

DIAGNOSIS AND TREATMENT OF DISEASES OF TACTICAL IMPORTANCE TO U.S. CENTRAL COMMAND



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SOURCES

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Original text for a large portion of this TG was contributed by the U.S. Army Medical Research and Materiel Command (<https://mrmc-www.army.mil/>) through four of its laboratories:

- Walter Reed Army Institute of Research
(<http://wrair-www.army.mil/>)
- U.S. Army Research Institute of Environmental Medicine
(<http://www.usariem.army.mil/>)
- U.S. Army Medical Research Institute of Infectious Diseases
(<http://www.usamriid.army.mil/>)
- U.S. Army Medical Research Institute of Chemical Defense
(<http://chemdef.apgea.army.mil/>)

Global epidemiologic information that served as a basis for selecting and appropriately outlining the diseases addressed herein was provided by the Armed Forces Medical Intelligence Center (<http://mic.afmic.detrick.army.mil/>), which can provide additional tools to U.S. military personnel for estimating the risk of specific diseases in specific geographic areas.

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AREA OF RESPONSIBILITY (AOR)



Introduction

This TG is compiled and edited for military physicians and physician assistants operating outside the continental United States (OCONUS) under the U.S. Central Command (USCENTCOM).

The geographic area of consideration includes parts of Central and Southwest Asia, and northeastern Africa. A reasonable degree of geographic specificity is provided for conditions that show significant variability in this regard. Certain tropical diseases (such as filariasis) will most likely not be encountered in Central Asia. However, this TG is not to be considered a definitive source of medical intelligence, and the reader is referred to the Armed Forces Medical Intelligence Center (AFMIC) for the most current medical intelligence.

Diseases outlined herein are primarily within the scope of primary care internal medicine. The following caveats bear mentioning:

- Focus is on initial, not definitive, management outside of the hospital. Assumed level of care is I or II, with a worst-case evacuation policy of 7 days. The format and distribution of this TG is aimed at providers who would generally lack laboratory support, and whose local supply of drugs, sterile supplies, and medical equipment would be extraordinarily limited. However, for most conditions, components of definitive care are briefly mentioned to help guide the reader through triage and clinical decisions that depend on feasibility of evacuation and predictability of an acceptable prognosis.
- Determination of physical profiles and specific duty limitations are left primarily to the provider, though some of the information in this guide may be helpful in decision support.
- This is not a complete guide for preventive countermeasures. Therefore, this is not the source for patient or command education. Use alternative, easily available, military sources for prevention guidance at the individual, small-unit, and command level. (see USACHPPM Web site).

- Certain categories of disease are not addressed, even though they would include conditions of military relevance. These categories were excluded for one or more of the following reasons:
 - ◆ Alternative, field-expedient sources are available to military providers (examples: for traumatic injuries, military dermatology, neuropsychiatric conditions, and care of local populations with and without malnutrition during relief operations).
 - ◆ Management of diseases in the category is an integral part of daily practice in the continental United States (CONUS) and would not be unique in the operational setting (example: sexually transmitted diseases (STDs)).
 - ◆ Morbidity or epidemic potential of the diseases, at least in the acute phase, would typically not be great (examples: intestinal helminth infections and human immunodeficiency virus infection).
 - ◆ Medical management of the conditions is typically conservative, with emphasis on self-care and unit-level prevention strategies (examples related to cold weather operations: cold urticaria, chilblains, solar keratitis, sunburn, and eczema).
 - ◆ Immunity in U.S. military populations (including vaccine-induced) is reliably universal (examples: tetanus, diphtheria, measles, and poliomyelitis).
- Within categories that are covered, some specific diseases are not included even though it is possible they will be encountered in the CENTCOMAOR. This was primarily in the interest of prioritizing to keep the manual reasonably sized, and to minimize delays in revision and publication. Examples include West Nile encephalitis, tickborne encephalitis, hemorrhagic fever with renal syndrome, and Rift Valley Fever. Still, some of these are included in notes regarding differential diagnosis.

CONSULTATIVE SOURCES AND REFERENCES

Global Emerging Infections System: <http://geis.fhp.osd.mil/> and
Directory of DOD Public Health Laboratory Services:
<https://afip-geis.afip.osd.mil/Vphl/Vphlasp/Loginadmin.asp>

Sphere Guidelines - Civilians (Pediatrics, Dislocated Civilians):
www.sphereproject.org

An email consultation service through AKO is available to deployed providers in the areas of infectious disease, dermatology, and ophthalmology at the following email addresses:

Infectious disease: id.consult@us.army.mil
Dermatology: derm.consult@us.army.mil
Ophthalmology: eye.consult@us.army.mil

The following references have been used to prepare this TG. It is strongly encouraged that you take these manuals during deployment.

Chemical Exposure Guidelines for Deployed Military Personnel, U.S. Army Center for Health Promotion and Preventive Medicine, May 2003 with January 2004 Addendum

Control of Communicable Diseases Manual (Army FM 8-33, NAVMED P-5038), 18th Edition, American Public Health Association, 2004

Emergency War Surgery, Third United States Revision, NATO Handbook

Field Operations Guide for Disaster Assessment & Response, Office of Foreign Disaster Assistance, U.S. Agency for International Development, 1998

Special Operations Medical Handbook, U.S. Special Operations Command & Center for Total Access, Teton NewMedia & The Geneva Foundation, 2001

Medical Management of Chemical Casualties Handbook, U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, Maryland, July 2000

Medical Management of Biological Casualties Handbook, U.S. Army Medical Research Institute of Infectious Diseases, Fort Detrick, Maryland, August 2004

Textbook of Military Medicine – Part III: Disease and the Environment, Military Dermatology, Office of The Surgeon General, Department of the Army, August 2004

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PART ONE: ENDEMIC INFECTIOUS DISEASES (see also appendices A thru E)

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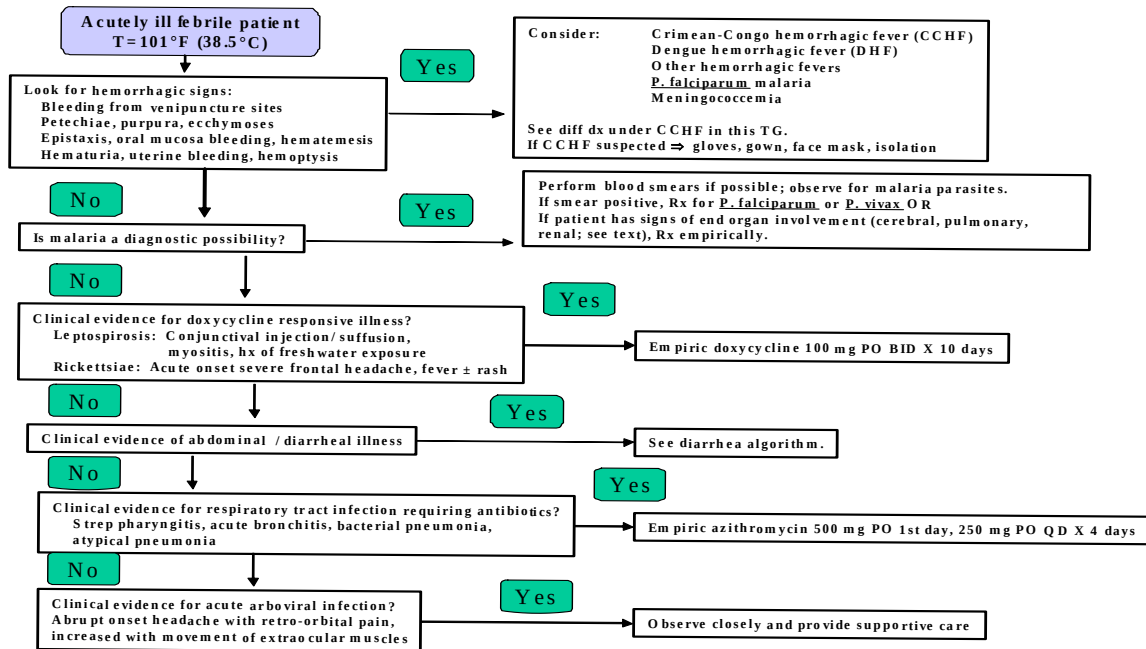
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APPROACH TO THE ACUTELY ILL FEBRILE PATIENT

When evaluating a patient with fever, consider first the possibility of highly transmissible infections that can pose a threat to health care providers in addition to the community, and second the need to identify diseases that may progress rapidly to death if not promptly treated. Keep your index of suspicion high for both malaria and viral hemorrhagic fevers. Remember, malaria MUST be considered immediately in all febrile patients who are, or were recently, in a malarious region; and that hemorrhagic manifestations of viral hemorrhagic fevers such as Crimean-Congo hemorrhagic fever (CCHF) and dengue hemorrhagic fever (DHF) may not appear until at or near the time of defervescence.

APPROACH TO THE ACUTELY ILL FEBRILE PATIENT



APPROACH TO THE PATIENT WITH DIARRHEA

Communicability of Pathogens Causing Acute Gastroenteritis:

- Route: oral ingestion of infectious organisms in contaminated food/water, particularly if inadequately cooked/purified. Inadequate personal hygiene, inadequate sanitary measures, and flies are the most likely contributory factors.
- Isolation: standard and contact precautions.
- Prophylaxis: Not recommended, except for short duration during high-risk missions, such as aircraft pilots who must eat on the local economy. Efficacy is of brief duration; inadequate for sustained operations. After initial 1- 2 weeks of protection, prophylaxis with antibiotics has been associated with increased incidence of diarrhea due to disruption of protective normal bowel flora and with emergence of drug-resistant pathogens. Furthermore, it is prudent to reserve the best prophylactic drugs (fluoroquinolones) for treatment to ensure its effectiveness when needed.

Public Health Measures - Command emphasis is essential:

- Adequate sanitary facilities.
- Personal hygiene, especially hand washing.
- Water purification and individual water discipline.
- Use of food obtained only from medically approved sources.

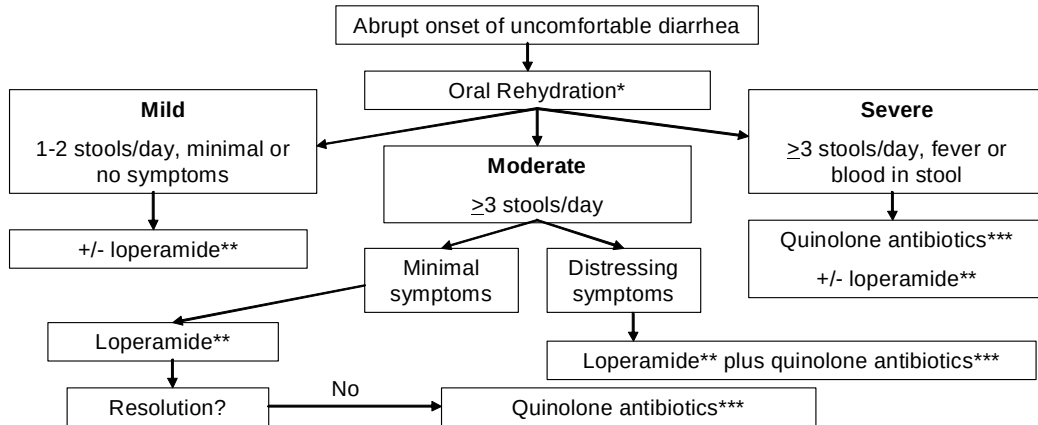
Incubation:

- Varies with specific pathogen. Ranges from hours (staphylococcal enterotoxins) to several weeks (giardiasis or amebiasis).

Diagnosis:

- Specific pathogen identification is not usually required for effective management of individual patients.
- The following algorithm provides an effective, efficient approach.

APPROACH TO THE PATIENT WITH DIARRHEA



Definition/Note:

- 1) Uncomfortable diarrhea = loose stools, abdominal cramps, urgency.
- 2) Antibiotic Rx is most effective when given ASAP after onset of symptoms.
- 3) Self Rx encouraged for mission essential personnel.
- 4) Significant nausea and vomiting at onset likely to be viral or exotoxin induced. Antibiotic Rx not useful.
- 5) More gradual onset and prolonged symptoms of greater than 1 week: empiric metronidazole (Flagyl) 250 mg PO TID X 7 days.
- 6) Consider malaria if systemic symptoms and signs are present and failure to improve quickly with antimicrobial.
- 7) Campylobacter may have high resistance rates to Ciprofloxacin; if a campylobacter is known to be the causative agent (e.g., identified outbreak), then azithromycin is preferred.
- 8) For continued bloody diarrhea, consider E. histolytica.

* For oral rehydration solution, see following page. ** For loperamide dosing, see following page. Loperamide may be substituted with other antidiarrheal medications. *** For quinolone antibiotic doses, see following page. If quinolones such as Ciprofloxacin are not available, use Azithromycin.

- Oral rehydration: 3.3 gm NaCl, 2.5 gm NaHCO₃, 1.5 gm KCl, 20 gm glucose (or 40 gm sucrose) in 1 liter H₂O. Intake should be sufficient to maintain 60 to 100 mL urine output per hour. Pre-mixed salts/glucose are available. (See end of Heat Illness section, Part Five, for simple measure and field expedient equivalents.)
- Antimotility agents:
 - ◆ Use loperamide (Imodium) 2 mg tablet, 2 tablets loading dose, followed by 1 tablet after each stool, not to exceed 8 tablets per day.
 - ◆ Kaopectate is ineffective.
 - ◆ Diphenoxylate with atropine (Lomotil) is less desirable than loperamide due to a higher incidence of side effects.
- Fluoroquinolone choices, in order of preference, include:
 - ◆ Ciprofloxacin 500 mg PO BID for 3 days.
 - ◆ Levofloxacin 500 mg PO QD for 3 days.
 - ◆ Gatifloxacin 400 mg PO QD for 3 days.
 - ◆ A single day of fluoroquinolone therapy has proven effective in relatively mild causes.
 - ◆ In event of fluoroquinolone clinical failure, 1 gram of Azithromycin as a single dose may be used.

APPROACH TO THE PATIENT WITH PNEUMONIA (PNEUMONITIS)

Background

- Beginning in March 2003, numerous troops have been medically evacuated out of the CENTCOM AOR to be treated for severe pneumonia requiring mechanical ventilation and progressing to Acute Respiratory Disease Syndrome (ARDS). At least half of these cases were determined to be confirmed or probable cases of acute eosinophilic pneumonia (AEP) which is discussed separately. The other cases were determined to be likely or confirmed cases of infectious etiology.
- A number of additional, uncomplicated pneumonias have been diagnosed across the CENTCOM AOR; all of these patients have recovered. The uncomplicated pneumonias have fallen into two distinct classes: an atypical (viral) process and a classic lobar pneumonia. The viral process is characterized by a 1-3 day history of typical upper respiratory infection (URI) symptoms including malaise, fever, cough, and headache. A chest radiograph (CXR) demonstrates subtle lobar or bilateral interstitial infiltrates, and a complete blood count (CBC) shows a low white blood cell (WBC) count ($< 5000/\mu\text{L}$) and thrombocytopenia ($< 120\text{K}/\mu\text{L}$). The vast majority of such cases quickly resolve. The other class is characterized by leukocytosis, fever, cough, and typical unilateral lobar alveolar infiltrates on CXR suggestive of (but usually not proven to be) a bacterial pneumonia. These latter cases respond well to antibiotics and recover fairly rapidly.

Case definition

For purposes of standardization, a case of pneumonia must be confirmed by presence of infiltrates on a CXR. The case definition for severe pneumonia includes a CXR characterized by the presence of bilateral alveolar infiltrates, with or without a requirement for mechanical ventilation. The remainder of this practice guideline is directed towards the management of patients who present with severe pneumonia.

Approach

The challenge confronting most clinicians is not in detecting the presence of the disease but rather in identifying its cause. This is critical to diagnose and treat the disease, but also to establish common or shared factors that indicate whether cases are related to a common exposure. Even so, in the best of medical centers, less than one-third of all pneumonias reveal a specific pathogen after extensive investigation. Establishing standardized operating procedures in the clinical and diagnostic evaluation of pneumonia is important for the control of disease for a population (military units) and the effective management of an individual patient. While empiric antibiotic therapy is usually necessary even in the absence of a specific etiology, it is vital that clinical specimens be obtained to link specific pathogens to clusters of cases, to exclude common causes of pneumonia, and to identify unusual pathogens.

- **History.** A detailed history of each case of pneumonia is required to document date of onset, duration of illness before seeking medical care, symptomatology, exposures (e.g., dust, chemicals, and smoke), MOS, work environment, and geographic location. The EPICON team has prepared a detailed questionnaire that includes a patient interview (clinical situation permitting) for each case of pneumonia (uncomplicated and severe) to establish links with other cases throughout the area of operation. Ideally, a health care worker (HCW) should administer this questionnaire (i.e., interview the patient). Additional data capture forms in the EPI Data Collection Instrument are included to record laboratory test results and radiologic findings for transfer to the U.S. Army Center for Health Promotion and Preventive Medicine (USACHPPM) in CONUS.
- **Diagnostic work-up.** Although definitive identification of a pathogen is often elusive, it is imperative to collect clinical specimens for epidemiologic purposes, especially when the cause of a suspected outbreak is unknown. The following recommendations are based upon medical and logistical assets available at Combat Support Hospitals (CSHs) in order to minimize the logistical burden to clinical staff and to facilitate the timely transfer of specimens from the clinical laboratory to Landstuhl Regional Medical Center (LRMC) and/or CONUS. **Severe cases of pneumonia, or**

uncomplicated cases with patterns with bilateral disease on a CXR, are best managed at a centralized medical treatment facility with advanced laboratory services and the capability to perform bronchoscopic examinations. Due to the potential for rapid progression to respiratory failure, patients with bilateral infiltrates on a CXR should be evacuated to LRMC immediately after the initial evaluation described below.

◆ **Laboratory**

- CBC with manual differential (numerate eosinophils)
- Sputum gram stain (all CSHs) and culture (military treatment facility (MTF)-specific, the CSH in Baghdad) for patients with cough characterized by productive sputum
- Throat swab for detection of Group A Streptococci (*S. pyogenes*) using rapid detection assay (all CSHs)
- Throat swab (Dacron) for viral culture—transport at 4°C to LRMC within 96 hours
- Urine sample for the detection of *S. pneumoniae* and *Legionella* using rapid antigen detection assay
- Group A Streptococcus (GAS) rapid diagnostic kits can be deployed to Echelons I and II facilities if providers want them

- ◆ **Radiology.** PA/LAT CXR documenting unilateral vs. bilateral disease, interstitial vs. alveolar pattern, and specific lobes involved

Treatment and Case Management

- Prognostic indicators to predict uncomplicated limited disease vs. progression to severe disease. Unfortunately, there are no good predictors to identify those patients who will progress to severe disease requiring mechanical ventilation or to distinguish between those who have bacterial versus viral infection. However, normal mental status, pulse <125 beats/minute, respiratory rate <24/minute, systolic blood pressure >90 mmHg, temperature >35°C and <40°C, and a “not very sick” appearance are fairly good predictors for outpatient therapy.

- Fluid management of patients with severe pneumonia. Many patients with severe disease have some degree of volume depletion, and adequate fluid replacement is essential. Replacement of fluids and electrolytes with normal saline or D5NS is recommended with the amount and rate carefully titrated to the patient's clinical condition so as to avoid exacerbating pulmonary leakage and pulmonary edema. Health care providers should avoid using Lactated Ringer's Solution, especially older racemic mixtures of Ringer's lactate.
- Strategies for treatment of pneumonia
 - ◆ Uncomplicated pneumonia. The empiric treatment of uncomplicated pneumonia using oral antibiotics such as Azithromycin (Zithromax) or Levofloxacin is common practice and often effective. Depending on the patient's clinical status, the physician may elect to initiate intravenous therapy followed by changeover to oral therapy when possible. It is imperative that combination broad-spectrum antibiotics be avoided in patients with uncomplicated pneumonia to prevent emergence of multi-drug resistant organisms.
 - ◆ Severe pneumonia. Without a definitive microbiologic diagnosis, combination antibiotic therapy administered intravenously is frequently required as empiric treatment:

3rd generation cephalosporin (ceftriaxone, cefotaxime)

AND

Respiratory tract quinolone (levofloxacin (500-750 mg IV QD) >> preferred over ciprofloxacin) **OR** azithromycin (500 mg IV QD)

AND

doxycycline (100 mg IV BID)

Alternatives to levofloxacin are gatifloxacin and moxifloxacin. Doxycycline is included because a number of cases of infection with *Coxiella burnetii* (causative agent of Q fever) have been reported. There is no strong recommendation for the addition of imipenem to the above regimen. However, if imipenem is used in a seriously ill patient requiring mechanical ventilation, this should be used as a substitute for the cephalosporin.

- ◆ Use of steroids. This clinical practice guideline neither recommends nor prohibits the use of steroids for patients with severe pneumonia. The use of steroids is NOT the standard of care for management of pneumonia or ARDS in the United States. However, under certain clinical conditions and depending upon the available laboratory data, the physician may elect to give steroids (in addition to broad spectrum antibiotics) to the severely ill patient who is being mechanically ventilated. This may be appropriate in those cases where AEP has been diagnosed or there is a strong clinical suspicion that the patient has AEP.
- ◆ Pressure-control ventilation. The use of a pressure-control ventilator should be limited to those experienced in its use. The device itself may need to be requested as special augmentation for the CSH. The advantages include proper management of patients that typically require high PEEP (>10) with high peak pressures (45-50) to maintain adequate oxygenation, particularly if prolonged care in theater is needed because of delays in medical evacuation.
- ◆ Evacuation from theater. **The clinical course of patients with severe pneumonia requires immediate evacuation from theater** (dependent upon the stability of the patient for transport). This is critical because: 1) most of these patients will require management in an ICU for > 7 days; and 2) the diagnostic work-up may require early bronchoscopy and detailed analysis of BAL fluid.

ACUTE EOSINOPHILIC PNEUMONIA (AEP)

Communicability:

- Etiology undetermined, but evidence suggests one or more inhalational exposures in a predisposed individual. Although not an infection, AEP may present in conjunction with, or as a result of, infectious agents.
- The initiation of tobacco smoking, or a significant increase in the quantity of tobacco smoked, appears to be a major risk factor in the development of AEP.
- Person-to-person transmission is not known to occur.

Incubation: Unknown

Diagnosis:

- Symptoms/Signs (abrupt onset; indistinguishable from community-acquired pneumonia (CAP)):
 - ◆ Shortness of breath (89%).
 - ◆ Fever/chills (67%).
 - ◆ Cough (67%).
 - ◆ Fatigue (67%).
 - ◆ Chest pain (56%).
 - ◆ Myalgias/arthralgias (56%).
 - ◆ Inspiratory crackles.
 - ◆ Hypoxemia.
 - ◆ Respiratory failure.
- Laboratory findings:
 - ◆ Hematology: Eosinophilia ($= 250 \text{ cells} \times 10^3/\text{mL}$).
 - ◆ Microbiology: Negative.
 - ◆ Serology: Negative.
 - ◆ BAL: Eosinophilia ($= 5\%$).
- Radiology:
 - ◆ CXR: Alveolar and/or interstitial infiltrates (bilateral in 56%); pleural effusions uncommon (6%); no adenopathy.
 - ◆ CT: Dense alveolar consolidation and pulmonary edema; injury usually follows a bronchovascular distribution; no adenopathy.

Duration: Abrupt onset of symptoms and rapid deterioration of lung function; may require intubation with increasing requirements for oxygen, positive end expiratory pressure (PEEP) >15 , and high peak pressures >40 . Untreated cases may recover with careful ICU

ventilator management in 1-2 weeks. Prompt recognition and early steroid therapy can reverse respiratory decompensation, preclude the need for intubation, and lead to more rapid resolution.

Complications: Respiratory infections, complications associated with mechanical ventilation.

Treatment:

- Cessation of tobacco smoking
- Empiric treatment of CAP (see above under Approach to the Patient with Pneumonia)
 - ◆ 3rd generation cephalosporin (ceftriaxone, cefotaxime)

AND

- ◆ Respiratory tract quinolone (levofloxacin 500-750 mg IV QD) or Azithromycin (500 mg IV QD)

AND

- ◆ Doxycycline (100 mg IV BID)
- Corticosteroids
 - ◆ Solumedrol 80 mg IV Q 8 hrs (or equivalent); begin taper over 2-4 weeks as symptoms resolve
- Pressure-control ventilation
- Evacuation from theater

Disposition: Evacuate from theater; anticipate return to full duty status in 4-6 weeks, but patient should NOT be returned to theater. Returning a Servicemember to the environment in which he/she was potentially exposed to the causative agent(s) of AEP is NOT recommended.

Prognosis: Most recover without long-term sequelae or relapse. Early administration of steroids—given only in conjunction with broad-spectrum antibiotics—often leads to a rapid recovery and can prevent respiratory failure requiring intubation.

Prevention and Public Health Measures:

- Although the exposure(s) necessary to cause AEP are not fully understood, tobacco smoking and inhalation of particulates (dust) are associated with this disease. Servicemembers should be encouraged to avoid smoking, especially non-smokers who are considering initiation of the habit and current smokers who may increase tobacco use during deployments.

- 
- Report case as a reportable event using theater medical surveillance reporting channels.

Continuing Epidemiologic Assessment:

- If AEP is suspected, two (2) serum separator tubes of blood should be drawn for serologic tests—and transported at 4°C (or on regular ice) with patient to LRMC.
- In addition, upon evacuation the following items should accompany the patient to LRMC: all medical notes, lab results, radiographs, and other clinical records; the patient's uniform(s) and any other equipment he/she may have been wearing; all medications the patient may have been taking, including over the counter (OTC); all cigarettes and other tobacco products in the patient's possession; and any other personal effects that can be acquired.

BRUCELLOSIS

(see also Brucellosis, Part Two: Exposure to Biological Warfare Agents)

Communicability:

- Route:
 - ◆ Ingestion of contaminated meat or dairy products.
 - ◆ Inhalation of infectious aerosols, including laboratory-specimens.
 - ◆ Direct contact of abraded skin or mucous membranes with infected tissues, blood or lymph.
- Isolation: standard precautions.
- Prophylaxis: none required, no evidence of communicability from person to person.

Incubation: 2-3 weeks (1 week to several months).

Diagnosis: systemic infection with many different manifestations; no diagnostic clinical findings. Exposure history is critical. Screen for:

- ◆ ingestion of unpasteurized milk products or consumption of cheese, and
- ◆ exposure to animals, livestock, meats.

Classic presentation of the patients with no history of prior brucellosis infection could be presentation of fever associated with peripheral arthritis, prominent sacroiliitis or spondylitis.

- Symptoms/Signs:
 - ◆ Systemic (almost 100%):
 - Fever as high as 104°F.
 - Weakness.
 - Night sweats.
 - Weight loss.
 - Malaise.
 - Lymphadenopathy (20%).
 - ◆ Gastrointestinal:
 - Nausea and vomiting.
 - Splenomegaly (50-70% in acute disease).
 - Constipation or diarrhea.
 - Abdominal pain.

- Liver and spleen.
- Hepatomegaly (up to 65% in acute disease).
- Abscesses.
- Anorexia.
- ◆ Osteoarticular (20-85%):
 - Arthralgias.
 - Tenosynovitis.
 - Myalgia.
 - Bursitis.
 - Arthritis.
 - Sacroiliitis.
 - Spondylitis.
 - Paravertebral abscess.
 - Osteomyelitis.
- ◆ Genitourinary (2-40%):
 - Unilateral epididymo-orchitis.
 - Pyelonephritis.
 - Acute interstitial nephritis.
 - Prostatitis (very uncommon).
- ◆ Pulmonary: cough (15-25%)
- ◆ Neurological (2-5%):
 - Meningitis, encephalitis.
 - Psychosis.
 - Meningoencephalitis.
 - Depression.
 - Myelitis.
 - Headaches.
 - Paresis.
- ◆ Cardiovascular:
 - Endocarditis - 2% (most common cause of death)
- ◆ Cutaneous (5%) - many nonspecific findings such as:
 - Red-violet papules.
 - Diffuse erythema.
 - Abscesses.
 - Erysipelas-like lesions.
 - Erythema nodosum.
- ◆ Ocular:
 - Keratitis.
 - Uveitis.
 - Papilledema.
 - Optic neuritis.

- Laboratory findings:
 - ◆ Hematology: anemia, leukopenia, thrombocytopenia; lymphopenia worse in more severe cases.
 - ◆ Chemistry: elevated alkaline phosphatase; mildly elevated transaminases.
 - ◆ Microbiology: culture of pathogen from blood, bone marrow, fluids or tissue; blood cultures 70% sensitive, bone marrow-cultures 92% sensitive. Cultures must be held 45 days. Special media and conditions helpful. Strict precautions to avoid aerosol exposure necessary.
 - ◆ Serology: very helpful; IgM elevated in first 3 weeks, followed by IgG elevation after 3 weeks; titer $\geq 1:160$ indicates past exposure.
- Radiology:
 - ◆ CXR abnormal in patients who acquired infection by aerosol: hilar adenopathy, perihilar infiltrates, nodular lesions, lung abscess, pleural effusions, and/or pneumothorax.
 - ◆ Spondylitis with disk-space narrowing and epiphysitis; erosion and rounding of anterior superior edge of vertebral body with syndesmophyte formation; lumbar involvement much greater than thoracic or cervical involvement.
- Invasive procedures: not required for diagnosis; only required in therapy for focal suppurative complications.
- “Gold Standard” for Diagnosis:
 - ◆ Isolation of pathogen, or
 - ◆ Titer $\geq 1:160$ with compatible epidemiologic and clinical findings.

Duration:

- Treated: week to months.
- Untreated: months, with up to 30% complications.

Complications: see Symptoms/Signs.

Treatment:

- Uncomplicated: doxycycline 100 mg PO BID plus rifampin 600 mg/day x 6 weeks.
- Complications: seek specialist consultation.

- Alternative treatment regimens: not well defined; obtain specialist consultation.
- Treatment failure and relapses occur in 5%; most not due to drug resistance; re-treat with initial regimen and obtain specialist consultation.

Disposition:

- Uncomplicated: limited duty (consider evacuation).
- Complications: hospitalization and evacuation.

Prognosis:

- Treated: excellent.
- Untreated: 30% complications, prolonged hospitalization and convalescence with occasional deaths due to endocarditis.

Prevention and Public Health Measures:

- Locate contaminated products, if implicated, and destroy.
- Educate commanders and troops not to drink or eat unpasteurized dairy products.
- Report case as a reportable event using theater medical surveillance reporting channels.

CRIMEAN-CONGO HEMORRHAGIC FEVER (CCHF)

Communicability:

- Route:
 - ◆ Ixodid tick (*Hyalomma* species) bite.
 - ◆ Exposure to blood, secretions, or excrement of infected patients. Aerosol transmission may occur, as transmission to hospital staff has been documented in the absence of direct patient contact.
 - ◆ Exposure to tissue or blood of infected animals. (Source animals do not appear to be sick.)
 - ◆ Onset of illness more than 3 weeks after last possible exposure rules out CCHF.
- Isolation: Standard, contact, and airborne precautions (see Standard Precautions and Isolation Procedures – appendix A).
- Prophylaxis:
 - ◆ No prophylaxis of proven efficacy available.
 - ◆ The antiviral drug Ribavirin may be effective as immediate, post-exposure prophylaxis; and, if available, it should be strongly considered for HCWs and transportation personnel involved in caring for patients with CCHF (400 mg PO Q6H for 24 hours, then 400 mg PO TID for 6 days).

Incubation: 1-3 days (range: 1-12 days).

Diagnosis:

- Symptoms:
 - ◆ Sudden onset of fever, chills, headache, myalgias (especially lower back).
 - ◆ Marked anorexia and irritability.
 - ◆ Flushing of face and chest with fine petechial rash.
 - ◆ Pharyngeal hyperemia, petechiae, and hemorrhage on soft palate.
 - ◆ Conjunctival injection.
 - ◆ Signs of hemorrhage on 4th or 5th day.
 - ◆ Ecchymoses or bleeding from mucous membranes (gums, nose, mouth, lungs, intestines, uterus).
 - ◆ Abdominal pain (90%).
 - ◆ Backache (90%).

- ◆ Arthralgia/myalgia (70%).
- ◆ Diarrhea (40-50%).
- ◆ Photophobia (50%).
- ◆ Cough (nonproductive) (16-40%).
- ◆ Chest pain (20%).
- ◆ Sore throat (16%).

■ Signs:

- ◆ Fever: to 104°F (40°C) (100%).
- ◆ Skin hemorrhages (petechiae, purpura) (100%).
- ◆ Jaundice (25-100%).
- ◆ Hematuria (90%).
- ◆ Tachycardia (70-90%).
- ◆ Hypotension (70-90%).
- ◆ Oliguria (80%).
- ◆ Hepatomegaly (80-100%).
- ◆ Disturbed consciousness (80%).
- ◆ GI bleeding (hematemesis or melena) (70%).
- ◆ Epistaxis (50%).
- ◆ Vaginal bleeding (>50% of women).
- ◆ Edema (50%).
- ◆ Meningeal irritation (40%).
- ◆ Bleeding gums (40%).
- ◆ Relative bradycardia (20%).
- ◆ Conjunctival injection (20%).
- ◆ Palmar erythema (20%).
- ◆ Gingival ulcers (16%).

■ Laboratory findings:

- ◆ Hematologic:
 - Anemia (as condition deteriorates).
 - Leukopenia (60%).
 - Thrombocytopenia (100%).
 - Atypical lymphocytes (60%).
- ◆ Chemistry:
 - Hyperbilirubinemia.
 - Elevated transaminases.
- ◆ Urinalysis:
 - Hematuria (90%).
 - Proteinuria (90%).
- ◆ Microbiologic:
 - Unavailable in most clinical laboratories.
 - Viral isolation possible by specialized laboratories with sophisticated containment and viral culture capabilities.

