Oblique section of brain showing extensive destruction of right diencephalon and deep parts of right parietal and occipital lobes.



AFIP 100829

FIGURE 121. Coronal section showing extensive damage to right diencephalon and involvement of the third ventricle.

possibly due to a beginning infection. In the region of the parieto-occipital wound were cortical areas of edematous necrosis, giving the picture of a foamy ground substance in which the highly shrunk necrotic nerve cells, glial elements, and polymorphonuclear cells were enclosed (fig. 78). The walls of the sylvian aqueduct were edematous and displayed so-called ameboid glia cells in the subependymal cell plate (fig. 122).



AFIP 106224

FIGURE 122.—Edema and hemorrhage in wall of sylvian aqueduct, with swollen astrocytes. Blood cells are also present in the lumen of the aqueduct. Hematoxylin and eosin stain. (× 400)

The cerebellar cortex in the region of the lacerations of the right cerebellar hemisphere showed dense infiltration with polymorphonuclear cells in the molecular layer (fig. 81). There was marked leptomeningeal reaction in the basal frontal and the cerebellomedullary regions, with large masses of polymorphonuclear leukocytes.

Comment

In the first of these cases (case 1), in addition to petechial hemorrhages and grosser hemorrhages from lacerated larger vessels into the missile track, subarachnoid space, and entire ventricular system, early changes of the ischemic or chronic type were conspicuous in both the cortex and the basal ganglia. There were also mild changes in the choroid plexus. The lesion of the cranial bones

included so-called discontinuous fractures of the lamina cribrosa and the sella turcica.

In the second case (case 10), the conspicuous features were widespread destruction of internal structures of the right hemisphere, basal lacerations of both frontal lobes, including the olfactory bulb, due to discontinuous fracture, and, in the histologic picture, very marked infiltration with polymorphonuclear cells. The temperature of 102° F. may have resulted from involvement of the centers in the anterior hypothalamic region but may also have resulted from infection. The fracture in the right cribriform plate and medial portion of the right sphenoid wing was discontinuous in a sense, although, in this instance, it may have been caused by direct impact of the missile.

In view of the intraventricular bleeding and the extensive destruction of the diencephalon, it is indeed remarkable that this patient did not die immediately after he had been wounded.

These two cases demonstrate, both clinically and pathologically, the intrinsically hopeless character of such head wounds. Both patients were practically moribund when they were admitted to the hospital. No experienced surgeon would have been tempted to operate upon either of them, for their vital signs were never stabilized. The most remarkable feature of both cases is that the patients survived the initial trauma.

The comparison between the anatomic areas of destruction in the two cases is interesting (table 19). In the first case, the destruction involved the frontal and temporal lobes, corpus striatum, corpus callosum, septum pellucidum, and both lateral ventricles. The patient survived 30 minutes. In the second case, the destruction was even more widespread than in the first. The injured structures included the frontal, parietal, and occipital lobes; the corpus striatum and corpus callosum; the lateral ventricles and the third ventricle; the thalamus and hypothalamus; and the mesencephalon and cerebellum. The brain showed far greater mutilation than the brain in the first case, yet this patient survived for about 31 hours. Both cases are excellent illustrations of the futility of attempting to postulate the lethality of mortal wounds of the brain solely upon the anatomic areas destroyed by the missile.

As has been pointed out previously, one can say with finality that a wound of the brain is mortal only when the lesion involves, directly or indirectly, the cardiac and respiratory centers of the medulla oblongata. Probably most of the wounded who died immediately on the battlefield had suffered direct damage to these structures. As table 19 shows, not a single case in this series presented gross evidence of damage to either the medulla or the pons.

GROUP II (SURGICAL)

In each of the 14 cases in the second group, the neurosurgeon, by operation, attempted to convert what appeared clinically to be a hopeless situation into a more favorable one. The following protocols illustrate both the type of cases in this group and the type of surgery performed in them.

Case 7

Clinical History

This soldier was found unconscious on the battlefield early in the morning; no definite history could be obtained, but it was believed that he had been wounded several hours before. When admitted to an evacuation hospital at 5:30 that afternoon, he was semicomatose but could, with assistance, carry out simple commands. He would not or could not phonate. It was recorded that he had left hemiplegia, left hemianesthesia, and, probably, left hemianopsia. The pulse rate was 120, the respiration 40, and the blood pressure 110/70. There was a penetrating wound in the medial portion of the left orbital ridge. The globe of the left eye was preserved.

A lengthy debridement of the missile track through bilateral osteoplastic flaps was commenced at 9 o'clock that evening. Troublesome bleeding was encountered from the longitudinal sinus. Both cribriform plates and the adjacent dura and brain were extensively involved in the wound of entry. Repeated attempts to tap the anterior horn of the lateral ventricles were unsuccessful. Because of the rapid deterioration of the patient's condition, efforts to find a hematoma farther back along the track were abandoned.

He died 25 hours after tagging, hence between 25 and 30 hours after wounding. Autopsy was performed 7 hours later.

Protocol

GROSS ANATOMY

Skull.—Few details were available concerning the skull injury. A bullet wound had its entry at the left nasal bridge, causing damage to the left frontal sinus and the wall of the left orbit. The tip of the bullet was found in the right occipital lobe of the brain. The ethmoid bone, including the cribriform plate and the crista galli, were shattered. The dura was extremely tense. The opened longitudinal sinus was free of thrombus formation. There was a considerable amount of clotted blood in the posterior cranial fossa, and blood surrounded the cervical part of the spinal cord. The source of this hemorrhage was not determined.

Brain.—The brain was rather firm. A small amount of subarachnoid hemorrhage was noted. The orbital surfaces of both frontal lobes, including the olfactory bulbs and tracts, were lacerated. Coronal sections of the brain showed damage to the medial and basal frontal cortex of both frontal lobes. The missile track (fig. 123) could be followed into the right hemisphere, where extensive destruction of the right striatum, pallidum, and internal capsule was found (fig. 124). The septum pellucidum was involved anteriorly, the laceration including both right and left lateral ventricles. The anterior parts of the right thalamus and hypothalamus were also involved, without direct damage to the periventricular diencephalic structures. Farther caudad, the

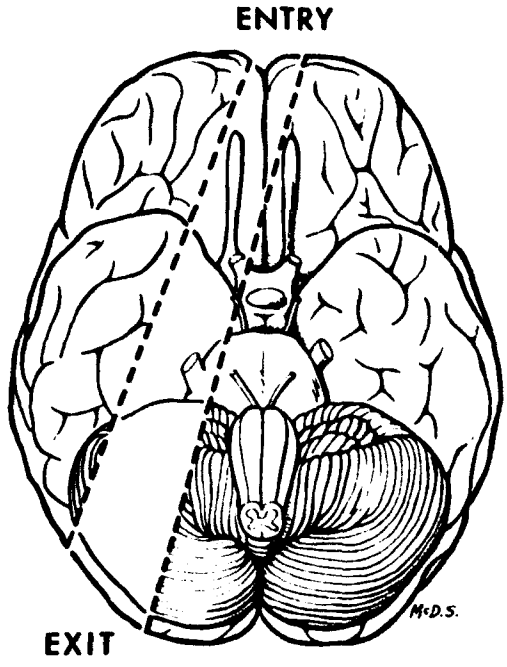


FIGURE 123.—Diagram of missile track through brain.



AFIP 103960-33b

FIGURE 124.—Coronal section showing damage to right striatum, pallidum, and internal capsule.

track ran laterally and spared the thalamic centers, destroying, however, the posterior limb of the right internal capsule and the caudal parts of the lenticular nucleus.

In the right occipital lobe, the track ran dorsal to the posterior horn of the lateral ventricle (fig. 125), almost reaching the occipital pole. There were



AFIP 103960-33a

FIGURE 125.—Coronal section showing missile track in right occipital lobe.

massive hemorrhages into the ventricular system, and numerous petechial hemorrhages were scattered throughout the entire brain substance.

MICROSCOPIC ANATOMY

The histologic findings corresponded, in general, to the findings described in the previous case in which survival time was about 31 hours. The usual zones of hemorrhage and edema were found around the large areas of destruction of the basal ganglia. Numerous polymorphonuclear cells and a few large mononuclear elements were scattered throughout the hemorrhagic regions (fig. 104). The zone of manifest changes extended to a depth of about 8 mm. along the missile track. In addition, the ependyma, including that of the fourth ventricle, showed edema. The choroid plexus displayed moderate edema, and in some of the villi the epithelial cells appeared flattened. In this case, as in others, the zone of complete destruction of nerve fibers was characterized by a disintegration of the axons, which had lost their property of being impregnated by silver salts. A fine granular detritus was interpreted as indicating immediate primary necrosis. In this case, however, the secondary changes in the nerve fibers in the marginal zones of the lesions had become very conspicuous, especially among the large fibers in the hemorrhagic

zones of the internal capsule. Affected axons displayed a tortuous course, with varicosities and occasional fragmentation. End bulbs, as described by Rand and Courville,[21] could be found (fig. 87). In some instances, the swollen myelin sheaths showed affinity for diffuse impregnation with silver salts (fig. 85A and B). The leptomeninges showed a mild reaction; the meshes of the arachnoid were in many regions filled with erythrocytes and white blood cells.

Case 12

Clinical History

This patient, as the result of small-arms fire, sustained a perforating wound running from the right frontal to the right occipital region. The huge compound comminuted fracture involved the right frontal, temporal, parietal, and occipital bones; lacerated cerebral tissue herniated through a large defect present in its center. The patient was said to have been in coma and in shock when he was admitted to the hospital, but no record of his vital signs was available. After 24 hours, he became restless, and left hemiplegia was evident. The right pupil was dilated and fixed. The neck was stiff. The left arm and leg were flaccid; the left biceps and triceps reflexes were hypoactive as compared to those on the right, while the left knee and left ankle jerks were relatively hyperactive. The Babinski and Hoffmann reflexes were positive bilaterally. Abdominal reflexes were not obtained on the left. Roentgenologic examination disclosed a large bony defect which involved all the bones on the right side of the calvarium. Widely separated fissures ran both forward and backward, as well as across into the left temporal region.

The condition remained essentially unchanged, and the wound was debrided 52 hours after injury. The operator's note follows:

> Under ether anesthesia the wound of the right cerebral hemisphere was debrided, the herniations of pulp brain at the wounds of entry and exit having first been excised. Within the cranium devitalized masses of right cerebral hemisphere were removed, together with numerous bone fragments and large blood clots which were found in the extradural, subdural, intracerebral and intraventricular portions of the wound. Wounds of entry and exit were thus connected. After removal of pulp brain, the futility of any procedure was seen. The extensiveness of destruction was markedly out of proportion to the clinical signs, save for depth of coma. The brain wound extended into the body of the lateral ventricle, superiorly to the longitudinal sinus, posteriorly into the occipital lobe, and deep into the temporal lobe. It was necessary to clip large branches of the right middle cerebral and the main trunk of the right posterior cerebral arteries. The open ventricle was washed as free of clots and debris as possible. The dura was not sutured; scalp closed by plastic procedure. Four thousand cubic centimeters of whole blood were required during the operation to compensate for the blood lost.

Death took place 12 hours after operation and 65 hours after wounding. Autopsy was performed 9 hours later.

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[21] See footnote 13, p. 348.

Protocol

GROSS ANATOMY

Skull.—There was an irregular opening in the parietal and temporal bones on the right side, measuring from 12 to 15 cm. in different diameters and involving adjacent parts of the occipital, frontal, and sphenoidal bones (fig. 126). Irregular fracture lines from the borders of this defect radiated in all directions.