- Exposure of laboratory personnel to aerosolized specimens is highly dangerous.
- ◆ Serology: Acute and convalescent paired sera can demonstrate virus-specific antibodies by 20-21 days using ELISA or IFA tests, but are of no immediate clinical diagnostic value.
- ◆ Coagulation studies:
 - Prolonged bleeding time (100%).
 - Prolonged PT (75%).
 - Prolonged PTT (67%).
 - Fibrinogen - decreased.
 - Fibrin split products (increased in 60%).
- Invasive procedures: not applicable.
- X-rays: nonspecific.
- Diagnostic confirmation: serologic or viral isolation.

Duration:

- Treated: undefined, but presumably shorter acute illness and markedly abbreviated convalescence.
- Untreated: 10-14 days with subsequent convalescence requiring several weeks.

Complications:

- Sepsis, shock, renal failure, death (20-40% mortality).
- Relapse does not occur.

Treatment:

- Treatment regimens of proven efficacy do not exist.
- Ribavirin may be beneficial for CCHF treatment if given intravenously over a 10-day course, and IF available as part of an investigational new drug (IND) protocol (LRMC, Germany).
- Ribavirin to be administered in a volume of 50-100 mL and infused over 30-40 minutes with the following dosing schedule:
 - ◆ Loading dose: 33 mg/kg (maximum dose: 2.64 gm),
 - ◆ Days 1-4: 16 mg/kg (maximum dose: 1.28 gm) IV Q6H,
 - ◆ Days 5-10: 8 mg/kg (maximum dose 0.64 gm) IV Q8H.

- Oral Ribavirin is NOT recommended for acute CCHF, and if used, would be considered off-label use (prompting informed consent): 400 mg PO Q4H for 24 hours, then 400 mg PO Q8H for 7-14 days.
- Human immune convalescent serum: isolated clinical reports suggest possible benefit, but efficacy has not been proven.
- No alternatives exist for treatment failure.
- Relapses are not known to occur.
- Aggressive supportive care and fluid management, emphasizing replacement of intravascular volume and blood products, is essential.
- Summary of immediate objectives:
 - ◆ Prevent secondary cases (institute barrier precautions against blood and secretions ASAP).
 - ◆ Initiate evacuation procedures.
 - ◆ Consider treatable diseases in differential diagnosis:

Falciparum malaria	Mefloquine 1250 mg (5 tabs) given in a divided PO dosing schedule with 3 tabs initially followed by 2 tabs 8-24 hours later OR Malarone 4 adult tabs PO QD for 3 days
Meningococcemia	Ceftriaxone 50 mg/kg per day (2 gm max) IM for 14 days
Leptospirosis	Doxycycline 100 mg PO BID for 10 days
Typhus	Doxycycline 200 mg PO single dose
Septicemia	Ceftriaxone 50 mg /kg per day (2 gm max) IM for 14 days
Plague	Doxycycline 100 mg PO BID for 10 days
Hemorrhagic Fever With Renal Syndrome	Ribavirin (Huggins et. al. <i>J Infect Dis</i> 164:1119-27, 1991)

◆ Clinical clues to differentiate from CCHF:	
CCHF	Pharyngeal hyperemia, petechiae, and hemorrhage on soft palate
Falciparum malaria	DIC and/or bleeding as a preterminal event occurring more than 7 days following onset of fever
Meningococemia	Purpura occurring very soon (within hours) after onset of illness
Leptospirosis	Hemorrhage, especially pulmonary, with jaundice
Typhus	Maculopapular rash development on days 4-7 with progression to purpura
Septicemia	DIC and/or bleeding less prominent than organ failure
Plague	Acral gangrene with septicemic plague, no adenopathy

Disposition:

- Local hospitalization is favored during acute illness if possible. If evacuation to larger facilities is unavoidable, strict isolation must be observed.
- Depending on clinical response, evacuation for extended convalescence after acute illness may be required.
- Rapidly recovered cases may return to duty.

Prognosis:

- Treated: unknown.
- Untreated: 10-70% mortality; nosocomial cases may be associated with higher mortality than sporadic cases.
- Survivors generally suffer no major sequelae.

Prevention and Public Health Measures:

- Investigate possible infection sources.
- Identify and clinically assess close contacts.
- Report case as a reportable event using theater medical surveillance reporting channels.

DENGUE FEVER AND DENGUE-LIKE DISEASES

Communicability:

- Route: Dengue is transmitted by the daytime biting *Aedes* mosquito.
- Isolation: not directly transmitted from patient to patient. Patients must be protected from exposure to day-biting mosquitoes until afebrile. Insect repellent, permethrin-treated uniforms, and permethrin-impregnated bed netting should be used.
- Prophylaxis: none.

Incubation period: 3-14 days, commonly 4-7 days.

Diagnosis: Primary infection of adults with dengue virus or similar viruses results in an undifferentiated febrile syndrome known as dengue fever (DF). A positive tourniquet test or spontaneous epistaxis may occur in DF. Secondary dengue cases in persons who may have previously had a dengue infection can manifest with critical hemorrhagic manifestations that require aggressive clinical management (dengue hemorrhagic fever, or DHF). Very rarely does primary dengue infection result in a significant hemorrhagic diathesis or capillary leakage and shock. Therefore, extensive petechiae, ecchymoses or significant bleeding should prompt immediate consideration of more serious diseases, such as DHF or CCHF; see Fever Algorithm.

- Symptoms (abrupt onset):
 - ◆ Fever, may be biphasic (saddleback).
 - ◆ Chills.
 - ◆ Headache, diffuse.
 - ◆ Eye pain.
 - ◆ Intense long bone and joint pain.
 - ◆ Myalgias.
 - ◆ Anorexia.
 - ◆ Nausea.
 - ◆ Vomiting.
 - ◆ Lassitude.
 - ◆ Cutaneous hyperesthesia.

- Signs:
 - ◆ Initially, a transient, generalized, flushing rash.
 - ◆ Bradycardia.
 - ◆ Rash, morbilliform or macular, erythematous and blanching on day 3, 4 or 5.
 - ◆ Petechiae, epistaxis or positive tourniquet test.
 - ◆ Generalized lymphadenopathy.
 - ◆ Significant bleeding and shock suggest alternate diagnoses (DHF, CCHF or complicated Rift Valley Fever. Also see Fever Algorithm).
- Tourniquet Test - assesses capillary fragility (www.cdc.gov/ncidod/dvbid/dengue)
 - ◆ Inflate blood pressure cuff to midway between systolic and diastolic for 5 minutes.
 - ◆ After deflating the cuff, wait for skin to return to its normal color.
 - ◆ Positive test: 20 or more petechiae per 1 inch² (6.25 cm²).
- Laboratory findings:
 - ◆ Hematologic:
 - Hb/Hct: normal.
 - WBC: leukopenia ($\leq 1,500$ WBC/mm³).
 - Platelets: normal or low.
 - ◆ Chemistry, Urinalysis, Microbiology, Coagulation: not applicable.
 - ◆ Serology: Diagnosis by single acute IgM serum ELISA OR paired acute and convalescent sera 10-14 days apart using ELISA.
- Diagnostic confirmation: Numerous arthropod-borne viruses endemic in the region cause dengue-like illnesses which are indistinguishable solely on clinical grounds. These can be definitively distinguished only by paired serology or demonstration of specific viral antigen. Exact diagnosis often has no specific vector control, treatment or prophylaxis consequences, but there are exceptions: Rift Valley Fever is transmitted by contact with infected animals or mosquitoes and there is a vaccine available. CCHF is a severe, tick-borne viral hemorrhagic fever, which poses the threat of nosocomial spread, and for which specific treatment and chemopro-

phylaxis exists. Arthropod or animal control would also have an impact in cases of infection with hantaviruses as well as West Nile, Karshi, and tickborne encephalitis viruses.

Duration: Usually 5-7, but up to 10 days.

Complications: Psychiatric depression may be severe.

Treatment:

- No specific therapy exists.
- Therapy is supportive; hydrate to maintain intravascular volume and correct electrolyte abnormalities, and administer analgesics and antipyretics (NSAIDs are not recommended for DHF). DO NOT OVERHYDRATE. Appropriate fluid hydration decreases DHF mortality; consider blood transfusion if Hct falls below 30%. Although platelet transfusions are generally not effective in reducing bleeding complications, consider if platelet count is less than 10,000.

Disposition: May be hospitalized in theater; most will be able to return to duty in 1 week, but there may be need for up to 30 days of convalescence. Development of severe systemic symptoms may require evacuation.

Prognosis:

- Mortality:
 - ◆ DF is a nonfatal disease; complete recovery is expected.
 - ◆ DHF is associated with a 0.5 to 15% mortality rate.
- Subsequent dengue virus infection is more likely to be complicated by hemorrhage and shock.
- Patients may experience fatigue, weakness, and depression for several weeks.

Prevention and Public Health Measures:

- Command emphasis on use of personal protective measures (use of insect repellents and impregnated mosquito netting, application of permethrin to clothes and netting if not previously treated).
- Insecticide applications to mosquito habitats.
- Eliminate mosquito breeding sites (standing water).
- Protection of patients from mosquito bites.
- Report case as a reportable event using theater medical surveillance reporting channels.

FILARIASIS

Filarial parasites are long thread-like roundworms that dwell in tissue and produce microfilariae. The microfilariae are immature larval forms found in blood and skin and are the infective forms for the insect vector. Bancroftian filariasis is caused by the mosquito-borne nematode Wuchereria bancrofti. Not seen in Central Asia.

Communicability:

- Route: Infective larvae transmitted by mosquito bite.
- Isolation: not required.

Incubation period: 3-12 months.

Diagnosis: Symptoms and signs can be grouped as inflammatory, chronic obstructive or atypically hypersensitive.

- Symptoms/Signs (acute inflammatory, most likely to be seen in our nonimmune troops):
 - ◆ An acute attack of localized pain, tenderness, swelling, and erythema is the hallmark of lymphatic filariasis. Genitalia (42%), arms (25%), legs (11%).
 - ◆ Fever, sweats, chills, headache, lethargy, weakness, myalgias, and arthralgias after the lymphadenitis.
 - ◆ A retrograde lymphangitis follows within hours and is strongly diagnostic for filariasis.
 - ◆ Painful, swollen, tender testicle and/or epididymis.
- Symptoms/Signs (chronic obstructive, requires prolonged exposure over years following multiple acute attacks; may be seen in endemic populations):
 - ◆ Chronic lymphedema.
 - ◆ Hydrocele/chylocele.
 - ◆ Lymph varices.
 - ◆ Lymph scrotum.
 - ◆ Elephantiasis.
 - ◆ Chyluria.
- Laboratory findings:
 - ◆ Routine labs are not necessary. Eosinophilia frequently accompanies the acute phase symptoms.

- ◆ Diagnosis established by identifying the microfilaria in peripheral blood.
 - Giemsa stain thick film of peripheral blood collected between 2000 and 0200 as this is a nocturnally periodic parasite.
 - Concentration of peripheral blood:
 - Collect 5 mL of heparinized peripheral blood (nighttime collection will increase sensitivity).
 - Pass it through a 3-5 mm Nucleopore® filter.
 - Pass 10 mL distilled water through the filter to remove debris.
 - Place membrane on slide and stain With Giemsa.
- ◆ Serology: only available in research labs.

Duration:

- Treated: Curative.
- Untreated: variable, recurrent attacks of acute lymphadenitis/lymphangitis frequently occur.

Complications: chronic obstructive phenomena if unrecognized and untreated.

Treatment: diethylcarbamazine (DEC) and ivermectin (Mectizan®) - not available in a field situation. Doxycycline may be beneficial in the absence of these antihelminthics.

Prognosis: Excellent.

Disposition: Evacuate all nonmission-essential individuals with suspected or confirmed filarial infection to a CONUS facility such as Walter Reed Army Medical Center (WRAMC) or National Naval Medical Center (NNMC) where tropical medicine expertise is available.

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Prevention and Public Health Measures:

- Command emphasis on use of personal protection measures (use of insect repellents and impregnated netting, application of permethrin to clothes and netting if not previously treated).
- Insecticide applications to mosquito habitats.
- Eliminate mosquito breeding sites (standing water).
- Protection of patients from mosquito bites.
- Report case as a reportable event using theater medical surveillance reporting channels.

HEPATITIS (VIRAL)

Communicability:

■ Route:

- ◆ Hepatitis A and hepatitis E (epidemic non-A, non-B hepatitis):
 - Usually contracted by ingestion of contaminated food or water, or after physical contact with an infected individual (i.e., fecal-oral transmission).
 - Hepatitis A is rarely spread by male homosexual activity, among IV drug abusers or by blood transfusions.
- ◆ Hepatitis B, hepatitis delta, and hepatitis C are contracted by exposure to infected blood, blood products, other infected bodily fluids, or by sexual activity. Hepatitis delta occurs as co-infection with acute hepatitis B or as superinfection with chronic hepatitis B.

■ Isolation:

- ◆ Hepatitis A, E: contact precautions, hand washing.
- ◆ Hepatitis B, delta, C: standard precautions.
- ◆ Infectiousness is generally greatest during incubation period and early icteric phase of illness, but may persist with hepatitis B or C for much longer periods.

■ Prophylaxis:

- ◆ Hepatitis A: Vaccinate with Hepatitis A vaccine.
- ◆ Hepatitis B: Vaccinate with Hepatitis B vaccine.
- ◆ Hepatitis C: No prophylaxis available.
- ◆ Hepatitis E: No prophylaxis available.

Incubation:

- Hepatitis A: 30 days (range: 15-45).
- Hepatitis B: 70 days (range: 30-180).
- Hepatitis C: 50 days (range: 15-150).
- Hepatitis D: less well defined; probably similar to hepatitis B.
- Hepatitis E: 40 days (range 15-60).

Diagnosis: the clinical manifestations of acute hepatitis caused by the various viral agents overlap. Specific diagnosis is based on serology. For any type of viral hepatitis, the spectrum of disease may range from inapparent to fulminant.

■ Symptoms:

- ◆ Malaise.
- ◆ Anorexia, including loss of taste for tobacco.
- ◆ Nausea and/or vomiting.
- ◆ Right upper quadrant pain/discomfort.
- ◆ Pruritus.
- ◆ Arthritis/arthralgia.
- ◆ Headaches.
- ◆ Fever (low grade).

■ Signs:

- ◆ Icterus/jaundice.
- ◆ Tender hepatomegaly (mild-moderate).
- ◆ Splenomegaly (uncommon).
- ◆ Palmar erythema.
- ◆ Spider angiomas.
- ◆ Jaundice.
- ◆ Dark urine.
- ◆ Light color (acholic) stools.
- ◆ Low-grade fever (although fever is usually absent).

■ Laboratory findings:

- ◆ Hematologic:
 - Hgb/Hct: usually normal; hemolysis occurs uncommonly.
 - WBC:
 - normal or mild leukopenia.
 - mild lymphocytosis with or without atypical lymphocytes may occur.
 - Platelets: normal.
- ◆ Chemistry:
 - Transaminases:
 - rise 5-100 times normal.
 - ALT (SGPT) > AST (SGOT).
 - ALT usually >1000 in acute viral hepatitis.
 - Bilirubin: rises 1-20 times normal.

- Alkaline phosphatase: raises mildly, 1-4x normal.
- Albumin/globulin: remains normal or near normal in uncomplicated acute hepatitis.
- ◆ Urinalysis:
 - Positive for bile.
 - Occasional microhematuria.
 - Occasional mild proteinuria.
- ◆ Microbiologic: not applicable.
- ◆ Serology:
 - Anti-hepatitis A IgM suggests acute hepatitis A.
 - Anti-hepatitis A IgG indicates prior infection with hepatitis A or immunity following vaccination.
 - Hepatitis B surface antigen (HBsAg) indicates active infection with hepatitis B, acute or chronic.
 - Hepatitis B “e” antigen indicates greater infectiousness.
 - Anti-hepatitis B surface antibody denotes immunity from prior infection or vaccination.
 - Anti-hepatitis B core IgM antibody indicates acute infection with hepatitis B, (IgG: past infection).
 - Anti-hepatitis C antibody indicates prior or ongoing infection with hepatitis C.
 - Anti-hepatitis E IgM suggests acute hepatitis E, (IgG past or current infection).
- ◆ Coagulation:
 - Generally normal in uncomplicated acute viral hepatitis.
 - Prothrombin time (PT) rises in fulminant hepatitis.
- X-rays: nonspecific.
- Invasive procedures: not indicated.
- Diagnostic confirmation: serologic.

Duration:

- Icteric phase: 1-3 weeks.
- Convalescent phase: may require up to several months.

Complications:

- Fulminant hepatitis:
 - ◆ Presentation: hepatic encephalopathy, asterixis, coma, coagulopathy, death.
 - ◆ Treatment:
 - Supportive to include bed rest, protein restriction.
 - Lactulose in sorbitol orally, if tolerated; by enema otherwise; or oral neomycin.
 - Pregnant women with hepatitis E are at particular risk for fulminant hepatitis associated with a 15-40% mortality.
- Progression to chronic hepatitis:
 - ◆ Hepatitis A or E: none.
 - ◆ Hepatitis B: 5-10%.
 - ◆ Hepatitis C: up to 50-70%.
- Pancreatitis.

Treatment:

- No specific treatment is available for acute viral hepatitis.
- Rest is important.
- Discontinue any nonessential medications.

Disposition:

- Mild cases may be hospitalized in theater, as some will be able to return to duty in 2-3 weeks.
- Evacuate moderate or severe cases.

Prognosis:

- Mortality: less than 1%, (except hepatitis E, up to 5% in normal hosts, greater in pregnancy).
- Chronic disease: see complications above.

Prevention and Public Health Measures:

- Hepatitis A:
 - ◆ Hepatitis A vaccine, if available.
 - ◆ Command emphasis on proper sanitation.
 - ◆ Proper food preparation/water purification.
 - ◆ Personal hygiene.
- Hepatitis B, delta, and C:
 - ◆ Vaccinate high-risk populations with hepatitis B vaccine. Vaccination series requires three injections at 0, 1, and 6 months.
 - ◆ Sexual abstinence or use of barrier (condom) protection.
 - ◆ Screening of blood products for hepatitis B and C.
 - ◆ Use of standard precautions by health workers when dealing with blood or other body fluids.
- Immune globulin (for hepatitis A) or hepatitis B immune globulin (HBIG) can be administered as post-exposure prophylaxis in previously unvaccinated persons.
- Report all cases using theater medical surveillance reporting channels.

LEISHMANIASIS, CUTANEOUS (OLD WORLD)

(see also appendix F)

Communicability:

- Route: parasite inoculated into skin by the bite of an infected sand fly.
- Isolation: not required.
- Prophylaxis: none.
- Highly endemic in Iran, Iraq, Afghanistan. Hundreds of *Leishmania major* (*L. major*) cases have been diagnosed in American military deployed to Iraq. *Leishmania tropica* (*L. tropica*) is more common with Afghanistan exposure although few cases have been found to date in American military personnel.

Incubation period: usually 2-8 weeks, but may be longer (unusual past 6 months).

Diagnosis:

- Symptoms/signs: Chronic inflammatory papule/nodule(s) that slowly increases in size and ulcerates over several weeks. Base will crust over but the ulcer spreads under the edge of a firm and raised border. Lesions are usually on exposed skin and are rarely seen on the palms and soles. Secondary bacterial infection with pus (otherwise unusual with leishmaniasis alone) can be seen which can delay healing and lead to lesion enlargement.
- Laboratory confirmation: requires parasite demonstration. Follow procedures as outlined in the Leishmania Scraping and Biopsy Procedures appendix. **Note:** Optimally, the parasite should be demonstrated for definitive diagnosis since clinical diagnosis is less accurate. Clinical judgement should be used in the situation of a lesion that seems to be healing (completely epithelialized) as to whether diagnostic procedures should be implemented.

Treatment: The diagnosis must be confirmed parasitologically before treatment is offered (see above).

- Small lesions (< 2 cm total diameter) that are few in number (less than 5-10) may not need treatment if *L. major*

(99% of cases in American soldiers in Iraq have been *L. major*). If sores are persistent or patient requests therapy, then localized measures such as cryotherapy or heat (using Thermomed™ device) may be considered.

- Larger lesions due to *L. major*, off-label fluconazole 200 mg PO QD for 6 weeks (liver function tests should be checked in mid course) may be tried in individual cases; expect an efficacy of about 60%.
- Large lesions, lesions on the face or other cosmetically concerning areas, over joints (especially elbows and fingers), on hands or feet, lesions with regional adenopathy or subcutaneous nodules, and lesions due to *L. tropica* or *L. infantum* should be referred to one of the two Leishmaniasis Treatment Centers for consideration of Pentostam® (parenteral pentavalent antimony). These treatment centers are located at Walter Reed (Washington, DC) and Brooke (San Antonio, Texas) Army Medical Centers (WRAMC/BAMC). See detailed service policies on-line at www.pdhealth.mil (search under leishmaniasis).

Disposition: Evacuate patients to WRAMC/BAMC CONUS treatment centers if Pentostam therapy is needed. This requires a 10-20 day course of daily intravenous therapy under a research protocol as the drug is not FDA approved, although it has been used for this condition for more than 50 years.

Complications: Secondary infection with staphylococcal and streptococcal bacteria may cause lymphangitis and cellulitis. Leishmaniasis is a scarring condition and early treatment may decrease the size of scar. Big scars over small joints may permanently affect mobility. Mucosal leishmaniasis is rarely reported from the Old World and then usually due to *L. tropica*.

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®Pentostam is a registered trademark of GlaxoSmithKline, Research Triangle Park, North Carolina.

Prognosis: Very good. Old world cutaneous leishmaniasis generally heals without specific treatment in 6-12 months. Chronic cutaneous lesions (> 6-12 months duration) are usually associated with *L. tropica* infections.

Prevention and Public Health Measures:

- Command emphasis on use of personal protective measures (use of insect repellents, application of permethrin insecticides to clothes and netting if not previously treated). Sandflies are night-biting insects and use of clothing to avoid exposed skin at night is needed. Sand-fly season peak in Iraq is May – September.
- Insecticide applications to sand-fly habitats.
- Reservoir host (rodents and canines) control.
- Protection of patients from sand-fly bites.
- Individuals who have a confirmed diagnosis of cutaneous or visceral leishmaniasis are prohibited from blood donation for life according to recent policies of both military and civilian blood banks. This policy is based on the motivation of blood banks to eliminate all possible transfusion-associated infection risk. The likelihood that an individual, who is healed of active disease, either naturally or by specific treatment, will remain infectious and able to transmit leishmaniasis by blood donation is rare to theoretical in the case of *L. major*. However, the simplest course of action to eliminate any possibility of transfusion-associated leishmaniasis is donor deferral. The number of persons affected by this policy is relatively few compared to the size of the donor pool. Thus, the impact of donor deferral for leishmaniasis to the donor pool is trivial. It is important to explain the reasons for donor deferral to those who have been infected with leishmaniasis; otherwise, they may think that they remain infectious. As stated above, almost all cases to date in the American forces in Iraq have been *L. major*, and there has never been a reported case of transfusion-associated leishmaniasis caused by *L. major*.
- Report case as a reportable event using theater medical surveillance reporting channels.



'Phlebotomus papatasi' or sand fly



Multiple sand fly bites.



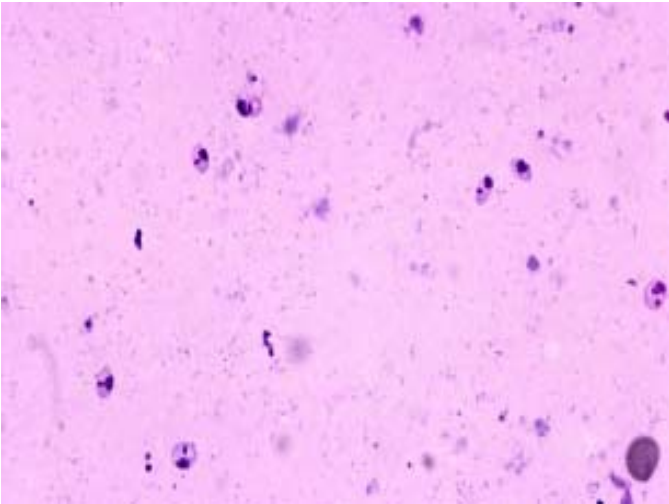
Impetiginous secondary infection of cutaneous leishmaniasis lesions.



Large ulcerated lesion.



A lesion after it has crusted and begun healing.



Skin scraping with giemsa staining.

LEISHMANIASIS, VISCERAL

Communicability:

- Route:
 - ◆ Sand fly (Phlebotomus species) bites.
 - ◆ Other transmission routes have been reported.
 - Sexual intercourse (two reports).
 - Infected blood transfusion (several reports).
 - Accidental inoculation in a laboratory.
 - Vertical transmission from mother to fetus.
- Isolation: generally not required. In forward areas or under field conditions where continued exposure to sand flies may occur, personal measures to protect the patient from sand fly bites, including insect repellents and permethrin-impregnated netting, should be used.
- Prophylaxis: no immunoprophylaxis, chemoprophylaxis or vaccine available.
- Has been reported from the CENTCOM AOR, particularly in Afghanistan, and is quite common in India.

Incubation: normally 3-8 months (range: from 10 days to more than 10 years).

Diagnosis: Acute visceral leishmaniasis in an immunologically naive adult will frequently present as an acute febrile illness that is often confused with malaria. There are no pathognomonic signs or symptoms that will distinguish this febrile illness from many other causes. Patients are often described as not toxic even with fever $> 40^{\circ}\text{C}$. The acute presentation can resolve, progress to kala-azar, or evolve to a chronic syndrome of abdominal complaints, diarrhea, night sweats, weight loss, and fatigue. Consider visceral leishmaniasis in the differential diagnosis of all acutely febrile patients.

- Symptoms:
 - ◆ Onset may be insidious or abrupt.
 - ◆ Fever: high intermittent or remittent, can be associated with chills or prostration.
 - ◆ Sweats.

- ◆ Nonproductive cough.
- ◆ Epistaxis.
- ◆ Abdominal discomfort and/or swelling.
- ◆ Weight loss.
- ◆ Diarrhea.
- ◆ Peripheral edema (late).
- ◆ Bleeding diathesis (late).
- ◆ Generalized weakness (as emaciation progresses).
- ◆ Headaches $\pm$ nuchal rigidity.

■ Signs:

- ◆ Weight loss/emaciation.
- ◆ Splenomegaly (presents early, progressively worsens).
- ◆ Hepatomegaly (less pronounced than splenomegaly).
- ◆ Lymphadenopathy (solitary, regional or generalized).
- ◆ Fever (39 to 40°C).
- ◆ Skin:
 - trophic changes (due to malnutrition): thinning, dryness, hair loss, hypopigmentation.
 - polymorphic lesions: papules, wart-like nodules, ulcers (rare).
 - petechiae, purpura, bruises.
- ◆ Eye: retinal hemorrhage, papilledema, eyelid nodules, anterior uveitis (rare and occur late).
- ◆ Jaundice (in advanced disease).
- ◆ Nodules or ulcers of oral and/or nasopharyngeal mucosa (rare).
- ◆ Edema (associated with hypoalbuminemia with proteinuria).
- ◆ Bleeding: epistaxis, gingival, vaginal, other sites.
- ◆ Ascites (advanced disease).

■ Laboratory findings:

Note: In early illness there may only be mild anemia and transaminase elevation. Leukopenia and thrombocytopenia seen in advanced disease with hepatosplenomegaly.

- ◆ Hematologic:
 - anemia (normochromic, normocytic).
 - leukopenia.
 - thrombocytopenia.

- Coombs test usually positive.
- marked decrease or absence of eosinophils.
- parasitemia may be occasionally detected on peripheral blood smear.
- buffy coat smears may be diagnostic.
- ◆ Chemistry:
 - polyclonal hypergammaglobulinemia.
 - positive rheumatoid factor.
 - hypoalbuminemia.
 - elevated transaminases.
 - hyperbilirubinemia (advanced disease).
- ◆ Urinalysis:
 - proteinuria (occasional).
 - hematuria (occasional).
- ◆ Microbiologic: standard microbiologic techniques are not applicable.
- ◆ Serologic: Serologic diagnosis available.
- ◆ Coagulation:
 - bleeding and clotting times are generally normal.
 - prothrombin time (PT) may be mildly prolonged (2-4 seconds more than control).
- X-ray:
 - ◆ Standard examinations are nonspecific.
 - ◆ Hepatomegaly and splenomegaly can be detected by appropriate imaging modalities (sonogram, CT, etc.).
- Invasive procedures:
 - ◆ Aspiration/biopsy of bone marrow, spleen, liver, or lymph nodes.
 - ◆ Attempt to visualize amastigotes on Wright or Giemsa stained smears of tissue and /or (+) culture.
- Skin testing: Leishmanin skin test will be negative in active disease and is not useful for diagnosis.
- Diagnostic confirmation:
 - ◆ Diagnosis must be confirmed by the demonstration of parasites in a tissue aspirate or biopsy.
 - ◆ Culture of organism from tissue aspirate specimens is possible with specialized technique (NNN or Schneider's media), but this should only be attempted in facilities with experience in culturing leishmania.

Duration:

- Treated: varies with therapeutic regimen; generally about 1 month with sodium stibogluconate (Pentostam) therapy; however, fever will respond within 48-72 hours of starting therapy, and patient will feel improved within first week.
- Untreated: indefinite.

Complications:

- Renal:
 - ◆ Renal amyloidosis with nephrotic syndrome.
 - ◆ Immune-complex mediated glomerulonephritis .
- Hepatic:
 - ◆ Acute liver failure may rarely occur.
 - ◆ Cirrhosis (rare).
- Disseminated intravascular coagulation (DIC).
- Hemorrhage.
- Secondary infections usually seen only in advanced cases with accompanying malnutrition.
 - ◆ Tuberculosis.
 - ◆ Pneumonia.
 - ◆ Dysentery.
 - ◆ Measles, in previously unvaccinated individuals.
 - ◆ Herpes Zoster.
- Persistent post-disease splenomegaly.

Treatment: visceral leishmaniasis is not a life threatening disease acutely. However, all patients with suspected or confirmed visceral leishmaniasis must be evacuated to CONUS facilities (WRAMC). These patients should not be treated in theater or in Europe.

- Standard therapy:
 - ◆ Liposomal amphotericin (AmBisome®) 3 mg/kg, days 1-5, day 14 and day 21.

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◆ Alternatives:

- Sodium stibogluconate (Pentostam), 20 mg/kg IV QD, for 30 days.
- Sodium stibogluconate is not a licensed product in the United States, and must only be given under a treatment IND protocol. Patients requiring stibogluconate treatment must be evacuated to a CONUS facility such as WRAMC or NNMC.

Prognosis: generally good; mortality usually occurs only in advanced disease, but even advanced disease may be successfully cured.

Disposition: evacuate all patients with suspected or confirmed visceral leishmaniasis. This disease is slowly progressive and should not be so far advanced in U.S. military personnel that emergency treatment is required.

Prevention and Public Health Measures:

- Command emphasis on use of personal protection (repellent, impregnated netting, application of permethrin to clothes and netting if not previously treated).
- Insecticide applications to sand fly habitats located near troop areas.
- Control of wild canids and feral dogs (foxes, jackals, dogs may be natural reservoirs of infection; probably includes domestic dogs as well).
- Protection of patients from further sand fly bites, thus aborting possibility of epidemics based on human reservoirs.
- Report all cases through preventive medicine channels.

LEPTOSPIROSIS

Communicability:

- Route:
 - ◆ Transmitted by contact of mucous membranes or skin with water or soil contaminated by urine from leptospiuric animals.
 - ◆ Transmitted by ingestion of food contaminated by leptospiuric animals.
- Isolation: Standard precautions.
- Prophylaxis: None: person-to-person transmission is rare.
- Chemoprophylaxis: Doxycycline 200 mg weekly is effective in areas and settings with potential of high exposure.

Incubation: 10 days, (range 2-26 days).

Diagnosis: Infection is typically inapparent or mild. Symptomatic leptospirosis usually presents as an anicteric, often biphasic illness. Less often, severe, icteric disease with renal failure and hemorrhage occurs. Exposure history including contact with contaminated water (swimming, wading, or drinking) or soil (digging, farming, or construction) may be helpful.

- Symptoms (abrupt onset):
 - ◆ Fever (100% -may be biphasic).
 - ◆ Myalgia (97% -especially calves and thighs).
 - ◆ Headache (95%).
 - ◆ Chills (85%).
 - ◆ Sore throat (14%).
 - ◆ Nausea/vomiting (40%).
 - ◆ Eye pain (50%).
 - ◆ Cough (25-50%).
 - ◆ Diarrhea (23%).
- Signs:
 - ◆ Conjunctival injection (30-100%).
 - ◆ Muscle tenderness (50-70%).
 - ◆ Hepatosplenomegaly (10-50%).
 - ◆ Lymphadenopathy (20-50%).
 - ◆ Pulmonary findings (11%).

- ◆ Jaundice (5-100%).
- ◆ Rash (0-7%).
- Laboratory findings:
 - ◆ Hematologic:
 - WBC variable, but neutrophilia common.
 - Thrombocytopenia.
 - ESR increased.
 - ◆ Chemistry:
 - BUN/creatinine increased.
 - ALT/AST mild elevations.
 - alkaline phosphatase mild elevations.
 - aldolase increased.
 - CPK increased.
 - ◆ Urinalysis:
 - proteinuria.
 - pyuria.
 - microscopic hematuria.
 - ◆ Microbiologic: Culture is seldom useful in initial diagnosis. Unique media needed is not routinely available, although some standard blood culture systems will support growth.
 - ◆ Serologic: Field diagnosis is limited to rapid diagnostic kits (dipstick and EIA) when available. Definitive diagnosis is obtained with microscopic agglutination testing (MAT). This test is performed only at reference labs and usually with paired acute and convalescent serum (2-3 weeks apart).
- X-ray: variable in those with symptomatic pulmonary disease, most often small patchy, peripheral infiltrates.

Duration: 3 days to 3 weeks.

Complications:

- Anicteric leptospirosis: Aseptic meningitis, meningoencephalitis, uveitis, myocarditis, and iridocyclitis.
- Icteric leptospirosis (Weil's disease): Jaundice, renal failure, hemorrhage (especially pulmonary), and death.

Treatment:

- All cases should receive either doxycycline 100 mg PO BID for 7 days; or if severely ill, high dose parenteral aqueous penicillin G 110,000 U/kg per day (6-8 million U/day) IV as four divided doses every 6 hours. Ceftriaxone and

cefotaxime have also been shown to be effective. Initiation of therapy may be accompanied by high fever and hypotension within 12 hours after starting therapy (Jarisch-Herxheimer reaction, see Relapsing Fever complications section for characteristics and treatment suggestions).

- In icteric leptospirosis (Weil's Disease), careful maintenance of intravascular volume will reduce the incidence of renal failure requiring dialysis.

Disposition:

- For mild cases, hospitalization in theater.
- For complicated cases with hemorrhage or jaundice or renal insufficiency, evacuation to third or fourth echelon medical facilities.

Prognosis:

- Anicteric leptospirosis: if treated, complete recovery.
- Icteric leptospirosis: untreated, 5-30% mortality rate, mostly due to renal failure, but survivors recover completely. Protracted recovery has been described.

Prevention and Public Health Measures:

- Command emphasis on educating troops to avoid swimming, wading, and exposure to contaminated soil.
- Control local rodent populations.
- Report case as a reportable event using theater medical surveillance reporting channels.
- Consider troop prophylaxis in endemic areas with confirmed cases: doxycycline 200 mg PO once weekly during periods of high exposure.

MALARIA: GEOGRAPHICAL RISK

Country	Transmission Period	Distribution	Potential Rate per Month Without Countermeasures	Type
Afghanistan	May – November	Countrywide	Up to 11 – 50%	80-90% <i>P. vivax</i> 10-20% <i>P. falciparum</i>
Bahrain			Risk does not currently exist	
Djibouti	November – March	Countrywide	Up to 11 – 50%	90% <i>P. falciparum</i> 2% <i>P. vivax</i>
Egypt	June – October	Variable – Risk only occurs in focal rural areas of Al Fayyum Governorate	Infrequent cases	99% <i>P. vivax</i> 1% <i>P. falciparum</i>
Eritrea	Year-round	Variable	Up to 11 – 50%	85% <i>P. falciparum</i> <i>P. vivax</i> and <i>P. malariae</i> also occur
Ethiopia	Year-round	Variable	Up to 11 – 50%	85% <i>P. falciparum</i> <i>P. vivax</i> , <i>P. malariae</i> , and <i>P. ovale</i> also occur
Iran	March – November	Variable	Up to 1%	<i>P. falciparum</i> , <i>P. vivax</i> , and <i>P. malariae</i> all occur at varying levels
Iraq	May – November	Variable	Up to 2 – 10%	99% <i>P. vivax</i> 1% <i>P. falciparum</i>
Jordan			Risk does not currently exist	
Kazakhstan	May – September	Variable	Infrequent cases	Primarily <i>P. vivax</i> , but <i>P. falciparum</i> and <i>P. malariae</i> also occur
Kenya	Year-round	Countrywide	11 – 50%	90% <i>P. falciparum</i> <i>P. malariae</i> , <i>P. ovale</i> , and <i>P. vivax</i> also occur
Kyrgyzstan	June – September	Variable	Infrequent cases	Primarily <i>P. vivax</i> , but <i>P. falciparum</i> and <i>P. malariae</i> also occur

MALARIA: GEOGRAPHICAL RISK (continued)

Country	Transmission Period	Distribution	Potential Rate per Month Without Countermeasures	Type
Kuwait			Risk does not currently exist	
Lebanon			Risk does not currently exist	
Oman	Year-round	Variable	Infrequent cases	90% <i>P. falciparum</i> 10% <i>P. vivax</i>
Pakistan	Year-round	Countrywide	Up to 2 – 10%	90% <i>P. falciparum</i> 10% <i>P. vivax</i>
Qatar			Risk does not currently exist	
Saudi Arabia	Year-round	Variable – Risk occurs in the southern and western provinces	Up to 1%	88% <i>P. falciparum</i> 12% <i>P. vivax</i>
Somalia	Year-round	Countrywide	Up to 11 – 50%	95% <i>P. falciparum</i> <i>P. vivax</i> , <i>P. malariae</i> , and <i>P. ovale</i> also occur
Sudan	Year-round	Countrywide	Up to 11 – 50%	90% <i>P. falciparum</i> <i>P. vivax</i> , <i>P. malariae</i> , and <i>P. ovale</i> also occur
Tajikistan	April – October	Variable	Up to 2 – 10%	84% <i>P. vivax</i> 16% <i>P. falciparum</i>
Turkmenistan	April – September	Variable	Infrequent cases	Primarily <i>P. vivax</i>
United Arab Emirates		Variable	Up to 1%	77% <i>P. vivax</i> 23% <i>P. falciparum</i>
Uzbekistan	June – September	Variable	Up to 1%	Primarily <i>P. vivax</i>
Yemen	Year-round	Countrywide	Up to 11 – 50%	95% <i>P. falciparum</i> <i>P. vivax</i> <i>P. malariae</i>

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MALARIA

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Communicability:

- Route:
 - ◆ The malaria parasite is transmitted by bites of infected anopheline mosquitoes (primarily nighttime biters, including evening and early morning).
 - ◆ Transfusion of malaria-infected blood will transmit infection.
 - ◆ IV drug abusers sharing contaminated needles have become infected.
- Isolation: Malarious patients may also have the sexual forms of the malaria parasite in their blood. To protect others in the hospital from being infected, permethrin-impregnated bed netting should be used. No other isolation is required.
- Prophylaxis: Individuals who have had contact with malaria patients do not require prophylaxis.
- Chemoprophylaxis:
 - ◆ Chemoprophylaxis of all individuals should be considered for specific destinations where malaria is endemic. In Central Asia, Plasmodium falciparum accounts for less than 5% of cases and P. vivax predominates. Chloroquine-resistant P. falciparum is described in the area (see table). There have been no confirmed cases of malaria acquired in Iraq. Multiple cases of P. vivax malaria have been acquired in Afghanistan.
 - ◆ Recommended regimen: Follow Commander in Chief (CINC) surgeon advice for theater of operations. FDA approved regimens include:
 - Chloroquine 1 adult tablet PO QD, starting 2 weeks before arrival in country and continuing for 4 weeks (4 doses) after departure,
OR
 - Mefloquine 250 mg PO weekly, preferably beginning 2 weeks before arrival in country and continuing for 4 weeks (4 doses) after departure,
OR
 - Doxycycline 100 mg PO QD, preferably beginning 1-2 days before arrival in

country and continuing for 4 weeks after departure,
OR

- Malarone 1 adult tablet PO QD starting 1-2 days before arrival in theater and continuing 7 days after departure from theater.
 - The choice of a particular regimen is based on a risk – benefit assessment of each regimen and the deployment-specific infection risk.
- ◆ Primaquine is the only available drug that can eradicate persistent hepatic parasites, called hypnozoites, of P. vivax malaria. Use of primaquine, concurrently with chloroquine, in symptomatic patients with documented smear positive P. vivax malaria is referred to as “radical cure.” Primaquine must not be given to pregnant or G6PD deficient individuals because of the risk of hemolytic anemia. Different strains of vivax parasites have varying tolerances to primaquine; therefore, dosage recommendations may vary by geographical region. Studies of Afghan refugees indicate that 30 mg daily for 14 days is needed to reduce relapse rates for vivax infections from that region. When used for radical cure, give primaquine phosphate (30 mg primaquine base) by mouth daily for 14 days to individuals known to be G6PD normal.
- Use of primaquine, concurrently with an anti-malarial drug, in asymptomatic patients when they leave an endemic area is referred to as anti-relapse therapy (also called terminal prophylaxis). Individuals unable to take primaquine because they are G6PD deficient should not be given primaquine for anti-relapse therapy.
 - The current FDA approved dose regimen for primaquine for both the radical cure and anti-relapse therapy indications is 15 mg (base) PO QD X 14 days. Recently, an expert panel recommended a higher dose of 30 mg (base) PO QD X 14 days for strains of P. vivax known to require higher dose for cure, and this is the current Center for Disease Control (CDC)-recommended, first-line regimen. Current DOD Force Health Protection (FHP) policy does not allow use of FDA approved drugs with unapproved dose regimens. Therefore, the use of primaquine for FHP should be limited to the approved

regimen of 15 mg (base) PO QD X 14 days. Individual physicians are free to prescribe the higher dose of primaquine to individual patients for radical cure indication as practice of medicine, not governed by FHP.

- ◆ Specific malaria chemoprophylaxis dosage recommendations from the CINC surgeon, although frequently identical to those used to treat a P. vivax malaria infection, will depend on the geographic area and season of exposure.
 - In areas where there is seasonal transmission of P. vivax malaria, chemoprophylactic medications should be discontinued at the end of transmission season. For those with heavy exposure to P. vivax, post-exposure chemoprophylactic medications such as primaquine should be considered for the appropriate duration after the last possible period of malaria transmission has occurred.

Incubation: Malaria can present as soon as 5 days after arrival in an endemic area, and anytime thereafter, under conditions of continuous exposure. Usual incubation periods are:

- P. falciparum: 12 days (range 9-30 days).
- P. vivax: 14 days (range 12 days - 10 months).
- P. malariae: 28 days (range 18-365 days).

Diagnosis: Malaria MUST be considered in all febrile patients.

If not diagnosed and treated promptly, P. falciparum is often fatal. Disease can occur before parasites are detectable by blood smear, but patients that are critically ill due to malaria will have a detectable parasitemia at some time in their illness. Patients with suspected malaria should have blood smear exams every 8-12 hours for 48 hours to exclude malaria. Persons on effective chemoprophylaxis may have very low parasitemias and atypical presentations.

- Symptoms:
 - ◆ Prodrome of malaise, fatigue, and myalgia may precede febrile paroxysm by several days.
 - ◆ Paroxysm characterized by abrupt onset of fever, chills, rigors, profuse sweats, headache, backache, myalgia, abdominal pain, nausea, vomiting, and diarrhea (may be watery and profuse) in P. falciparum.

- Signs: Intermittent fever to $\geq 40^{\circ}\text{C}$ (105°F). Fever may be almost continuous in *P. falciparum* malaria; classic “periodicity” is usually absent. Profuse sweating between febrile paroxysms. Tachycardia, orthostatic hypotension, tender hepatomegaly, moderate splenomegaly, and delirium (during fever; see “Cerebral malaria”).
- Laboratory findings:
 - ◆ Hematologic:
 - CBC
 - Anemia (normochromic, normocytic, hemolytic).
 - Leukopenia.
 - Monocytosis ($>10\%$).
 - Eosinophilia not seen.
 - Thrombocytopenia (to $<150,000/\text{mm}^3$).
 - Blood smears intra-erythrocytic parasites on smears of peripheral blood.
 - SMEAR MUST BE PREPARED AND EXAMINED 2-3 TIMES DAILY FOR 48 HOURS TO RULE OUT MALARIA.
 - Thick smears are more sensitive (about 20X) for finding parasites; thin smears are more accurate for identifying parasite species.
 - Thick smear: place one drop of blood on a slide; with the corner of another glass slide, spread drop until it is about dime size, and newspaper below slide can barely be read; wait until thoroughly dry. DO NOT METHANOL FIX; stain with Giemsa stain.
 - Thin smear: prepare film as for normal CBC, fix in methanol, use Giemsa stain.
 - ◆ Chemistry:
 - Hypoglycemia (may be severe, especially with quinine therapy, and may be recurrent).
 - Electrolyte abnormalities, including hyperkalemia (from RBC lysis), and hyponatremia (from reduced free water clearance).
 - Elevated transaminases (alkaline phosphatase normal).
 - Azotemia (pre-renal).
 - Hyperbilirubinemia.
 - ◆ Urinalysis: may be normal; but increased protein, urobilinogen, and conjugated bilirubin may occur.

- ◆ Microbiologic: standard techniques are not applicable.
- ◆ Coagulation:
 - Normal in uncomplicated disease, but prolonged prothrombin time (PT) and partial thromboplastin time (PTT) with disseminated intravascular coagulation (DIC) may be seen in late stage disease.
- X-ray: nonspecific.
- Diagnostic confirmation: Identification of parasite on blood smears.

Duration:

- Treated: 3-5 days in uncomplicated cases. May recrudescence within 4 weeks if parasite is drug resistant.
- Untreated:
 - ◆ P. falciparum rapidly fatal in untreated nonimmune patients.
 - ◆ P. vivax rarely fatal but relapses can occur in up to 50% of cases. Majority of cases occur within weeks to 1 year, but there are case reports of latency up to 8 years if persistent liver forms are not eliminated with primaquine (see Terminal prophylaxis).
 - ◆ P. malariae is rarely fatal but may persist for years if not treated.

Complications: The following complications strongly indicate infection with P. falciparum:

- Hyperparasitemia: $\geq 5\%$ of RBCs on thin smear parasitized; correlates with other complications in a non-immune patient, though complications can be seen with lower degrees of parasitemia.
- Cerebral malaria:
 - ◆ Altered mental status, personality changes, lethargy, stupor, coma or delirium.
 - ◆ Neurologic impairment: hyperpyrexia, monoplegia, hemiplegia, cerebellar signs, seizures (assess for hypoglycemia).
 - ◆ Treatment is with appropriate antimalarials and supportive care.
 - ◆ Mortality is high (20-50%), but survivors rarely show neurologic sequelae.

- Renal failure:
 - ◆ May be prerenal or intrarenal (ATN-like) in origin.
 - ◆ Treatment:
 - Assure adequate intravascular volume replacement.
 - Supportive care to include dialysis if needed.
- Adult respiratory distress syndrome (ARDS, noncardiogenic pulmonary edema):
 - ◆ Pathogenesis: due to increased capillary permeability and fluid extravasation. Avoiding excessive intravascular fluid administration may reduce incidence.
 - ◆ Treatment is supportive, to include mechanical ventilation with positive pressure.
- Splenic rupture/hemorrhage:
 - ◆ Spontaneous or from palpation of the spleen, especially with VIVAX malaria.
 - ◆ Treatment is emergent blood replacement and surgical control of hemorrhage.

Treatment:

- Treatment of choice:
 - ◆ Chloroquine plus primaquine for P. vivax malaria.
 - ◆ Initial treatment for patients with P. falciparum who are able to tolerate oral medication should be Malarone 4 tablets PO QD for 3 days (if not already on Malarone prophylaxis).
 - If mefloquine is the only anti-malarial drug available, then it should be used at a dose of 1250 mg PO (5 tablets). The FDA approved dose is 5 tablets as a single dose. This regimen is poorly tolerated. The dose should be split and given at least 8-12 hours apart to reduce toxicity. Mefloquine should not be the first choice for treatment because other, better tolerated drugs are currently available.
 - ◆ Alternatives:
 - Quinine 650 mg PO TID for 3 days PLUS doxycycline 100 mg PO BID X 7 days or Fansidar® 3 tablets PO one time.

®Fansidar is a registered trademark of Hoffman-La Roche, Inc., Nutley, New Jersey.

- ◆ Depending on response, prophylaxis can then be resumed, or patient can be evacuated and terminal prophylaxis given if needed.
- Critically ill patients who require IV medication can receive one the following regimens:
 - ◆ Quinidine gluconate in normal saline, 10 mg/kg (max 600 mg) IV loading dose over 1-2 hours, followed by 0.2 mg/kg/minute constant IV infusion for a maximum of 72 hours. Monitor ECG and switch to oral agents when mental status clears and parasitemia < 1%. Suitable oral agents to complete 7 days of therapy include: doxycycline 100 mg PO Q12H, or tetracycline 250 mg PO Q6H.
 - ◆ Quinidine gluconate, 15 mg/kg (max 650 mg) IV loading dose over 4 hours, followed by 7.5 mg/kg over 4 hours Q8H for 7 days. Monitor ECG and switch to oral agents as above.
 - ◆ Quinine dihydrochloride, 650 mg IV over 4 hours, Q8H for 7 days. Monitor ECG and switch to oral agents as listed above (if IV quinine is not available, use IV quinidine as listed above). The intravenous formulation of quinine is not FDA approved but may be available in some locations within the AOR.
- Specific Precautions for IV Quinidine and IV Quinine:
 - ◆ Monitor patient with ECG. Either IV quinidine or IV quinine should be slowed or temporarily stopped if the QRS complex widens by $\geq 50\%$ of baseline, or if the QT interval exceeds 0.6 seconds, or if hypotension develops which is unresponsive to fluid challenge.
 - ◆ Monitor patient for hypoglycemia and treat it immediately if it is noted during either IV quinidine or IV quinine therapy. Failure to monitor and treat promptly can result in potentially lethal or severe complications.

Disposition for uncomplicated cases: local hospitalization for 48-72 hours with limited duty for several days (until drug therapy is completed). In P. vivax cases, the patient should be gently examined to ensure that splenomegaly has resolved before allowing the patient to return to full duty.

MENINGOCOCCAL DISEASE

Communicability:

- Route: person to person by respiratory droplets.
- Isolation: droplet precautions for first 24 hours of antibiotic therapy; disinfect nasal and pharyngeal secretions and material contaminated with them.
- Prophylaxis:
 - ◆ Intimate and household contacts, including barracks and tent-mates should receive:
 - rifampin 600 mg PO Q12H for 4 doses, OR ceftriaxone 250 mg IM one dose, OR Ciprofloxacin 500 mg PO one dose,
 - PLUS
 - meningococcal vaccine, unless this has been received within 2 years prior to the incident exposure.
 - ◆ Casual contacts need not receive prophylaxis.
 - ◆ Prophylaxis is not uniformly effective. Even after receiving prophylaxis, close contacts with individuals who develop symptoms suggestive of meningococcal disease should be rapidly evaluated.

Incubation: 3-4 days (range 1-10 days).

Diagnosis: meningococcal infection may be asymptomatic, or may present either as a self-limited flu-like illness (without sequelae), as meningitis, as fulminant septicemia (meningococcemia), or as combined meningitis-septicemia. Clinical signs and symptoms will vary with the type of presentation. Serogroup B meningococcus is prevalent in Asia, therefore, vaccination history should not preclude a possible diagnosis of acute meningitis.

- Symptoms:
 - ◆ Meningococcemia (very abrupt onset with fulminant course):
 - Fever.
 - Headache.
 - Malaise.
 - Diarrhea (occasionally may be severe).

- ◆ Meningitis [onset may be abrupt or subacute (several days)]:
 - Headache.
 - Fever.
 - Malaise.
 - Photophobia.
 - Nausea/vomiting.
 - Backache.
- Signs:
 - ◆ Meningococcemia:
 - Fever.
 - Tachycardia.
 - General muscular tenderness.
 - Altered mental status.
 - Petechiae/purpura/ecchymoses (skin & mucosal) develop in one-third to one-half of cases. May progress to ecchymosis and ischemic necrosis. Rarely just a faint, morbilliform rash. Development of purpura or ecchymoses within a few hours of onset of illness is pathognomonic for meningococcemia.
 - ◆ Meningitis:
 - Headache.
 - Fever.
 - Meningismus/stiff neck.
 - Cranial nerve palsies (most commonly III, VI, and VIII).
 - Altered mental status.
 - Seizures.
 - Positive Kernig's sign.
- Laboratory findings:
 - ◆ Hematologic:
 - Meningococcemia:
 - HGB/HCT: nonspecific.
 - WBC: leukocytosis or leukopenia (leukopenia implies more fulminant illness).
 - thrombocytopenia: common.
 - Meningitis:
 - HGB/HCT: nonspecific.
 - WBC: leukocytosis more typical, leukopenia suggests sepsis/meningococcemia.

- platelets: usually normal: thrombocytopenia suggests sepsis/ meningococcemia.
 - CSF: see below.
- ◆ Chemistry: nonspecific; serum glucose and protein should be obtained for comparison against CSF values.
- ◆ Urinalysis: nonspecific.
- ◆ Microbiologic:
 - CSF Gram stain: positive in 50-90%, including meningococcemia without clinical meningitis. Organisms may be present prior to WBCs.
 - CSF culture: positive in 50-90%, including meningococcemia without clinical meningitis.
 - Blood culture: positive in 50-60%.
 - Organisms are fragile: smear and cultures should be prepared as soon as CSF is obtained from patient.
- ◆ Serology: not applicable.
- ◆ Coagulation: prothrombin time (PT) and partial thromboplastin (PTT) time may be prolonged in meningococcemia. Evidence of DIC, including decreased fibrinogen levels, and elevated levels of fibrin degradation products may be seen.
- X-ray: nonspecific.
- Invasive procedure:
 - ◆ In presence of meningitis or suspected meningococcemia, lumbar puncture for CSF should be performed immediately, unless papilledema or other focal neurologic signs suggestive of either intracranial mass or increased intracranial pressure are present.
 - ◆ CSF should be tested for glucose, cell count, Gram stain, and culture. Abnormal CSF results include but are not limited to:
 - glucose ≤ 40 mg/dl (in 75% of cases).
 - protein > 150 mg/dl (range 25-800).
 - WBC > 1000 cells/mm, neutrophils predominant (range 10- 65,000; lymphocyte predominance is seen in $<10\%$; poor prognosis associated with absence of WBCs in CSF, as may be caused by WBC lysis from large amounts of meningococcus).
- Diagnostic confirmation: culture of organism from clinical specimen (from CSF, or petechial aspirate). Chocolate agar plates required.

Duration:

- Treated: clinical response should occur within 48 hours. Duration of convalescence depends on severity of illness and its complications.
- Untreated: death may occur within minutes to hours. Mortality is 5-15%, even with the best of care.

Complications: shock, DIC, ARDS, pericarditis including tamponade, pneumonia, diabetes insipidus, cranial nerve palsies, prolonged mental status changes.

Treatment:

Treatment must be initiated immediately upon suspicion of meningococcal disease, with continued diagnostic evaluation to proceed simultaneously with initiated treatment regimen.

- ◆ Obtain rapid history and physical exam, identifying contraindications to lumbar puncture.
- ◆ While establishing IV line access, obtain blood for hemoglobin, chemistry, coagulation and culture studies.
- ◆ Perform LP if not contraindicated.
- ◆ Administer one of the following regimens of antibiotic. Duration of therapy for meningitis is specifically 10-14 days.
 - cefotaxime 2 gm IV Q4-6H.
 - ceftriaxone 2 gm IV Q12H.
 - penicillin G 4 mil units IV Q4H.
 - ampicillin 2 gm Q4H.

*For PCN allergic patients:

- gatifloxacin or moxifloxacin 400 mg IV Q day
- chloramphenicol 1 gm IV Q6H
- ◆ Provide hemodynamic and respiratory support as needed.
- ◆ Proceed with more detailed history and examination, and evaluate results of laboratory tests.
- If pneumococci are identified in CSF, add vancomycin 500-750 mg IV Q6H* to ceftriaxone 2 gm IV Q12H.
 (*Note: This dose is double a usual Vancomycin dose for other indications, but is NOT a typographical error for meningitis.)
- If *H. influenzae* are identified in CSF, treat with ceftriaxone 2 gm IV Q12H.

- In addition, consider dexamethasone 0.15 mg/kg IV Q6H X 2-4 day for any bacterial meningitis.

Disposition:

- Milder cases or cases that recover rapidly may be treated at hospitals in theater in anticipation of return to duty.
- Cases that initially appear more severe, become complicated, or convalesce more slowly should be evacuated after initial stabilization.

Prognosis:

- Treated: In properly treated cases, residual morbidity is not unusual, hearing loss may persist, and mortality may be as high as 5-10%.
- Untreated: Mortality may range from 50-85%.

Prevention and Public Health Measures:

- Vaccination of susceptible populations. An effective quadrivalent vaccine (affording protection against serotypes A, C, Y, and W-135) is available. Vaccination within the past 3 years is regarded as protective. An approved vaccine for serogroup B meningococci is not available.
- Antibiotic prophylaxis of close contacts, as above.
- Prevent overcrowding in troop shelters, and provide them

Q FEVER

(see also Q Fever, Part Two: Exposure to Biological Warfare Agents)

Communicability:

- Route:
 - ◆ Inhalation of contaminated aerosols or handling of infected material—organisms found in urine, feces, milk, birth products of infected cattle, goats, sheep. Rare ingestion of contaminated milk.
 - ◆ Person-to-person transmission has rarely occurred.

Incubation: 20 days (range 14-39 days).

Diagnosis:

- Symptoms (gradual or abrupt onset):
 - ◆ Fever to 40°C (100%).
 - ◆ Headache, severe (75%).
 - ◆ Fatigue (98%).
 - ◆ Chills (88%).
 - ◆ Myalgia (68%).
 - ◆ Nausea, emesis (25-50%).
 - ◆ Pleuritic chest pain (28%).
 - ◆ Diarrhea (21%).
 - ◆ Retroorbital pain.
 - ◆ Nonproductive cough.
- Signs:
 - ◆ Inspiratory crackles.
 - ◆ Hepatomegaly, splenomegaly, or both (~50%).
 - ◆ Mental status changes (2-5%).
 - ◆ Relative bradycardia.
 - ◆ Hypoxemia, in cases with rapidly progressive pneumonia rash is rare, except in cases of endocarditis with morbilliform rash.
- Laboratory findings:
 - ◆ Hematology: Leukocytosis in 30%.
 - ◆ Chemistry: hepatic transaminases elevated 2-3 times normal.

- ◆ Microbiology: not available under field conditions, requires specialized facility with high level containment.
- ◆ CXR: rounded opacities, pleural effusions (35%), patchy interstitial disease.
- ◆ Serology: fourfold rise in antibody titer between acute and convalescent samples usually measured by complement fixation or IFA.
- ◆ Invasive procedures: LP to rule out pyogenic meningitis if symptoms warrant.
- ◆ Diagnostic confirmation: serology (see above).

Duration: clinical manifestations usually resolve within 2-4 weeks in absence of specific treatment. However, therapy is indicated to prevent development of complications (i.e., chronic infection).

Complications: endocarditis (chronic Q fever), granulomatous hepatitis, and rarely, osteomyelitis, aseptic meningitis/encephalitis, hemolytic anemia, pericarditis.

Treatment:

- Doxycycline 200 mg PO BID for 2 weeks.
- Tetracycline 250 mg PO QID for 10 days.
- Ciprofloxacin 500 mg PO TID for 10 days.

Disposition: local hospitalization, anticipate return to duty after 4-6 weeks. Complicated cases should be evacuated.

Prognosis: most recover uneventfully and without relapse. Fatalities rare in acute disease.

Prevention and Public Health Measures:

- Avoidance of slaughterhouses and birth products of animals.
- Ingestion of only pasteurized dairy products.
- Report case as a reportable event using theater medical surveillance reporting channels.

RABIES

Communicability:

- Route: virus laden saliva of an infected animal introduced by a bite (or, rarely, by non-bite contact).
- Isolation: contact isolation for saliva and respiratory secretions. Transmission to attending personnel has not been documented.
- Prophylaxis: contacts with an open wound or mucous membrane that has been exposed to patient's saliva should receive post-exposure prophylaxis.

Incubation period: 14-60 days (range: 10 days to 1 year); 95% are within 1 year.

Diagnosis:

- Symptoms/Signs: nonspecific syndrome of malaise, fatigue, headache, and fever lasting 2-10 days with pain and paresthesia at the bite site in over 50%. Syndrome merges to an acute encephalomyelitis with apprehension and hyperactivity progressing to spasm of the swallowing muscles and hydrophobia.
- Laboratory: diagnosis confirmed by specific fluorescent antibody staining of brain tissues. No useful antemortem diagnostic findings that would change management, although corneal impression smears or a skin biopsy of the neck above the hair line, stained with immunofluorescent antibody, can confirm the diagnosis.

Duration:

- Treated: death in weeks to months.
- Untreated: death in days to weeks of clinical symptoms.

Complications: usual multiple complications of comatose ICU patient.

Treatment:

- No specific antirabies chemotherapy available; treatment is directed solely at supportive care.
- Pre-exposure prophylaxis to be determined by CINC surgeon.

- Local treatment of wound:
 - ◆ IMMEDIATE and THOROUGH washing of all bite wounds and scratches with SOAP and WATER.
 - ◆ Debridement as indicated, under medical supervision, leaving wound open if possible.
 - ◆ Tetanus prophylaxis and measures to control bacterial infection should be given as indicated.
- Post-exposure prophylaxis (Both HRIG *and* HDCV):
 - ◆ Indicated for anyone who is bitten by a dog, cat, fox or jackal, and should be considered in nonbite exposures (though risk is much lower).
 - ◆ Single dose of Human Rabies Immune Globulin (HRIG), 20 IU/kg or 9 IU/lb body weight; all of the dose should be infiltrated into the bite site if anatomically possible. Any residual dose can be given intramuscularly in the gluteal area. (HRIG should NEVER be given in the same syringe or into the same site as the vaccine.) AND
 - ◆ Give Human Diploid Cell Vaccine (HDCV) in five 1.0 mL doses, IM (deltoid area), on days 0, 3, 7, 14, and 28. *HDCV administration concurrent with malaria chemoprophylaxis with chloroquine may result in decreased HDCV vaccine efficacy.*
 - ◆ Other approved rabies vaccines may be available.

Disposition:

- Exposure: full duty with supervised HRIG and HDCV.
- Clinical illness: EVAC.
- Nonavailability of HRIG and HDCV: EVAC.

Prognosis:

- Treated potential exposure: excellent.
- Treated or untreated clinical illness: uniformly fatal.

Prevention and Public Health Measures:

- Safely capture and sacrifice implicated animal; submit intact head packed in ice (not frozen) to laboratory.
- Command emphasis and education of troops to avoid stray or feral dogs, cats, and wild fox.
- Report case as a reportable event using theater medical surveillance reporting channels.

RELAPSING FEVER

Communicability:

- Route:
 - ◆ Louse-borne relapsing fever (due to Borrelia recurrentis) is acquired when infected lice are crushed or injured and the spirochetes thus released are able to enter abrasions, scratches, or other skin wounds. Not transmitted by louse bites or louse feces. Lice become infective 4-5 days after ingesting an infected blood meal.
 - ◆ Tick-borne relapsing fever (due to Borrelia duttonii and the Borrelia spp.) is transmitted by the bite of infected nymph or adult soft ticks of the genus Ornithodoros. Transmission of infection may occur within minutes of tick attachment.
 - ◆ Transfusion of infected blood transmits disease.
 - ◆ Transplacental infection occurs.
- Isolation:
 - ◆ Delouse patients with insecticide, taking care to avoid crushing lice or abrading skin.
 - ◆ Direct person-to-person transmission does not occur; isolation other than measures to avoid transfer of lice is not needed.
 - ◆ Ticks should be identified and carefully removed.
 - ◆ Standard precautions.
- Prophylaxis: Following tick or louse bites in a high-risk setting, a single dose of tetracycline 500 mg PO may be administered.

Incubation:

- Louse-borne relapsing fever: 4-8 days (range 2-10 days).
- Tick-borne relapsing fever: 2-14 days.

Diagnosis:

- Symptoms: The characteristic feature of relapsing fever is abrupt severe fever lasting several days, terminating in a crisis by rapid defervescence, severe sweating and weakness ; then, after an interval of days (rarely weeks), recurring one or more times.

- ◆ Louse-borne relapsing fever:
 - Fever (98%): $> 39^{\circ}\text{C}$ in over 70% of cases. Initial episode of fever usually lasts 5-7 days, then ends abruptly. Death may occur suddenly and unexpectedly during febrile crisis. Approximately 65% of patients will have a relapse after 5-9 days, similar to but less severe than the first. A third relapse is seen in about 25% of patients; more than three relapses are uncommon.
 - Body pain (80-90%): severe and generalized, especially back, chest, abdominal, legs (especially calves), joints.
 - Chills/rigors (90%).
 - Severe headache (87%).
 - Dizziness (74%).
 - Nausea (65%).
 - Vomiting (30-35%).
 - Cough (50%).
 - Prostration dysphagia (45-50%).
 - Severe dyspnea (15-20%).
 - Anorexia.
- ◆ Tick-borne relapsing fever:

Initial episode of fever averages 3.5 days, but may persist up to 17 days. Relapses are more common and more frequent than in louse-borne disease, with at least one relapse in almost all, 8 relapses about average, and up to 17 relapses being reported. Relapses average about 2 days each. Neurologic symptoms more common than in louse-borne disease. Other symptoms similar to, but less severe than, those in louse-borne disease.

■ Signs:

- ◆ Louse-borne relapsing fever:
 - Petechiae, ecchymoses (up to 54%), more pronounced on trunk than extremities.
 - Jaundice.
 - Fine erythematous rash early in illness.
 - Liver tenderness (60%).
 - Spleen tenderness (55%).
 - Iritis.
 - Meningismus.

- Delirium.
- No local skin lesion or eschar at site of entry of infection.
- ◆ Tick-borne relapsing fever: Signs are similar to those of louse-borne disease, but less severe. Neurologic signs common, including encephalitis, subarachnoid hemorrhage, aseptic meningitis, aphasia, hemiplegia, sciatica, iritis, iridocyclitis, optic atrophy, and palsies of cranial nerves III, IV, V, VI, VIII, and especially VII (Bell's Palsy). No local skin lesion or eschar at site of entry of infection.
- Laboratory findings:
 - ◆ Hematologic:
 - Wright or Giemsa-stained blood smears should be carefully examined for extracellular spirochetes. In louse-borne disease, spirochetes should be visible on smears, but they may too low in concentration to detect by this method in tick-borne disease. Thick and thin malaria smears are satisfactory for examination. Spirochetes are not detectable during intervals between fevers.
 - Anemia.
 - Thrombocytopenia, often $< 50,000$.
 - WBC: usually WNL, but may rise to 15,000-30,000.
 - ◆ Chemistry: elevated transaminases; elevated bilirubin, both conjugated and unconjugated.
 - ◆ Urinalysis: albuminuria, microhematuria, pyuria.
 - ◆ Microbiologic:
 - Dilute one drop of blood with one drop of normal saline, cover with cover slip, and examine by darkfield or phase contrast microscopy. Typical corkscrew rotation of spirochetes can be seen.
 - Specialized culture techniques or animal inoculation are required for isolation.
 - ◆ Serologic: not clinically available.
 - ◆ Coagulation: PT and PTT may be prolonged.
- Radiology: nonspecific.
- Invasive Procedures: lumbar punctures show elevated opening pressure; pleocytosis, and elevated CSF protein are typical. Both polys and mononuclear cells may be seen.