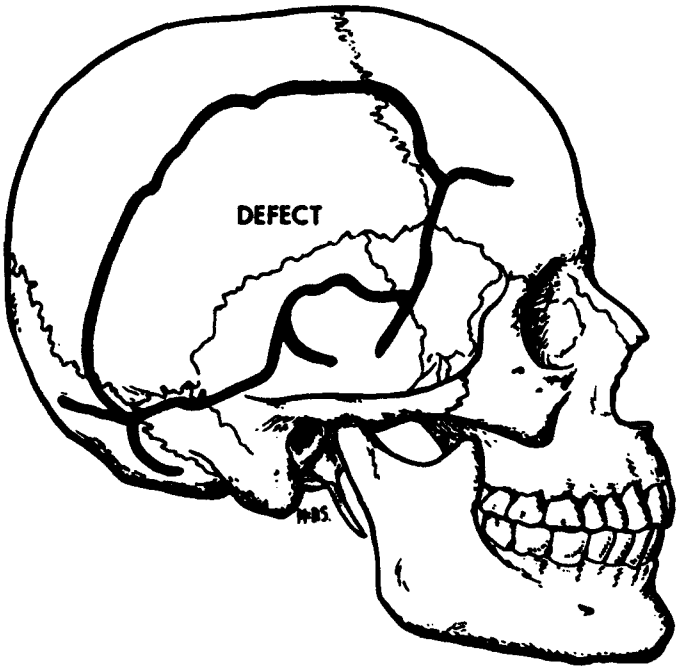
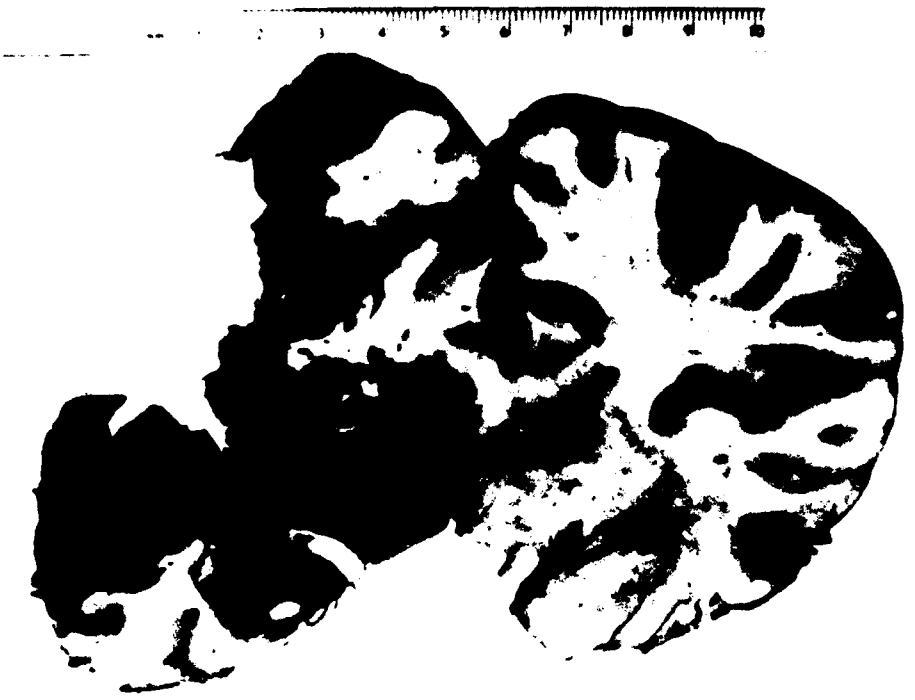


FIGURE 126.—Approximate outline of skull defect.

The large opening was covered by adjacent mobilized tissues held together by sutures, and there was free and interstitial hematoma formation in the wound area. In the dura mater was a large, irregular opening, and several silver wire clips were in the edge of the dura. In the occipital region, the subdural space contained a massive, loose hematoma.

Brain.—The right parietal lobe of the brain was severely lacerated, and the shreds of brain tissue were covered by blood coagulum. The brain wound extended dorsad into the occipital lobe and rostrad along the sylvian fissure into the temporal lobe and the base of the frontal lobe.

Coronal sections of the brain showed destruction of the greater part of the right parietal lobe (fig. 127), except for the region of the gyrus cinguli and neighboring structures on the medial surface. The right occipital and temporal lobes were also severely damaged, while the lesion of the frontal lobe was chiefly cortical and for the most part involved the right inferior frontal gyrus. The lesion reached the body and the inferior horn of the right lateral ventricle. The destructive lesion inolved also the right half of the diencephalon, including



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FIGURE 127. Coronal section showing laceration and extensive destruction of right parietal lobe. The right temporal lobe is also severely damaged and the right diencephalon is included in the hemorrhagic zone.

pretectal structures, but did not reach the third ventricle. There was hemorrhage into most parts of the ventricular system, and solid blood clots were found in the third and fourth ventricles.

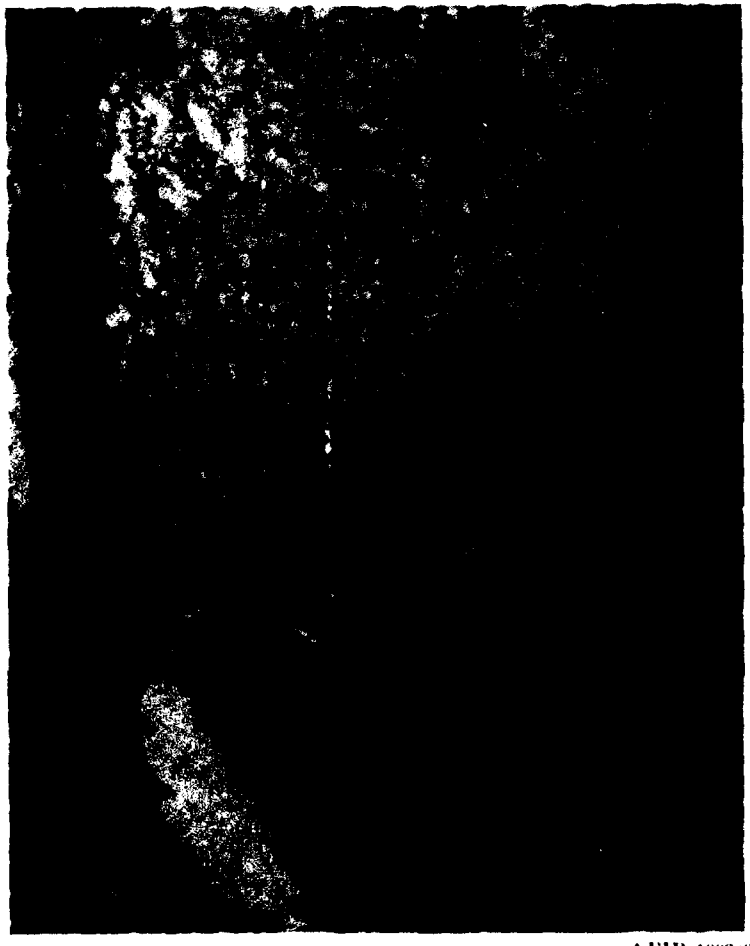
MICROSCOPIC ANATOMY

In the neighborhood of the cortical lacerations, the ganglion-cell changes were far advanced and had resulted in hyalinized shrunk remnants and ghost cells (fig. 79). Glial reaction was barely noticeable, but infiltration with polymorphonuclear and large mononuclear cells was extremely conspicuous (fig. 105). In the white substance of the occipital and parietal lobes, there were, in addition to areas of interstitial hemorrhage, several areas of necrosis with beginning demyelinization, presumably perivascular, characterized by swelling as well as by partial loss of tinctorial properties (fig. 128). Clusters of polymorphonuclear cells, as well as small and large mononuclear cells, were seen in the central parts of the necrotic patches (fig. 129); compound granular cells, however, were nowhere in evidence. In some subcortical regions of the white matter of the damaged occipital lobe, and in the neighborhood of hemorrhages, were microglia cells which showed indications of transformation into rod cells. Nerve fibers, especially those of larger caliber, displayed marked sinuosity of the swollen axon with varicosities, possible fragmentation, and so-called end bulbs (fig. 88). It was difficult to ascertain whether the frag-



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FIGURE 128.—Perivascular infiltration, necrosis, hemorrhage, edema, and demyelinization in white matter of damaged parietal lobe. Hematoxylin and eosin stain. (× 210)



AFIP 100200

FIGURE 129.—Necrotic area with demyelinization in white substance of damaged parietal lobe. Clusters of polymorphonuclear leukocytes and mononuclear cells can be seen in the center of the demyelinized area. Hematoxylin and eosin stain. (× 210)

mentation was the result of actual interruption of the axon or of extension of the loops beyond the plane of the sections (fig. 86A and B). Marked edema and hyalinization were observed in the choroid plexus.

Case 17

Clinical History

This soldier was struck in the right temple, right flank, and buttocks by shell fragments. When he was admitted to an evacuation hospital 5 hours later, he was dazed, very restless, and uncooperative. The blood pressure was 62/40. He moved all four limbs, in each of which moderate extensor spasm was observed. The deep reflexes were equally hyperactive; the superficial reflexes were absent. No pathologic reflexes were present. The pupils were equal and reacted promptly to light. Roentgenologic examination disclosed a metallic foreign body measuring 3 by 2 by 1.5 cm. embedded in, and depressing, the right temporal bone, just above the external auditory canal, for approximately 1.5 cm. Small shell fragments were also visible in the buttocks and in the right side of the twelfth dorsal vertebra.

After repeated whole-blood transfusions, the patient had improved to such an extent that the wounds in the flank and buttocks could be debrided, and the right kidney, which was found to be shattered, could be removed. By this time, he had received 2,500 cc. of type O blood. The temperature was 99.2° F., the pulse rate 100, the respiration 28, and the blood pressure 116/76.

The following day, the vital signs were unchanged, but the patient remained disoriented and confused. The pupils were equal and reacted to light. Spasticity of all extremities had increased, as had hyperreflexia.

Fifty-one hours after wounding, the head wound was debrided, the indriven bone and metallic fragments were removed, and the dural defect was repaired by a graft of temporal fascia. An additional 2,000 cc. of whole blood were administered during the 2-hour operation. The right trigeminal nerve was found to be lacerated. No note was made concerning the ventricle.

By the following day, the temperature had risen to 102°–104° F., the pulse rate was 120, and the respiration was 20–26. The blood pressure was 135/84. The patient became more stuporous. The lungs were filled with coarse rales, and cyanosis was quite evident. His condition continued to worsen; that night, the lumbar spinal fluid pressure was 580 mm. H_2O, although the temporal flap was said to have been pulsating.

Death occurred 85 hours after wounding. Autopsy was performed 2 hours later.

Protocol

GROSS ANATOMY

Skull.—A defect in the right temporal squama measured 2.5 by 4.5 cm.; its posterior margin involved the external anditory canal (fig. 130). There

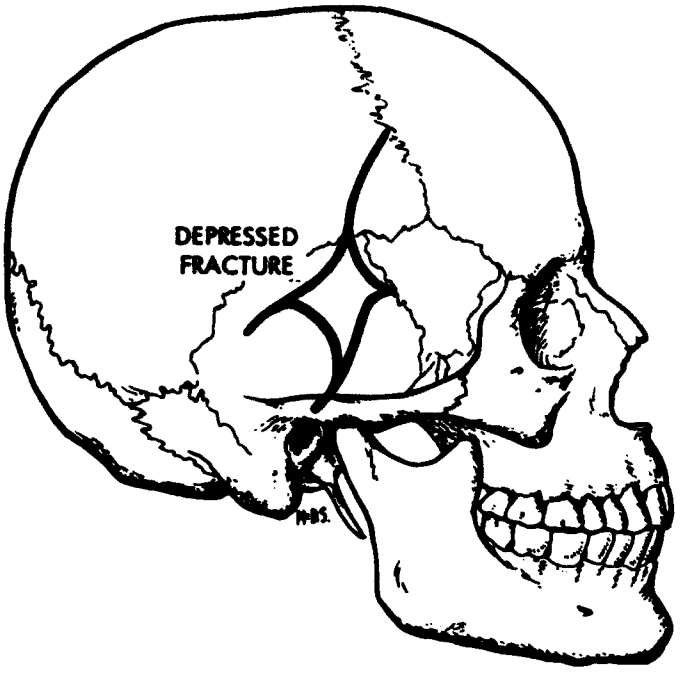


FIGURE 130.—Approximate location of defect in right temporal squama.

was also a defect resulting from craniotomy in the right parietal bone. Its anterior margin was at the coronal suture, and its medial margin was 6 cm. from the midline. A bone flap was found in the defect. A large defect in the dura of the floor and lateral wall of the right middle cranial fossa had been incompletely repaired with a fascial graft.

Brain.—A laceration measuring about 7 by 7 cm. and covered by a blood clot was found in the inferior and lateral surface of the right temporal lobe.

Coronal sections of the brain showed extensive damage to the right temporal lobe, with involvement of the inferior horn of the lateral ventricle and ventricular hemorrhage. The brain appeared generally hyperemic, and there were many scattered petechial hemorrhages throughout the brain substance. There was marked generalized swelling of the brain.

MICROSCOPIC ANATOMY

The histologic changes in this case did not differ essentially from those found in other cases with a survival period of more than 3 days. In the vicinity of the laceration, there was still considerable edema with severe ischemic changes in the ganglion cells and a distribution of glia cells suggesting so-called neuronophagia (fig. 74). Occasional compound granular cells and numerous ameboid glia cells were found in the areas of hemorrhage and necrosis.

Polymorphonuclear cells were also widely distributed through the damaged areas and their surroundings. Blood vessels in some regions of cortex and white substance were filled with rod-shaped micro-organisms, a few of which were also found in the brain substance around the vessels. Some of these

micro-organisms had round spores at one end. It is likely that they represented post mortem contamination. This view is supported by the fact that the tissue reaction in contaminated areas did not show any difference from that of noncontaminated areas or that of other cases in which micro-organisms were not found in the brain.

Comment

In the first of these cases (case 7), the histologic picture did not show new details or further developments of the reaction of the tissues to trauma except for the very conspicuous secondary changes in the nerve fibers. From the standpoint of localization, the extensive defect of the corpus striatum throughout the length of the right hemisphere is noteworthy. Left hemiplegia and left hemianesthesia were doubtless the result of the complete involvement of the right internal capsule, including the retrolenticular portion. Exact data concerning the temperature were not available, but a note made upon the patient's admission to the evacuation hospital was to the effect that he was warm. It is possible that this statement referred to a hyperthermia resulting from a lesion of the rostral hypothalamus.

While nothing could have saved this patient, many experienced neurosurgeons would question the wisdom of using bilateral osteoplastic flaps to expose the intracranial structures, as was done in this case. Such a procedure is far more time consuming than the more conventional procedure of rongeuring away enough bone at the site of entry of the missile to expose the traumatized areas.

Both ventricles were filled with clotted blood, which explains why neither could be tapped. While the lethal outcome in this case would appear to have been both inevitable and merciful, it would probably be better, when a ventricular clot is suspected at such an operation, to expose the ventricle for direct inspection and for debridement, if necessary. Wounds of the ventricle could sometimes be diagnosed, or their presence at least suspected, by the apparent path of the missile or by the escape of cerebrospinal fluid from a wound in the cerebrum.

The second of these cases (case 12) represents a noble effort on the part of a surgeon who dealt skillfully with what was perhaps a lethal wound from the beginning. It is another extremely striking example of relatively long survival (2½ days) following a very extensive wound of the right diencephalon with intraventricular hematoma. In view of the damage to the right pretectal area, it is significant that the right pupil was found dilated and fixed.

In this case, very definite foci of beginning demyelinization could be recognized in the white matter. Compound granular corpuscles, however, had not yet been formed.

The third of these cases (case 17) is the only one in the series in which the evidence at post mortem examination might indicate that with more skillful surgery, applied at the right time, the patient's chances of survival might have been materially enhanced. Here, again, the surgeon elected to employ a bone

lap for exposure, whereas the conventional practice is to enlarge the cranial lefect at the point of entry of the missile. Furthermore, the patient's condition vas allowed to deteriorate without any measures being employed to control the obviously mounting intracranial pressure. This criticism, however, is made vithout knowledge of the circumstances under which the surgeon was working, ior is there any possibility of knowing just what part the associated injuries olayed in the fatal outcome.

The histologic changes present in this case corresponded to those present n other cases of more than 3 days' duration. The intravascular and extravas-ular presence of micro-organisms was interpreted as post mortem contamnation.

The clot found at autopsy over the temporal lobe and the wound entering he right temporal horn of the ventricle with the contained blood suggested hat hemostasis at operation had not been complete. Inadequate hemostasis ould also account for the steadily downhill course, the mounting intracranial ressure, and the final fatal outcome.

Since retrospect is always better than prospect, it is easy for the neurourgical armchair strategist to sit back, read the clinical and pathologic records f these 14 surgical cases, and remark that they represent a perfectly useless ffort. They do. But they seem less useless when one remembers that experinced neurosurgeons were handling them, surgeons who had seen and operated n dozens of similar patients who presented similar clinical findings. Then the *notivation of the surgical effort becomes* apparent. These surgeons had seen nany similar patients survive appropriate surgery. This is not a fact which an be substantiated statistically, but it is well known to every neurosurgeon vho gained his experience in World War II.

The conclusion of the whole matter is that there must be an extraordinarily arrow zone which divides nonmortal from mortal wounds of the brain. Many actors influence the outcome. Some are known. Some are not. Among the nown facts, the three which follow seem particularly pertinent to this study:

1. Speed of transport to a hospital installation in which proper equipment nd experienced personnel are available for expert medical and surgical care is sually essential for the best results in serious craniocerebral injuries. In 14 f these 24 cases, it is known that from 5 hours to 3 days were required for the atients to reach the level of the evacuation hospital. In 4 other cases, the xact hour of wounding was not recorded, but the evidence indicated that the imelag was more than 5 hours. Thus, in 19 of the 24 fatal cases in this series, he timelag was unduly prolonged, so prolonged in 3 cases, in fact, that the atients arrived in evacuation hospitals with active meningitis and cerebritis.

It is the first hours after wounding which are often of the greatest imporance in craniocerebral injuries because it is during this period that intracranial leeding is occurring and intracranial pressure is mounting, often to the point f irreversible cerebral damage from anoxia. Speed of evacuation, however, is ometimes not controllable. In every military campaign, it depends to a large xtent upon the character of the fighting and the terrain. These factors were

undoubtedly responsible for much of the delay in transit observed in these and other cases during the last phase of the Italian campaign.

2. The skill of the surgical team is unquestionably an important factor in the outcome of craniocerebral injuries. Surgery of this kind requires teamwork. Every member of the team— the surgeon, the assistant surgeon, the sterile nurse, the circulating assistant—must know his or her job and must perform it with speed and skill. The surgeon must have learned that many of the time-consuming rituals of the civilian neurosurgical operating room have no place in a military hospital. He must operate with great accuracy, but he must lose no time about it. Most important of all, he must have learned how to achieve perfect hemostasis before the wound is closed. The most frequent evidence of surgical error observed in these 24 autopsies was the presence of intracranial blood clots, undoubtedly to be explained by imperfect hemostasis at operation.

3. A capable anesthetist ranks in importance second only to the surgeon. His skill in maintaining a free airway, in using just the right amount of the anesthetic drug, and in administering intravenous fluids and blood in just the right amounts at just the right time is a factor of inestimable value in the end results of intracranial surgery for combat-incurred wounds.

SUMMARY AND CONCLUSIONS

This chapter is based upon a clinicopathologic analysis of 24 mortal craniocerebral injuries caused by high-velocity missiles. The survival after wounding varied from 30 minutes to approximately 10 days. The analysis of this series of cases warrants the following conclusions:

1. The majority of the cerebral lesions in this series directly involved the ventricular system. Damage of the cerebral hemispheres was present in all cases, but comparatively few lesions of the diencephalon and the cerebellum were observed, and the mesencephalon was involved in only a single case. Penetrating missile wounds of the brain caused local damage of the lacerating and contusing type along the track of the missile and in its immediate vicinity, as well as additional damage of the contusion type at varying, and often at considerable, distances from the wound.

2. In the studies made of the reaction to trauma and the histopathologic changes of traumatic encephalitis affecting the various ectodermal and mesodermal components of the central nervous system, of particular note was the relatively sluggish reaction of the brain to injury as compared with the reaction of other organs of the body to trauma.

3. Hemorrhage, edema, degenerative processes, and infiltration with hematogenous and histogenous elements were the predominant features during the first 4 days after wounding. After the fourth day, proliferation of capillaries marked the beginning of reparative processes. Compound granular corpuscles began to appear after 3 days.

4. In cranial lesions connected with penetrating and perforating wounds of the head, so-called discontinuous fractures are an important complicating

feature. When such fractures occur in the region of the cribriform plate and orbital roof, which is a common location, the suprajacent dura and brain are frequently damaged. It is thus easy for cerebrospinal fluid and blood to escape and easy, at the same time, for infection, air, or both, to gain entrance. The overlying dura and brain may be torn or contused. These injuries, together with contusions, hemorrhages, and occasionally lacerations at some distance from the primary wound, serve to emphasize the fact that, in injuries caused by high-velocity missiles, the resultant lesions are by no means always confined to the missile track.

5. Severe injury of the brain tissue and of its envelope is not necessarily confined to the missile track but may occur at a considerable distance from it, in the form of laceration, contusion, and hemorrhage, alone or in combination. In this series, clots of varying size were almost invariably present in the missile track. If the ventricles were injured, blood in varying amounts was almost invariably found in the ventricular wounds also. Extradural hemorrhages were common but in most instances were small and of little clinical significance. Subdural hematomas, while sometimes large, were for the most part relatively small.

6. The pathway of a high-velocity missile is not smooth but is ragged and irregular and is often made multiple by the fragmentation of the missile or the propulsion of bone fragments. These circumstances usually render complete operative removal of devitalized tissue a practical impossibility. In this same connection, it is noteworthy that much of the brain tissue destroyed by injury is not immediately softened and is not readily distinguishable from uninjured tissue if operation is performed promptly or even within a period of 24 hours. Areas of this kind probably correspond to zones of delayed disintegration.

7. The primary purpose of this investigation was to establish the clinical criteria of mortal brain wounds. Neither its results nor the results of any other military experience, so far as is known, have established criteria to indicate to the clinical neurosurgeon the particular craniocerebral wounds which are apt to be lethal.

ADDENDUM

The following case history is as good an illustration as could be found of the practical impossibility of determining when an injury of the head is lethal and when it is not. It was related, on request, by Dr. Francis A. Carmichael, Jr. (formerly Major, MC, AUS), who served in the European theater, and it is published as he related it, without editorial revision.[22]

As I recall, I encountered this patient, who was then an infantry lieutenant, during the Battle of the Bulge. This particular evening I was making rounds of the new casualties, as I had instructed my various assistants to do, and in order to set a good example I thought I would make a few myself on the way toward my hut in the evening.

This patient presented a striking picture from the start, inasmuch as he was swathed in a very voluminous turban type of bandage completely encircling his head, the bandage being

[22] This patient visited me in 1955, and he is, as Dr. Carmichael intimates in his case history, a most remarkable illustration of what can be accomplished by neurosurgery and plastic surgery.—R. G. S.

liberally soaked in blood. Upon removing this turban, I was utterly and completely horrified at the underlying mess which was a conglomeration of mud and blood, but by gentle palpation with the gloved finger I was able to ascertain that there was virtually no skull in this mass of debris since everything beneath the finger was soft over a very wide area. Having no idea what to do about the situation and wishing some time to regroup my own thoughts on the matter, I quickly applied a fresh turban and retreated to the privacy of my nissen hut for meditation on the subject.

The patient himself was able to talk, although he was hemiplegic on the left side. He was disoriented as to time and thought that he had been wounded a day or two previously in France. As a matter of fact, as I ascertained later from Colonel Spurling, he had been wounded that very morning, and Colonel Spurling had come upon him in France and ordered his immediate evacuation, not knowing, of course, at which hospital he might arrive.