- Diagnostic confirmation:
 - ◆ Demonstration of spirochetes in blood smear.

Duration:

- Treated: immediate cure, with residual exhaustion lasting several days.
- Untreated: see above for discussion of duration of febrile episodes, afebrile intervals, and number of relapses.

Complications:

- Jarisch-Herxheimer reactions (see leptospirosis as well):
 - ◆ Characteristics: within 1-3 hours of administration of antibiotics, a severe rigor and transient blood pressure elevation occur. These are followed by a sharp rise in fever and marked hypotension. Severe headaches and myalgias occur. Vomiting and urinary incontinence may develop. Abrupt hypoglycemia, leukopenia and thrombocytopenia occur. The severity of Jarisch-Herxheimer reactions is greater if antibiotics are given IV.
 - ◆ Treatment of Jarisch-Herxheimer reactions:
 - Meptazinol (an opioid agonist-antagonist) 100 mg IV given with antibiotic dose, repeated 30 minutes after antibiotic dose, repeated again at onset of febrile reaction, and again if systolic BP drops below 70 mm Hg; has been shown to ameliorate the Jarisch-Herxheimer reaction in relapsing fever. This drug is neither approved for use nor available in the U.S.
 - Pretreatment with steroids or antipyretic agents is of questionable value.
 - Naloxone is ineffective. Hemodynamic support with isotonic IV fluid administration (e.g., normal saline) is essential. Vasopressors may be necessary, but should be reserved for hypotension refractory to adequate volume replacement.
- Myocarditis with prolonged QT interval, T wave changes, and congestive heart failure.
- Coincident typhus may occur simultaneously with louse-borne relapsing fever.

Treatment:

Lethal Jarisch-Herxheimer reactions may rapidly follow administrations of appropriate antibiotics and must be anticipated, particularly in louse-borne disease. Frequency and severity of these reactions is higher when IV antibiotics are given. See above.

Antibiotics (in order of preference):

■ Louse-borne:

◆ Oral regimens:

- tetracycline 500 mg PO, single dose (*avoid in pregnant women or children < 8 years old*).
- doxycycline 200 mg PO, single dose.
- erythromycin 500 mg PO, single dose.

◆ Parenteral regimens:

- tetracycline 250 mg IV, single dose.
- erythromycin, 250 mg IV, single dose.

■ Tick-borne:

◆ Oral regimens:

- tetracycline 500 mg PO Q6H for 6 days (*avoid in pregnant women or children < 8 years old*).
- doxycycline 100 mg PO BID for 10 days.
- erythromycin 500 mg Q6H for 10 days.

◆ Parenteral regimens:

- penicillin G 3 million units IV Q4H for 10 days.
- ceftriaxone 2 gm IV Q6H for 10 days.

Disposition: Local hospitalization, with subsequent return to duty. Consider restricted duty with no hard physical activity for 30 days to allow associated splenomegaly to resolve. Evacuation may be needed for cases with complications.

Prognosis:

■ Louse-borne:

- ◆ Treated: complete recovery in > 95% of cases.
- ◆ Untreated: mortality variable; usually 2-10%, but may reach 70% in epidemic conditions.

■ Tick-borne:

- ◆ Treated: complete recovery in $\geq$ 95% of cases.
- ◆ Untreated: mortality low, less than 10%.

Prevention and Public Health Measures:

- Vector control:
 - ◆ Delousing, personal hygiene. Use permethrin impregnated clothing and bedding.
 - ◆ Tick control/avoidance:
 - Avoidance of tick-infested locales: old housing, old campsites.
 - Insect repellents and other personal protection measures including permethrin-impregnated clothing and bedding; avoid sleeping on ground.
 - Insecticide application to tick-infested sites.
- Reservoir eradication:
 - ◆ Louse-borne disease: human reservoir; identify and treat.
 - ◆ Tick-borne disease: human and animal reservoirs; treat and control rodents.
- Report case as reportable event using theater medical surveillance reporting channels.

SAND FLY FEVER

Communicability:

- Route:
 - ◆ Sand fly (*Phlebotomus papatasi*) bites.
 - ◆ No direct human-to-human transmission.
- Isolation: not required. Protection of patients from further sand fly bites will interrupt transmission. Human viremia is present from about 24 hours prior to onset of fever until about 24 hours after fever resolves. Very fine mesh for screens or bed net (10-12 mesh/cm) required. Permethrin treatment of larger mesh mosquito nets will also make effective barriers for sand flies.
- Prophylaxis: none required.

Incubation: 3-6 days.

Diagnosis: Widespread outbreaks have occurred in military campaigns when nonimmune soldiers entered endemic areas.

- Symptoms:
 - ◆ Fever to 40°C.
 - ◆ Headache.
 - ◆ Myalgia.
 - ◆ Supraorbital pain (intense) or retrobulbar pain with eye movement.
 - ◆ Limb stiffness.
 - ◆ Malaise.
 - ◆ Gastrointestinal symptoms (nausea, vomiting).
 - ◆ Facial congestion.
 - ◆ Neck stiffness.
- Signs:
 - ◆ Fever.
 - ◆ Conjunctival injection.
 - ◆ Papilledema (occasional).
 - ◆ **No adenopathy.**
 - ◆ Erythematous flush but **no rash.**
- Laboratory findings:
 - ◆ Hematologic: leukopenia on day 4 - 5 of fever.
 - ◆ Chemistry, urinalysis, microbiology, and coagulation: not applicable.

- ◆ Serology: paired sera for hemagglutination-inhibition (HI) and neutralizing antibodies (retrospective only).
- Invasive procedures: lumbar puncture shows increased opening pressure and CSF pleocytosis.
- X-ray: not applicable.
- Diagnostic confirmation: serologic.

Duration: 2-4 days, convalescence may be a week or longer.

Complications: none; though patients may have lethargy, depression, and easy fatigability for weeks after recovery.

Treatment:

- No specific treatment available yet.
- Provide supportive care.

Disposition: limited duty or local hospitalization until fever resolves, then full duty; occasionally, convalescence may be prolonged and some patients may require EVAC.

Prognosis: full recovery. Single infection confers lasting immunity against same serotype.

Prevention and Public Health Measures:

- Insecticide spraying of troop quarters, emplacements and entrenchments.
- Troop education.
- Command emphasis on use of personal protective measures (use of insect repellents, application of permethrin insecticides to clothes if not previously treated). Mosquito netting such as bednets are not effective controls against sand flies *unless mesh hole density is no larger than 10-12 mesh holes/cm*.
- Report outbreaks to higher echelon medical authorities.

SCHISTOSOMIASIS, ACUTE (KATAYAMA FEVER)

Communicability:

- Route: human-to-human spread not seen. Disease acquired by contact with infected fresh water (swimming, wading, washing, etc.). Not seen in Central Asia.
- Isolation: not required.
- Prophylaxis: not required.

Incubation: schistosomiasis dermatitis (swimmer's itch) occurs within 24 hours of penetration of skin by the infective, forked-tailed cercariae. Clinical syndrome of acute schistosomiasis occurs after 2 weeks to 3 months.

Diagnosis:

- Characteristically associated with S. japonicum and sometimes with S. mansoni. S. haematobium is rarely associated with an acute syndrome.
- While schistosomiasis is not a threat in most of Central Asia, it does occur in the Arabian Peninsula and parts of Southwest Asia. Outbreaks of swimmer's itch that have occurred in Central Asia are attributable to animal-dependent schistosomes for which humans are not primary hosts.
- Symptoms:
 - ◆ Fever (all).
 - ◆ Chills.
 - ◆ Sweating.
 - ◆ Headache.
 - ◆ Cough (most).
 - ◆ Diarrhea (50%).
 - ◆ Weight loss.
- Signs:
 - ◆ Lymphadenopathy.
 - ◆ Hepatomegaly (50%).
 - ◆ Splenomegaly (10%).
- Laboratory findings:
 - ◆ Hematology: eosinophilia very common.
 - ◆ Microbiology: stool or urine exam may but usually does not show schistosoma eggs in patients with acute schistosomiasis.

- ◆ Serology: not useful in acute cases.
- Radiology: not useful acutely.
- **Note:** exposure history is essential to consider the diagnosis. Absence of eosinophilia (>500 cell/mm³) does not usually support the diagnosis.

Duration:

- Treated: aborts chronic sequelae but may not limit acute disease.
- Untreated: 2-4 weeks for resolution of acute symptoms.

Complications:

- Rare reports of death in nonimmune with a heavy primary infection.
- If not recognized or treated, could present later as chronic manifestations of schistosomiasis; so all infections must be treated, whether asymptomatic or not.

Treatment:

- Praziquantel (Biltricide®): single oral dose of 40 mg/kg following a meal; may also be given in two divided doses on the same day.
- Praziquantel may cause malaise, headache or dizziness; side effects fewer if given as two divided doses.
- Treatment of acute schistosomiasis with schistosomicidal drugs may result in acute, severe, possibly life-threatening clinical deterioration. This transient complication may be prevented by the co-administration of prednisone 40 mg PO QD for 5 days, or by an equivalent course of another corticosteroid.
- Swimmer's itch can be treated with a topical steroid and an antipruritic.

Disposition: limited duty or hospitalization depending on illness severity; evacuation may be indicated with severe disease.

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Prognosis: excellent if diagnosed and treated early.

Prevention and Public Health Measures:

- Command emphasis and education of soldiers to avoid exposure (swimming or wading with bare skin contacting fresh water — especially lakes, marshes and slow-moving waters). Vigorous towel-drying or application of rubbing alcohol can prevent penetration of parasites after water contact.
- Report case as a reportable event using theater medical surveillance reporting channels.

STREPTOCOCCAL INFECTIONS

Communicability:

- Route:
 - ◆ Person to person, via respiratory or salivary droplets; crowded living arrangements enhance transmission.
 - ◆ Food and waterborne outbreaks have occurred.
- Isolation: not warranted.
- Prophylaxis: generally not warranted. In an outbreak of streptococcal disease associated with rheumatic fever or glomerulonephritis, culture and treatment of culture-positive household contacts (barracks or tent mates) can be considered. Alternatively, prophylactic benzathine penicillin can be employed to interrupt an outbreak.

Incubation: 2-4 days for pharyngitis.

Diagnosis: clinical streptococcal disease may present as pharyngitis, scarlet fever, erysipelas (superficial cellulitis), or pyoderma (impetigo). A streptococcal toxic shock-like syndrome occurs, but is uncommon.

- **Pharyngitis**:
 - ◆ Symptoms: sore throat, headache, fever, malaise.
 - ◆ Signs: pharyngeal redness, edema, and lymphoid hyperplasia; enlarged reddened tonsils with exudate (in 50%), tender submandibular lymphadenopathy; fever $\geq 101^{\circ}\text{F}$ (38.3°C); palatine petechiae.
 - ◆ Laboratory: mild leukocytosis, positive pharyngeal cultures. Antigen detection tests are very specific, but somewhat insensitive. They may be helpful if laboratory support is minimal.
- **Scarlet fever**:
 - ◆ Usually occurs with pharyngitis, but may be seen with streptococcal skin infections.
 - ◆ Symptoms: those of primary infected site, plus fever, rash, and occasionally marked systemic toxicity or a toxic shock-like syndrome.

- ◆ Signs: diffuse rash consisting of 1-2 mm, blanching, red papules, darker at skin creases. Begins on neck, spreads to trunk then extremities, normal sparing face, palms, and soles. "Sandpaper" texture of skin is due to sweat gland occlusion. Linear petechial eruption in antecubital fossae and other skin folds (Pastia's lines). Palatal petechiae. Yellowish-white coating of tongue followed by beefy red appearance of tongue.
- Cellulitis:
 - ◆ Symptoms: fever and malaise.
 - ◆ Signs: intense erythema, pain, pitting edema, warmth in a well-defined area. Often lymphangitis or lymphadenitis. Progresses rapidly. May evolve to abscesses, gangrene, or septicemia.
- Erysipelas:
 - ◆ Symptoms: chills, fever, systemic toxicity.
 - ◆ Signs: red, edematous, sharply demarcated, advancing skin lesion.
- Impetigo:
 - ◆ Signs: pustules that rupture easily and enlarge into shallow skin ulcers or erosions, typically with honey-colored crusts. Usually occur on exposed skin areas, such as the mid-face or beard area or at sites of insect bites or other preexisting rashes. Highly contagious.
- Streptococcal toxic shock:
 - ◆ Signs: sepsis syndrome, shock, acute renal failure, ARDS.

Duration:

- Pharyngitis: treated 1-4 days; untreated 3-5 days.
- Scarlet fever: rash persists 4-5 days; subsequent desquamation persists 2-4 weeks.
- Erysipelas/cellulitis:
 - ◆ Treated: improvement in 24-48 hours.
 - ◆ Untreated: may proceed to fatality.

- Impetigo:
 - ◆ Treated: improvement within 2-3 days.
 - ◆ Untreated: may persist several weeks.
- Streptococcal toxic shock: 30% mortality rate even with intensive care and antibiotics.

Complications:

- Immunologic:
 - ◆ Rheumatic fever (from pharyngeal disease).
 - ◆ Acute glomerulonephritis (most commonly from skin infections).
- Infection: septicemia, otitis media, sinusitis, mastoiditis, meningitis, brain abscess, toxic shock syndrome, necrotizing fascitis (all uncommon).

Treatment:

- Pharyngitis:
 - ◆ Benzathine Penicillin G 1.2 million units IM one dose; preferred; OR
 - ◆ Penicillin V 250 mg PO TID for 10 days (avoid due to compliance problem); OR
 - ◆ Erythromycin 250 mg PO QID for 10 days (for penicillin allergic patients).
- Scarlet fever:
 - ◆ Treat primary source of infection (e.g., pharyngitis, skin) as appropriate.
 - ◆ Supportive care.
- Erysipelas/cellulitis:
 - ◆ Penicillinase-resistant penicillin (to cross cover possible staphylococcal etiology) IV or PO depending on severity of infection. May switch to oral agent 1-2 days after initiating therapy if response is good. Minimum 10-day course; OR
 - ◆ Erythromycin 0.5 to 1 gm IV Q6H; when response occurs, transition to PO dosing with 500 mg PO Q6H to complete full 10-day course of Erythromycin; OR
 - ◆ Vancomycin 1 gm IV Q12H for a 10-day antibiotic course.

- Impetigo - must cover for both Staphylococcal aureus and Streptococcal pyogenes organisms:
 - ◆ Dicloxacillin.
 - ◆ Cephalexin (Keflex®).
 - ◆ Trimethoprim-Sulfamethoxazole (Septra®).
 - ◆ Doxycycline.
 - ◆ Topical Mupirocin (Bactroban®).
- Streptococcal toxic shock, or necrotizing fascitis:
 - ◆ Penicillin G 3 million U IV Q4H for 10-14 days PLUS Clindamycin 900 mg IV Q8H.
 - ◆ Surgical debridement and compartment pressure monitoring required for necrotizing fascitis.
 - ◆ Intensive supportive care.
- For penicillinase producing streptococcal organisms that are resistant to penicillin, alternate therapies include macrolides such as clarithromycin or the second generation quinolones such as ofloxacin.

Disposition:

- Local hospitalization required for scarlet fever, erysipelas, or severe pharyngitis.
- Mild pharyngitis or impetigo may be returned to duty.
- Evacuation required for rheumatic fever, glomerulonephritis, toxic shock, necrotizing fascitis or other advanced infectious complications.

Prognosis:

- Treated: excellent prognosis.
- Untreated: complication will be associated with serious sequelae in some cases.
 - ◆ Scarlet fever and erysipelas: may be fatal if not properly treated.

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- ◆ Toxic shock-like syndrome: 30% mortality and may be associated with lasting sequelae in survivors.
- ◆ Necrotizing fascitis: any therapy delay associated with markedly increased mortality.

Prevention and Public Health Measures:

- No specific measures warranted under most circumstances. Investigation of outbreaks is mandatory; intervention with prophylactic antibiotics may be required. Occurrence of any cases of streptococcal toxic shock-like syndrome indicates presence of toxin-producing strain with potential for additional cases.
- Food handler precautions and good hygiene by all personnel will minimize incidence of streptococcal skin infections.

TUBERCULOSIS (TB)

Communicability:

- Route:
 - ◆ Inhalation of airborne droplet nuclei from coughing patients with active pulmonary or laryngeal TB.
 - ◆ Ingestion of infected unpasteurized dairy products.
- Isolation: airborne precautions and early evacuation for suspected cases.

Incubation:

- For development of primary lesion: 4-12 weeks.
- For progressive, reactivation or extrapulmonary disease: Progressive disease can occur soon after exposure, although this is unlikely unless the exposed person is immunosuppressed. Lifetime risk of reactivation is about 10%. Half of this (5%) is the first 2 years after initial exposure (infection).

Diagnosis:

- Symptoms:
 - ◆ Initial infection is usually asymptomatic.
 - ◆ Fever (may be intermittent) night sweats, anorexia, weight loss, fatigue, cough (productive or nonproductive for more than 3 weeks), hemoptysis, chest pain (pleuritic), dyspnea.
 - ◆ Symptoms produced by extrapulmonary tuberculosis depend on the organ system involved. Extrapulmonary sites, listed here in order of decreasing frequency, include lymphatics, pleura, genitourinary tract, bone/joint, meninges, peritoneum, liver, pericardium, middle ear, and brain.
- Signs:
 - ◆ Signs may be absent, especially in early disease. In general, they are nonspecific and less significant than would be expected from extent of disease.
 - ◆ Rales, especially post-tussive: dullness to percussion; and diminished breath sounds.
 - ◆ Other signs depend on the site(s) of extrapulmonary involvement.

- Laboratory findings:
 - ◆ Hematologic:
 - May be normal.
 - Anemia, mild leukocytosis or monocytosis ($\geq 10\%$).
 - ESR may be elevated.
 - ◆ Chemistry:
 - Usually normal.
 - Hypercalcemia.
 - Hyponatremia.
 - Other abnormalities may represent specific effects of extrapulmonary involvement.
 - ◆ Urinalysis:
 - Usually normal.
 - In presence of genitourinary tuberculosis may see sterile pyuria, proteinuria and/or hematuria.
 - ◆ Microbiologic:
 - Observation of acid-fast bacteria in smears (sputum, gastric aspirate) may be used for presumptive diagnosis (although other acid-fast organisms also rarely cause pulmonary diseases). A single organism on a slide may be significant, though usually 3 to 5 organisms per slide are considered a true positive.
 - Fluorochrome staining is most efficient.
 - Alternatives include Ziehl-Neelsen, Kinyoun, or blue-light fluorescent stains.
 - Radiometric culture system (i.e., BACTEC®) can reveal presence of organisms in 2-6 days.
 - Standard mycobacterial cultures may take up to 12 weeks to recover the organism.
 - In presence of urinary sediment abnormalities, obtain AFB smears and cultures on centrifuged urine.
 - ◆ Serology: not in general use.
 - ◆ Coagulation: generally normal.

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- X-ray: findings depend on the character and extent of disease.
 - ◆ Early or primary TB may present in any lobe (more typically lower) as pneumonic infiltrate, atelectasis or mass, with or without ipsilateral hilar adenopathy.
 - ◆ Later, chronic, or reactivation TB typically shows patchy or nodular infiltrates in the apices or superior segments of lower lobes; cavitation may or may not be present.
 - ◆ Pleural effusions may be seen.
- Invasive procedures:
 - ◆ Gastric aspirate for smear and culture may be useful if no sputum can be produced.
 - ◆ Bronchoscopy, with washings for cultures, may be diagnostic when TB is a consideration but organisms cannot be recovered by less invasive means.
 - ◆ The choice of other specific invasive procedures, including thoracentesis, lumbar puncture, or biopsies, is guided by clinical evidence of extrapulmonary TB.
- Skin testing:
 - ◆ In previous nonreactors TB skin test (PPD) may become positive in 4-10 weeks.
 - ◆ PPD may be negative in early or primary disease, in overwhelming disease, or in patients with immunosuppression from other disease.
 - ◆ Up to 25% of patients with pulmonary TB may have negative skin tests; 5% of patients may have a selective anergy (negative PPD and positive anergy panel).
 - ◆ In persons exposed to others with active pulmonary TB, and those with abnormal chest x-rays (but not active disease) or immunosuppression, >5 mm induration is considered positive.
 - ◆ In an area of high prevalence, PPD skin tests of >10 mm induration are considered positive.
 - ◆ Prior BCG vaccination should not be considered when interpreting PPD results.
- Diagnostic confirmation: successful culture of mycobacteria from clinical specimens.

Duration:

- Treated: variable, depending on extent of disease. Treatment regimens are typically 6-9 months. Clinical response should be seen after 2-4 weeks of therapy.
- Untreated: indefinite; 50% die, 25% develop chronic TB which can remain active for years, and 25% spontaneously resolve.

Complications:

- Pulmonary: hemoptysis, massive hemorrhage, and major parenchymal lung damage with permanent impairment of respiratory function.
- Extrapulmonary: ranges from minor damage to destruction of the involved organ.
- Recurrence, possibly with resistant organisms, may occur in inadequately treated patients. Recurrence in adequately treated patients is very uncommon, but may occur.

Treatment:

- Treatment should not be started in theater unless patient shows evidence of disseminated disease, meningitis, or is otherwise acutely unstable. Culture recovery of the organism to determine susceptibility is important for correct clinical management and will not be available in theater.
- If initiated, therapy should include standard four drug regimen of rifampin, isoniazid (INH), pyrazinamide, and ethambutol. Initiation of nonstandard regimens in the field is discouraged.

Disposition: EVAC.**Prognosis:** excellent in properly treated cases.**Prevention and Public Health Measures:**

- Isolation and treatment of infectious patients.
- Prophylaxis of contacts per policy of CINC surgeon.
- Persons with suspected or confirmed infection should wear a surgical mask when contact with other personnel is likely.

- Personnel in contact with persons with suspected or confirmed infection should wear N-95 masks or other devices to prevent inhalation of droplet nuclei. When not available, consider conducting evaluations outdoors where UV light will kill the organism.
- Latent TB infection (LTBI): Those found to have LTBI (positive PPD without signs or symptoms of active disease) can be managed in theater or upon redeployment. Evaluation should include chest xray to rule out active disease. Therapy is usually 9 months of INH (to be completed within 1 year).
- Avoid use of local (unpasteurized) dairy products.
- Report case as a reportable event using theater medical surveillance reporting channels.

TYPHOID AND PARATYPHOID ENTERIC FEVER

Communicability:

- Route: oral ingestion of organisms, typically in contaminated food or water.
 - ◆ Patients excrete organisms in stool, urine, pus and/or emesis. Asymptomatic carriage and excretion of organisms in stool is common.
 - ◆ Viable organisms can contaminate food and water via spread by hands, flies, fomites, or direct contamination.
- Isolation:
 - ◆ Standard and contact precautions.
 - ◆ Disinfection of contaminated articles.
 - ◆ Since excretion of organisms typically persists for several weeks after resolution of illness, and persists more than 1 year in up to 3% of patients, convalescing patients should be evacuated rather than returned to field setting.
- Prophylaxis:
 - ◆ For household (barracks or tent mate) contact, administer vaccine if this has not been received within 3 years.
 - ◆ Household contact should not be used as food handlers unless both stool and urine are each negative for salmonella on two occasions at least 24 hours apart.

Incubation:

- Average: 1 week.
- Range: 3 days to 8 weeks.
- Larger inoculum is associated with briefer incubations.

Diagnosis:

- Symptoms: insidious onset (note that brucellosis may present with similar symptoms; see Brucellosis section for considerations):
 - ◆ Fever (75-100%).
 - ◆ Headache* (59-90%).
 - ◆ Anorexia (39-91%).
 - ◆ Cough (28-86%).

- ◆ Myalgia (12-91%).
- ◆ Constipation (10-79%).
- ◆ Weakness (10-87%).
- ◆ Diarrhea (37-57%) *may NOT be present*.
- ◆ Vomiting (24-54%).
- ◆ Nausea (23-54%).
- ◆ Sore throat (6-84%).
- ◆ Chills (16-37%).
- ◆ Abdominal pain (19-39%).
- ◆ Sweats (33%).

**Headache associated with enteric fever is frequently intense, frontal, and not bitemporal.*

■ Signs:

- ◆ Fever: remittent, 104°F (40°C); (75-100%); less likely in early phase.
- ◆ Pulse slow relative to fever.
- ◆ Rose spots: 2-8 mm blanching, erythematous, maculopapular lesions; occur in crops of about 10 located on upper abdomen; persist several hours to several days; appear 2-4 weeks into illness, (13-46%).
- ◆ Hepatomegaly: (15-50%).
- ◆ Splenomegaly: often tender; (40-64%).
- ◆ Neurological/mental status changes: lethargy, stupor, coma seizures, delirium, and meningismus; (10%).

■ Laboratory findings:

- ◆ Hematologic:
 - HGB/HCT: anemia common, worsens progressively over first 3 weeks.
 - WBC: normal in 75% (range 1,200-20,000).
 - Platelets: usually normal, occasionally low.
 - ESR: typically elevated.
- ◆ Chemistry:
 - SGOT, LDH: mild/moderate elevation in about 33%.
 - Alkaline phosphatase: mild elevation common.
 - Bilirubin: mild elevation (twofold) common; sufficient to cause jaundice, uncommon.
 - CPK: occasionally elevated.
- ◆ Urinalysis: nonspecific.

- ◆ Microbiologic: causative organisms include Salmonella typhi (typhoid), other salmonella species (paratyphoid) and other bacteria including Yersinia enterocolitica, Yersinia pseudotuberculosis and Campylobacter fetus.
 - Blood cultures: first week 80% positive; by third week 20-30% positive. Obtain 2 to 3 sets for optimal yield.
 - Bone marrow aspirate cultures: 90-95% positive.
 - Stool cultures: occasionally positive during incubation; 33-67% positive during weeks 2-4 of illness.
 - Urine culture: intermittently positive after second week of illness in 25%. Multiple specimens should be sent.
 - Skin snips of rose spots may be positive when cultures of other sites fail to isolate organism.
- ◆ Serologic: limited value; insensitive and nonspecific.
- ◆ Coagulation: usually normal. Occasionally coagulopathy, with prolonged PT and PTT may be seen.
- X-Ray: chest x-ray normal (infiltrates in <10%).
- Invasive procedures:
 - ◆ Bone marrow aspiration, for culture, as above.
 - ◆ Skin snip or biopsy of rose spot, for culture, as above.
- Diagnostic confirmation: isolation of organism from blood, marrow, or skin. Isolation from stool of a typical case is presumptive evidence, but is not definitive.

Duration:

- Treated: 3-10 days, until fever resolves.
- Untreated: 4-week acute illness, if not complicated.

Complications:

- Intestinal perforation:
 - ◆ Incidence: 1-10%, typically during second or third week of illness.
 - ◆ Mortality: 25%.

- ◆ Signs:
 - Classic peritoneal signs often absent.
 - Abdominal x-ray shows air below diaphragm.
 - Absent bowel sounds and vomiting, suggesting ileus, may be most prominent clinical features.
- ◆ Perforations may be single or multiple.
- ◆ Ileum is most common location.
- ◆ Treatment is surgical.
- GI hemorrhage:
 - ◆ Incidence: 1-20% depending on initiation of antibiotics.
 - ◆ Mortality: low if recognized and treated.
 - ◆ Typically occurs during second or third week of illness.
 - ◆ Treatment is supportive, including transfusion. Surgical intervention should be reserved for massive or persistent bleeding.
- Local abscess/infection:
 - ◆ Incidence: $\leq 1\%$.
 - ◆ May occur in any tissue, notably bone, soft tissue, meninges, heart, pericardium, lungs, liver, spleen, kidneys, thyroid, or breast.
- Other complications:
 - ◆ Hemolytic anemia (2%).
 - ◆ Typhoid pneumonia (8-10%).
 - ◆ Peripheral neuropathy.
 - ◆ Relapse (5-20%).

Treatment:

- Preferred regimens:
 - ◆ Ciprofloxacin 500 mg PO BID for 10-14 days (if disease detected early); OR
 - ◆ Ceftriaxone (Rocephin®) 1-2 g IV QD for 7 days.
- Supportive fluid and nutritional therapy is essential.
- Avoid heparin and antipyretics.

- In critically ill patients (i.e., shock, delirium, stupor, or coma), IV dexamethasone improves survival from 45-90%: dexamethasone, loading dose 3 mg/kg IV, then 1 mg/kg IV Q8H for 48 hours.

Disposition: evacuation, once stabilized.

Prognosis:

- Treated: ≤ 1 mortality.
- Untreated: 10% mortality.

Prevention and Public Health Measures:

- Vaccinate all military personnel.
- Command emphasis:
 - ◆ Strict sanitation.
 - ◆ Hand washing/personal hygiene.
 - ◆ Strict water purification/food preparation.
 - ◆ Fly control:
 - Insecticide spraying.
 - Screening.
 - Proper garbage disposal.
- Epidemiologic investigation of each case is required.
- Report case as a reportable event using theater medical surveillance reporting channels.

TYPHUS, ENDEMIC (MURINE, FLEA-BORNE)

Communicability:

- Route:
 - ◆ Bite of infected rat flea (Xenopsylla cheopis).
 - ◆ No evidence of person-to-person transmission.
- Isolation: not required.
- Prophylaxis: not required.

Incubation: 12 days (range 4-15 days).

Diagnosis: overall similar to epidemic typhus but milder, briefer.

- Symptoms: onset variable, but more commonly sudden.
 - ◆ Fever (90-100%).
 - ◆ Chills.
 - ◆ Headache (severe) (85% or more).
 - ◆ Myalgia (85%).
 - ◆ Nonproductive cough (50-60%).
 - ◆ Nausea.
 - ◆ Vomiting.
 - ◆ Marked weakness/prostration.
 - ◆ Sore throat.
 - ◆ Chest pain.
- Signs:
 - ◆ Fever (100%) up to 105°F (40°C) for 12-16 days duration.
 - ◆ Rash (60-80%).
 - Initial: pink macules on upper thorax and abdomen, rapidly spreads to the rest of the body, but spares face, palms, and soles; appears on days 3-5 of illness.
 - Later: remains central, macules become hemorrhagic and gangrene may ensue, duration 4-8 days.
 - ◆ Conjunctival injection (50%).
 - ◆ Splenomegaly (30%).
 - ◆ Mental status changes (20%).
 - ◆ Photophobia (10-20%).
 - ◆ No eschar present.

- Laboratory findings:
 - ◆ Hematology: WBC usually normal.
 - ◆ Chemistry: nonspecific.
 - ◆ Microbiology: not available, except in special facilities with containment capability.
 - ◆ Urinalysis: proteinuria (15-20%).
 - ◆ Serology: available but cross-reacts with other rickettsial organisms.
 - ◆ Coagulation: nonspecific.
- Invasive procedures: not indicated.
- X-ray: findings nonspecific.
- Diagnostic confirmation: clinical diagnosis generally is sufficient for patient care. If specific confirmation is required for epidemiologic purposes, either culture or specialized application of indirect immunofluorescent antibody (after cross-absorption of patient's serum with specially prepared antigen from other Rickettsial species) may be done.

Duration:

- Treated: 2-3 days, until defervescence.
- Untreated: up to 16 days until defervescence.

Complications: very uncommon.

Treatment:

- Standard: Doxycycline 100 mg PO BID until 3 days after defervescence.
- Alternatives: Tetracycline 250 mg PO QID until 3 days after defervescence.
- Relapse: rare in murine typhus; retreat with original regimen.

Disposition: local hospitalization, anticipate return to duty in 1-2 weeks.

Prognosis: excellent; even untreated cases should recover without sequelae.

Prevention and Public Health Measures:

- Insecticide application to rat runs and rat-infested areas to kill fleas.
- After effective insecticide applications, rat elimination measures including poisoning and trapping are indicated.
- Rat-proofing human quarters.

TYPHUS, EPIDEMIC (LOUSE-BORNE)

Communicability:

- Route:
 - ◆ Body louse (*Pediculus humanis*) infestation; inoculation with louse feces through skin abrasions or excoriations.
 - ◆ No evidence of person-to-person transmission.
- Isolation: contact isolation required until after delousing (by insecticide) of patients clothing, bedding, quarters, and household contacts. Options: lindane, malathion, carbaryl, DDT.
- Prophylaxis: Doxycycline, single dose, 200 mg.

Incubation: 12 days (range 5-23 days).

Diagnosis:

- Symptoms (abrupt onset):
 - ◆ Sustained fever $\geq 40^{\circ}\text{C}$.
 - ◆ Severe headache.
 - ◆ Prostration.
 - ◆ Back pain.
 - ◆ Limb pain.
 - ◆ Nonproductive cough.
 - ◆ Photophobia.
 - ◆ Anorexia.
 - ◆ Constipation.
 - ◆ Nausea (uncommon).
 - ◆ Vomiting (uncommon).
 - ◆ Diarrhea (uncommon).
- Signs:
 - ◆ Rash (90%); onset on 5th or 6th day of illness. Initially in axillary folds, on abdomen and chest. Centrifugal spread later. Initially roseolar, macular; becomes petechial. Rarely involves palms, soles or face. No eschar is seen. Rash is similar to endemic typhus but less severe. Also:
 - ◆ Profound lethargy/stupor.
 - ◆ Delirium.
 - ◆ Facial congestion.
 - ◆ Conjunctival injection.
 - ◆ Splenomegaly.
 - ◆ Hypotension.
 - ◆ Tachycardia.

- ◆ Jaundice (uncommon).
- ◆ Oliguria.
- ◆ Meningismus.
- ◆ Cranial nerve palsies, including deafness/tinnitus.
- Laboratory findings:
 - ◆ Hematologic: leukopenia early; no eosinophilia; anemia and thrombocytopenia seen as disease advances.
 - ◆ Chemistry: azotemia, hypoalbuminemia, hyponatremia.
 - ◆ Urinalysis: proteinuria.
 - ◆ Microbiology: culture may be possible in large centers but not under field conditions or in small hospitals.
 - ◆ Serology available.
 - ◆ Coagulation: prolonged prothrombin time (PPT).
- Invasive procedures: CSF may show pleocytosis.
- X-ray: CXR may show pulmonary infiltrate.