On the subsequent days, the lieutenant was able to tell me this story; namely, he was going down a road with his platoon and a German tank came into view. It was terrifically muddy and slushy that day, and he remembers that the turret gun of the tank was pointed directly at him at rather close range. The gun was discharged, there was a deafening concussion, and the patient remembered nothing until some time later when he was awakened by the stretcher bearer. He was unable to say definitely whether the 88-mm. tank gun discharged or struck him directly or whether it concussed him and he fell into the muddy road where his head was run over by the treads of the German tank. From the resulting trauma, I would judge that the latter theory was probably the more correct.

The following morning, having the lieutenant on the schedule, I uncovered the wound again under better circumstances in the operating room. Here, after copious lavaging with saline and cotton pledget manipulation, I was able to uncover the wound in its entirety and effect some sort of a toilet of the entire debris.

The situation was as follows: The scalp, the skull, and the dura had been completely and cleanly avulsed from an area about three-fourths of an inch above the superciliary ridge on the right side, extending to beneath the zygoma laterally up to the sagittal suture in the midline and back to encompass the lambdoidal suture behind so that this area of the brain was completely exposed, being denuded of the dura, of the skull, and of the scalp, all of which were missing so that, in essence, the rather intact cortex was presenting itself to the atmosphere. The cortex was not badly damaged but was ecchymotic and bruised here and there with minor lacerations.

On this occasion, I accomplished nothing more than debridement; there was nothing to repair, nothing to sew to, and no way to cover the wound. So, again, I enclosed the patient in the customary turban dressing, after having overlaid the cortex with petrolatum-impregnated strips of gauze.

Another onslaught to the problem was made the following morning, at which time, with the patient in the upright position, I uncovered the wound and prepared his back, thinking that perhaps I should be able to swing some sort of a covering flap up from the intrascapular or deltoid area of one side in order to cover the wound. This also proved to be a failure, since there was no way to acquire enough coverage or skin by my meager methods to even start to close the wound.

On the third morning, another attempt was made, and this time, by some scalp-releasing incisions and splitting of the scalp areas, it was possible to cover approximately one-third of the denuded area but no more. There was nothing with which to make a dural repair and, again, a similar dressing was applied.

This patient was with us some time, I should say over all at least 6 weeks, and in the course of this time there was, as might be expected, some rather superficial abscess formation in various sectors of the cortex, which responded quite well to incision and drainage and to continuous wet packs of Azochloramid (chlorazodin).

Finally, in utter despair and panic, on one occasion, with the aid and abetment of the plastic surgery department, we took off split-thickness grafts from the abdomen and applied them directly to the exposed and now granulating surface of the cortex, there being no

intervening arachnoid or dura. To my astonishment, these grafts took, and we were able in a relatively short time to accomplish complete closure, flimsy as it was, by this method of split-thickness grafting.

As might be expected, as soon as coverage was obtained, the problem of edema and abscess formation vanished quite rapidly under the antibiotic treatment and local packs of Azochloramid which had been instituted. Coincidentally, with this favorable turn of events, the patient's memory cleared quite well and he showed some recovery of strength in the heretofore hemiplegic left extremities. I do remember a personal touch at this time, when we were in the throes of getting this patient over such a grievous injury, that word arrived at the hospital that his father had suddenly died in the States. For this reason, we redoubled our efforts and without any hesitation sent the patient to the Zone of Interior. I was, of course, at this time worrying what the plight of the next surgeon would be when he attempted to remove the split-thickness grafts which we had applied directly to the cortex of the brain. There had been, in my experience, no precedent for this type of frantic operation such as we had done.

Dr. Malvin F. White (formerly colonel, MC, AUS) had the courtesy to write me some years later, saying that when it came his turn to remove the split-thickness grafts which we had applied to very great areas of the right cerebral hemisphere, he found the subarachnoid space had re-formed, that the brain or cortex on that side was bathed in a layer of spinal fluid, and that the grafts were removed with considerable ease, more or less as if they had been designed in that fashion. Dr. White was thereafter to carry out some very brilliant reconstructive work on this patient.

The patient had apparently been quite grateful to all concerned and, I understand (as he does to me) remembers us with a kindly letter each Christmas. He once visited Kansas City, Mo., and called me, but unfortunately, I was out of town that weekend and did not get to see him.

The patient now has almost virtually recovered from the left hemiplegia; he is quite well and happy, has been promoted to captain, and has been retired on full disability. It has been deeply gratifying to me and I know of interest to you, that this patient has not in any way suffered any psychiatric complication from this mutilating and somewhat horrifying wound, the most spectacular that I encountered during my military career.

APPENDIX A

Course of Instruction in Neurosurgery

Phase I

Fundamentals of Neurosurgery (192 hours), Neurological Institute, College of Physicians and Surgeons, Columbia University

1. Neuroanatomy, 64 hours, didactic lectures, amphitheater exercises.

Scope: Relations of skull to brain; of spinal column to spinal cord and nerve roots; external and internal anatomy of brain; blood supply of brain; dissection of brain, spinal column, and principal nerve trunks.

References: Mettler, F. A.: Neuroanatomy, St. Louis, C. V. Mosby Company, 1942; Krieg, W. J. S.: Functional Neuroanatomy, Philadelphia, Blakiston Company, 1942.

2. Neurophysiology, 16 hours, didactic lectures, amphitheater demonstrations.

Scope: Fundamental activity of nervous tissue; results of lesions of various regions of brain and at various levels of spinal cord.

Reference: Fulton, J. F.: Physiology of the Nervous System, New York, Oxford Press, 1938.

3. Cadaver surgery, 24 hours, didactic lectures, amphitheater demonstrations.

Scope: Simulated types of head and nerve injuries; surgical approaches and techniques of repair.

4. Neuropathology, 14 hours, didactic lectures, amphitheater demonstrations.

Scope: Reaction of nervous tissues to injury; examination of gross specimens of brain and cord injuries; suppuration; histopathology of traumatic lesions.

5. Medical examiners' demonstration, 4 hours.

Scope: Autopsies on head and nerve injuries from accident cases at large city hospitals. Officers will attend in small groups.

6. Neurological examination, 10 hours, lectures, amphitheater demonstrations.

Scope: Physiologic significance of pathologic signs; individual drill in standard neurologic examination; principles of localization.

References: Spurling, R. G.: Practical Neurological Diagnosis, with special reference to the Problems of Neurosurgery, Springfield, Charles C Thomas, 1940; Monrad-Krohn, G. H.: The Clinical Examination of the Nervous System, London, H. K. Lewis, 1938.

7. Principles of clinical neurosurgery, 16 hours, lectures, demonstrations.

Scope: Physiologic principles underlying neurosurgical technique; didactic instruction in care and treatment of acute head injuries and of injuries to spinal column; prophylaxis of meningitis; care of bladder, tidal drainage; physiology and pathology of spinal fluid.

References: Neurosurgery (Military Surgical Manuals, National Research Council, vol. 6), Philadelphia, W. B. Saunders Company, 1943; Rowbotham, G. F.: Acute Injuries of the Head, Baltimore, William Wood and Co., 1942; Ehrlich, W., and Gross, S. W.: Diagnosis and Treatment of Head Injuries, New York, Paul B. Hoeber, 1940.

8. Neurologic and neurosurgical clinics, 16 hours, lectures, demonstrations.

Scope: Demonstration of wide variety of medical and surgical cases exhibiting nerve and brain injuries.

9. Examinations, tests and inspections, 10 hours.

10. Open time, 18 hours.

Phase II

Technical Training in Neurosurgery (576 hours), Selected Army General Hospitals

1. Pathology, 36 hours, conferences, amphitheater exercises.

Scope: Autopsies and examination of tissues, with particular attention to traumatic lesions of nervous system. Study of gross surgical specimens with an accurate written description of each. Microscopy of all neurosurgical specimens studied under the direction of the Chief Pathologist.

References: Rand, C. W., and Courville, C. B.: Histological studies of the brain in cases of fatal injury to the head, Arch. Neurol. & Psychiat. 27:1342–1379 (June) 1932 (reprints supplied).

2. Clinical pathologic conferences and staff conferences, 24 hours.

Scope: Review of neurologic and neurosurgical cases. Officers to present and take part in discussion of neurosurgical material at weekly staff and clinicopathologic conferences.

Special assigned reading, directed by preceptor, covering the important data of each case presented at conference.

3. Neurologic examination and care of patients, 264 hours, conferences, amphitheater exercises.

Scope: Students will be stationed upon the neurosurgical wards, where they will act as clinical clerks and dressers. They will perform complete neurologic examinations of all assigned cases. Records will be carefully checked and graded by neurosurgical preceptor. No student will have fulfilled the requirements of the course until he shall have demonstrated to the preceptor his ability to perform, record and interpret a complete neurologic examination. Postoperative care of patients.

References: Spurling, as listed; Fulton, as listed.

4. Neurologic clinic, 24 hours, conferences, amphitheater exercises.

Scope: Study of neurologic cases. The preceptor will hold once each week a 2-hour conference or ward walk clinic in which the important phases of diagnostic neurologic problems are stressed.

5. Neurosurgery, 144 hours, amphitheater exercises.

Scope: Operating room assignments. Each student will be a member of the operating team in the capacity of an assistant in his proportional number of neurosurgical cases. Particular stress will be placed upon the technical problem of hemostasis, the use of the electrosurgical unit, suction, etc. No student will have finished his course until he shall have demonstrated to the preceptor his technical ability to perform a *thorough* debridement.

References: Davis, L. E.: The Principles of Neurological Surgery, Philadelphia, Lea & Febiger, 1942; Bailey, Hamilton: Surgery of Modern Warfare, chapters 42, 43, 47–50, Baltimore, Williams & Wilkins, 1941; Neurosurgery, in Military Surgical Manuals, as listed.

6. Roentgenology, 36 hours, conferences, amphitheater exercises.

Scope: Study of X-ray films of head and spine injuries. The Chief of Roentgenology will teach all students a minimum of 3 hours weekly the interpretation of skull and spine films, particular attention being placed upon the localization of foreign bodies, depressed fragments of bone and linear fractures into the accessory nasal sinuses.

References: Technical Manual (TM) 8–275, paragraphs 27–34; Technical Manual (TM) 8–240.

7. Physiotherapy, 12 hours, conferences, amphitheater exercises.

Scope: Physiotherapy in peripheral nerve lesions. The Chief of Physical Therapy will teach all students the care of paralyzed muscles, and splinting for prevention of deformity in group muscle paralysis.

8. Open time, 36 hours.

Scope: Assigned reading. It is expected that the students will follow the example of the preceptor in answering all night emergencies and attending such off-hour exercises as would contribute to their knowledge of neurosurgery.

APPENDIX B

Extracts From Manual of Therapy European Theater of Operations

* * * * * * *

SURGICAL EMERGENCIES. SECTION A

PRIMARY SURGICAL TREATMENT

* * * * * * *

II. WOUNDS OF THE CENTRAL AND PERIPHERAL NERVOUS SYSTEM

a. CRANIOCEREBRAL INJURIES

1. *Diagnosis:*

a. Signs of critical brain injury.