Complications: sepsis, parotitis, and pneumonia; rarely myocarditis, CHF, venous thromboses.

Treatment:

- Doxycycline, 200 mg PO, single dose.
- Tetracycline, 250 mg PO QID, until 3 days post deferescence (avoid if renal failure is present).
- Relapse: repeating initial treatment is effective.

Disposition:

- Initial: hospitalization.
- Post treatment:
 - ◆ prompt responders: return to duty.
 - ◆ complicated cases or those with inadequate response to treatment: evacuate.

Prognosis:

- Mortality:
 - ◆ Treated: very little, if any, mortality.
 - ◆ Untreated: 10-40% depending on clinical situation.
- Prompt recovery with therapy: usually better in 24-48 hours.
- Untreated: rapid fever defervescence after about 2 weeks, mentation rapidly returns to normal; 2-3 months may be required for return of strength.
- Relapses rarely occur, but are more likely if tetracycline or chloramphenicol is prematurely stopped.

Prevention and Public Health Measures:

- Insecticides:
 - ◆ Application of insecticide to clothing of all personnel at risk of exposure.
 - ◆ Use of persistent insecticide for application to clothing of individuals at particular risk.
- Hygiene: command emphasis on personal hygiene and cleanliness of clothing.
- Disease reporting to higher echelon medical authorities.
- Report case as a reportable event using theater medical surveillance reporting channels.

TICK TYPHUS

Communicability:

- Route:
 - ◆ Bite of infected tick (Ixodid).
 - ◆ No evidence of person-to-person transmission.
- Isolation: not required.
- Prophylaxis: not required.

Incubation: 7 days.

Clinical manifestations and diagnosis: usually abrupt onset.

- Symptoms:
 - ◆ Fever (100%).
 - ◆ Headache, severe (60%).
 - ◆ Myalgia (35%).
 - ◆ Dyspnea.
 - ◆ Weakness, fatigue.
 - ◆ Cough.
- Signs:
 - ◆ Fever (100%) up to 39 °C for 7-14 days if untreated.
 - ◆ Rash (>95%).
 - Initial: painless eschar (tache noire) in 70% at site of infected tick bite (lower limbs, groin, abdomen) with tender regional lymphadenopathy followed by systemic symptoms.
 - Later: maculopapular rash on 3rd-5th day of fever, located on extremities (including palms/soles) then trunk. Eruption lasts 7-14 days if untreated. May develop petechial appearance in severe cases.
 - ◆ Conjunctival injection, chemosis (10%).
 - ◆ Hepatomegaly (13%).
- Laboratory findings:
 - ◆ Hematology: thrombocytopenia (35%).
 - ◆ Chemistry: hyponatremia, azotemia, elevated SGOT.
 - ◆ Microbiology: not available under field conditions.
 - ◆ Urinalysis: proteinuria.
 - ◆ Serology: available.

- ◆ Coagulation: prolonged prothrombin time (PPT) and bleeding time in severe cases.
- Invasive procedures: not indicated.
- Diagnostic confirmation: clinical diagnosis sufficient for patient care. Diagnosis may be confirmed by demonstrating rickettsiae in skin biopsy of macules using immunofluorescent techniques.

Duration: 7-14 days if untreated. Treatment leads to clinical improvement after 48 hours, with rapid resolution of symptoms and rash.

Complications: deep venous thrombosis, digital gangrene, myocarditis, renal failure, hemorrhage.

Treatment: Doxycycline 100 mg PO BID or Tetracycline 250 mg PO Q6H until 1 day after defervescence.

Disposition: local hospitalization, anticipate return to duty in 1-2 weeks. Evacuate cases with complications.

Prognosis: excellent, most show rapid clinical improvement after 46 hours of therapy.

Prevention and Public Health Measures:

- Avoid contact with ticks or tick-infested animals if possible.
- Use of DEET repellent and permethrin impregnated clothing by all personnel will reduce risk of exposure.
- Report case as a reportable event using theater medical surveillance reporting channels.

PART TWO: EXPOSURE TO BIOLOGICAL WARFARE AGENTS

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ANTHRAX

Symptoms/Signs: Incubation period is generally 1-6 days, although longer periods have been noted. Patients with inhalational anthrax would typically have fever, malaise, fatigue, dry cough and mild chest discomfort progressing to severe respiratory distress or dyspnea, diaphoresis, stridor, cyanosis, and shock. Death typically occurs within 24-36 hours after onset of severe symptoms. Other forms of anthrax include cutaneous (papule progressing to dark-colored eschar or ulcer), and gastrointestinal (nausea, vomiting, abdominal pain, bloody diarrhea; and including oropharyngeal anthrax with sore throat, dysphagia, and oral / tonsillar lesions). Significant lymphadenopathy (mediastinal in inhalational disease, regional in others) is characteristic.

Diagnosis: Physical findings are nonspecific. A widened mediastinum may be seen on CXR in later stages of illness. The organism is detectable by Gram stain of the blood and by blood culture late in the course of illness.

Prophylaxis: Oral Ciprofloxacin or doxycycline for known or imminent exposure. An FDA-licensed vaccine is available. Soldiers going to areas of increased threat should already be vaccinated. Vaccine schedule is 0.5 mL SC at 0, 2, 4 weeks, then 6, 12, and 18 months (primary series), followed by annual boosters.

Isolation and Decontamination: Standard precautions for HCWs. After an invasive procedure or autopsy is performed (avoid if possible), the instruments and area used should be thoroughly disinfected with a sporicidal agent (hypochlorite). Inhalation anthrax does not seem to be transmissible from person to person.

Treatment: Although effectiveness may be limited after symptoms are present, high dose antibiotic treatment with penicillin, and Ciprofloxacin or doxycycline should be undertaken. Penicillin should never be used as monotherapy for inhalational anthrax until strain sensitivities are known. It has been commonly used alone for cutaneous forms. Supportive therapy may be necessary.

- Almost all inhalational anthrax cases in which treatment was begun after patients were significantly symptomatic have been fatal, regardless of treatment. Military policy (FM 8-284) currently recommends Ciprofloxacin (400 mg IV Q12H) or doxycycline (200 mg IV load, followed by 100 mg IV Q12H) as initial therapy, with penicillin (4 million U IV Q4H) as an addition once sensitivity data is available. Published recommendations from a public health consensus panel recommends Ciprofloxacin as initial therapy. Therapy may then be tailored once antibiotic sensitivity is available to penicillin G or doxycycline. Tetracyclines and erythromycin have been recommended in penicillin allergic patients. Recommended treatment duration is 60 days, and should be changed to oral therapy as clinical condition improves. Supportive therapy for shock, fluid volume deficit, and adequacy of airway may all be needed.

Note: More information available at www.bt.cdc.gov/agent/anthrax/

BOTULINUM

Symptoms/Signs: Usually begins with cranial nerve palsies, including ptosis, blurred vision, diplopia, dry mouth and throat, dysphagia, and dysphonia. This is followed by symmetrical descending flaccid paralysis, with generalized weakness and progression to respiratory failure. Symptoms begin as early as 12-36 hours after inhalation, but may take several days after exposure to low doses of toxin.

Diagnosis: Diagnosis is primarily a clinical one. Biowarfare attack should be suspected if multiple casualties simultaneously present with progressive descending flaccid paralysis. Lab confirmation can be obtained by bioassay (mouse neutralization) of the patient's serum. Other helpful labs include: ELISA for antigen in environmental samples, PCR for bacterial DNA in environmental samples, or nerve conduction studies and electromyography.

Prophylaxis: Pentavalent toxoid vaccine (types A, B, C, D, and E) is available as an IND product for those at high risk of exposure.

Isolation and Decontamination: Standard precautions for HCWs. Toxin is not dermally active and secondary aerosols are not a hazard from patients. Decontaminate with soap and water. Botulinum toxin is inactivated by sunlight within 1-3 hours. Heat (80°C for 30 minutes, 100°C for several minutes) and chlorine (>99.7% inactivation by 3 mg/L free available chlorine (FAC) in 20 minutes); also destroy the toxin.

Treatment: Early administration of trivalent licensed antitoxin or heptavalent antitoxin (types A, B, C, D, E, F and G), available only as an IND product, may prevent or decrease progression to respiratory failure and hasten recovery. Intubation and ventilatory assistance for respiratory failure. Tracheostomy may be required.

- Supportive care, including prompt respiratory support, can be lifesaving. Respiratory failure due to paralysis of respiratory muscles is the most serious effect and, gener-

ally, the cause of death. Reported cases of botulism prior to 1950 had a mortality rate of 60%. With tracheotomy or endotracheal intubation and ventilatory assistance, fatalities are less than 5% today. Prevention of nosocomial infections is a primary concern, along with hydration, nasogastric suctioning for ileus, bowel and bladder care, and prevention of decubitus ulcers and deep venous thromboses. Intensive and prolonged nursing care may be required for recovery, which may take up to 3 months for initial signs of improvement, and up to 1 year for complete resolution of symptoms.

Antitoxin: Early administration of botulinum antitoxin is critical, since the antitoxin can only neutralize the circulating toxin in patients with symptoms that continue to progress. When symptom progression ceases, no circulating toxin remains, and the antitoxin has no effect. Antitoxin may be particularly effective in food-borne cases, where presumably toxin continues to be absorbed through the gut wall. Animal experiments show that after aerosol exposure, botulinum antitoxin is very effective if given before the onset of clinical signs. If the antitoxin is delayed until after the onset of symptoms, it does not protect against respiratory failure.

- Three different antitoxin preparations are available in the United States. A licensed trivalent (types A, B, E) equine antitoxin is available from the Centers for Disease Control and Prevention for cases of food-borne botulism. This product has all the disadvantages of a horse serum product, including the risks of anaphylaxis and serum sickness. A monovalent human antiserum (type A) is available from the California Department of Health Services for infant botulism. USAMRIID (1-888-872-7443) offers a heptavalent antitoxin available under IND status.
- Use of the equine antitoxin requires skin testing for horse serum sensitivity prior to administration. Skin testing is performed by injecting 0.1 mL of a 1:10 dilution (in sterile physiological saline) of antitoxin intradermally in the patient's forearm with a 26 or 27 gauge needle. Monitor the injection site and observe the patient for allergic reaction for 20 minutes. The skin test is positive if any of

these allergic reactions occur: hyperemic areola (colored ring) at the site of the injection > 0.5 cm; fever or chills; hypotension with decrease of blood pressure > 20 mm Hg for systolic and diastolic pressures; skin rash; respiratory difficulty; nausea or vomiting; generalized itching. DO NOT administer equine-derived Botulinum F(ab)₂ antitoxin if the skin test is positive. If no allergic symptoms are observed, the antitoxin is administered as a single dose intravenously in normal saline solution, 10 mL over 20 minutes.

- With a positive skin test, desensitization can be attempted by administering 0.01 - 0.1 mL of antitoxin subcutaneously, doubling the previous dose every 20 minutes until 1.0 - 2.0 mL can be sustained without any marked reaction. Preferably, desensitization should be performed by an experienced allergist. Medical personnel administering the antitoxin should be prepared to treat anaphylaxis with epinephrine, intubation equipment, and IV access.

BRUCELLOSIS

(see also Brucellosis, Part One: Endemic Infectious Diseases)

2

Symptoms/Signs: Illness, when manifest, typically presents with fever, headache, myalgias, arthralgias, back pain, sweats, chills, and generalized malaise. Other manifestations include depression, mental status changes, and osteoarticular findings (i.e., Sacroiliitis, vertebral osteomyelitis). Fatalities are uncommon.

Diagnosis: Diagnosis requires a high index of suspicion, since many infections present as nonspecific febrile illnesses or are asymptomatic. Laboratory diagnosis can be made by blood culture with prolonged incubation. Bone marrow cultures produce a higher yield. Confirmation requires phage-typing, oxidative metabolism, or genotyping procedures. ELISA, followed by Western blot are available.

Prophylaxis: There is no human vaccine available against brucellosis, although animal vaccines exist. Chemoprophylaxis is not recommended after possible exposure to endemic disease. Treatment should be considered for high-risk exposure to the potentially infected livestock tissues and body fluids and aerosols, inadvertent laboratory exposure, or confirmed biological warfare exposure.

Isolation and Decontamination: Brucellosis is readily spread by body fluids and aerosols. Standard precautions are appropriate for HCWs. Person-to-person transmission has been reported via tissue transplantation and sexual contact. Environmental decontamination can be accomplished with a 0.5% hypochlorite solution.

Treatment: Antibiotic therapy with doxycycline + rifampin for 6 weeks is usually sufficient in most cases. More prolonged regimens may be required for patients with complications of meningoencephalitis, endocarditis, or osteomyelitis.

- Oral antibiotic therapy alone is sufficient in most cases of brucellosis. Exceptions involve uncommon cases of localized disease, where surgical intervention may be required (e.g., valve replacement for endocarditis). A combination of Doxycycline 200 mg/d PO + Rifampin 600 mg/d PO is generally recommended. Both drugs should be administered for 6 weeks. Doxycycline 200 mg/d PO for 6 weeks in combination with 2 weeks of Streptomycin (1 g/d IM) is an acceptable alternative. Regimens involving Doxycycline + Gentamicin, TMP/SMX + Gentamicin, and Ofloxacin + Rifampin have also been studied and shown effective. Long-term triple-drug therapy with rifampin, a tetracycline, and an aminoglycoside is recommended by some experts for patients with meningoencephalitis or endocarditis.

GLANDERS AND MELIOIDOSIS

Symptoms/Signs: These are two distinct diseases with different epidemiology but similar diagnosis and treatment. Incubation period ranges from 10-14 days after inhalation. Onset of symptoms may be abrupt or gradual. Inhalational exposure produces fever (common in excess of 102 °F), rigors, sweats, myalgias, headache, pleuritic chest pain, cervical adenopathy, hepatosplenomegaly, and generalized papular/pustular eruptions. Acute pulmonary disease can progress and result in bacteremia and acute septicemic disease. Both diseases are almost always fatal without treatment.

Diagnosis: Methylene blue or Wright stain of exudates may reveal scant small bacilli with a safety-pin bipolar appearance. Standard cultures can be used to identify both *B. mallei* and *B. pseudomallei*. CXR may show military lesions, small multiple lung abscesses, or infiltrates involving upper lungs, with consolidation and cavitation. Leukocyte counts may be normal or elevated. Serologic tests can help confirm diagnosis, but low titers or negative serology does not exclude the diagnosis.

Prophylaxis: Currently, no pre-exposure or post-exposure prophylaxis is available.

Isolation and Decontamination: Standard precautions for health care person-to-person airborne transmission are unlikely, although secondary cases may occur through improper handling of infected secretions. Contact precautions are indicated while caring for patients with skin involvement. Environmental decontamination using a 0.5% hypochlorite solution is effective.

Treatment: Therapy will vary with the type and severity of the clinical presentation. Patients with localized disease may be managed with oral antibiotics for 60-150 days. More severe illness may require parenteral therapy and more prolonged treatment.

- The recommended therapy will vary with the type and severity of the clinical presentation. The following oral regimens have been suggested for localized disease: Amoxicillin / clavulanate 60 mg/kg/day in three divided doses; Tetracycline 40 mg/kg/day in three divided doses; or Trimethoprim / sulfa (TMP 4 mg/kg/day-sulfa 20 mg/kg/day) in two divided doses. The duration of treatment should be for 60-150 days.
 - ◆ If the patient has localized disease with signs of mild toxicity, then a combination of two of the oral regimens is recommended for a duration of 30 days, followed by monotherapy with either amoxicillin / clavulanate or TMP / sulfa for 60-150 days. If extrapulmonary suppurative disease is present, then therapy should continue for 6-12 months. Surgical drainage of abscesses may be required.
- For severe disease, parental therapy with Ceftazidime 120 mg/kg/day in three divided doses combined with TMP/sulfa (TMP 8 mg/kg/day – sulfa 40 mg/kg/day) in four divided doses for 2 weeks, followed by oral therapy for 6 months.
- Other antibiotics that have been effective in experimental infection in hamsters include doxycycline, rifampin, and Ciprofloxacin. The limited number of infections in humans has precluded therapeutic evaluation of most of the antibiotic agents; therefore, most antibiotic sensitivities are based on animal and *in vitro* studies. Various isolates have markedly different antibiotic sensitivities; therefore, each isolate should be tested for its own resistance pattern.

PLAGUE

2

Symptoms/Signs: Pneumonic plague begins after an incubation period of 1-6 days, with high fever, chills, headache, malaise, followed by cough (often with hemoptysis), progressing rapidly to dyspnea, stridor, cyanosis, and death. Gastrointestinal symptoms are often present. Death results from respiratory failure, circulatory collapse, and a bleeding diathesis. Bubonic plague, featuring high fever, malaise, and painful lymph nodes (buboes) may progress spontaneously to the septicemic form (septic shock, thrombosis, DIC) or to the pneumonic form. Septicemia may also occur without evidence of a bubo, and plague meningitis is not uncommon.

Diagnosis: Suspect plague if large numbers of previously healthy individuals develop fulminant Gram negative pneumonia, especially if hemoptysis is present. Presumptive diagnosis can be made by Gram, Wright, Giemsa or Wayson stain of blood, sputum, CSF, or lymph node aspirates. Definitive diagnosis requires culture of the organism from those sites. Immunodiagnosis is also helpful.

Prophylaxis: For asymptomatic persons exposed to a plague aerosol or to a patient with suspected pneumonic plague, give doxycycline 100 mg orally twice daily for 7 days or the duration of risk of exposure plus 1 week. Alternative antibiotics include Ciprofloxacin, tetracycline, or chloramphenicol. No vaccine is currently available for plague prophylaxis. The previously available licensed, killed vaccine was effective against the most likely BW form, bubonic plague, but not against aerosol exposure.

Isolation and Decontamination: Use standard precautions for bubonic plague, and respiratory droplet precautions for suspected pneumonic plague. *Y. pestis* can survive in the environment for varying periods, but is susceptible to heat, disinfectants, and exposure to sunlight. Soap and water is effective if decon is needed. Take measures to prevent local disease cycles if vectors (fleas) and reservoirs (rodents) are present.

Treatment: Early administration of antibiotics is critical, as pneumonic plague is invariably fatal if antibiotic therapy is delayed more than 1 day after the onset of symptoms. Choose one of the following: streptomycin, gentamicin, Ciprofloxacin, or doxycycline for 10-14 days. Chloramphenicol is the drug of choice for plague meningitis.

- Streptomycin, gentamicin, doxycycline, and chloramphenicol are highly effective, if begun early. Plague pneumonia is almost always fatal if treatment is not initiated within 24 hours of the onset of symptoms. Dosage regimens are as follows:
 - ◆ streptomycin, 30 mg/kg/day IM in two divided doses;
 - ◆ gentamicin, 5 mg/kg IM or IV once daily, or 2 mg/kg loading dose followed by 1.75 mg/kg IM or IV every 8 hours;
 - ◆ doxycycline 200 mg initially, followed by 100 mg every 12 hours.

Duration of therapy is 10-14 days. While the patient is typically afebrile after 3 days, the extra week of therapy prevents relapses. Results obtained from laboratory animal, but not human, experience, indicate that quinolone antibiotics, such as Ciprofloxacin and ofloxacin, may also be effective. Recommended dosage of Ciprofloxacin is 400 mg IV twice daily. Chloramphenicol, 25 mg/kg IV loading dose followed by 15 mg/kg IV four times daily for 10-14 days, is required for the treatment of plague meningitis.

- Usual supportive therapy includes IV crystalloids and hemodynamic monitoring. Although low-grade DIC may occur, clinically significant hemorrhage is uncommon, as is the need to treat with heparin. Endotoxic shock is common, but pressor agents are rarely needed. Finally, buboes rarely require any form of local care or surgical drainage, but instead recede with systemic antibiotic therapy. In fact, incision and drainage poses a risk to others in contact with the patient; aspiration is recommended for diagnostic purposes and may provide symptomatic relief. It is standard practice to first inject saline to mobilize aspirated contents.

Q FEVER

(see also Q Fever, Part One: Endemic Infectious Diseases)

Symptoms/Signs: Fever, cough, and pleuritic chest pain may occur as early as 10 days after exposure. Patients are not generally critically ill (BW incapacitating agent), and the illness lasts from 2 days to 2 weeks.

Diagnosis: Q fever is not a clinically distinct illness and may resemble a viral illness or other types of atypical pneumonia. A high suspicion is necessary. The diagnosis is confirmed serologically.

Prophylaxis: Chemoprophylaxis begun too early during the incubation period may delay but not prevent the onset of symptoms. Therefore, tetracycline or doxycycline should be started 8-12 days post exposure and continued for 5 days. This regimen has been shown to prevent clinical disease. An inactivated whole cell IND vaccine is effective in eliciting protection against exposure, but severe local reactions to this vaccine may be seen in those who already possess immunity. Therefore, an intradermal skin test is recommended to detect presensitized or immune individuals.

Isolation and Decontamination: Standard precautions are recommended for HCWs. Person-to-person transmission is rare. Patients exposed to Q fever by aerosol do not present a risk for secondary contamination or re-aerosolization of the organism. Decontamination is accomplished with soap and water or a 0.5% chlorine solution on personnel. The M291 skin decontamination kit will not neutralize the organism.

Treatment: Q fever is generally a self-limited illness even without treatment, but tetracycline or doxycycline should be given orally for 5-7 days to prevent complications of the disease. Q fever endocarditis (rare) is much more difficult to treat.

- Standard precautions are recommended for HCWs. Most cases of acute Q fever will eventually resolve without antibiotic treatment, but all suspected cases of Q fever should be treated to reduce the risk of complications. Tetracycline 500 mg every 6 hours or doxycycline 100 mg every 12 hours for 5-7 days will shorten the duration of illness, and fever usually disappears within 1-2 days after treatment is begun. Ciprofloxacin and other quinolones are active *in vitro* and should be considered in patients unable to take tetracycline or doxycycline.

RICIN

2

Symptoms/Signs: Acute onset of fever, chest tightness, cough, dyspnea, nausea, and arthralgias occurs 4-8 hours after inhalational exposure. Airway necrosis and pulmonary capillary leak resulting in pulmonary edema would likely occur within 18-24 hours, followed by severe respiratory distress and death from hypoxemia in 36-72 hours.

Diagnosis: Acute lung injury in large numbers of geographically clustered patients suggests exposure to aerosolized ricin. The rapid time course to severe symptoms and death would be unusual for infectious agents. Serum and respiratory secretions should be submitted for antigen detection (ELISA). Acute and convalescent sera provide retrospective diagnosis. Nonspecific laboratory and radiographic findings include leukocytosis and bilateral interstitial infiltrates.

Prophylaxis: There is currently no vaccine or prophylactic antitoxin available for human use, although immunization appears promising in animal models. Use of the military chemical protective mask is currently the best protection against inhalation.

Isolation and Decontamination: Standard precautions for HCWs. Ricin is nonvolatile, and secondary aerosols are not expected to be a danger to health care providers. Decontaminate with soap and water. Hypochlorite solutions (0.1% sodium hypochlorite) can inactivate ricin.

Treatment: Management is supportive and should include treatment for pulmonary edema. Gastric lavage and cathartics are indicated for ingestion, but charcoal is of little value for large molecules such as ricin.

- Management of ricin-intoxicated patients depends on the route of exposure. Patients with pulmonary intoxication are managed by appropriate respiratory support (oxygen, intubation, ventilation, PEEP, and hemodynamic monitoring) and treatment for pulmonary edema, as indicated. Gastrointestinal intoxication is best managed by vigorous gastric lavage, followed by use of cathartics such as magnesium citrate. Superactivated charcoal is of little value for large molecules such as ricin. Volume replacement of GI fluid losses is important. In percutaneous exposures, treatment would be primarily supportive.

SMALLPOX

2

Symptoms/Signs: Clinical manifestations begin acutely with malaise, fever, rigors, vomiting, headache. Two to three days later, lesions appear which quickly progress from macules to papules, and eventually to pustular vesicles. They are more abundant on the extremities and face, and develop synchronously.

Diagnosis: Neither electron nor light microscopy are capable of discriminating variola from vaccinia, monkeypox or cowpox. The new PCR diagnostic techniques may be more accurate in discriminating between variola and other *Orthopoxviruses*. (See “CDC Protocol for Evaluating Patients for Smallpox” at www.bt.cdc.gov/agent/smallpox/diagnosis/evalposter.asp)

Prophylaxis: Immediate vaccination or revaccination should be undertaken for all personnel exposed.

Isolation and Decontamination: Droplet and airborne precautions for a minimum of 17 days following exposure for all contacts. Patients should be considered infectious until all scabs separate and quarantined during this period. In the civilian setting, strict quarantine of asymptomatic contacts may prove to be impractical and impossible to enforce. A reasonable alternative would be to require contacts to check their temperatures daily. Any fever above 101°F (38°C) during the 17-day period following exposure to a confirmed case would suggest the development of smallpox. The contact should then be isolated immediately, preferably at home, until smallpox is either confirmed or ruled out, and remain in isolation until all scabs separate.

Treatment: At present, there is no effective chemotherapy, Cidofovir is under investigation, and treatment of a clinical case remains supportive.

STAPHYLOCOCCAL ENTEROTOXIN B

Symptoms/Signs: Latent period of 3-12 hours after aerosol exposure is followed by sudden onset of fever, chills, headache, myalgia, and nonproductive cough. Some patients may develop shortness of breath and retrosternal chest pain. Patients tend to plateau rapidly to a fairly stable clinical state. Fever may last 2-5 days, and cough may persist for up to 4 weeks. Patients may also present with nausea, vomiting, and diarrhea if they swallow the toxin. Presumably, higher exposure can lead to septic shock and death.

Diagnosis: Diagnosis is clinical. Patients present with a febrile respiratory syndrome without CXR abnormalities. Large numbers of patients presenting in a short period of time with typical symptoms and signs of SEB pulmonary exposure would suggest an intentional attack with this toxin.

Prophylaxis: Use of military chemical protective mask. There is currently no human vaccine available to prevent SEB intoxication.

Isolation and Decontamination: Standard precautions for HCWs. SEB is not dermally active and secondary aerosols are not a hazard from patients. Decontaminate with soap and water. Destroy any food that may have been contaminated.

Treatment: Treatment is limited to supportive care. Artificial ventilation might be needed for very severe cases, and attention to fluid management is important.

- Currently, therapy is limited to supportive care. Close attention to oxygenation and hydration is important, and in severe cases with pulmonary edema, ventilation with positive end expiratory pressure, vasopressors and diuretics might be necessary. Acetaminophen for fever, and cough suppressants may make the patient more comfortable. The value of steroids is unknown. Most patients would be expected to do quite well after the initial acute phase of their illness, but generally would be unfit for duty for 1-2 weeks. Severe cases risk death from pulmonary edema and respiratory failure.

T-2 MYCOTOXINS

Symptoms/Signs: Exposure causes skin pain, pruritus, redness, vesicles, necrosis, and sloughing of the epidermis. Effects on the airway include nose and throat pain, nasal discharge, itching and sneezing, cough, dyspnea, wheezing, chest pain, and hemoptysis. Toxin also produces effects after ingestion or eye contact. Severe intoxication results in prostration, weakness, ataxia, collapse, shock, and death.

Diagnosis: Should be suspected if an aerosol attack occurs in the form of "yellow rain" with droplets of variously pigmented oily fluids contaminating clothes and the environment. Confirmation requires testing of blood, tissue, and environmental samples.

Prophylaxis: The only defense is to prevent exposure by wearing a military chemical protective mask and clothing (or topical skin protectant) during an attack. No specific immunotherapy or chemotherapy is available for use in the field.

Isolation and Decontamination: Outer clothing should be removed and exposed skin decontaminated with soap and water. Eye exposure should be treated with copious saline irrigation. Secondary aerosols are not a hazard; however, contact with contaminated skin and clothing can produce secondary dermal exposures. Contact precautions are warranted until decontamination is accomplished. Then, standard precautions are recommended for HCWs. Environmental decontamination requires the use of a hypochlorite solution under alkaline conditions such as 1% sodium hypochlorite and 0.1M NaOH with 1 hour contact time.

Treatment: There is no specific antidote. Treatment is supportive. Soap and water washing, even 4-6 hours after exposure can significantly reduce dermal toxicity; washing within 1 hour may prevent toxicity entirely. Superactivated charcoal should be given orally if the toxin is swallowed.

- No specific antidote or therapeutic regimen is currently available. All therapy is supportive. If a soldier is unprotected during an attack, the outer uniform should be removed within 4 hours and decontaminated by exposure to 5% hypochlorite for 6-10 hours. The skin should be thoroughly washed with soap and uncontaminated water if available. This can reduce dermal toxicity, even if delayed 4-6 hours after exposure. The M291 skin decontamination kit can also be used to remove skin-adherent T-2. Standard burn care is indicated for cutaneous involvement. Standard therapy for poison ingestion, including the use of superactivated charcoal to absorb swallowed T-2, should be administered to victims of an unprotected aerosol attack. Respiratory support may be necessary. The eyes should be irrigated with normal saline or water to remove toxin.

TULAREMIA

Symptoms/Signs: Ulceroglandular tularemia presents with a local ulcer and regional lymphadenopathy, fever, chills, headache and malaise. Typhoidal tularemia presents with fever, headache, malaise, substernal discomfort, prostration, weight loss, and a nonproductive cough. The ulceroglandular form is the most common in nature, but the typhoidal form is the most likely to be used in a BW attack.

Diagnosis: Clinical diagnosis. Physical findings are usually nonspecific. Chest x-ray may reveal a pneumonic process, mediastinal lymphadenopathy or pleural effusion. Routine culture is possible but difficult. The diagnosis can be established retrospectively by serology.

Prophylaxis: A live, attenuated vaccine is available as an investigational new drug from USAMRIID (1-888-872-7443). It is administered once by scarification. Doxycycline or Ciprofloxacin given as a 2-week course is effective as prophylaxis when given after exposure.

Isolation and Decontamination: Standard precautions for HCWs. Organisms are relatively easy to render harmless by mild heat (55°C for 10 minutes) and standard disinfectants.

Treatment: Administration of antibiotics (streptomycin or gentamicin) with early treatment is very effective.

- Appropriate therapy includes one of the following antibiotics:
 - ◆ Gentamicin 3 - 5 mg/kg IV daily for 10-14 days.
 - ◆ Ciprofloxacin 400 mg IV every 12 hours, switch to oral Ciprofloxacin (500 mg every 12 hours) after the patient is clinically improved; continue for completion of a 10- to 14-day course of therapy.
 - ◆ Ciprofloxacin 750 mg orally every 12 hours for 10-14 days.
 - ◆ Streptomycin 7.5 - 10 mg/kg IM every 12 hours for 10-14 days.

- Streptomycin has historically been the drug of choice for tularemia; however, since it may not be readily available immediately after a large-scale BW attack, gentamicin and other alternative drugs should be considered first. Requests for streptomycin should be directed to the Roerig Streptomycin Program at Pfizer Pharmaceuticals in New York (800-254-4445)*. The strain was sensitive to gentamicin. Gentamicin offers the advantage of providing broader coverage for gram-negative bacteria and may be useful when the diagnosis of tularemia is considered but in doubt. Tetracycline and chloramphenicol are also effective antibiotics; however, they are associated with significant relapse rates.

* This number may only be answered during regular office hours: Monday-Friday, 8:30 AM to 5:30 PM, EST.

VENEZUELAN EQUINE ENCEPHALITIS

2

Symptoms/Signs: Incubation period 1-6 days. Acute systemic febrile illness with encephalitis developing in a small percentage (4% children; < 1% adults). Generalized malaise, spiking fevers, rigors, severe headache, photophobia, and myalgias for 24-72 hours. Nausea, vomiting, cough, sore throat, and diarrhea may follow. Full recovery from malaise and fatigue takes 1-2 weeks. The incidence of CNS disease and associated morbidity and mortality would be much higher after a BW attack.

Diagnosis: Clinical and epidemiological diagnosis. Physical findings nonspecific. The white blood cell count may show a striking leukopenia and lymphopenia. Virus isolation may be made from serum, and in some cases throat swab specimens. Both neutralizing or IgG antibody in paired sera or VEE-specific IgM present in a single serum sample indicate recent infection.

Prophylaxis: A live, attenuated vaccine is available as an investigational new drug. A second, formalin-inactivated, killed vaccine is available for boosting antibody titers in those initially receiving the first vaccine. No post-exposure immunoprophylaxis. Alpha-interferon has been effective in animal models. There are no human clinical data.

Isolation and Decontamination: Patient isolation and quarantine is not required. Standard precautions augmented with vector control while the patient is febrile. There is no evidence of direct human-to-human or horse-to-human transmission. The virus can be destroyed by heat (80°C for 30 minutes) and standard disinfectants.

Therapy: Treatment is supportive only. Treat uncomplicated VEE infections with analgesics to relieve headache and myalgia. Patients who develop encephalitis may require anticonvulsants and intensive supportive care to maintain fluid and electrolyte balance, ensure adequate ventilation, and avoid complicating secondary bacterial infections. Patients should be treated in a screened room or in quarters treated with a residual insecticide for at least 5 days after onset, or until afebrile, as human cases may be infectious to mosquitoes for at least 72 hours.

VIRAL HEMORRHAGIC FEVERS (VHF)

Viral hemorrhagic fevers are a diverse group of illnesses caused by RNA viruses from four viral families. The *Arenaviridae* include the etiologic agents of Argentine, Bolivian, and Venezuelan hemorrhagic fevers, and Lassa fever. The *Bunyaviridae* include the members of the *Hantavirus* genus, the CCHF virus from the *Nairovirus* genus, and the Rift Valley fever virus from the *Phlebovirus* genus; the *Filoviridae* include Ebola and Marburg viruses; and the *Flaviviridae* include dengue and yellow fever viruses. These viruses are spread in a variety of ways; some may be transmitted to humans through a respiratory portal of entry. Although evidence for weaponization does not exist for many of these viruses, they are included in this TG because of their *potential* for aerosol dissemination or weaponization, or likelihood for confusion with similar agents that might be weaponized.