(1) Profound unconsciousness with failure to respond to *deep* supraorbital pressure.
(2) Rapid or irregular respirations.
(3) Rapid pulse with a rising blood pressure.
(4) Decerebrate rigidity.
(5) Dilated fixed pupils.

b. Intracranial hemorrhage is a potential factor in every case of head injury. The common signs of acute localized intracranial hemorrhage are:—

(1) Deterioration in condition of patient.
(2) Convulsive seizures, particularly when localized to one side of body.
(3) Hemiplegia or hemiparesis.
(4) Unilateral dilated fixed pupil, particularly after a lucid interval of consciousness.
(5) Signs of increasing intracranial pressure (slowing of the pulse, elevation of blood pressure, changes in the respiratory rate and deepening unconsciousness).

c. Small lacerations of the scalp may be associated with penetrating wounds of the skull and extensive intracranial injury. *All scalp wounds should have serious implications until proved otherwise.*

2. *Treatment:*

a. Early evacuation to field or evacuation hospital where a neurosurgical team is available. The sole objective of emergency treatment should be to improve the condition of the patient sufficiently to permit transportation.

b. Treatment of cerebral concussion and edema by spinal fluid drainage and intravenous hypertonic solution are of questionable value and should only be used in exceptional cases.

c. The wound should be treated as follows:—

(1) Clip hair for several inches about lacerations.
(2) Control bleeding from scalp wounds with pressure dressings. If bleeding cannot be controlled with pressure, use through-and-through mattress suture.
(3) Scalp wounds should not be irrigated, debrided or *sutured.*
(4) Dust sulfanilamide crystals into the wound, care being taken to distribute them uniformly. More than 5 gms. is seldom required.
(5) Do not place sulfathiazole or sulfadiazine directly into the wound, particularly if there is brain tissue exposed.

(6) Once the dressing is placed, do not change it until the patient reaches a hospital where definitive treatment can be given. The dressing may be reinforced as necessary.

d. Blood loss from scalp wounds is seldom severe enough to require early transfusion.

e. Bleeding from the ear should be treated only with a dressing over the external ear. Do not attempt to stop the bleeding. Do not explore, cleanse or irrigate the auditory canal.

f. Shock rarely results from uncomplicated cranial injuries. If the patient is in shock, search carefully for associated injuries, particularly chest and abdominal wounds, or extensive injury to the long bones. *When shock is present, the usual treatment of this state takes precedence over all other considerations.*

g. An unconscious patient should not be placed on his back or in an upright position. Aspiration of secretions from the nose and throat into the bronchial tree is promoted in these positions and the patient may literally drown in his own secretions. He should be placed on his side, the foot of the litter or bed elevated so that secretions run from his mouth by gravity (postural drainage).

h. Medication. Morphine suppresses the state of consciousness, and depresses the respiratory centers, and its use should be discouraged in craniocerebral trauma. Head wounds accompanied by painful wounds of other parts may require morphine, but it must be used judiciously.

i. Early recording of the following data is important for the most efficient future treatment of the patient:—

(1) Time of injury.
(2) State of consciousness when first seen.
(3) Paralysis of one or more extremities.
(4) Pulse rate counted for one-half minute.
(5) Respiratory rate and rhythm.
(6) Blood pressure.

3. *Evacuation:*

a. Head injury patients without serious associated injuries stand transportation well. The usual dictums of good first aid care should be followed whether evacuation is by air, ambulance, boat or hospital train. High priority evacuation is always indicated.

b. FRACTURES OF THE SPINE WITH NEUROLOGICAL INVOLVEMENT

Cervical.

1. *Diagnosis:*

a. When an injured man complains of severe pain in his neck, suspect a fracture or fracture-dislocation of the cervical spine; if his arms and legs are paralyzed the diagnosis is certain.

b. Cervical spine fractures are usually associated with forward dislocation and anterior angulation at the level of the lesion. Determination of the exact level of the fracture is unimportant for emergency treatment.

Do not manipulate the head in an attempt to establish the level of the injury.

2. *Treatment:*

a. The most important principle of treatment is to prevent an increase in the bony deformity. Do not move the patient until the conditions are favorable. *Make every move count.* Do not raise the head under any circumstances. Do not put a rolled blanket or pillow beneath the head.

b. With the patient on his back, place a folded blanket (not more than 3 inches thick) beneath the shoulders, thus permitting the head to fall backward and downward. Place

and fix folded blankets or articles of clothing on either side of the head to reduce lateral movement.

c. When patient is picked up to transfer to a litter or solid support, maintain strong traction at both the head and feet in the longitudinal axis of the body.

d. Keep pressure off the sacrum and heels, and where possible transfer patient to soft mattress to minimize danger of pressure sores.

e. An indwelling catheter should be introduced before distention of the bladder occurs, especially before evacuation of patient to distant points. *Never clamp the catheter.*

f. Give morphine cautiously to cervical injuries for fear of respiratory paralysis; in many cases the diaphragm will have been paralyzed by the damage to the cervical spinal cord.

3. *Evacuation:*

a. High priority evacuation to General or Evacuation hospital is indicated.

Lumbar.

1. *Diagnosis:*

a. If an injured patient complains of severe local pain in the lumbar region, fracture of the spine in this area should be suspected. Paralysis of the legs below the knees makes the diagnosis certain.

2. *Treatment:*

a. Again the fundamental principle in handling fractures or fracture-dislocations of the lumbar spine is to prevent an increase in the bony deformity, thus avoiding further injury to the spinal cord.

b. There are two acceptable positions for transportation of patients with lumbar spinal fractures:—

(1) With the patient on his back, make a roll with folded blankets, about 12 to 18 inches in circumference, and place beneath the mattress near the site of the injury. This position produces hypertension of the spine.

(2) With the patient face down, some extension of the spine is automatically produced; in all circumstances further flexing is prevented.

c. When transferring patient from ground to litter, traction should be maintained at the feet and axilla and he should be slid or rotated to the litter and not lifted.

d. Keep pressure off sacrum and heels and wherever possible transfer to a soft mattress.

e. An indwelling catheter should be introduced but it should not be clamped.

f. Do not attempt to reduce the fracture in the field or in the intermediate dressing stations.

3. *Evacuation:*

a. High priority to a Field, Evacuation or General hospital is indicated.

c. INJURY TO PERIPHERAL NERVES

1. *Diagnosis:*

a. Fifteen percent of extremity wounds are complicated by injury to one or more major nerve trunks. Simple tests for motor and sensory function will usually indicate accurately which nerve is involved.

b. At the primary inspection of the wound, observe carefully the exposed nerves and record their condition on the EMT tag or on the cast. This information greatly facilitates later treatment.

2. *Treatment:*

a. Extremity wounds with nerve injuries should receive exactly the same treatment as soft tissue wounds elsewhere * * *. Most patients with nerve injury will have

injuries to one or more of the long bones, so splinting will be a preliminary requirement for transportation. In those cases with bone injury, the extremity should be immobilized in the most favorable position, to prevent deformity. This can usually be accomplished with bandages or rolls of clothing and blankets. Casts or elaborate splints will usually not be required for satisfactory evacuation. Remember to protect from pressure a part that has been deprived of sensation.

3. *Evacuation:*

a. Low priority evacuation is usually indicated.

* * * * * * *

SURGICAL EMERGENCIES. SECTION B

DEFINITIVE SURGICAL TREATMENT

* * * * * * *

II. WOUNDS OF THE CENTRAL AND PERIPHERAL NERVOUS SYSTEM

a. CRANIOCEREBRAL INJURIES

The following statements are intended as a guide for medical officers who, by force of circumstances, are required to treat neurosurgical patients before they arrive at a hospital where trained neurosurgical teams are available. (See also section on Emergency Treatment * * * .)

1. *Care of the Unconscious Patient:*

a. Keep the patient in optimal position for postural drainage. Never permit him to lie on his back or be in an upright position. Place on side with face rotated forward and, *if necessary, elevate the foot of the bed or litter to facilitate drainage of secretions* from mouth by gravity.

b. Maintain fluid intake to 1500 cc. daily and increase proportionately during hot weather or in case of high fever. Fluids should never be put into the mouth of a stuporous or semiconscious patient. They should be given per rectum, subcutaneously or intravenously. Never use more than 1000 cc. of physiological saline in 24 hours because of the danger of salt retention in the tissues of the brain. If unconsciousness persists after 24 hours feed nourishing liquids by nasal tube.

c. Turn the patient from side to side every 3 hours to prevent bed sores and hypostatic pneumonia.

d. Avoid morphine and other opium derivatives for restlessness. Paraldehyde, administered by nasal tube, rectum or intravenously is the safest and most satisfactory sedative for the patient with head trauma.

2. *Intracranial Hemorrhage:*

a. Extra Dural: The classic signs of extra-dural hemorrhage are:—

(1) A lucid interval of consciousness.
(2) Deepening stupor.
(3) Unilateral dilation and fixation of the pupil on the side of the lesion.
(4) Slowing of the pulse rate.
(5) Rising blood pressure.
(6) Focal convulsions or paralysis.

Extra-dural hemorrhage requires immediate surgical evacuation with ligation of the main trunk of the middle meningeal artery. A straight incision extending from the midportion of the zygoma upward into the temporal region should be used. The temporal bone exposed by separating the fibres of the temporal muscle is trephined. The bony opening may be enlarged as necessary to evacuate the clot and ligate the middle meningeal artery. The dura should not be opened, except when a sub-dural hemorrhage is suspected. If the

main artery cannot be found and small bleeding points completely controlled with the facilities available, place a light gauze pack over the dura and compress until all bleeding has stopped. Dust sulfanilamide crystals throughout the wound and close the incision with the tip of the packing presenting through the lower angle. Fractional removal of the pack may be begun after 48 hours.

b. Sub-dural hemorrhage: Acute sub-dural hematomas of size sufficient to cause profound symptoms are frequently arterial in origin. They are usually associated with profound brain injury and prognosis is always grave. They are often clinically indistinguishable from extra-dural hematomas. The technical details for evacuating the clot are similar to those described for extra-dural hematomas.

Chronic sub-dural hematomas are of late occurrence and should always be suspected when recovery from a relatively minor closed head injury is retarded. These are not emergency lesions and can always await evacuation to hospitals where trained neurosurgeons are available.

c. Intracerebral bleeding, if arterial, is apt to be quickly fatal. If venous, localized intracerebral hematoma may form and stop spontaneously. The usual location is in the anterior half of the temporal lobe. These clots produce characteristic neurological symptoms and signs. They are seldom emergencies and will usually await evacuation to the rear.

In general, surgical exploration for intracranial hemorrhage should be carried out early only as a life-saving measure when the patient is deteriorating rapidly.

d. Scalp wounds: Carefully debride the scalp to conserve as much skin and subcutaneous tissues as possible, otherwise the wound cannot be closed. Sulfanilamide powder (not over 5 gms.) should be dusted into the wounds and the edges of the skin meticulously approximated. *Do not use drains.* If there is extensive loss of tissue and the edges of the scalp cannot be approximated, a split-thickness skin graft should be placed over the exposed bone (fig. 1).

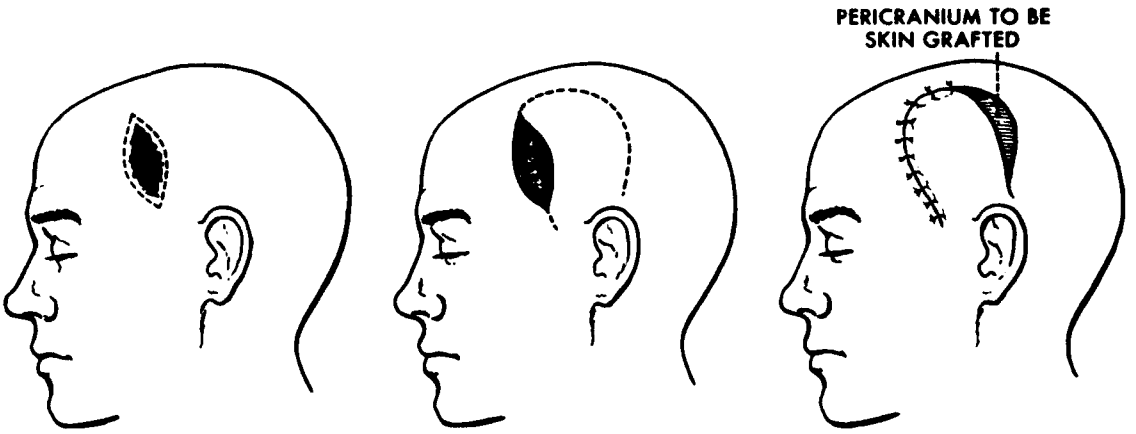


FIGURE 1.—Method of closing defect in scalp.