Symptoms/Signs: VHFs are febrile illnesses which can feature flushing of the face and chest, petechiae, bleeding, edema, hypotension, and shock. Malaise, myalgias, headache, vomiting, and diarrhea may occur in any of the hemorrhagic fevers.

Diagnosis: Definitive diagnosis rests on specific virologic techniques. Significant numbers of military personnel with a hemorrhagic fever syndrome should suggest the diagnosis of a VHF.

Prophylaxis: The only licensed VHF vaccine is yellow fever vaccine. Prophylactic ribavirin (available only as IND under protocol) may be effective for Lassa fever, Rift Valley Fever, CCHF, and possibly hemorrhagic fever with renal syndrome (HFRS).

Isolation and Decontamination: Contact isolation, with the addition of a surgical mask and eye protection for those coming within 3 feet of the patient, is indicated for suspected or proven Lassa fever, CCHF, or filovirus infections. Respiratory protection should be upgraded to airborne isolation, including the use of a fit-tested HEPA filtered respirator, a

2 battery-powered air purifying respirator, or a positive pressure-supplied air respirator, if patients with the above conditions have prominent cough, vomiting, diarrhea, or hemorrhage. Decontamination is accomplished with hypochlorite or phenolic disinfectants.

Treatment: Intensive supportive care may be required. Antiviral therapy with ribavirin may be useful in several of these infections (available only as IND under protocol). Convalescent plasma may be effective in Argentine hemorrhagic fever (available only as IND under protocol).

- General principles of supportive care apply to hemodynamic, hematologic, pulmonary, and neurologic manifestations of VHF, regardless of the specific etiologic agent. Only intensive care will save the most severely ill patients. Health care providers employing vigorous fluid resuscitation of hypotensive patients must be mindful of the propensity of some VHFs (e.g., HFRS) for pulmonary capillary leak. Pressor agents are frequently required. The use of intravascular devices and invasive hemodynamic monitoring must be carefully considered in the context of potential benefit versus the risk of hemorrhage. Restlessness, confusion, myalgia, and hyperesthesia should be managed by conservative measures, and the judicious use of sedatives and analgesics. Secondary infections may occur as with any patient undergoing intensive care utilizing invasive procedures and devices, such as IV lines and indwelling catheters.
- The management of clinical bleeding should follow the same principles as for any patient with a systemic coagulopathy, assisted by coagulation studies. Intramuscular injections, aspirin, and other anticoagulant drugs should be avoided.
- The investigational antiviral drug ribavirin is available via compassionate use protocols for therapy of Lassa fever, HFRS, CCHF, and Rift Valley fever. Separate Phase III efficacy trials have indicated that parenteral ribavirin reduces morbidity in HFRS, and lowers both the morbidity and mortality of Lassa fever. In the HFRS field trial, treatment was effective if begun within the first 4 days of fever, and continued for a 7-day course. A compassionate use protocol, utilizing IV ribavirin as a treatment for Lassa fever,

is sponsored by the CDC. Doses are slightly different, and continued for a 10-day course; treatment is most effective if begun within 7 days of onset. The only significant side effect of ribavirin is a modest anemia due to a reversible inhibition of erythropoiesis, and mild hemolysis. Although ribavirin is teratogenic in laboratory animals, the potential benefits must be weighed against the potential risks to pregnant women with grave illness due to one of these VHFs. Safety in infants and children has not been established. Ribavirin has poor *in vitro* and *in vivo* activity against the filoviruses (Ebola and Marburg) and the flaviviruses (dengue, yellow fever, Omsk HF and Kyasanur Forest disease).

- Argentine HF responds to therapy with 2 or more units of convalescent plasma containing adequate amounts of neutralizing antibody and given within 8 days of onset. This therapy is investigational, and available only under protocol.

DECONTAMINATION

Contamination is the introduction of an infectious agent on a body surface, food or water, or other inanimate objects. Decontamination involves either disinfection or sterilization to reduce microorganisms to an acceptable level on contaminated articles, thus rendering them suitable for use. Disinfection is the selective reduction of undesirable microbes to a level below that required for transmission. Sterilization is the killing of all organisms.

Decontamination methods have always played an important role in the control of infectious diseases. However, we are often unable to use the most efficient means of rendering microbes harmless (e.g., toxic chemical sterilization), as these methods may injure people and damage materials which are to be decontaminated. BW agents can be decontaminated by mechanical, chemical, and physical methods:

- Mechanical decontamination involves measures to remove but not necessarily neutralize an agent. An example is the filtering of drinking water to remove certain water-borne pathogens (e.g., *Dracunculus medinensis*), or in a BW context, the use of an air filter to remove aerosolized anthrax spores, or water to wash agent from the skin.
- Chemical decontamination renders BW agents harmless by the use of disinfectants that are usually in the form of a liquid, gas or aerosol. Some disinfectants are harmful to humans, animals, the environment, and materials.
- Physical means (heat, radiation) are other methods that can be employed for decontamination of objects.

Dermal exposure to a suspected BW aerosol should be immediately treated by soap and water decontamination. Careful washing with soap and water removes nearly all of the agent from the skin surface. Hypochlorite solution or other disinfectants are reserved for gross contamination (i.e., following the spill of solid or liquid agent from a munition directly onto the skin). In the absence of chemical or gross biological contamination, these will confer no additional benefit, may be caustic, and may predispose to coloniza-

tion and resistant superinfection by reducing the normal skin flora. Grossly contaminated skin surfaces should be washed with a 0.5% sodium hypochlorite solution, if available, with a contact time of 10-15 minutes. Place a damp cloth over potential reaerosolization hazards.

Ampules of calcium hypochlorite high test hypochlorite (HTH) are currently fielded in the Chemical Agent Decon Set for mixing hypochlorite solutions. The 0.5% solution can be made by adding one 6-ounce container of calcium hypochlorite to five gallons of water. The 5% solution can be made by adding eight 6-ounce ampules of calcium hypochlorite to 5 gallons of water. These solutions evaporate quickly at high temperatures, so if they are made in advance, they should be stored in closed containers. Also, the chlorine solutions should be placed in distinctly marked containers because it is very difficult to tell the difference between the 5% chlorine solution and the 0.5% solution.

To mix a 0.5% sodium hypochlorite solution, take one part Clorox and nine parts water (1:9), since standard stock Clorox is a 5.25% sodium hypochlorite solution. The solution is then applied with a cloth or swab. The solution should be made fresh daily with the pH in the alkaline range.

Chlorine solution must NOT be used in (1) open body cavity wounds, as it may lead to the formation of adhesions, or (2) brain and spinal cord injuries. However, this solution may be instilled into noncavity wounds and then removed by suction to an appropriate disposal container. Within about 5 minutes, this contaminated solution will be neutralized and nonhazardous. Subsequent irrigation with saline or other surgical solutions should be performed. Prevent the chlorine solution from being sprayed into the eyes, as corneal opacities may result.

For decontamination of fabric clothing or equipment, a 5% hypochlorite solution should be used. For decontamination of equipment, a contact time of 30 minutes prior to normal cleaning is required. This is corrosive to most metals and injurious to most fabrics, so rinse thoroughly and apply oil to metal surfaces after completion.

2 BW agents can be rendered harmless through such physical means as heat and radiation. To render agents completely harmless, sterilize with dry heat for 2 hours at 160°C. If autoclaving with steam at 121°C and 1 atmosphere of overpressure (15 pounds per square inch), the time may be reduced to 20 minutes, depending on volume. Solar ultraviolet (UV) radiation has a disinfectant effect, often in combination with drying. This is effective in certain environmental conditions, but hard to standardize for practical usage for decontamination purposes.

The health hazards posed by environmental contamination with biological agents differ from those posed by persistent or volatile chemical agents. Aerosolized particles in the 1-5 μm size range will remain suspended due to Brownian motion; suspended BW agents would be eventually inactivated by solar UV light, desiccation, and oxidation. Little, if any, environmental residues would occur. Possible exceptions include residues near the dissemination line, or in the immediate area surrounding a point-source munition. BW agents deposited on the soil would be subject to degradation by environmental stressors and competing soil microflora. Simulant studies at Dugway Proving Ground suggest that secondary reaerosolization would be difficult, and would probably not pose a human health hazard. Environmental decontamination of terrain is costly and difficult and should be avoided, if possible. If grossly contaminated terrain, streets, or roads must be passed, the use of dust-binding spray to minimize reaerosolization may be considered. If it is necessary to decontaminate these surfaces, chlorine-calcium or lye may be used. Otherwise, rely on the natural processes which, especially outdoors, lead to the decontamination of agent by drying and solar UV radiation. Rooms in fixed spaces are best decontaminated with gases or liquids in aerosol form (e.g., formaldehyde). This is usually combined with surface disinfectants to ensure complete decontamination.

BW Agent Characteristics

2

Disease	Transmit Man to Man	Infective Dose (Aerosol)	Incubation Period	Duration of Illness	Lethality (approx. case fatality rates)
Inhalation anthrax	No	8,000-50,000 spores	1-6 days	3-5 days (usually fatal if untreated)	High
Brucellosis	No	10 -100 organisms	5-60 days (usually 1-2 months)	Weeks to months	<5% untreated
Cholera	Rare	10-500 organisms	4 hours - 5 days (usually 2-3 days)	≥ 1 week	Low with treatment, high without
Glanders	Low	Assumed low	10-14 days via aerosol	Death in 7-10 days in septicemic form	> 50%
Pneumonic Plague	High	100-500 organisms	2-3 days	1-6 days (usually fatal)	High unless treated within 12-24 hours
Tularemia	No	10-50 organisms	2-10 days (average 3-5)	≥ 2 weeks	Moderate if untreated
Q Fever	Rare	1-10 organisms	10-40 days	2-14 days	Very low
Smallpox	High	Assumed low (10-100 organisms)	7-17 days (average 12)	4 weeks	High to moderate
Venezuelan Equine Encephalitis	Low	10-100 organisms	2-6 days	Days to weeks	Low
Viral Hemorrhagic Fevers	Moderate	1-10 organisms	4-21 days	Death between 7-16 days	High for Zaire strain, moderate with Sudan
Botulism	No	0.001 µg/kg is LD ₅₀ for type A	1-5 days	Death in 24-72 hours; lasts months if not lethal	High without respiratory support
Staph Enterotoxin B	No	0.03 µg/ person incapacitation	3-12 hours after inhalation	Hours	< 1%
Ricin	No	3-5 µg/kg is LD ₅₀ in mice	18-24 hours	Days - death within 10-12 days for ingestion	High
T-2 Mycotoxins	No	Moderate	2-4 hours	Days to months	Moderate

BW AGENTS – DRUG THERAPY AND PROPHYLAXIS

2

DISEASE	CHEMOTHERAPY (Rx)	CHEMOPROPHYLAXIS (Px)	COMMENTS
Anthrax	Ciprofloxacin 400 mg IV Q12H or Doxycycline 200 mg IV, then 100 mg IV Q12H	Ciprofloxacin 500 mg PO BID x 4 wk. If unvaccinated, begin initial doses of vaccine.	Potential alternates for Rx: gentamicin, erythromycin, and chloramphenicol
	Penicillin 4 million units IV Q4H	Doxycycline 100 mg PO BOD x 4 wk plus vaccination	PCN for sensitive organisms only
Cholera	Oral rehydration therapy during period of high fluid loss	NA	Vaccine not recommended for routine protection in endemic areas (50% efficacy, short term)
	Tetracycline 500 mg Q6H x 3 d		Alternates for Rx: erythromycin, trimethoprim and sulfamethoxazole, and furazolidone
	Doxycycline 300 mg once, or 100 mg Q12H x 3 d		Quinolones for tetra/doxy resistant strains
	Ciprofloxacin 500 mg Q12H x 3 d		
	Norfloxacin 400 mg Q12H x 3 d		
Q Fever	Tetracycline 500 mg PO Q6H x 5-7 d continued at least 2 d after afebrile	Tetracycline 500 mg PO QID x 5 d (start 8-12 d post-exposure)	Currently testing vaccine to determine the necessity of skin testing prior to use.
	Doxycycline 100 mg PO Q12H x 5-7 d continued at least 2 d after afebrile	Doxycycline 100 mg PO BID x 5 d (start 8-12 d post-exposure)	
Glanders	Antibiotic regimens vary depending on localization and severity of disease - refer to text	Post-exposure prophylaxis may be tried with TMP-SMX	No large therapeutic human trials have been conducted owing to the rarity of naturally occurring disease.
Plague	Streptomycin 30 mg/kg/d IM in 2 divided doses x 10-14 d or Gentamicin 5mg/kg IM or IV once daily x 10-14 d or Ciprofloxacin 400mg IV Q12H until clinically improved then 750 mg PO BID for total of 10-14 d	Doxycycline 100 mg PO BID x 7 d or duration of exposure Ciprofloxacin 500 mg PO BID x 7 d	Chloramphenicol for plague meningitis is required 25 mg/kg IV, then 15 mg/kg QID x 14d
	Doxycycline 200 mg IV then 100 mg IV BID, until clinically improved then 100mg PO BID for total of 10-14 d	Tetracycline 500 mg PO QID x 7 d	Alternate Rx: trimethoprim-sulfamethoxazole

DISEASE	CHEMOTHERAPY (Rx)	CHEMOPROPHYLAXIS (Px)	COMMENTS
Brucellosis	Doxycycline 200 mg/d PO plus rifampin 600 mg/d PO x 6 wks	Doxycycline 200 mg/d PO plus rifampin 600 mg/d PO x 6 wks	Trimethoprim-sulfamethoxazole may be substituted for rifampin; however, relapse may reach 30%
	Ofloxacin 400/rifampin 600 mg/d PO x 6 wks		
Tularemia	Streptomycin 7.5-10 mg/kg IM BID x 10-14 d	Doxycycline 100 mg PO BID x 14 d	
	Gentamicin 3-5 mg/kg/d IV x 10-14 d	Tetracycline 500 mg PO QID x 14 d	
	Ciprofloxacin 400 mg IV Q12H until improved, then 500 mg PO Q12H for total of 10 - 14 d	Ciprofloxacin 500 mg PO Q12H x 14 d	
	Ciprofloxacin 750 mg PO Q12H for 10 - 14 d		
Viral encephalitides	Supportive therapy: analgesics and anticonvulsants prn	NA	
Viral Hemorrhagic Fevers	Ribavirin (CCHF/Lassa) (IND) 30 mg/kg IV initial dose; then 16 mg/kg IV Q6 H x 4 d; then 8 mg/kg IV Q8H x 6 d Passive antibody for AHF, BHF, Lassa fever, and CCHF	NA	Aggressive supportive care and management of hypotension very important
Smallpox	No current Rx other than supportive; Cidofovir (effective in vitro); animal studies ongoing	Vaccinia immune globulin 0.6 mL/kg IM (within 3 d of exposure, best within 24 h)	Pre and post exposure vaccination recommended if > 3 years since last vaccine
Botulism	DOD heptavalent equine despeciated antitoxin for serotypes A-G (IND): 1 vial (10 mL) IV CDC trivalent equine antitoxin for serotypes A, B, E (licensed)	NA NA	Skin test for hypersensitivity before equine antitoxin administration
Staphylococcus Enterotoxin B	Ventilatory support for inhalation exposure	NA	
Ricin	Inhalation: supportive therapy G-I : gastric lavage, superactivated charcoal, cathartics	NA	
T-2 Mycotoxins		Decontamination of clothing and skin	

INTENTIONALLY LEFT BLANK

PART THREE: EXPOSURE TO CHEMICAL WARFARE AGENTS

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CHLORINE

General: Chlorine is found as a greenish-yellow gas. There is a pungent, acrid, characteristic odor. Sensitivity to the odor is below toxic levels; however, since some sensory adaptation occurs, repeat exposures are more likely to produce toxic effects. Chlorine affects both the central (conducting) and distal (respiratory) surfaces. Exposures irritate eyes and central (upper) airways within minutes. Low doses produce some cough and choking sensation. Moderate doses also produce a sense of suffocation, hoarseness, and substernal pain. High doses also produce a severe dyspnea, with pulmonary edema, nausea, vomiting, headache, syncope also seen. Very high doses may produce sudden death without obvious pulmonary lesions—possibly via laryngospasm. All recognized exposures should be referred for direct observation/care.

Patient Evaluation:

- Victim should be immediately removed from the toxic environment by fully masked personnel. The M40 mask is insufficient respiratory protection at the point of release where chlorine concentrations may be very high. Chemically protective clothing is required for liquid/solution exposures.
- Liquid contamination causes eye and skin burns on contact. Contaminated clothing should be removed/disposed of.

Treatment:

- General: Persons with inhalational exposures must be put at immediate rest. Exertion increases the extent of injury and hastens the onset of pulmonary edema. Symptoms of trouble breathing are experienced before signs manifest. Casualties must be observed for 6 hours after exposure before they can be cleared for discharge.
- Eyes: Liquid exposures should be flushed with copious quantities of water; medical attention should be sought. Vapor exposures, if symptomatic, should be flushed with water. Medical attention should be sought if symptomatic.
- Skin: Liquid exposures should be flushed with copious quantities of water. Contaminated clothing should be removed/disposed of. Vapor exposures require no specific therapy unless symptomatic. Intense exposure produces burns; wash with water.

- **Breathing:** Symptoms of noncardiogenic pulmonary edema precede signs. Patients must be put at rest in order to minimize the degree of injury. Patients who demonstrate trouble breathing at rest, particularly with audible crackles or diminished breath sounds, in 4 hours or less after exposure are severely injured and must be evacuated for hospital level respiratory care. Evaluate respiration, cyanosis, and bronchospasm. Bronchospasm should be treated as asthma with B-agonists and bronchodilators.

If apnea: CPR with intubation. Be aware that laryngospasm may be present with intense exposures, hence intubation may be very difficult and tracheostomy could be required. Medical attention should be sought.

If stridorous/hoarse: Consider intubation under direct vision since laryngospasm may be imminent (see above). Medical attention should be sought.

If dyspnea/cough/chest tightness: Consider intubation for impending pulmonary edema. Also consider possible bronchospasm sufficiently severe to have so little air exchange that wheezes are absent. Medical attention should be sought. Codeine-containing demulcents may help. Be wary of sedation. Oxygen therapy with positive pressure ventilation and PEEP are appropriate for severe pulmonary edema.

Fluid management: Patients may become hypotensive due to fluid shifts from the vasculature to the lungs. Rehydration using appropriate solutions is advisable should hypotension develop.

If bronchospasm: Provide aggressive bronchodilation:

Adult:

Inhaled albuterol: unit dose Q2H.

Steroids: methylprednisolone, load 120 mg, then 60 mg Q6H.

Theophylline: load 150 mg, then 30 mg/hr.

If asymptomatic: Maintain direct observation for at least 1 hour.

If becomes symptomatic, treat as above.

If still asymptomatic, lesser observation for additional 6 hours, since some bronchospasm may appear late.

If hypoxic from bronchospasm: bronchodilators and supplemental oxygen (oxygen may be utilized with positive pressure; e.g., PEEP 5-7 cm or intubation).

If pulmonary edema (occurs with moderate to severe exposures): Treat as noncardiac pulmonary edema (ARDS) with PEEP 5-7 cm and/or intubation. Control hypotension with fluids, not vasoactive drugs. Diuretic therapy risks severe hypotension if intubation is required.

If infection: Inhalational exposures may produce pulmonary infiltrates, fever, and white blood cell elevations leading to an erroneous diagnosis of (presumed bacterial) pneumonia. Prophylactic antibiotics are not indicated. Surveillance bacteriologic cultures are obtained anticipating an approximate 50% risk of nosocomial pneumonia at days 3-6.

If pain: Airway discomfort may benefit from codeine. Be wary of sedation.

HYDROGEN CYANIDE AND CYANOGEN CHLORIDE

3

General:

- Rescuers must don protective mask and clothing prior to rescuing the casualty. Cyanides are dermally active.
- Patient should be removed from the toxic environment immediately. Their clothing (underwear included) should be removed (vapor or liquid exposure).
- Clothing should be removed. If liquid cyanide is the contaminant, wash the patient with water while instituting medical measures.
- The effects of severe vapor exposure from either form of cyanide appear within seconds to a minute. If vapor exposed person has no or only mild effects when seen 5-30 minutes after exposure, he/she will need no treatment. Liquid exposed persons may take 30-60 minutes to manifest even a severe poisoning, longer for a milder poisoning.
- Clues to cyanide intoxication include: respiratory depression (cyanide stops the central respiratory center and apnea ensues rapidly), unconsciousness, convulsions, pink color, almond smell on the breath, and history of exposure. Cyanide intoxications can be difficult to distinguish from nerve agent intoxications. Hydrogen sulfide (rotten egg smell) is clinically and therapeutically identical.
- Severe cyanide poisoning produces metabolic acidosis. If cyanide poisoning is suspected in a patient who does not have moderate or severe acidosis, treatment for cyanide poisoning should not be delayed, but the diagnosis should be reconsidered.

Patient Evaluation: level of consciousness, respiratory rate, heart rate, convulsive activity.

- Exposure to a high concentration: transient hyperpnea, followed by convulsions (30 seconds after exposure), gradual decrease in respiratory rate and depth to apnea (3-5 minutes) and cessation of cardiac activity (5-8 minutes).

- Exposure to lower concentration: flushing, headache, anxiety, agitation, vertigo, feeling of weakness, nausea, muscular trembling (cyanogen chloride may cause bronchospasm/laryngospasm and irritation of eyes, nose, and airway). Prolonged exposure may lead to effects listed above.
- Odor of bitter almonds may be detected (half of the population cannot smell this); normal pupils (may be dilated in terminal stage); “cherry-red” skin (may not be present); diaphoresis; venules in fundus are same color as arterioles; cyanosis occurs only after circulatory collapse and apnea.

Treatment:

- For a mild exposure (conscious and breathing): remove from further exposure and observe; no antidotes; oxygen may be given to young or old or in presence of heart disease in a patient with mild symptoms.
- Severe exposure (unconscious, decreased respiratory rate, and acidotic): immediately receive 100% oxygen and CPR as needed. Antidotes should be administered as soon as possible (see below). It is important to note that pulse oximeter results are completely unreliable in the setting of methemoglobinemia, which is induced by amyl nitrite or sodium nitrite therapy. Rapid initiation of therapy is crucial.
- Cyanide antidotes are packaged in kits containing methemoglobin formers (amyl and sodium nitrite) and sulfur donor (thiosulfate). Kit contents are appropriate as packaged for adults. Antidote is used for severely affected persons but not for mildly affected persons.
- If casualty is seriously affected, and IV/IO line is not immediately available, use amyl nitrite ampules while establishing access. Crush the ampule for casualty to inhale. It may be wrapped in gauze and inserted into a nostril, put into the ventilation mask, or inserted into the ET tube. Monitor for hypotension and temporarily discontinue use if excessive. Once blood pressure responds, restart the nitrite (ampule or IV). Amyl nitrite is irritating to naïve users and as they recover they will push it away.

- Administer 300 mg (10 mL) of sodium nitrite IV over 5 minutes. Flush line. Be aware: Nitrites produce orthostatic hypertension, but a patient who can stand does not need them. *If there is a chance of serious oxygen carrying disability, such as carboxyhemoglobinemia or methemoglobinemia from another exposure, it may be best to skip the nitrite administration and go directly to IV thiosulphate administration.* Use care giving nitrite in a patient with hypertension or heart disease. [Amyl nitrite*, sodium nitrite, and sodium thiosulfate are in the Taylor (formerly Pasadena or Lilly) Cyanide Antidote Kit, the latter two in ampules of 300 mg/10 mL and 12.5 gm/50 mL].
- Follow with 12.5 gm (50 mL) of sodium thiosulfate IV over 20 minutes. Too rapid infusion causes vomiting.
 - ◆ If initial dose is ineffective, re-dose with one-half the initial dose. It is best to first ensure methemoglobin levels are well under 25%. Use one-half dose in 20 minutes if no improvement. See instructions on top of Antidote Kit box. Clinical response to antidote therapy is usually rapid (under 5 minutes).
 - ◆ Seizing patients should receive anticonvulsant therapy with benzodiazepines such as the CANA, lorazepam, midazolam, etc.
 - ◆ The earliest signs of recovery include reversal of acidosis toward normal (indicating resumption of aerobic metabolism), and stabilization of blood pressure. Alertness may take several hours or days to recover.
- If patient continues to remain apneic, intubate and continue oxygen through tube with assisted ventilation.
- Transfer apneic or unconscious patients to medical facility.
- Patients often recover rapidly unless CNS hypoxia has occurred. Headache from the nitrites responds to acetaminophen (Tylenol). Hypoxic injury to central or peripheral nerves may not be evident for several weeks to months.

Laboratory Issues:

- Metabolic acidosis is common. Sodium bicarbonate IV may be helpful.
- Monitor arterial pO_2 ; should be normal until near-terminal stage.

* For all ages, crush amyl nitrite ampule and allow it to be inhaled for up to 3 minutes. If patient is endotracheally intubated, place ampule or some of its contents in the large end of the ET tube where it connects to the Ambu bag or the ventilator. If amyl nitrite use is to continue beyond 3 minutes, use a new vial approximately every 3 minutes until patient recovers or until sodium nitrite can be administered. Once venous access is established and sodium nitrite is available, administer sodium nitrite and discontinue use of amyl nitrite as soon as possible.

MUSTARD (SULFUR MUSTARD)

3

General:

- The initial clinical effects of mustard (which usually involve the eyes, the skin, and the airways) appear 2-24 hours (usually 4-8 hours) after exposure to liquid mustard or to mustard vapor. However, liquid or vapor mustard penetrates the skin and mucous membranes and damages cells within minutes of exposure, so decontamination must be done immediately after exposure.
- The patient should be immediately removed from the toxic environment.
- *Physical removal by the fastest available method, even with just copious amounts of water, is far better than wasting time to prepare a special mixture.* Clothing must be removed, and skin decontaminated with whatever is immediately available that is clean. The best combination is a dry absorbent plus liquid using mild friction. The M291 kit followed by a wet wipe is good for gross decontamination. Warm soapy water followed by warm water rinse is excellent. 0.5% hypochlorite (1 part household bleach mixed with 9 parts water), soap and cool water, or thoroughly flushed with water alone are acceptable alternatives. Other options include bread plus wet wipes and 10% dilute solution of bleach water. Full strength bleach must not be used for it will enhance mustard penetration and further injure the skin. Eyes should be flushed with large amounts of saline. If exposure is to vapor alone, remove clothing and thoroughly wash as above.
- If there is a history of definite exposure and, either the patient becomes symptomatic OR later evacuation might become impossible, consider evacuation to a medical facility for observation and indicated treatment.

Patient Evaluation: Initial effects (usually 2-24 hours after exposure):

- Eyes: irritation, feeling of grit in eye, redness. First affected organ. Severe conjunctivitis with massive lid swelling may ensue. Duration: often 2 weeks or longer. Severe eye injury may result in globe perforation or corneal destruction resulting in permanent blindness.

- GI: expect patients to vomit. Vomiting starts 3-5 hours after significant systemic exposure. The vomiting is not stopped with anti-emetics. It will stop in time. If vomiting occurs, this patient is required to have a baseline and 24 hour post-exposure monitoring for serious bone marrow effects (see below).
- Skin: erythema and itching (will progress to blisters 1-4 hours later if exposure was large enough).
- Airways: irritation of nose, voice change, sinus pain, hacking cough. (Very rarely a patient might inhale an extremely large amount and start to have these effects plus dyspnea within 2 hours. This patient should be intubated, and assisted ventilation with oxygen should be started. This patient should be evacuated to an intensive care unit as quickly as possible.)

Treatment:

- There is nothing to do for these patients until effects appear except to decontaminate. Tissue is damaged within minutes, so decontamination must be done immediately.
- Eyes: Acute mustard (both vapor and liquid) casualties should ALWAYS have their eyes copiously irrigated with a sterile solution PRIOR to administration of any additional ophthalmic solutions or medications. *If copious irrigation is not done initially, it may well not be possible later if the eyelids have swollen shut over the eye.* Any commercial eye solution may relieve the irritation from a mild exposure. If significant eye exposure is expected, initiate topical and/or systemic steroid and antibiotic therapy immediately, redosing frequently (every 15 minutes for topical) for the first day with earliest evacuation to ophthalmic specialist. More severe effects: a mydriatic BID or QID (depending on the length of action of the drug) may provide pain relief; vaseline on lid edges BID; sunglasses if photophobia is present. Control pain with systemic, not topical, analgesics. Visual loss is usually due to lid edema and blepharospasm, not eye damage.
- Skin: A soothing lotion (e.g., calamine) for erythema and itching. Leave small blisters intact. Do not unroof blisters of any size until it can be done under sterile conditions (Echelon III). If blisters are flaccid, debride and redress with topical antibiotic. It is very critical to avoid infection as it will increase the depth of injury. Any burns on the face,

joints, in the genital area, or expanding over large area require earliest evacuation. Watch for infection. Fluid requirements are much less than those for thermal burns; do not overhydrate. Give full dose Motrin or other NSAID and apply ice packs liberally (1-2 hours contact time) to diminish the inflammatory reaction.

- Airways: Steam inhalation, mucomyst, and cough suppressants will generally relieve mild airway symptoms. A chemical pneumonitis (increased temperature, WBC; chest x-ray findings) may develop after large exposure: intubation, assisted ventilation with oxygen (and possibly with PEEP or CPAP); bronchodilators; watch sputum at least daily for organisms (no antibiotics until organism is identified). Secondary pneumonia typically takes 2-5 days to develop.
- Systemic absorption of a large amount of mustard may cause bone marrow and gastrointestinal tract damage. Monitor lymphocyte (not total WBC) counts of moderately to severely intoxicated patients (vomiting or burns severe enough to require medical care) every 12-24 hours starting as soon as possible. Lymphocyte drops of 50% within the first 24-36 hours predict bone marrow failure and those patients should be evacuated for definitive management. Within about 5 days they may become granulocytopenic and septic, and within 7 days they may bleed from low platelet counts. Administration of granulocyte colony stimulating factor (GCSF) within 1-3 days will reduce the magnitude of granulocytopenia and shorten its duration. Granulocyte counts initially rise rendering the total WBC count less valuable.

NERVE AGENTS (GA, GB, GD, GF, VX)

General: Nerve agents are extremely toxic chemicals that cause effects by inhibiting the enzyme acetylcholinesterase, allowing excess acetylcholine to accumulate. This excess neurotransmitter then produces overstimulation and causes hyperactivity in muscles, glands, and nerves. The nerve agents are GA (tabun), GB (sarin), GD (soman), GF, and VX. Their routes of exposures are skin, respiratory system, and GI.

Protect yourself. Remove patient from contaminated atmosphere. *Physical removal by the fastest available method, even with just copious amounts of water, is far better than wasting time to prepare a dilute bleach solution.* If liquid contact, clothing should be removed and skin decontaminated with whatever is immediately available that is clean. Use friction plus a liquid. The M291 kit followed by a wet wipe is good for gross decontamination. Warm soapy water followed by warm water rinse is excellent. 0.5% hypochlorite (1 part household bleach mixed with 9 parts water), soap and cool water, or thoroughly flushed with water alone are acceptable alternatives. Full strength bleach must not be used for it will enhance mustard penetration and further injure the skin. Eyes should be flushed with large amounts of saline. If exposure is to vapor alone, remove clothing and thoroughly wash as above. All casualties require complete nakedness and full washing to protect the treating personnel.

Patient Evaluation: If conscious, note ventilatory status and ask about nausea. If unconscious, note ventilatory status and heart rate (heart rate may be high, low, or normal in a nerve agent casualty). Initial effects differ depending on whether exposure was to vapor or to liquid.

- Vapor: Effects start within seconds to a minute or two.
 - ◆ Mild to moderate: Miosis (possible redness in eye, eye pain, and complaints of dim or blurred vision, nausea), rhinorrhea, excess secretions, dyspnea (mild to severe). Nerve agents cause severe bronchospasm and bronchorrhea, the worst conceivable asthma attack.

- ◆ Severe: Include all the effects from mild exposure PLUS loss of consciousness, seizures, apnea, flaccid paralysis; systemic distribution is indicated by affect on two or more body systems.
- Liquid: Effects start in minutes (large exposure) to 18 hours (small exposure) after an asymptomatic interval. Miosis may be absent.
 - ◆ Mild to moderate: Sweating and fasciculations at site of exposure; nausea, vomiting, diarrhea; weakness.
 - ◆ Severe: Same as for vapor, but after a 1- to 30-minute asymptomatic interval.

Treatment:

- Initial management.
 - ◆ Mild to moderate: Dyspnea should be treated with one or two doses of atropine IM or IV and one dose of pralidoxime (IV drip) initially, depending on severity of the dyspnea. See “Recommended adult doses” below for size of dose. This should be supplemented with oxygen, if available. Atropine dose should be repeated at 5- to 10-minute intervals until improvement is noted. Failure to respond (i.e., no dry mouth, no decrease in secretions) confirms the need to administer additional doses of atropine. Gastrointestinal effects after liquid exposure are treated in the same manner. Do not treat for miosis (unless eye pain is severe) or rhinorrhea (unless severe).
FOR SELF-CARE OR BUDDY CARE.
 - Mild exposure: no treatment required.
 - Moderate exposure with moderate respiratory symptoms: one to two MARK 1 kits. Repeat atropine alone every 3-5 minutes until respiratory symptoms are relieved.
 - Severe exposure or severe symptoms: three MARK 1 kits and one CANA (diazepam). If already seizing, will need three to four CANA. Repeat atropine alone every 3-5 minutes until respiratory symptoms are relieved. Repeat three MARK 1's in 1 hour if needed. Titrate atropine to respiratory effect. Do not give more than three 2-PAM per hour and do not give more than six

unless BP can be monitored. It can cause hypertension. Treat convulsions with benzodiazapines. Other anticonvulsants do not work.