3. *Fractures:*

a. Simple linear fractures require no treatment.

b. Compound linear fractures into the accessory nasal sinuses or external auditory canal are important, chiefly because they afford portals of entry for infection. In such cases sulfadiazine therapy (administered intravenously or by mouth) should be instituted at once.

c. Simple comminuted depressed fractures of bone should be removed and the edges of the defect smoothed with rongeurs. These defects can be most satisfactorily repaired with tantalum plate at a subsequent operation. In certain instances the bony fragments will be large and may be elevated into position, thus restoring the contour of the skull.

d. In compound comminuted fractures the wound should be thoroughly cleansed of dirt and foreign matter. If the bony fragments have torn the dura and damaged the brain,

all devitalized tissue, including the brain, should be meticulously removed. Openings in the dura should be closed, if necessary by free transplants of fascia from the temporal muscle or fascia lata. Drains should never be used unless available facilities do not permit thorough control of hemorrhage. All tissues, including the brain, should be dusted with sulfanilamide crystals. Primary closure should be attempted up to 72 hours in wounds treated locally with sulfanilamide at time of injury. All compound skull fractures should receive adequate sulfadiazine therapy systematically.

In penetrating wounds of the brain it is important that all fragmented bone be removed. Small metallic fragments, unless producing symptoms, may be left, especially if their removal requires traversing relatively normal brain tissue.

b. SPINAL INJURIES WITH NEUROLOGICAL INVOLVEMENT

(Refer to section on Emergency Treatment, * * * .)

1. *Fracture Dislocation of the Cervical Spine:*

a. The best form of treatment is skeletal traction applied by Crutchfield tongs. If tongs are not available, skeletal traction may be applied by wires passed through two small burr holes made on either side of the skull, or by barbless fish hooks placed beneath the midportions of the zygomatic arches. In the absence of all facilities for skeletal traction, halter traction is the method of choice. At least 15 pounds of weight should be applied. Open or closed reduction by manipulation should never be attempted, for fear of further damaging the spinal cord.

2. *Compression Fractures of the Lumbar Spine:*

a. Reduction is best accomplished with the patient on his back and a blanket roll, approximately 12 inches in diameter, placed beneath the mattress at the point of fracture. (fig. 2.)

b. *Care of the Bladder.* Treat the paralyzed bladder by tidal drainage as soon as practicable (fig. 3). If after 6 weeks there is no evidence of recovery, a cystostomy should be done. Keep urine acidulated with ammonium chloride or ammonium mandelate. Change catheter at least once a week.

c. INJURY TO PERIPHERAL NERVES

In view of the irreparable degenerative changes that occur in the end plates of severed nerves, early surgical repair is essential for best results. Primary suture is theoretically desirable but experience has shown that in battle wounds where there is contusion of the nerve, immediate suture is not desirable. However, it is essential that the severed nerve ends be approximated as nearly as possible to prevent retraction. This greatly facilitates early end-to-end secondary suture.

When the wound is debrided, exposed nerve ends will often be visualized. Approximate the ends if possible but if the gap does not permit approximation, anchor the nerve ends snugly to surrounding soft tissues to prevent subsequent retraction. Thin stainless steel wire or tantalum wire should be used for nerve repair. These suture materials are ideal as they serve a useful purpose in identifying by subsequent X-ray examinations the location of the nerve injury or the extent of the nerve defect. *Do not attempt elaborate primary nerve suture.*

Dust the wound thoroughly with sulfanilamide crystals and close the muscles or fascia loosely over the exposed nerve trunks. *Never place a pack on a minor nerve trunk.* Do not attempt primary closure of the wound. Delayed or secondary sutures are recommended.

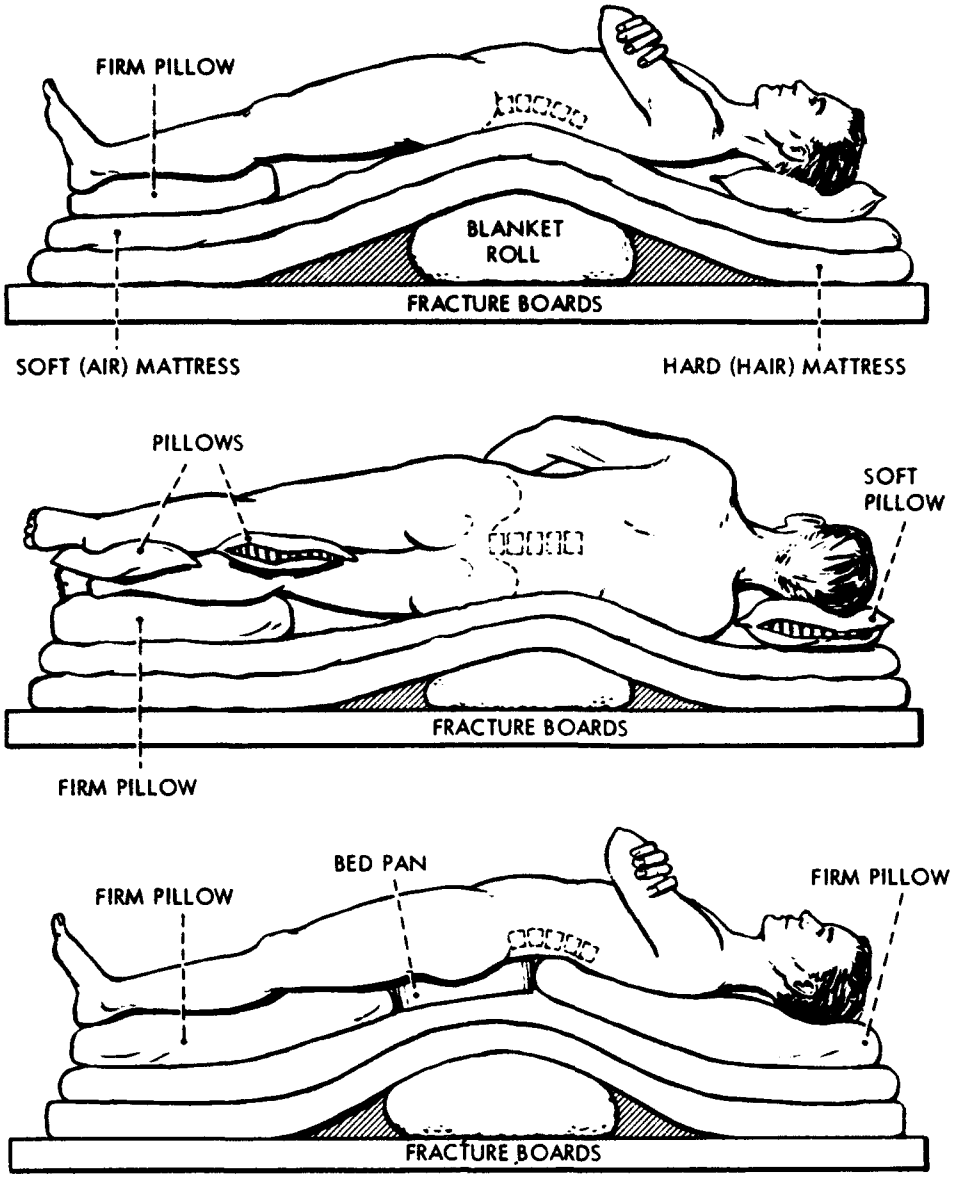


FIGURE 2.—Proper position for patient with compression fracture of the lumbar spine.

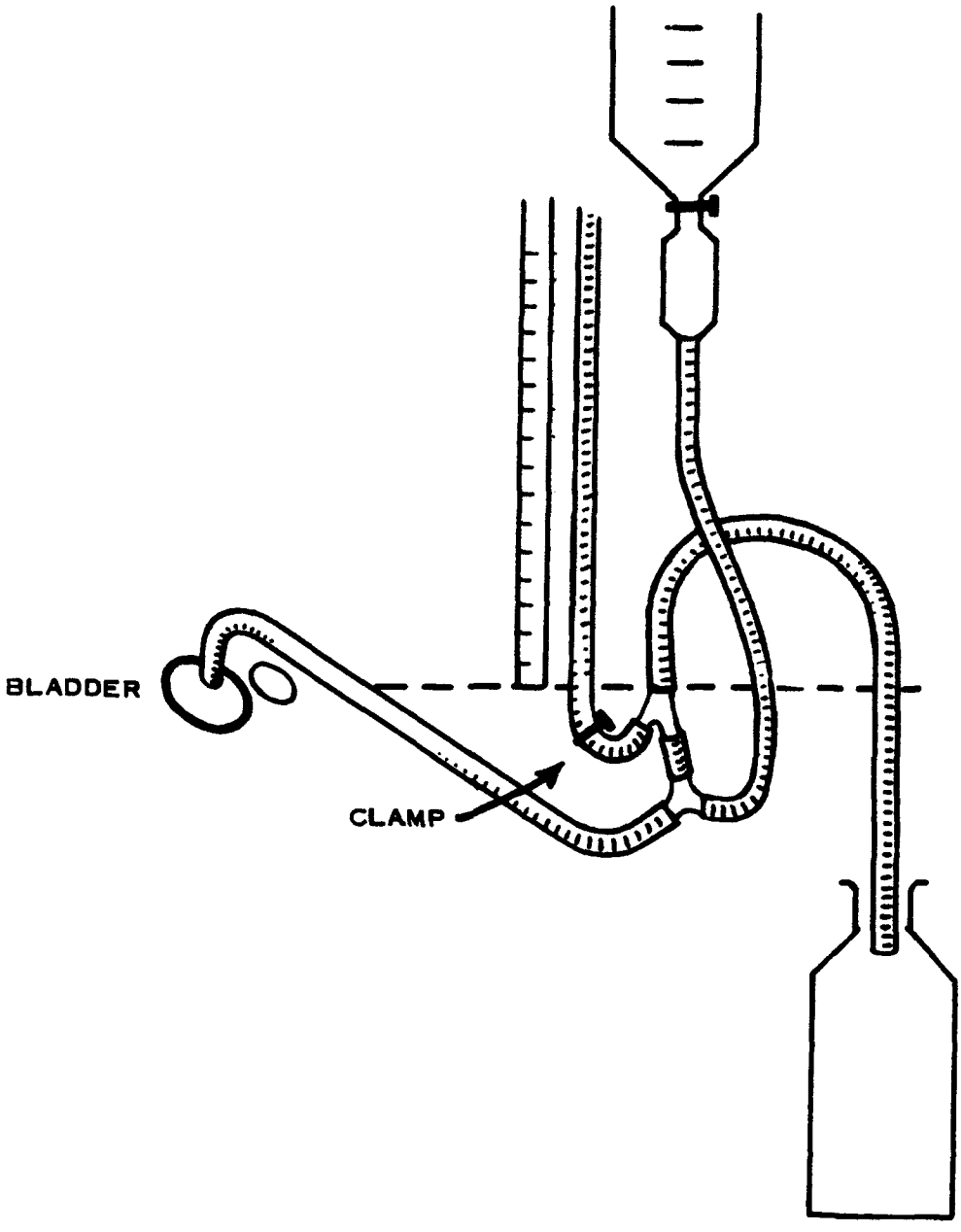


Figure 3.—Simple type of tidal drainage. The essential point in successful operation is that the rubber tubing alongside the yardstick empty more slowly than the bladder. This is accomplished by means of a small screw clamp shown in the drawing.