- *NOTE: Military doctrine advises diazepam (CANA) administration for any nonseizing patient with severe nerve agent poisoning. In both field and remote-site clinical settings, it is difficult to differentiate between grossly visible convulsions and electrical seizing which can appear either as flaccid paralysis (due to nonavailability of ATP) or as a post-ictal state; therefore, diazepam (CANA) must be administered in ALL of these situations.*

- ◆ Severe: Administer three doses of atropine IM (not IV in hypoxic patient) and start one dose of pralidoxime by slow IV drip over 20 minutes. (More rapid administration will cause hypertension.) See “Recommended adult doses” below for size of dose. Intubate and ventilate with oxygen. Initial ventilation will be difficult because of airway resistance; atropine will relieve this. Administer diazepam if convulsing. Suction for secretions. Repeat one dose of atropine (IM until hypoxia is improved, then IV) every 5 minutes until:

- secretions diminish or
- airway resistance is less or is normal.

Failure to respond (i.e., no dry mouth, no decrease in secretions) confirms the need to administer additional doses of atropine. Monitor via pulse oximeter; cardiac monitoring should also be done. Cardiac arrhythmias are uncommon after atropine is given. Acidosis may develop after seizures or after period of hypoxia and will require therapy. Patient should be medically evacuated to a hospital after stabilization (adequate drug therapy and initiation of ventilation).

- ◆ Eyes: Do not treat miosis unless eye/head pain is severe. Use topical, not systemic, anticholinergic to relieve pain. Papillary constriction may persist for up to 2 months.

- Recommended adult doses:

Atropine: 2 mg

Pralidoxime: 1 gram

Pralidoxime can cause hypertension when given rapidly IV. Slow administration over 20 minutes will minimize the hypertension effect. After rapid administration, hypertension can be rapidly but transiently reversed by phentolamine (adult: 5 mg IV). Pralidoxime is also available for IM administration as an autoinjector formulation (600 mg autoinjector) to be repeated twice at 15-minute intervals.

- Further care.

- ◆ Give atropine repetitively until the patient is breathing comfortably on his/her own and respiratory secretions are no longer excessive. In a liquid exposure, this may require treatment for hours or even days.
- ◆ Mild to moderate: After vapor exposure, a patient who is breathing normally does not need to be hospitalized as he will not worsen. However, miosis should be followed until eyes are normal (4-6 weeks). After liquid exposure, a patient should be observed in hospital for 18 hours until all agent is absorbed from skin.
- ◆ Severe: Continue to ventilate and to administer atropine following guidelines above. Treat acidosis if present. If patient has not had prolonged hypoxia, recovery of an unconscious patient will be gradual over 1-3 hours.

PHOSGENE - CARBONYL CHLORIDE

3

General: Will only be encountered on the battlefield in the gaseous state. Even a terrorist release will produce a gas. Phosgene is a colorless-to-white gas and has an odor of newly-mown or moldy hay. Sensitivity to the odor may degrade, making individuals unaware of toxic inhalation. Lethal inhalations may be asymptomatic at the time of exposure, although very high-intensity exposure irritates eyes and upper airways within minutes. Symptoms of severe intoxication appear in 2–4 hours. Less severe injury may take more than 4 hours to manifest. Phosgene is a peripheral acting pulmonary intoxicant and causes noncardiogenic pulmonary edema. Development of pulmonary edema within 4 hours of exposure is a very poor prognostic sign.

Patient Evaluation:

- Victim should be immediately removed from the toxic environment by fully masked personnel (full-face positive pressure apparatus).

Treatment: Maintain at rest at least 6 hours.

- Eyes: Gas exposures, if symptomatic, should be flushed with water.
- Skin: Gas exposures require no specific therapy unless symptomatic.
- Breathing: symptoms precede signs. Casualties may first complain of dyspnea on exertion. Evaluate respiration, cyanosis. Oxygen always used –

If apneic: CPR with intubation. Be aware that laryngospasm may be present with intense exposures; hence, intubation may be very difficult and tracheostomy required.

If stridorous/hoarse: Consider intubation under direct vision since laryngospasm may be imminent (see above).

If dyspnea/cough/chest tightness: Consider intubation for impending pulmonary edema.

Also consider possible bronchospasm sufficiently severe to have so little air exchange that wheezes are absent. Codeine-containing demulcents may help. Be wary of sedation.

Note: cough may presage pulmonary edema.

If bronchospasm: Individuals with underlying asthma may suffer bronchospasm. Treat as any asthmatic: Inhaled albuterol, parenteral steroids, theophylline. Watch for hypoxia.

Adult:

Inhaled albuterol: unit dose Q2H.

Steroids: methylprednisolone, load 120 mg, then 60 mg Q6H.

Theophylline: load 150 mg, then 30 mg/hr.

If asymptomatic: Maintain direct observation for at least 6 hours;

If becomes symptomatic, treat as above.

If still asymptomatic, lesser observation for additional 36 hours.

If hypotensive (will occur rapidly with pulmonary edema):

Immediate volume replacement should be undertaken. Colloid or crystalloid may be used to maintain adequate tissue perfusion.

If infection: Inhalational exposures may produce pulmonary infiltrates, fever, and WBC elevations—leading to an erroneous diagnosis of (presumed bacterial) pneumonia. Prophylactic antibiotics are not indicated. Surveillance bacteriologic cultures are obtained anticipating an approximate 50% risk of nosocomial pneumonia at days 3-6.

If hypoxia: Commonly from pulmonary edema, treat as above; occasionally from bronchospasm, treat as above.

If pain: Airway discomfort may benefit from codeine. Be wary of sedation.

DOSAGES FOR CHEMICAL AGENT TREATMENTS

<u>Drug</u>	<u>Adult Dose</u> <u>[> 50 kg (110 lb)]</u>
ATROPINE at 0.1 mg/mL drug concentration (0.02 mg/kg Pediatric, 2 mg adult)	20 mL
ATROPINE at 0.4 mg/mL drug concentration (0.02 mg/kg Pediatric, 2 mg adult)	5 mL
ATROPINE at 1 mg/mL drug concentration (0.02 mg/kg Pediatric, 2 mg adult)	2 mL
ATROPINE at 2 mg/mL drug concentration (0.02 mg/kg Pediatric, 2 mg adult)	1 mL
PRALIDOXIME (2-PAM, Protopam®) at 50 mg/mL (for IV use) (50 mg/kg Pediatric, 1000 mg adult)	20 mL
PRALIDOXIME (2-PAM, Protopam) at 300 mg/mL (for IM use) (40 mg/kg Pediatric, 600 mg adult) (Reconstitute by adding 3 mL sterile water to a 1 g vial of pralidoxime)	20 mL
SODIUM NITRITE at 3% (300 mg/ 10 mL) (Pediatric 0.3 mL/kg for Hgb 11 g/dL, adult 10 mL)	10 mL
SODIUM THIOSULFATE at 25% concentration (Pediatric 1.65 mL/kg, adult 50 mL)	

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PART FOUR: TOXIC INDUSTRIAL CHEMICALS (TICs)

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BASIC PRINCIPLES

- TICs are routinely used at many different types of work-places, including industrial sites. They are often stored in bulk and transported (e.g., trucks and railcars) through populated areas. Workers can be occupationally exposed if proper controls are not in place. In addition, when there are accidental releases of large amounts of TICs as a result of explosion or fire (products of combustion), or when they are used as “agents of opportunity” by terrorists, they can cause catastrophic injuries and many deaths in surrounding areas.
- Uncontrolled or poorly controlled occupational exposures in deployed military personnel, and in specific occupational groups, is quite possible. The resulting health effects may not be clearly linked with such exposures unless an event (fire, ruptured tanks, etc.) occurs that makes the possibility of exposure evident. Based on the presenting clinical signs or symptoms, health care practitioner (HCP) should remember to inquire about the possibility of occupational exposures to TICs to their patients and to others they work with. The “Chemical Exposure Questionnaire” located at the end of Part Four should be used as a screening tool for patients whose complaints or clinical signs are possibly related to an occupational or environmental exposure during deployment. The practitioner should attempt to link positive answers to the questions with signs and symptoms found during the clinical encounter, thus prompting any appropriate interventions. Known exposures should be documented in the medical record.
- When a TIC release is either accidental or due to a terrorist or warfare event, and casualties are obvious, it is not always practical to consider every specific chemical that may have been present for early casualty management. One can, however, consider major classes of chemicals that were likely and how they are expected to affect the exposed population, thus guiding general treatment measures. This part of the TG is organized around classes of chemicals and their toxic syndromes (“toxidromes”).
 - ◆ Toxidrome classes include irritant gases, corrosives, asphyxiants, organic vapors (hydrocarbons and chlorinated hydrocarbons), and cholinergics.

- ◆ The presenting symptoms and initial treatments for chemicals within a toxidrome are similar. Some toxic chemicals will fit into more than one toxidrome, depending on effects or physical state. As noted below, many toxic chemicals a practitioner may encounter in his/her patients' exposures do not fall into any of these toxidromes. This is simply a tool for early casualty management of the many that do.
- ◆ The importance of these toxidromes and specific agents lies in their characteristics of acute, high toxicity, exposure potential (appreciable vapor pressures, airborne route), and availability (widely used in commerce with high production volumes).
- ◆ Inhalation and skin exposures are emphasized, as most chemical accidents would not involve ingestion. However, terrorists can potentially contaminate food and water supplies intentionally in an attack.
- ◆ The Table (Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure), located at the end of Part Four, summarizes essential information for some common acutely toxic industrial chemicals (TICs) from inhalation exposure. Its use is intended to assist practitioners to recognize and characterize the exposure and its severity as well as provide expedient treatment and protection. Acute health effects are discussed in this table, but not more chronic effects such as potential for cancer or teratogenicity.
- ◆ A few chemicals have specific life-saving antidotes which will be presented in this chapter.
- ◆ There are chemicals which fit into the toxidromes for initial treatment but in addition also have unique toxicities (e.g., systemic toxicities). Some of these will be pointed out, but in general, the HCP should look up information on the specific chemicals as soon as time permits after the crisis is over.
- Products of combustion contain a mixture of many unknown and toxic chemicals and are commonly encountered in a wartime/deployed situation. Important toxidrome categories to consider in combustion products are irritant gases and asphyxiants.

- There are many other TICs that military personnel could be exposed to besides those that fit into the toxidromes and into this TG. Specific chemicals can be looked up in various resources available on-line, or USACHPPM can assist in characterizing significant exposures in military populations as needed.
- Exposure incidents are often complicated by concurrent injuries such as thermal burns of the skin, mucous membranes and lungs, and trauma from projectiles and the force of explosions. Radioactive releases should also be considered and looked for, although, in general, early treatment of radioactive exposure is secondary to treatment of injuries from trauma and chemical exposures.
- Other important basic principles to bear in mind:
 - ◆ The incident site should be organized to provide immediate decontamination, triage and care for injured persons, but at the same time protect the rescuers from exposure/injury themselves.
 - Organize control zones (“hot,” “warm,” “cold” zones).
 - Firefighters and other rescuers should wear protective equipment to avoid becoming casualties themselves.
 - Except for immediate life-saving procedures (the ABCs), decontamination of chemically contaminated patients is the priority in the warm zone. (Removal of contamination prevents further absorption, as well as secondary contamination of others.)
 - Finish the primary survey (ABCDE), resuscitate, triage and transport in the cold zone.
 - ◆ Characterize what happened, types of exposure, and document who the exposed personnel are as soon as possible.
- Acute and chronic effects can vary among different sub-populations (e.g., occupational groups, including military personnel, are typically healthier than many of the local residents who may have more preexisting sensitivities/susceptibilities).

- In deployed settings, the potential significance or severity of chemical exposures can be evaluated and expressed to commanders through the Operational Risk Management Process and use of USACHPPM TG 230. Vulnerabilities should be addressed. Should an attack or accident occur, the Military Exposure Guidelines (MEGs) in TG 230 can provide some context to understand the actual exposure levels experienced but should not be the only resource consulted to evaluate the potential for short- or long-term effects of an exposure. USACHPPM is available to assist in this evaluation as needed.

IRRITANT GASES

- Highly water soluble irritant gases include ammonia, sulfur dioxide, hydrogen chloride, and formaldehyde. Intermediate water soluble irritant gases include chlorine, hydrogen sulfide and acrolein. Lower water soluble irritant gases include oxides of nitrogen, phosgene, and ozone.

Toxidrome: irritation, inflammation, edema, and possibly chemical burns of the exposed mucous membranes, airways, and lungs.

- It is difficult to determine if a substance is a corrosive or irritant in a particular concentration or circumstance.
- Irritants are substances that cause inflammation and swelling, but not cellular death and tissue damage. A corrosive would cause cellular damage and death.

Acute Effects:

- Throat, eye, nose or skin irritation (rhinorrhea, lacrimation), choking feeling, burning feeling, coughing, tachypnea, and wheezing.
- Headache, and nasal dryness and hemorrhage.
- Skin: Redness, swelling, and pain may occur.
- To determine potential severity of the exposure, characterize the circumstances (e.g., what particular substance was involved, duration of exposure, determination of if exposure occurred in a confined space, and if the patient had a loss of consciousness).
- Highly water soluble irritant gases cause immediate irritation of the upper respiratory mucosa. Most people will find this unpleasant enough to get away before great damage is done. However, if they are trapped and must breathe or be in contact with high concentrations for long enough periods, damage will occur. Eyes and skin can burn and necrose. Upper airway will be affected most, but prolonged high concentration will affect lower airways as well. Patient may have trouble with breathing and oxygen absorption, hyperreactive airways (asthma), glottic and esophageal edema, hoarseness, stridor and laryngospasm (danger of airway obstruction). Development of pulmonary edema may be delayed by 1-3 days. Circulatory failure can occur in severe cases.

- Gases with lower water solubility can injure the lower airways and alveoli, with pulmonary edema that may be delayed up to 72 hours. Exposure may not manifest immediately with symptomatic upper respiratory tract irritation, so the recipient may not try to escape—thus increasing exposure. Hypoxemia may result.
- Gases with intermediate water solubility cause damage throughout the respiratory tract and have intermediate tendency to irritate the upper airway.
- Most irritant gases will exert their effects at the point of contact and not systemically, unless very severe. However, one must be alert to possible systemic effects.
- Cardiovascular (e.g., tachycardia, myocardial ischemia) and nervous system (e.g., anxiety, agitation, confusion, seizures, decreased level of consciousness) effects will likely be due to the hypoxemia due to respiratory system damage (e.g., pulmonary edema).

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Chronic Effects:

- Reactive airways dysfunction syndrome (RADS) is persistent asthma after irritant exposure:
 - ◆ Onset within 24 hours of high-level irritant exposure;
 - ◆ Positive methacholine challenge test;
 - ◆ Persistence of respiratory symptoms and airway hyperreactivity for at least 3 months;
 - ◆ Symptoms simulating asthma, with cough, wheezing, and dyspnea; and
 - ◆ Airflow obstruction as measured by pulmonary function tests.
- Skin: Allergic contact dermatitis may arise after repeated exposure to irritants.

Treatment:

- Using wide-range pH paper test for pH of irritant substance if possible, and pH of ocular cul de sac.
- Move patient to fresh air. Monitor for respiratory distress. If symptoms not relieved by exposure to fresh air, administer oxygen until blood gases can be measured. Use 100% humidified supplemental oxygen with assisted ventilation as needed if symptoms are severe or prolonged. Consider early intubation if there is impending upper airway obstruction.

- Decontamination by diluting and irrigating is critical.
 - ◆ Copiously flush exposed skin with water, and wash with soap and water.
 - ◆ Irrigate exposed eyes with copious amounts of tepid water or sterile saline, initially for at least 15 minutes at the scene, and then in the MTF until the lower lid cul-de-sac is returned to neutrality.
 - ◆ Some alkali exposures may require prolonged irrigation.
 - ◆ Application of an ophthalmic local anesthetic will increase patient comfort.
 - ◆ If irritation, pain, swelling, lacrimation, or photophobia persist, an ophthalmologic examination should be performed including a slit lamp exam after thorough irrigation.
 - ◆ Administration of topical antibiotics, cycloplegics, mydriatics, and patching may be necessary in rare instances of abrasion.
- Support respiratory and cardiovascular function. If cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Carefully observe patients with inhalation exposure for the development of any systemic signs or symptoms and administer symptomatic treatment as necessary. Especially with low solubility irritant exposures, if there is evidence of pulmonary edema within 4 hours, intensive care will likely be needed. If no symptoms are observed within 8 hours and the CXR is normal, acute lung injury is unlikely to develop. If acute lung injury is possible, the patient must have strict bedrest without exertion. Physical exertion can exacerbate the severity of the lung inflammation.
- Evacuate patients with moderate symptoms, chest signs, or impaired oxygenation. Observe for at least 48 hours if exposure has been to substances known to cause delayed onset of pulmonary symptoms. Chest x-ray may be needed. Crystalloid solutions must be administered cautiously to avoid a net positive fluid balance. The use of corticosteroids is controversial.

CORROSIVES

- Examples of corrosives include acids, bases, oxidizers, and allotropes of phosphorus. Corrosives can exist either as dusts, mists, fumes, aerosols, vapors or liquids, and cause injury either by inhalation or direct contact with skin or mucous membranes. Irritant gases, which dissolve in water to produce an acid or a base, are also considered to be potential corrosives. (Note: Ingestion is not discussed here.)
 - ◆ It is difficult to determine if a substance is a corrosive or irritant at a particular concentration.
 - ◆ Irritants are substances that cause inflammation and swelling, but not cellular death and tissue damage. A corrosive would cause cellular damage and death.
 - ◆ Examples of acids are acetic, hydrochloric, nitric, phosphoric, and sulfuric.
 - ◆ Examples of bases are ammonium, potassium, and sodium hydroxide.
 - ◆ Examples of corrosive oxidizers include chloride dioxide, hydrogen peroxide, methyl ethyl ketone peroxide, and sodium chlorate.
 - ◆ Examples of allotropes of phosphorus include black, red, and white phosphorus.

Toxidrome: local chemical burns of the skin and mucous membranes that come into contact with the corrosive.

Acute Effects:

- Swelling, redness and pain, burns at any site, especially at mucous membranes. Mouth, nose, and eyes are very susceptible. Lung damage is possible if inhaled.
- Headache, nasal dryness and hemorrhage, and/or rhinorrhea, lacrimation.
- Lung irritation symptoms include coughing, burning, and difficulty breathing.
- Upper airway signs and symptoms include dysphonia and throat tightness, which can be progressive to hoarseness, stridor, and aphonia (signals impending upper airway obstruction). The lower airway can manifest wheezing and crackles. These are manifestations of laryngospasm, bronchospasm, and edema of the upper

and lower airway (glottic, esophageal, or pulmonary edema). Irritation of the oropharynx can reflexively cause gagging, nausea, and vomiting.

- ◆ The more highly water soluble corrosives affect the upper airway more. Acids, bases, and oxidizers are generally water soluble.
- ◆ Respiratory effects depend on the size of the particles and how deeply they penetrate the lung.
- Cardiovascular effects are usually due to hypovolemia from intravascular volume depletion and third-spacing. This can lead to hypoxemia and shock, and myocardial ischemia. Oxidizers can cause actual or functional anemia due to hemolysis or methemoglobin formation if absorbed systemically (e.g., chromates, nitrates/nitrites). Tachycardia and other reflexive cardiovascular reactions would be expected.
 - ◆ Cardiac effects (e.g., pump failure or dysrhythmias) are possible with white phosphorus (WP) and hydrogen fluoride (HF) due to hypocalcemia (both) or direct cardiotoxicity (WP).
- Nervous system effects can result from hypoxemia, hypovolemia or anemia (anxiety, confusion, agitation, seizures, decreased level of consciousness, coma, death) or hypocalcemia (tetany, seizures).
- Skin and eyes: Pain and irritation, or corrosion at the burn site (acids produce a coagulation burn, bases produce a liquifactive necrosis (slippery burn or saponification)).
- Systemically absorbed WP can cause kidney or liver damage.

Chronic Effects: Scarring; RADS (see irritant gases).

Treatment:

- Move patient to fresh air. Administer 100% humidified supplemental oxygen with assisted ventilation as required. Airway intubation if needed – sooner versus later if signs of impending airway obstruction are present (see above). Cardiac monitoring or support as indicated and available.
- Decontamination by diluting and irrigating is critical.
 - ◆ Remove clothing and promptly irrigate skin with copious amounts of water.

- ◆ In contrast to thermal blisters, chemical burn blisters SHOULD be broken, as the offending chemical is usually inside.
- ◆ Exposed eyes should be irrigated with copious amounts of tepid water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist perform an ophthalmologic examination. If indicated, continue irrigation at the MTF.
- ◆ If in a medical facility, sterile saline should be used to irrigate the eyes until the lower lid cul-de-sac is returned to neutrality. Some alkali exposures may require prolonged irrigation. Application of an ophthalmic local anesthetic will increase patient comfort.
- Monitor for respiratory distress. If cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, pneumonitis and/or pulmonary edema.
- Consider and treat systemic toxicity in addition to local effects (see notes below).
- Labs as indicated and available (e.g., ABGs, CBC, electrolytes, liver or kidney function tests, calcium, or urinalysis).
- Evacuate patients with moderate symptoms, chest signs, or impaired oxygenation. Observe for at least 48 hours if exposure has been to substances known to cause delayed onset of pulmonary symptoms.
- Specific systemic toxicities and treatment:
 - ◆ Acids produce coagulative necrosis (thick and hardened surface that can somewhat limit deeper penetration). Systemic effects are possible with some acids as sometimes the anion is more toxic or injurious than the hydrogen ion. For example, hydrofluoric acid is a relatively weak acid but the fluoride ion is very toxic systemically (see below). Chromic acid is a strong acid and an oxidizer (and can cause hemolysis based on this), and can also cause chromium poisoning.
 - ◆ Bases produce liquefactive necrosis (liquefies and destroys tissue), so burns tend to penetrate deeper and be more severe; however, bases are less likely to produce systemic effects.

- ◆ Oxidizers, on the skin or on mucous membranes, oxidize cellular components indiscriminately, producing a burn with rapid tissue destruction. Mostly local effects are expected, but some such as chromates and methemoglobin formers have systemic toxicity with hemolysis or methemoglobin formation.
- For significant and symptomatic methemoglobin formation, use methylene blue (see asphyxiants). For serious hemolysis, blood transfusion and/or hemodialysis may be needed if hemolysis leads to acute renal failure (secondary to pigment release).
- White phosphorus spontaneously combusts on contact with air. It produces both chemical and thermal burns. Immersion in water stops the burning. It is unlikely to be inhaled, but its reactive combustion products, such as phosphorus pentoxide and related oxyacids, could be inhaled.
 - ◆ On the skin, it causes serious burns and systemic toxicity. It can be absorbed through the burned skin to produce kidney and/or liver damage.
 - ◆ Liver damage and even failure, and renal insufficiency and even failure, can be delayed developments.
 - ◆ Before liver or kidney damage is apparent, there can be rapid cardiovascular collapse due to hypovolemia from the burns, and direct cardiotoxicity with pump failure (cardiogenic shock) and dysrhythmias.
 - ◆ It also combines with endogenous calcium to produce hypocalcemia (can lead to prolonged QT, torsade de pointes, and negative inotropy with decreased cardiac output).
 - ◆ It is damaging to eyes and mucous membranes and can cause blindness.
- White phosphorus is often embedded in skin and should be removed. To better visualize:
 - ◆ Use a black light in a dark room (phosphoresces).
 - ◆ Apply 1-3% copper sulfate solution (color change to bluish copper phosphide). Use sparingly as systemic absorption can cause hemolysis.
 - ◆ Immerse removed WP in water to prevent spontaneous combustion.

- Hydrogen fluoride gas and hydrofluoric acid deserve special mention: the fluoride ion is very toxic systemically; it combines with endogenous calcium and magnesium to produce hypocalcemia, hypomagnesemia, and hyperkalemia (release of intracellular potassium). It is also a cytotoxicant (impairs oxidative phosphorylation and glycolysis). There can be severe burning neuropathic pain at the site of contact, twitching and tetany, nervous system irritability and seizures, and myocardial irritability with prolonged QT, decreased cardiac output, etc. Skin effects may be delayed up to 6-24 hours with more dilute solutions. Eye exposure is also very serious and could lead to blindness.
- Hydrogen fluoride and hydrofluoric acid exposure: administration of calcium may be required. A specialist should be contacted to advise and support the HCP in the procedures below.
 - ◆ Inhalation exposures: 2.5 - 5 mL of a dilute (2.5%) solution of calcium gluconate through a hand-held nebulizer. (Dilute a 10% solution 3 to 1.)
 - ◆ Topical application: 2.5-10% calcium gluconate in gel form, or undiluted 10% in a glove if hand is involved.
 - ◆ Local subcutaneous injection: calcium gluconate titrated to relieve pain with 0.5 mL per square cm of skin surface area. (DO NOT inject calcium chloride subcutaneously – tissue necrosis.)
 - ◆ IV injection for systemic poisoning: calcium gluconate 10-30 mL adult dose, titrated for effect to control cardiac and electrolyte disturbances; or calcium chloride 5-10 mL IV slowly.

ASPHYXIANTS

- Simple asphyxiants displace oxygen from the ambient atmosphere so there is less oxygen available for respiration. Examples include: acetylene, argon, butane, carbon dioxide, ethane, ethylene, helium, hydrogen, liquefied petroleum gas, methane, neon, nitrogen, propane, and propylene.
- Systemic (or tissue) asphyxiants act chemically in the body after absorption and include:
 - ◆ Methemoglobin-forming compounds which interfere with hemoglobin-mediated oxygen transport: aryl amines, organic nitro compounds, or inorganic nitrite or nitrate salts such as amyl nitrite, aniline, isobutyl nitrite, nitrobenzene, nitroglycerine, and sodium nitrite.
 - ◆ Carbon monoxide (CO) interferes with hemoglobin-mediated oxygen transport and oxygen utilization by mitochondrial cytochrome oxidase.
 - ◆ Cyanides, cyanogenic compounds (acetonitrile, acrylonitrile, cyanogens, cyanogen chloride), sulfides (such as hydrogen, potassium, and sodium sulfide), and azides (such as hydrogen, lead, and sodium azide) interfere with oxygen utilization by mitochondrial cytochrome oxidase.

Toxidrome: Inadequate oxygenation of tissues, especially those with high aerobic metabolic demand (e.g., cardiovascular and central nervous system).

Acute Effects:

- The neurologic and cardiopulmonary systems will be the first to show effects as they are the most dependent on oxygen.
 - ◆ Mild symptoms include dizziness, weakness, fatigue, headache, nausea.
 - ◆ Vision changes can include decreases in night vision, visual acuity, and visual fields (tunnel vision).
 - ◆ More severe symptoms include dyspnea, tachycardia, tachypnea, air hunger, hyperventilation, and

altered mental status (belligerence, agitation, euphoria, numbness and tingling of the extremities, sleepiness, mental confusion, memory loss, speech impairment, and decreased coordination and judgment).

- ◆ In severe cases, after initial agitation, respiratory depression and arrest may follow. There may be chest pain, palpitations, dysrhythmias, hypotension, myocardial ischemia, myocardial infarction, and eventually asystole. Neurologically there can be syncope, seizures, coma, and death.
- Skin appearances:
 - ◆ Cyanosis is not universal, as systemic asphyxiants may interfere with utilization of oxygen, but not its transport in blood, therefore keeping the blood bright red, but causing lactic acidosis (e.g., cyanide or azide poisoning).
 - ◆ Methemoglobinemia is characterized by a chocolate brown-colored blood on contact with the air. The skin appears bluish (cyanotic) when there is about 7-10% methemoglobin in the blood.
 - ◆ Skin appearance from carboxyhemoglobin (from CO exposure) does not appear cyanotic but possibly cherry red.
 - ◆ There may also be signs of increased sympathetic activity such as cool, pale, diaphoretic skin.
- Effects of lack of oxygen can be factors in impaired ability to escape from the toxic environment.
- Some agents causing asphyxia are stored and transported in compressed or liquid form and can cause frostbite on direct skin contact.

Chronic Effects: If hypoxia is severe and prolonged, potential exists for anoxic damage to various organ systems: CNS injury, eye/vision damage (mydriasis, proptosis, yellow vision, transient blindness, retinal cell damage); heart, and kidney (acute tubular necrosis, kidney failure).

Treatment:

- Removal from exposure and administration of 100% humidified oxygen is absolutely crucial, along with whatever other ventilatory or circulatory support is required. Arterial blood gases are useful to assess both the

degree of hypoxemia, as well as the acid/base balance. With CO poisoning, a pulse oximeter will give a falsely elevated oxygen reading. Other tissue asphyxiants (e.g., CN) may continue to show oxygenated blood with a pulse-oximeter since oxygen is not being utilized by the body as it should be.

- Exposed skin and eyes should be copiously flushed with water if indicated (or soap and water for skin, and normal saline for eyes).
- Monitor for respiratory distress. If cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Carefully observe patients with inhalation exposure for the development of any systemic signs or symptoms and administer symptomatic treatment as necessary (e.g., seizures).
- Patients with severe or prolonged exposure should be carefully evaluated for neurologic and other sequelae.
- Freeze injury associated with dermal exposure to compressed or liquid forms (i.e., propane) is unlike frostbite in that the damage occurs within seconds and rewarming is not beneficial. Freeze injuries of this nature should be managed much like a thermal burn.
- If a patient appears with characteristic symptoms of a particular asphyxiant (or is known to have been exposed) which has an antidote, administration of this in a timely manner can be lifesaving.
- Specific antidotes:
 - ◆ There are specific antidotes to some of the systemic asphyxiant exposures (e.g., a cyanide poisoning antidote kit, or hyperbaric oxygen for CO), but it may be difficult in the field setting to make specific diagnoses regarding a mixed exposure.
 - ◆ **Note:** This list is to remind you that these antidotes exist. There are specific instructions and caveats regarding how to administer these antidotes which should be obtained from your local consultants, packaging inserts, or medical texts:
 - Carbon monoxide: hyperbaric oxygen (HBO) may be lifesaving.
 - Methemoglobin-forming compounds: methylene blue. Methemoglobinemia results in a chocolate brownish-colored blood when viewed by

phlebotomy but looks bluish through the skin, therefore, making the person look cyanotic. This is visible at 1.5 g/dL of metHb in the blood (about 7-10%). However, there is no indication to treat methemoglobinemia below a level of 30% unless there is cardiorespiratory distress. DO NOT administer in someone with G6PD deficiency.

- Cyanide and cyanogenic compounds: an antidote kit, usually contains amyl nitrite and sodium nitrite (to form methemoglobin), followed by sodium thiosulfate (to form thiocyanate).
- Sulfides: MAYBE amyl nitrite and sodium nitrite (to form methemoglobin), or hyperbaric oxygen.

■ Special notes:

- ◆ Hydrogen sulfide can cause a characteristic and immediate loss of consciousness called a “knock down” (needs to be differentiated from cyanide or nerve gas knock down). It is also an irritant gas (see irritant gases) and will inflame the eye, upper and possibly lower respiratory tract.
- ◆ Hydrogen sulfide and some other compounds, though they have a characteristic odor, cause “olfactory fatigue” where it is not perceived (smelled) after a short period of time, even though it is present.
- ◆ Hydrogen azide will act as a low water soluble irritant gas (see irritant gases). It will irritate the lower respiratory tract and lung, possibly delayed, without as much upper respiratory effect.
- ◆ Nitrites and nitrates, and azides can also cause direct vasodilation and can produce a throbbing headache, hypotension, reflex tachycardia, syncope, and cerebral or myocardial ischemia or infarction due to hypoperfusion.
- ◆ Smoke/products of combustion (e.g., in a fire, burning building) may have a number of asphyxiants as components, depending on what was burning. Examples: cyanide compounds, CO. (Irritant gases are also components.)
- ◆ Some asphyxiant compounds, such as methane and hydrogen sulfide, are heavier than air and will settle in low places.

- ◆ All the simple asphyxiant agents included are colorless gases. Argon, carbon dioxide, ethane, helium, hydrogen, methane, neon, and nitrogen are odorless. Acetylene has a faint garlic odor. Ethylene has a sweet odor and taste. Butane, liquefied petroleum gas, propane, and propylene have a faint petroleum-like odor and may be stench with mercaptans to increase casual observer nasal detection during transport and storage.
- ◆ Some of the simple asphyxiants are hydrocarbons (see “hydrocarbons” as well), and some have abuse potential (e.g., when supplied as propellants). Some hydrocarbons such as methane and propane are also dangerous because they are highly flammable and can form explosive mixtures with air.
- Stages of exposure for simple asphyxiants: Four stages are described, depending on the arterial oxygen saturation. All early effects may decrease ability for self-rescue from the toxic environment.
 - ◆ INDIFFERENT STAGE:
 - %O₂ Saturation: 90%
 - Night vision: decreased
 - ◆ COMPENSATORY STAGE:
 - %O₂ Saturation: 82 to 90%
 - Respiratory rate: compensatory increase
 - Pulse: compensatory increase
 - Night vision: decreased further
 - Performance ability: somewhat reduced
 - Alertness: somewhat reduced
 - Above symptoms may begin earlier in those with significant preexisting cardiac, pulmonary, or hematologic diseases.
 - ◆ DISTURBANCE STAGE:
 - %O₂ Saturation: 64 to 82%
 - Compensatory mechanisms become inadequate (air hunger; fatigue, tunnel vision, dizziness, headache, belligerence, euphoria, reduced visual acuity, numbness and tingling of extremities, hyperventilation, poor judgment, memory loss, cyanosis, decreased ability for escape from toxic environment).

- ◆ CRITICAL STAGE:
 - %O₂ Saturation: 60 to 70% or less
 - Deterioration in judgment and coordination may occur in 3-5 minutes or less; total incapacitation and unconsciousness follow rapidly.

ORGANIC VAPORS [HYDROCARBONS (HC) AND HALOGENATED HYDROCARBONS (H-HC)]

- Examples of HCs include—
 - ◆ aliphatic: methane, ethane, propane, and butane.
 - ◆ aromatic: benzene, ethylbenzene, toluene, xylene, phenol, and aniline.
 - ◆ petroleum aliphatic mixtures: gasoline, kerosene, naphtha, mineral spirits, and mineral seal oil.
 - ◆ pine tree distillation mixtures: turpentine, pine oil, pine tar.