APPENDIX C

Special History and Examination Forms

HEAD INJURY—HISTORY

Patient — Date of examination
Date of birth — Marital state: S M W D
Home address
Family history

Patient's parents	At present	History of: Seizures	Fainting spells	Migraine	Other nervous or mental disease
Father	Living	Yes	Yes	Yes	
	Dead	No	No	No	
Mother	Living	Yes	Yes	Yes	
	Dead	No	No	No	

Patient's siblings	Total number	Number with history of: Seizures	Fainting spells	Migraine	Other nervous or mental disease
Living Male					
Living Female					
Dead Male					
Dead Female					
Spouse					
Patient's children					
Living Male					
Living Female					
Dead Male					
Dead Female					

Patient's number in order of birth (excluding foetal deaths)
Seizures in other relatives

Past History
- Birth date
- Delivery (normal-prolonged-instrumental-injury-circumstances unknown)
- Development
  - Walked at
  - Talked at
  - Stuttering
- Accidents
  - Head injuries
  - Broken bones
- Operations
- Illnesses
  - Migraine

Past History—Continued
Illness—Continued
Meningitis
Chorea
Other illnesses
Personal habits
Tobacco — Number Per Day
Cigars
Cigarettes
Chewing tobacco
Alcohol — Amount Per Week
Beer
Hard liquor
Frequency of intoxication
Sleep
Personal hygiene (care of teeth, body and clothing, bowel movements)
Nature of Head Injury: Open
Closed
Type of missile
Place (street, field, etc.)
Location (frontal, occipital, parietal, etc.)
Period of unconsciousness
Period of confusion and disorientation
Other wounds
Bleeding
Time debrided after injury
Extent of debridement (scalp-skull-dural laceration-brain laceration-ventricle opened)
Local drug therapy
General chemotherapy
Time of healing
Secondary procedures (debridement-abscess-cranioplasty-scalp plastic)
Immediate neurological sequelae (weakness of arm or leg-aphasia-motor and sensory disturbance)
Convulsive Seizures (list all attacks)
Dates (Day or month and/or day) | Night | Under medication? | Type | Remarks (alcohol, excitement, other factor)
Status epilepticus (duration and date)
Typical Attack
Prodromal symptoms
Aura
Olfactory
Gustatory
Auditory
Visual
Sensory
Gastric paresthesia
Motor
Vertiginous
Dreamy state
Automatism
States of confusion or furor
Petit mal
Automatic seizures (pallor—flushing)
Cry

Typical Attack—Continued
- Attack
  - —tonic (cry—loss of consciousness)
  - —clonic
- Clonic
  - Beginning with
    1) turning of head and eyes
    2) movements of face
    3) movement of arm
    4) movement of leg
  - Generalized
  - Unilateral
  - Biting of tongue
  - Frothing at mouth
  - Wounds (location)
  - Urinary incontinence
  - Cyanosis (duration)
  - Duration
- Post-epileptic states
  - Period of confusion
  - Sleep
  - Excitement
  - Headache
  - Weakness of arm or leg
  - Aphasia
  - Vomiting

Minor Attacks
- Pallor
- Staring
- Loss of consciousness
- Blackouts

Epileptic Equivalents
- Dreamy state
- Sleepwalking
- Emotional fits

Allergies	Personal	Familial
Hay fever		
Asthma		
Migraine		
Urticaria		
Food		

Childhood Manifestations of Convulsions
- Enuresis
- Fugues
- Nocturnal crying
- Temper tantrums
- Fainting spells
- Petit mal attacks
- Seizures

Present Complaints (spontaneous):
- Question regarding:
  - Attacks (frequency, severity, and duration)
  - Headache (local, generalized, associated with emotional upset, type, frequency and duration)

Present Complaints (spontaneous)—Continued
Question regarding—Continued
Dizziness (type, frequency, aggravated)
Irritability (constant, aggravated)
Impaired mentation (give examples)
Lack of concentration
Easy fatigability
Insomnia (initial, nightmares)
Effect of alcohol
Other changes

Previous Medication	Amount	Duration	Effect
Bromides			
Phenobarbital			
Dilantin			
Tridione			
Other drugs			

HEAD INJURY—EXAMINATION

Height Weight
Psychiatric Evaluation
General behavior (appearance, activities, attitude, cooperation)
Emotional reaction and mood tendencies:
Manner (distant. expansive, hopeless, indifferent)
Mood (depressed, moody, unstable, seclusive, suspicious, anxiety, fear)
Judgment (discrimination)
Insight
Orientation
Memory address, 4962, 53817, 738294, 82961475
Mental ability and intelligence:
100–7; 93, 86, 79, 72, 65, 58, 51. 44, 37, 30, 23, 16, 9. 2.
Name months backward
Began school at
Age and grade at finishing school
Occupational training
Social adjustment (arrests, AWOLS, occupational, sexual, sociability)
Neurological Examination

Cranial Nerves		Right	Left
I	Perceives odors (use cigarette)		
	Identifies odors		
II	Vision		
	Fundi		
	Fields		
III	IV and VI		
	Extraocular movements		
	Convergence		
IV	Nystagmus		
	Ptosis		
	Endophthalmos or exophthalmos		
	Pupils		
	Size		
	Shape		
	React to light		
	React to convergence		
	Consensual		

Neurological Examination—Continued

Cranial Nerves—Continued

		Right	Left
V	Sensation		
	Cotton		
	Pin prick		
	Corneal reflex		
	Motor		
	Masseters		
	Lateral motion of jaw		
	Deviation of jaw		
VII	Facial weakness		
	Forehead		
	Eyes		
	Mouth		
VIII	Acuity		
	Weber		
	Rinné		
IX	and X		
	Sensation of palate and pharynx		
	Movement of palate		
	Pharyngeal reflex		
	Phonation		
	Swallowing		
XI	Sternocleidomastoids		
	Trapezius		
XII	Movements of tongue		
	Tremor		
	Atrophy		
	Fibrillations		
	Motor		
	Involuntary movements		
	Atrophy		
	Fibrillations		
	Tonus		
	Upper extremities		
	Lower extremities		
	Strength and motility		
	Upper extremities		
	Lower extremities		
	Co-ordination		
	Finger to nose		
	Heel to knee		
	Each finger to thumb		
	Rapidly alternating movements		

Reflexes	Right	Left	
Biceps			Abdominal
Triceps			Upper
Radial			Lower
Knee			Hoffmann
Ankle			Rossolimo
Plantar			Oppenheim

Sensation	Right	Left
Pin prick		
Cotton		
Warm		
Cold		
Deep pain		
Vibration		
Localization		
Position sense		
Fingers		
Toes		
Two point discrimination		
Figure writing		
Palms		
Legs		
Stereognosis		
Nerve status		
Tenderness		
Excitability		
Size		
Station and gait		
Romberg		
Gait		
Associated movements		
Hopping		
Walking tandem		
Head, spine and neck		
Scalp (position, size and condition scalp wounds, depressed areas, etc.)		
Stiff neck		
Kernig		
Laségue		
Patrick		
Macewen		
Naffziger		
Additional observations		

EXAMINATION FOR APHASIA AND RELATED DISTURBANCES

Name _____ Grade _____ ASN _____ Ward _____
Age _____ Date _____

1. *Sidedness:* Hand (Catches ball) _____ (Kicks ball) _____
   Eye (Cone) _____
2. *Spontaneous speech:* "How did you get here?" (Stenographic record of one minute)
3. *Automatic speech:* Greetings _____
   Cursing _____
4. *Repetition:* "I am at Cushing General Hospital." _____
   "This is a wonderful day." _____
   "Man" _____ "Dog" _____ "Chair" _____ "Table" _____
   A _____ E _____ I _____ O _____ U _____ P _____
   B _____ T _____ D _____ M _____ N _____
   7 _____ 11 _____ 5 _____ 24 _____ 341 _____
5. *Singing:* (My Country) _____ Humming: _____
   Whistling: _____
6. *Serial speech:* a—alone; b—with help
   1–21
   Week days
   Months
   Seasons
   Alphabet
   Lord's prayer
   Poem: (Mary Had a Little Lamb)
7. *Naming:* *Seeing:* *Feeling:* *Handling:*
   Key
   Pencil
   Button
   Comb
   Hammer
   Safety pin
   Scissors
   Eraser
8. Selecting objects: Tactile Auditory Visual
   Key
   Pencil
   Button
   Comb
   Hammer
   Safety pin
   Scissors
   Eraser
9. *Commands*

	Visual	Auditory
1. Point to your teeth	_____	_____
2. Point to your chin.	_____	_____
3. Point to your eyebrows.	_____	_____
4. Point to your eyelashes.	_____	_____
5. Point to your eyelids.	_____	_____
6. Smile.	_____	_____
7. Get up, close the door, turn on the light, and then come back and sit down.	_____	_____
8. Point with right hand to left eye.	_____	_____
9. Point with left hand to left ear.	_____	_____
10. Point with right hand to right elbow.	_____	_____

10. *Reading out loud:*
    Story: (Page 58, Mental Examiner's Handbook,[1] par A)
    Words: (Page 50, Mental Examiner's Handbook [1])
    Letters: A _____ F _____ Z _____ Q _____ L _____
    a _____ f _____ z _____ q _____] _____
    Numbers: 7 _____ 11 _____ 5 _____ 24 _____ 341_____
11. *Writing:* a. Spontaneous: "How were you injured?"_____
    b. Dictation: "I am at Cushing Hospital."
    "This is a wonderful day."
    "Man" _____ "Dog" _____ "Chair" _____ "Table" _____
    A _____ E_____ I _____ O _____ U _____ P _____
    B _____ T _____ d _____ m _____ n _____
    7 _____ 11 _____ 5 _____ 24 _____ 341 _____
    c. Copying: "Don't count your chickens before they are hatched."
11A. Drawing of a man
12. Calculation: (Oral) 2 + 2 = 7 + 8 = 12 − 5 = 10 − 2 =
    3 × 4 = 6 − 9 = 8 ÷ 2 = 24 ÷ 4 =
    (Written) 14 + 13 = 18 + 76 = 382 − 241 =
    234 − 169 = 72 × 8 = 21 × 14 =
    148 ÷ 4 = 357 ÷ 17 =
13. Spelling: (From dictation) (From sight of object)
    Key
    Pencil
    Button
    Comb
    Hammer
    Safety pin
    Scissors
14. Rhythm: Right Left



15. Stereognosis: (See Number 8)
16. Apraxia: (See attached form)
17. Agnosia: (Single objects) (See Number 7) (Pictorial representation)
18. *Optic Constructional Agnosia:* (Apraxia)
    Right Left
19. *Spatial orientation:* "How do you get to the lab from this ward?"
20. *Right—Left Differentiation:* (See Number 9)
21. *Finger agnosia:* Name fingers:
    Index finger
    Ring finger
    Thumb
    Little finger
    Middle finger
    Show me the index finger of your hand. My hand.
    Ring finger
    Thumb
    Little finger
    Middle finger

Summary and evaluation of language function:

[1] Wells, F. L., and Ruesch, J., Mental Examiner's Handbook, New York, N. Y., The Psychological Corporation, 1st Edition.

SUGGESTIONS FOR SOCIAL HISTORY IN CASES WITH SEIZURES

About the patient:

1. Birth (normal, prolonged, instrumental, injury, circumstances unknown)
2. Early development
   Walked at—
   Talked at—
3. Schooling
   Began school at—
   Finished _____ grade at _____ years
4. Occupational training
   Number of jobs in _____ years
   Longest job
5. Social adjustment: arrests, number of friends: male, female
6. As a child or later:
   Bedwetting (frequency, up to what age)
   Temper tantrums
   Fainting spells
   Blackouts or dreamy states (did something which he did not remember)
   Fits (describe them: onset in face? Does one arm or leg move first? Are they unilateral or generalized? Convulsive? Duration? Unconscious? Incontinent? Tongue-biting? Face pale or flushed? Injuries during attacks?)
   After attacks: Confused? Excited? Sleepy?
   Weakness of arm or leg?
7. Head injuries, diseases?
8. Drinking
9. Personality: depressed, overactive, moody, unstable, seclusive, egocentric, suspicious, irritable, rigid, worrisome, carefree, overreligious

About the family:

Immediate—mother's and father's family and their children

1. Fits, attacks.
2. Is any member of the family institutionalized?
3. Alcholism.
4. Drug addiction.

OCCUPATIONAL COUNSELING

Head Injury Patients With Post-Traumatic Epilepsy

Section I Patient Identity

1. Name of patient
2. ASN
3. Rank
4. Branch of service
5. Location of home or residence (City and state)
6. Age
7. Marital status (Single, married, divorced, separated, widowed)
8. Children (specify number, age, and sex)
9. Does family visit patient? (Yes or no)
10. Frequency

Section II Physical and Mental Status

A. Physical status
   1. Motor impairment
      a. Not incapacitated by loss of motor power or by incoordination
      b. Paralysis of: Right Left Mild Severe
         (1) One leg
         (2) One arm
         (3) Arm and leg
         (4) Two legs and one arm
         (5) Other
      c. *Ambulation* Well Poorly
         (1) Walks:
            (a) Without support
            (b) With cane
            (c) With crutches
         (2) Stands indefinitely
         (3) Sits indefinitely
         (4) Wheelchair patient
         (5) Bed patient
      d. Handedness
         (1) Right-left handed
         (2) Paralyzed hand is right-left
         (3) Uses paralyzed hand (well, poorly, not at all)
         (4) Uses non-paralyzed hand (well, poorly, for writing only, not at all)
   2. *Impairment of speech* Mild Severe
      a. None
      b. Impairment of
         (1) Speaking
         (2) Understanding
         (3) Finding words
         (4) Writing
         (5) Reading
         (6) Arithmetic
   3. *Loss of senses*
      a. None
      b. Blindness
         (1) In one eye
         (2) In both eyes
         (3) To one side (right-left)
      c. Deafness
         (1) One side (right-left)
         (2) Bilateral

A. Physical status—Continued
   3. *Loss of senses*—Continued
      d. Loss of sensation (location)
      e. Astereognosis (right-left)
   4. *Attacks*
      a. None
      b. Petit mal
      c. Convulsive attacks
         (1) Frequency
         (2) Aura (does patient have forewarning)
         (3) Loss of consciousness
         (4) Duration
         (5) After effect (type, time)
         (6) Controlled by medication
      d. Psychomotor attack
      e. Headaches, dizziness
   5. *Operations*
      a. Cranioplasty
      b. Craniotomy
      c. Plastic
   6. *Associated injuries*
      a. (describe)
      b. Operations — Done — Indicated
         (1) Nerve suture or neurolysis
         (2) Orthopedic
         (3) Plastic

B. Mental status
   1. *Previous records show:*
      a. No neuropsychiatric disorder
      b. Psychoneurosis
      c. Psychosis
      d. Other
   2. *Present psychiatric condition*
      a. Good adjustment
      b. Psychoneurosis
      c. Post-traumatic behavior disorder
      d. Mental deterioration
      e. Mental deficiency
   3. *Present emotional status*
      a. Normal affect
      b. Indifferent
      c. Euphoric
      d. Depressed
      e. Resentment toward Army, society
   4. *Attitude toward present disability*
      a. Insight, with constructive plans
      b. Partial insight—no plans
      c. No insight into condition

C. Work tolerance
   1. *Normal*
   2. *Easily fatigued*
   3. *Concentration (good-poor)*

Section III Probable Disposition

A. Eventual Disposition
   1. *Discharge*
      a. To his own care
      b. To his family
      c. To VAF
   2. *Ability of family to care for patient on a domiciliary basis*
      a. Can care for the patient satisfactorily
      b. Cannot care for patient. Reason:
      c. Ability indeterminable
   3. *Probable date of final disposition*

Section IV Education

Education	Highest grade	Graduation, degree or diploma	Major course	Age at completion
Elementary				
Secondary				
College				
University				

Other training or schooling (other than occupational)

Course	*Date*	*Duration*

Interests in further schooling:

Section V Occupational Background and Interests

1. Trade school, art school, apprenticeship
2. Major civilian occupations
3. Number of jobs in years
4. Longest job
5. Usual reason for leaving
6. Army training
7. Army assignments
8. Does he want further training in a suitable occupation?
9. What kind of work is the patient interested in doing?

Section VI Aptitudes

1. Army General Classification Test
2. Mechanical Aptitude Test
3. General Educational Development Test
4. Radio Operators Aptitude Test
5. Other aptitude or ability tests
   a. Wechsler-Bellevue
   b. Rorschach
   c. Bender
   d. Kuder
6. Types of work patient is now doing in Occupational Therapy
   a. Diversional
   b. Functional
7. Attitude toward present work
8. Hobbies and other avocational interests

Section VII Summary

1. By attending medical officer and clinical psychologist:
   a. Evaluation of current physical and mental condition
   b. Prognosis and probable disposition
2. By vocational counselor:
   a. Appraisal of aptitudes, abilities and interests
   b. Evaluation of vocational potentialities
   c. Specific types of training recommended as suitable

APPENDIX D

Battle Wounds and Battle Injuries of the Head and Brain

The statistical data presented in tables 1, 2, 3, and 4 were prepared by the Medical Statistics Division, Office of the Surgeon General.

The term "admission," as used in these tables, refers to cases in which a battle wound or a battle injury of the specified site was reported as the primary cause of admission to hospital or quarters. With respect to multiple wounds recorded on admission, only one, the more serious, would have been recorded as the admission diagnosis. Data pertaining to patients who incurred head or spinal injuries but were admitted to treatment primarily for wounds or injuries of other sites are not available. With respect to KIA (killed in action) data, the anatomical region indicated is that associated with the primary cause of death. The variables, causative agent, nature of traumatism, and anatomical location pertain to the primary diagnosis for admissions and CRO (carded for record only) cases and to the principal cause of death for the KIA.

TABLE 1.—*Battle wounds and battle injuries of the head (including brain and ear) in the United States Army, by causative agent, combined years, 1942, 1943, and 1945*

[Preliminary data based on tabulations of individual medical records]

Causative agent	KIA[1]	Nonfatal CRO cases[2]	Admissions[3]	Deaths among those admitted[4]	Case fatality ratio
	Number	*Number*	*Number*	*Number*	*Percent*
Bomb and bomb fragments	130	171	657	35	5.3
Shell, shell fragment, and flak	5,779	2,011	12,500	886	7.1
Bullet, machinegun, rifle, etc	3,945	465	2,714	465	17.1
Land mine, boobytrap	226	232	1,406	83	5.9
Grenade and grenade fragments	56	88	490	23	4.7
Explosion of ammunition, weapon, etc	99	87	1,076	11	1.0
Incendiaries	2	22	41	--------	--------
Firearms, mechanism or effects of discharge of	10	24	272	13	4.8
Aircraft, excluding aircraft weapons	459	12	334	36	10.8
Parachute jump	28	4	50	2	4.0
Boat sinking and accident	3	--------	50	1	2.0
Tank, tractor, caisson	21	4	66	2	3.0
Motor vehicle, passenger and cargo	13	2	102	5	4.9
Vehicle, other and unspecified	1	3	20	3	15.0
Cutting or piercing instruments	32	5	26	--------	--------
Fire, hot liquid, or objects	--------	3	5	--------	--------
Fall or jump, twisting, turning, lifting, slipping, etc	2	3	112	1	.9
Other and unspecified	967	141	532	119	22.4
Total	11,773	3,277	20,453	1,685	8.2

[1] KIA for whom a wound or injury of the head was reported as the primary cause of death.

[2] CRO cases with a wound or injury of the head as the primary diagnosis.

[3] Due to wounds or injuries of the head.

[4] Consists of all cases which ended in death, not necessarily due to wound or injury of the head nor necessarily occurring during the years indicated.

TABLE 2.—*Battle wounds and battle injuries of the head (excluding brain and ear) in the United States Army, by causative agent, combined years, 1942, 1943, and 1945*

[Preliminary data based on tabulations of individual medical records]

Causative agent	KIA[1]	Nonfatal CRO cases[2]	Admissions[3]	Deaths among those admitted[4]	Case fatality ratio
	Number	*Number*	*Number*	*Number*	*Percent*
Bomb and bomb fragments	124	121	240	35	14.6
Shell, shell fragment, and flak	5,754	1,291	4,667	882	18.9
Bullet, machinegun, rifle, etc	3,929	343	1,689	462	27.4
Land mine, boobytrap	217	96	305	80	26.2
Grenade and grenade fragments	54	49	188	22	11.7
Explosion of ammunition, weapons, etc	79	35	68	5	7.4
Incendiaries	2	14	21	----	----
Firearms, mechanism or effects of discharge of.	10	7	59	12	20.3
Aircraft, excluding aircraft weapons	451	10	213	33	15.5
Parachute jump	28	3	18	2	11.1
Boat sinking and accident	2	----	22	1	4.5
Tank, tractor, caisson	21	3	34	2	5.9
Motor vehicle, passenger and cargo	13	2	49	2	4.1
Vehicle, other and unspecified	1	2	11	3	27.3
Cutting or piercing instruments	31	4	17	----	----
Fire, hot liquid or objects	----	2	4	----	----
Fall or jump, twisting, turning, lifting, slipping, etc.	2	2	51	1	2.0
Other and unspecified	949	94	225	117	52.0
Total	11,667	2,078	7,881	1,659	21.1

[1] KIA for whom a wound or injury of the head was reported as the primary cause of death The number of KIA cases is markedly affected by the lack of detail in describing the anatomical location In most of the KIA cases shown in this table, the anatomical location of the wound or injury causing death has been recorded as "head, generally."

[2] CRO cases with a wound or injury of the head as the primary diagnosis.

[3] Due to wounds or injuries of the head.

[4] Consists of all cases which ended in death, not necessarily due to wound or injury of the head nor necessarily occurring during the years indicated.

TABLE 3.—*Battle wounds and battle injuries of the brain in the United States Army, by causative agent, combined years, 1942, 1943, and 1945*

[Preliminary data based on tabulations of individual medical records]

Causative agent	KIA [1]	Nonfatal CRO cases [2]	Admissions [3]	Deaths among those admitted [4]	Case fatality ratio
	Number	*Number*	*Number*	*Number*	*Percent*
Bomb and bomb fragments	6	6	248		
Shell, shell fragment and flak	20	100	5, 345	2	0. 0
Bullet, machinegun, rifle, etc	8	2	721	2	. 3
Land mine, boobytrap	9	29	447	3	. 7
Grenade and grenade fragments	2	3	150		
Explosion of ammunition, weapon, etc	20	19	747	5	. 7
Incendiaries			1		
Firearms, mechanism or effects of discharge of.		3	66	1	1. 5
Aircraft, excluding aircraft weapons	8		97	3	3. 1
Parachute jump			32		
Boat sinking and accident	1		17		
Tank, tractor, caisson			23		
Motor vehicle, passenger and cargo			46	3	6. 5
Vehicle, other and unspecified			9		
Cutting or piercing instruments	1		5		
Fire, hot liquid or objects					
Fall or jump, twisting, turning, lifting, slipping, etc.		1	54		
Other and unspecified	17	11	242	2	. 8
Total	92	174	8, 250	21	. 3

[1] KIA for whom a wound or injury of the brain was reported as the primary cause of death. The number of KIA cases is markedly affected by the lack of detail in describing the anatomical location. This accounts for the relatively small number of KIA cases shown.

[2] CRO cases with a wound or injury of the brain as the primary diagnosis.

[3] Due to the wounds or injuries of the brain.

[4] Consists of all cases which ended in death, not necessarily due to a wound or injury of the brain nor necessarily occurring during the years indicated.

NOTE.— The low case fatality ratios are probably due to the relatively minor nature of many of the cases which reached medical treatment facilities; many serious cases are killed instantly or die before arriving at a treatment facility.

TABLE 4.—*Admissions for battle wounds and battle injuries of the head, by location of traumatism, United States Army, 1942–43*

[Preliminary data based on tabulations of individual medical records]

Anatomical location	Nonfatal CRO cases	Admissions	Deaths among those admitted [1]
	Number	*Number*	*Number*
Brain and coverings	29	1, 295	21
Cranial nerve and pituitary gland	----------	2	----------
Ear	61	295	1
Skull	----------	387	94
Scalp, occipital, parietal, or temporal regions	225	947	10
Head, generally	37	228	133
Total	352	3, 154	259

[1] Not necessarily due to the wound or injury causing admission nor necessarily occurring during 1942–43.

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