- Examples of H-HCs include (“halogenated” can be with chloride, bromide, fluoride, iodide)—
 - ◆ methylene chloride.
 - ◆ chloroform.
 - ◆ carbon tetrachloride.
 - ◆ trichloroethane (TCA).
 - ◆ trichloroethylene (TCE).
 - ◆ perchloroethylene (PCE).
 - ◆ vinyl chloride.

Toxidrome: sleepiness, even to the point of narcosis (deep stupor or coma), and cardiac irritability with premature ventricular contractions (PVCs) and even ventricular tachycardia (VT) or ventricular fibrillation (VF).

Acute Effects:

- Exposures in confined spaces with poor ventilation can be deadly.
- Neurologic: CNS depression (headache, dizziness and a sense of intoxication, confusion, decreased levels of consciousness, sleepiness), general anesthesia and narcosis, possibly to respiratory depression and arrest, coma, and death. (Some H-HCs used to be used for general inhalational anesthesia.)
 - ◆ Most likely from vapors of halogenated, aromatic, other higher molecular weight hydrocarbons, and volatile petroleum distillates.
 - ◆ Halogenated and aromatic hydrocarbons can produce CNS depression following ingestion.

- **Cardiac:** Heart myocardium can be sensitized to endogenous catecholamines, epinephrine and norepinephrine, lowering the threshold for ventricular irritability – PVCs, tachycardia, fibrillation. The fatal ventricular arrhythmias, popularly labelled “sudden sniffing death” syndrome (with inhalant abuse) may be a result. Avoid sympathomimetics when treating these patients.
 - ◆ Most likely from volatile HCs and H-HCs. HCs vary in degree of sensitization.
H-HCs are the greatest risk.
- **Pulmonary:** Prolonged exposure to inhaled fine HC mists and some longer chain HCs like gasoline (but not short chain molecules like methane, ethane, propane, and butane) can also result in chemical pneumonitis, with dyspnea, cough, sputum production, crackles, and hypoxemia.
- **Skin:**
 - ◆ Liquid HCs and H-HCs are excellent organic solvents and can dissolve skin and mucous membranes with acute but prolonged contact – irritation, defatting dermatitis (skin inflammation with drying and cracking), chemical burns.
 - ◆ With extensive or prolonged dermal exposure they can be absorbed through the skin and may contribute to systemic toxic effects.
 - ◆ Frostbite can result from contact with some liquefied gases (e.g., propane, methane, ethane).
 - ◆ HC and H-HCs vary in their degree of irritation and drying to the mucous membranes of the respiratory tract.
- **Eyes** can be injured by direct contact: irritation, lacrimation, blurred vision, conjunctival injection, corneal ulceration. Mild to moderate eye irritation and reversible ocular injury may occur after contact with most hydrocarbons.
- **Ingestion** is not the usual route of exposure in chemical “accidents” but keep in mind that liquid HCs and H-HCs can have some stomach absorption, and also cause aspiration pneumonitis. The HC odor will likely be on the patient’s exhaled breath. Patients with aspiration pneumonia may have fever, dyspnea, tachypnea, cough, sputum production, rales, rhonchi, and decreased breath sounds, hypoxemia, cyanosis.

- ◆ Initial signs and symptoms of aspiration most frequently occur during the act of swallowing and may include coughing, choking, and gagging or persistent coughing occurring immediately following ingestion.
- ◆ Hydrocarbons with low viscosity, low surface tension, and high volatility (e.g., kerosene, mineral seal oil, gasoline, petroleum naphtha) are most likely to cause aspiration pneumonitis.
- Simple asphyxia: gaseous HCs and H-HCs can also be simple asphyxiants (see asphyxiants). Lack of oxygen can cause headache, dizziness, weakness, confusion, agitation, seizures, coma, and even death. Tachycardia and tachypnea would be expected.
- Liver or kidney toxicity: Many H-HCs are metabolized in the liver and kidney and can cause toxicity and failure. Kidney effects can also occur due to hypoxia.
 - ◆ Liver: elevated transaminases possible. Significantly hepatotoxic H-HCs: carbon tetrachloride, chloroform, 1,1,2-trichloroethane (vinyl trichloride). Less hepatotoxic: trichloroethylene, tetrachloroethylene and 1,1,1-trichloroethane. Carbon tetrachloride can cause fatty liver.
 - ◆ Renal effects (acute renal tubular necrosis, proteinuria, or hematuria) occur infrequently following acute exposure to petroleum distillates and other unsubstituted hydrocarbons. Many H-HCs are potentially nephrotoxic.
- Disseminated intravascular coagulation, hemolytic anemia, and pancytopenia have occasionally been reported following vapor inhalation, aspiration, or ingestion of HCs. Rhabdomyolysis has been reported.

Chronic Effects:

- Long-term or repeated exposure to certain aromatic and chlorinated HCs can result in hematologic (e.g., benzene), hepatotoxic (e.g., chlorinated HCs), renal (e.g., chlorinated HCs), neuropsychiatric (e.g., toluene), neurological (e.g., n-hexane) and carcinogenic (e.g., known: benzene, vinyl chloride; suspected: carbon tetrachloride, chloroform, methylene chloride, methyl

chloride, tetrachloroethylene, tetrachloroethane, trichloroethane, etc.) effects.

- Chronic exposure or abuse of some HCs can result in chronic encephalopathy and residual neurological impairment: cerebellar abnormalities including ataxia, nystagmus and dysarthria; incoordination, memory deficits poor concentration, poor abstract reasoning, and emotional lability.
- Biopsy confirmed glomerulonephritis and nephrotic syndrome has been reported following long-term inhalation or dermal exposure to various HCs.

4

Treatment:

- Treatment involves removal from the exposure, decontamination, oxygen, and ventilatory and circulatory support as needed.
- Move patient to fresh air. Administer 100% humidified supplemental oxygen with assisted ventilation as required. Monitor for respiratory distress. If cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Respiratory tract irritation, if severe, can progress to pulmonary edema, which may be delayed in onset up to 24-72 hours after exposure.
- Support respiratory and cardiovascular function. Epinephrine and other sympathomimetics should only be used with caution due to possible increased sensitivity of the myocardium to catecholamines.
- Decontaminate:
 - ◆ Remove all contaminated clothing to prevent further absorption.
 - ◆ Wash all exposed areas of the body thoroughly with soap and water.
 - ◆ Exposed eyes should be irrigated with copious amounts of tepid water for at least 15 minutes (to normal pH if the agent is acidic or basic – e.g., phenol or aniline). If eye irritation, pain, swelling, lacrimation, or photophobia persists, the patient should have an ophthalmologic exam.
- A physician may need to examine the affected skin areas if irritation or pain persists. Consultation with a clinician experienced in burn therapy or a burn unit

should be obtained if larger area or more severe burns than the following are present:

- ◆ Minor chemical burns (first or second degree) less than 15% body surface area in adults or less than 10% body surface area in children; or
- ◆ Minor third degree: less than 2% body surface area.
- ◆ Neutralizing agents should NOT be used.
- Some chemicals can produce systemic poisoning by absorption through intact skin. Carefully observe patients with dermal exposure for the development of any systemic signs or symptoms and administer symptomatic treatment as necessary.
- In cases of significant inhalational abuse, chlorinated HC exposure, prolonged unconsciousness and hypoxia, prolonged and extensive dermal exposure to liquid HC, or HC injection:
 - ◆ Monitor CBC, urinalysis, and liver and kidney function tests, and serum CPK in patients with significant exposure.
 - ◆ Monitor fluids and electrolytes.
 - ◆ Monitor arterial blood gases, pulmonary function, and chest x-ray for patients with significant exposure.
- Benzene may particularly produce abnormalities of the hematopoietic system. Monitor the complete blood count for patients with significant exposure.
- Monitor methemoglobin level in cyanotic patients who do not respond to oxygen and who may have been exposed to aniline or nitrobenzene.
- Special notes:
 - ◆ Natural gas and petroleum are the sources of most HCs, and they can be aliphatic (straight or branched chained), aromatic (with at least one benzene ring), or substituted with a chemical constituent in place of the hydrogen, such as hydroxyl or nitro or amine groups or halogenated (having a halide constituent – such as chlorine, fluorine, bromine or iodine). There are many HC, substituted HC, and H-HC compounds and besides having certain effects in common, many have their own unique toxicities.
 - ◆ HCs can be gaseous, liquid or solid (physical state is dependent upon chain length/molecular weight with shorter chains/lower MWs tending to exist as gases, and longer chains/higher MWs tending to exist as

either liquids or solids). Their physical state will affect exposure and toxicity potentials. However, many HCs and H-HCs have a significant vapor pressure, and therefore, their potential for inhalation is substantial.

- ◆ HCs also tend to be lipid soluble, so are able to locally affect the skin and permeate either intact or non-intact skin to exert systemic effects.
- ◆ Flammability and significant vapor pressure create explosivity. HCs can form dangerous explosive mixtures in the air. HCs tend to be flammable, but H-HCs tend to be less or not flammable.
- Other specific characteristics and toxicities:
 - ◆ N-hexane: long-term exposure produces peripheral neuropathy, which would not otherwise be expected from a single exposure.
 - ◆ Phenol is rapidly and significantly absorbed through the skin leading to systemic toxicity. It is anesthetic on the skin but severe burns (depigmented) can result along with multi-organ injury, cardiac dysrhythmias and pump failure, hepatotoxicity, and death.
 - ◆ Aniline produces methemoglobinemia and direct hepatotoxicity.
 - ◆ Pentachlorophenol and dinitrophenol uncouple oxidative phosphorylation to disallow ATP formation and allow subsequent development of hyperthermia, tachypnea, hypoxia, hypoglycemia, and initial CNS and cardiac irritability followed by CNS depression and cardiovascular collapse.
 - ◆ Benzene is a known human carcinogen. Long-term exposure also causes bone marrow toxicity with pancytopenia, aplastic anemia, and leukemia.
 - ◆ Methylene chloride is endogenously metabolized to CO and can produce CO poisoning.

CHOLINERGICS

- Pesticides that are cholinesterase inhibitors, such as the organophosphates (OPs) and the carbamates, cause the cholinergic toxidrome by causing excess acetylcholine at the cholinergic synapses and nerve endings.
 - ◆ Organophosphate examples include: acephate, azinphos-methyl, chlorpyrifos, demeton, diazinon, dichlorvos, EPN, ethion, malathion, parathion, ronnel, and tetraethyl pyrophosphate.
 - ◆ Carbamate examples include: aldicarb, carbaryl, carbofuran, methomyl, and propoxur.
 - ◆ Chemical warfare nerve agents (CWA) are also cholinesterase inhibitors.
 - ◆ Major differences between OP pesticides and nerve agents involve vapor pressure, timing for “aging” [making a permanent bond with acetyl cholinesterase (Ach-ase) – becoming irreversible] and amount of antidote needed. OP pesticides are, in general, more oily and less volatile, take a longer time for aging, and require much more antidote over time (even though the nerve agents are acutely more toxic. OPs have a slower onset of toxicity but effects last longer.) Carbamates do not age (are always reversibly bound to Ach-ase).

Toxidrome: PNS muscarinic: diarrhea, urination, miosis, bradycardia, bronchorhea, bronchospasm, emesis, lacrimation, salivation, secretion, sweating (DUMBELS) and/or PNS nicotinic: mydriasis, tachycardia, weakness, hypertension, hyperglycemia and fasciculations (MTWTF), and/or CNS: confusion, convulsions, coma.

Acute Effects:

- Cholinergic nerve endings or synapses (nicotinic and muscarinic) occur in the central nervous system (CNS) and peripheral nervous system (PNS). PNS includes, at the sympathetic and parasympathetic ganglia, neuromuscular junctions and neuroeffector junctions (parasympathetic). Some of the effects can be contradictory and competing.
 - ◆ PNS muscarinic effects are also remembered as SLUDGE (salivation, lacrimation, urination, defeca-

tion, gastroenteritis, and emesis). Also rhinorrhea and abdominal cramps. Lungs can be wet, crackling, from secretions.

- ◆ PNS nicotinic effects include MTWTF: mydriasis, tachycardia, weakness, hypertension, hyperglycemia, and fasciculations (of the muscles). Muscle weakness can progress to paralysis. Muscle fasciculations can be seen through the skin but are best seen on the face and tongue. Hyperglycemia is stimulated sympathetically (increased epinephrine and norepinephrine).
 - Initially there will be tachypnea, but slow respirations and respiratory arrest may follow, due to CNS effects or muscle weakness/paralysis.
 - Cardiovascular effects may waiver between sympathetic (tachycardia and hypertension) and parasympathetic (bradycardia and bradydysrhythmias). Usually sympathetic predominates at first and parasympathetic later.
 - Other effects waiver similarly [e.g., miosis (pupillary constriction) and mydriasis (dilation), but usually miosis predominates].
- ◆ Ophthalmic effects besides miosis include dim vision, headache, and eye pain.
- ◆ CNS effects include headache, anxiety, dizziness, confusion, agitation, convulsions, coma, and even death.
- ◆ Skin: most of these chemicals cause no or mild irritation, except Ronnel which is corrosive. Most OPs absorb well through the skin and mucous membranes (as well as GI and by inhalation of mists or dusts). Carbamates are usually less well absorbed. Sweat may be prominent.

Chronic Effects:

- Intermediate syndrome is characterized by paralysis of respiratory, cranial motor, neck flexor, and proximal limb muscles 12 hours to 7 days after exposure and following resolution of cholinergic symptoms. It occurs prior to development time of delayed peripheral neuropathy. Treatment is supportive (e.g., respiratory support). Atropine and pralidoxime are ineffective. Recovery begins 5-15 days after onset.
- Delayed polyneuropathy [from inhibition of neuropathic target esterase (NTE) by some OP]: Distal sensory-motor polyneuropathy may develop 6-21 days following exposure. It manifests as burning or tingling, then progressive distal weakness and ataxia in the lower limbs. Flaccid paralysis, spasticity, ataxia or quadriplegia may ensue. Recovery may be slow or incomplete.
- Sequelae may also include subtle neuropsychological deficits. Decreased vigilance, defects in expressive language and cognitive function, impaired memory, depression, anxiety or irritability and psychosis have been reported as delayed effects.
- The HC diluent may contribute to the overall toxicity. Refer to HCs management for further information.

Treatment:

- Remove to fresh air. Ensure adequate ventilation and oxygenation (100% oxygen as indicated). Airway suction and/or intubation may be needed. Cardiac monitoring and support as needed. Treat seizures with IV valium or lorazepam. Provide specific antidotes as needed, promptly.
- Decontaminate:
 - ◆ Remove all contaminated clothing to prevent further absorption.
 - ◆ Wash all exposed areas of the body thoroughly with soap and water.
 - ◆ Exposed eyes should be irrigated with copious amounts of tepid water for at least 15 minutes.

- RBC cholinesterase activity levels can be measured and serve as a reflection of nervous system acetylcholinesterase levels.
 - ◆ “Pseudocholinesterase” (plasma cholinesterase) can be measured but is not as reflective of the nervous system.
 - ◆ Depression in excess of 50% of baseline is generally associated with severe symptoms.
 - ◆ Correlation between cholinesterase levels and clinical effects in milder poisonings may be poor.
- Monitor electrolytes, ECG, and serum pancreatic isoamylase levels in patients with significant poisoning. Patients who have increased serum amylase levels and those who develop a prolonged QTc interval or PVCs are more likely to develop respiratory insufficiency and have a worse prognosis. Other lab/x-rays as indicated.
- Specific antidotes:
 - ◆ Atropine:
 - Symptomatic antidote for muscarinic effects only. It will not stop the nicotinic effects of fasciculations, weakness, flaccid paralysis, or respiratory arrest. It will not regenerate the acetylcholinesterase.
 - Initial dose: 0.5 - 2.0 mg IV (adult). Repeat every 5 minutes until muscarinic effects (bronch-orrhea, bronchospasm, bradycardia) resolve. Severely poisoned patients may require large doses – up to several grams over days to weeks.
 - ◆ Pralidoxime (2-PAM):
 - Reactivates acetylcholinesterase (that has not “aged”) if given early enough and in enough dosage. Use for nicotinic and CNS effects. Not needed for carbamate poisoning, but use if not sure what the agent is.
 - Infuse SLOWLY over 5-10 minutes. Initial dose: 1-2 grams IV (adult). Follow with continuous infusion of 500 mg/h for at least 24 hours. If RBC cholinesterase activity levels drop with discontinuation, then continue.

- Special notes:
 - ◆ Carbamates bind reversibly to cholinesterase. OPs will bind irreversibly unless pralidoxime (2-PAM) is provided before aging occurs. Some OPs can take up to 2 days to age.
 - ◆ Dichlorvos can vaporize sufficiently to be a danger in an enclosed space.

RESPIRATORY PROTECTION

- Respirators are of two main types: air-purifying and supplied-air. The latter are not logistically practical for most military units, especially in a tactical setting.
- Air-purifying respirators are masks that filter ambient air and use different cartridge types (with color coded canisters) to protect against specific exposures [e.g., acid gases, organic vapors, ammonia gas, pesticides, and particulates (HEPA)]. Limitations: not protective against some common toxic gases such as CO and NO_x nor against a lack of ambient oxygen.
- Soldiers are fit-tested and instructed in using the mask issued to them for protection against NBC (e.g., M-40 mask with the C2A1 canister, which has carbon with other reactive materials, and HEPA filters).
- The M-40 series military protective mask was designed to be used by soldiers in tactical situations against various potential warfare agents on the battlefield including CWA, BWA, radiological/nuclear agents, and some TICs that are also considered CWA. The M-40 series mask filters are tested according to military specification and are required to provide protection against other CWAs besides nerve and blister agents such as blood agents (hydrogen cyanide, cyanogens chloride), choking agents (phosgene, chloropicrin), and riot control agents (ortho-chlorobenzylidene malonitrile, CS; chloroacetophenone, CN). The blood agents, hydrogen cyanide and cyanogens chloride, and the choking agent, phosgene, are also considered TICs. The M-40 series military protective mask is very effective in providing protection against the substances indicated above.
- The only TICs in the table that the M-40 mask was specifically designed for protection in tactical situations are hydrogen cyanide, cyanogens chloride, and phosgene; for tactical situations involving these chemicals, doctrine should be followed. However, breakthrough simulation studies by the Army have shown the M-40 series mask filter to be "effective" against a number of other TICs (e.g., see Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure table). For various reasons, only a NIOSH-approved respiratory

should be used against TICs in non-military unique settings (e.g., painting, welding, and other industrial type settings including response to spills, emergency response, etc.). In military-unique situations and deployed settings, a NIOSH-approved respirator should be used against TICs, as well, particularly in industrial-type settings. However, there are instances where a soldier only has an M-40 mask and may be in a situation where they could use it to help protect themselves (e.g., find themselves downwind from a plume that periodically drifts from an industrial site to their base camp, etc.). This should be considered on a case-by-case basis, taking into consideration what all of the confounding factors are in a deployed situation. See footnote I of Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure table.

- When NIOSH-approved commercial respirators are available and indicated, each type has specific guidance on fit testing, proper use, and maintenance. Soldiers should be trained and properly fit tested. When respirators are indicated for occupational duties, as much as is practicable in the military-unique setting, a respiratory protection program should be in place.

DEPLOYMENT CHEMICAL EXPOSURE QUESTIONNAIRE

List medicines or immunizations taken for this deployment**Job-related factors:**

- ☐ Where have you been located during this deployment? (Places, dates, jobs)
- ☐ Are you performing your usual job?
- ☐ Is your deployment job different? If yes, how?
 - Increased hours per day or days per week? How many?
 - Change of hours spent Indoors/Outdoors? What is the change?
 - New or different job duties/procedures?
 - New or different exposures?
(chemical/biological/radiation/physical)

(Inquire from list below about all on-the-job exposures)

- Is ventilation adequate where you are working?
- Increased stress?
- Do you get the material(s) on your skin or clothing?
- Can you smell the chemical or material you are working with?
- Do you wash your hands with solvents?
- Do you use—or were you advised to use—protective equipment such as gloves, masks, respirator, or hearing protectors?
(Specify the equipment.)
- Were you instructed in the use of protective equipment?

Environmental factors at deployment site:

- ☐ Is your deployment location next to or near an industrial plant, commercial business, dump site, or other nonresidential property of concern?
- ☐ Is air pollution a problem?
- ☐ Are pesticides or herbicides used to control weeds and pests?
- ☐ What is your source of drinking water? (private well, city water, bottled water, other)
- ☐ Is the water purified prior to drinking? Tested for pollutants?
- ☐ Any unusual or irritating exposures in the living or work areas?

[If answered 'yes' to any question, please explain.]

Health Issues:

- ☐ Do you smoke? Do you smoke at the workplace?
- ☐ Have you or co-workers experienced any symptoms?
If yes.....Aggravated by a specific activity?
Get either worse or better at the workplace?
Get either worse or better in your living area?
- ☐ Have you been off work more than 1 day due to a work-related illness during the deployment?
- ☐ Have you changed jobs or work assignments due to health problems or injuries during the deployment?

[Review of systems as appropriate.]

Exposures about which to inquire (and examples in parentheses):

During your deployment, have you been exposed to the following?

- ☐ Metals? (arsenic, beryllium, cadmium, chromates, lead, mercury, nickel)
- ☐ Dust or fibers? (asbestos, coal dust, fiberglass, rock dust, silica dust, talc)
- ☐ Fumes or mists? (welding fumes)
- ☐ Workplace chemicals? [gasoline, solvents, oils, acids, alkalis, industrial alcohols, ketones, pesticides – try to name specific chemicals (see list below)]
- ☐ Chemical warfare agents?
- ☐ Army-specific chemicals? [explosives, fogs (trinitrotoluene)]
- ☐ Radiation? (x-rays, lasers)
- ☐ Biologicals? (molds, viruses, insects, Anthrax, Smallpox, Bot tox, Ricin, tularemia)
- ☐ Loud noise, vibration, extreme heat or cold?

Specific chemicals for query: ammonia, benzene, carbon tetrachloride, chlorinated naphthalenes, chloroform, dichlorobenzene, ethylene dibromide, ethylene dichloride, halothane, isocyanates, ketones, methylene chloride, perchlorethylene, phenol, phosgene, styrene, toluene, TDI or MDI, trichloroethylene, vinyl chloride, PCBs, PBBs.

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ⁱ	Source/Use
			Impairment ^k	Fatality		
Acrolein 107-02-8	Irritant vapor/ corrosive	Colorless or yellow liquid. (1.8 ppm GM d, pungent odor) ^M (0.5 ppm eye) ^O	0.1 ^c	1.4 ^c	Poor	Formed from burning of trees, tobacco (cigarettes), plants, gasoline, and oil. Used as a pesticide to control algae, weeds, bacteria, and mollusks and used to make other chemicals. ^h
Acrylonitrile 107-13-1	Asphyxiant (cyanide; antidote!) (this effect can be delayed) Irritant vapor/corrosive; other systemic effects possible	Colorless to pale- yellow liquid. (1.6 ppm GM d, onion/garlic odor) ^{M, N}	35 ^f	75 ^f	Poor	Used to make other chemicals such as plastics, synthetic rubber, and acrylic fibers. Previously used as a pesticide. ^h
Allyl alcohol 107-18-6	Irritant vapor/corrosive; other systemic effects (liver, kidney)	Colorless liquid. (1.7 ppm GM d, mustard odor) ^M (6 ppm eye, 0.78 nasal)	4.2 ^c	67 ^c	Marginal	Used in chemical production for resins and plasticizers, pharmaceuticals, military poison gas, herbicide, and pesticide. ^j

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ⁱ	Source/Use
			Impairment ^k	Fatality		
Ammonia 7664-41-7	Irritant gas	Colorless gas. (17 ppm GM d, pungent/irritating odor) ^{M, N} (140 ppm eye, 55- 100 ppm nasal) ^O	110 ^c	1100 ^c	Poor	Naturally-occurring and applied directly to soil on farm fields. Used to make fertilizers for farm crops, lawns, plants and household and industrial cleaners. ^h
Arsine 7784-42-1	Primary effect: hemolysis Secondary renal failure; Effects may be delayed for hours. (arsenic)	Colorless gas with a mild, garlic-like odor. (0.5 ppm) ^j	0.17 ^d	0.5 ^d	Effective	Used in electronics manufacturing, glass dyes, possible poison gas for military purposes. ^j
Boron trifluoride 7637-07-2	Irritant gas. Treat as hydrogen fluoride; also boron toxicity	Colorless gas with a pungent, suffocating odor. (4.50 mg/m ³) ^j	30 mg/m ³ ^f	100 mg/m ³ ^f	Effective	Electronics industry, chemical production, fumigant, food packaging material manufacturing. ^j

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ⁱ	Source/Use
			Impairment ^k	Fatality		
Bromomethane 74-83-9	(aka methyl bromide); irritant gas; halogenated hydrocarbon. Alkylating agent. Multisystem effects – neuro/psych (seizures); liver, skin vesicant etc. Effects can be delayed by hours.	Colorless gas with a chloroform-like odor at high concentrations. (20 ppm) ^h	210 ^e	740 ^e	Poor	Naturally-occurring in the ocean and manufactured for use as a fumigant/pesticide, chemical production, and as a solvent to get oil out of nuts, seeds, and wool. ^h
Carbon monoxide 630-08-0	Asphyxiant (antidote!)	Colorless, odorless gas. (odorless)	83 ^c	330 ^c	Poor	Production of chemical intermediates, metal purification, fuels; motor vehicle emissions, combustion/burning of organic matter, by-product in some chemical reactions, natural sources. ^j

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Chlorine 7782-50-5	Irritant gas	Greenish-yellow gas. (0.080 ppm GM d, suffocating/sharp bleach odor) ^{M,N} (3-6 ppm eye, nasal, & throat) ^O	2 ^d	20 ^d	Effective	Used as a bleach in the manufacture of paper and cloth. ^h WWI war gas, cleaner/disinfectant, water treatment.
Cyanogen chloride 506-77-4	Asphyxiant (cyanide, antidote!); irritant gas	Colorless gas or liquid (below 55°F) with an irritating odor. Solid (below 20° F. (1 ppm)	0.4 ^f	4 ^f	Effective	Military poison gas, metal cleaner, ore refining, rubber production, fumigant for ships and warehouses, former pesticide. ^j
Diborane 19287-45-7	Irritant gas; (possible neuro, liver, kidney) (boron)	Colorless gas with a repulsive, sweet odor. General population not exposed; very toxic and flammable. ^h (3.3 ppm) ^{j,N}	1 ^d	3.7 ^d	Effective	Used in rocket propellants, as a reducing agent, as a rubber vulcanizer, catalyst for hydrocarbon polymerization, flame-speed accelerator, doping agent. Also used in electronics. ^h

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Dimethylamine 124-40-3	Irritant gas/corrosive	Colorless gas with an ammonia- or fish- like odor. (0.047 ppm) ^j	100 ^f	350 ^f	ND	Used as a photographic chemical, plasticizer, ion exchange agent, antiknock agent in fuels, pesticide production, solvent, surfactants, rocket propellants, missile fuels, textile chemicals, rubber processing, pharmaceutical. ^j
Ethylene oxide 75-21-8	Irritant gas; corrosive; alkylating agent. neuro (seizures, irreversible damage), GI- N, V, etc., skin irritant/ vesicant	Colorless gas or liquid (below 51°F) with an ether-like odor. (420 ppm GM d, 490 ppm GM r, sweet/olefinic odor) ^M	45 ^c	200 ^c	Poor	Used primarily to make ethylene glycol (for antifreeze and polyester), insecticide for stored agricultural products, sterilization of medical equipment and supplies. ^h

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ¹	Source/Use
			Impairment ^k	Fatality		
Fluorine 7782-41-4	Strong irritant gas	Pale-yellow to greenish gas with a pungent, irritating odor. Highly reactive ^h . (0.035 ppm) ^h (25-100 ppm eye & nasal) ^o	5 ^c	13 ^c	Effective	Naturally-occurring (volcanoes), coal, aluminum production, phosphate fertilizer plants. ^h
Formaldehyde 50-00-0	Irritant gas; corrosive; (poss.neuro, kidney, metab.acidosis acutely); (allergic reactions)	Nearly colorless gas with a pungent, suffocating odor. (0.5 – 1.0 ppm) ^h (0.005-2.0 ppm eye) ^o	14 ^e	56 ^e	Poor	Used in the production of fertilizer, paper, plywood, cosmetics, sugar, photographic film, and latex. Also used in leather tanning, wood preservation, and laboratory preservative. ^h

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Hydrazine 302-01-2	Irritant vapor/corrosive; (can have delayed effects) [asphyxiant (metHb poss.)] Hemolysis; CNS excitation/ seizures; liver; kidney; GI-n,v,d; blood sugar Antidotes! :pyridoxine-Vit B6; methylene blue prn	Colorless, fuming, oily liquid. Solid below 36° F. (3.7 ppm GM d, ammonia odor) ^M	13 ^c	35 ^c	ND	Naturally-occurring in plants and manufactured for use in rocket propellants and fuels, boiler water treatments, chemical reactants, medicines, and cancer research. ^h
Hydrogen bromide 10035-10-6	Irritant gas	Colorless gas with a sharp, irritating odor. (2 ppm) ^j (5 ppm nasal & throat) ^O	22 ^e	120 ^e	Effective	Chemical production, solvent for ore minerals, petroleum industry, reagent in medicine. ^j

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Hydrogen chloride 7647-01-0	Irritant gas	Colorless to slightly yellow gas with a pungent, irritating odor. (4.7 ppm) ^j (35 ppm throat) ^o	22 ^d	100 ^d	Effective	Chloride production, ore refining, laboratory reagent, preparation of food products, pharmaceutic aid, metal treating agent. Manufacturing of fertilizers, dyes, paints, textiles, rubber, soap, leather tanning, photographic. ^j
Hydrogen cyanide 74-90-8	Asphyxiant (antidote!)	Colorless or pale- blue liquid or gas (above 78°F) with a bitter, almond-like odor. (0.8 ppm) ^j	7.1 ^d	15 ^d	Effective	Manufacturing of acrylates, cyanide salts, dyes, herbicides. Formerly used as a fumigant. ^j
Hydrogen fluoride 7664-39-3	Irritant gas/corrosive (hydrofluoric acid); systemic effects – hypocalcemia, etc – antidote!).	Colorless gas or fuming liquid (below 67°F) with a strong, irritating odor. (0.5 – 3 ppm) ^h (5 ppm eye & nasal) ^o	24 ^d	44 ^d	Effective	Naturally-occurring (volcanoes), coal, aluminum production, phosphate fertilizer plants, chemical production, municipal incinerators. ^h

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Hydrogen selenide 7783-07-5	Irritant gas; (poss. delayed 2-12 hrs: GI – n,v,d, chills, malaise, etc.; ?cor) (garlic odor on breath)	Colorless gas with an odor resembling decayed horse radish. (0.3 ppm) ^{j, N} (1.5 ppm eye) ^O	0.73 ^e	2.2 ^e	Poor	Metal production, preparation of semiconductor materials. ^j
Hydrogen sulfide 7783-06-4	Irritant gas; asphyxiant ("knock down"); possible antidote!	Colorless gas. (0.0094 ppm GM d, 0.0045 ppm GM r, rotten egg odor) ^{M, N} (50-100 ppm eye) ^O	27 ^c	50 ^c	Effective	Naturally-occurring in crude petroleum, natural gas, volcanic gases, and hot springs. Produced during food processing, coke ovens, kraft paper mills, tanneries, petroleum refineries, and waste. ^h

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure

Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Mercury 7439-97-6 (see other resources for most mercury cmpds. Here discussed: elemental mercury vapor-acute effects)	pulmonary irritant (pneumonitis), kidney (ATN); neuro (mostly with subacute and chronic exposures); gingivitis (delayed few days); within hours: weak, chills, GI-n,v,d etc. (consider chelation)	Metal: Silver-white, heavy, odorless liquid. (odorless) ^h	0.25 ^f	0.5 ^f	ND	Used to produce chlorine gas and caustic soda, thermometers, dental fillings, and batteries. Released from incinerators and industries burning fuels containing mercury. Naturally-occurring. ^h
Monomethylamine 74-89-5	Irritant gas/corrosive	Colorless gas or liquid (below 21° F). (4.7 ppm GM d, fishy odor) ^{M,N}	100 ^f	500 ^f	ND	Intermediate for accelerators, dyes, pharmaceuticals, insecticides, tanning; dyeing of acetate textiles; fuel additive; component of paint removers; solvent; photographic developer; rocket propellant. ^j

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Methyl hydrazine 60-34-4	Irritant vapor/corrosive; (can have delayed effects) [asphyxiant (metHb poss.)] Hemolysis; CNS excitation/ seizures; liver; kidney; GI-n,v,d; blood sugar Antidotes! :pyridoxine-Vit B6; methylene blue prn	Fuming, colorless liquid with an ammonia-like odor. (0.93 ppm) ^j	0.9 ^d	2.7 ^d	Marginal	Rocket fuel, antibiotic synthesis, solvent. ^j
Methyl isocyanate 624-83-9	Strong irritant vapor/corrosive; (cyanide poisoning unlikely) (poss. liver, kidney, sensitization)	Colorless liquid with a sharp, pungent odor. (2-5 ppm) ^{h, N} (2 ppm eye) ^o	0.067 ^d	0.2 ^d	Poor	Used in the production of pesticides, polyurethane foam, and plastics. ^h

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure

Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Methyl mercaptan 74-93-1	Irritant gas; CNS depression, asphyxiant- similar to Hydrogen Sulfide, poss. MetHB	Colorless gas. (0.00054 ppm GM d, 0.0010 ppm GM r, rotten cabbage odor) ^{M, N}	47 ^c	68 ^c	Poor	Released from decaying organic matter and present in natural gas, coal tar, some crude oils. Used in plastics industry, pesticides, and as a jet fuel additive. ^h
Nitric acid 7697-37-2	Irritant vapor/Corrosive; [poss. MetHb (see asphyxia) and hemolysis if nitric oxides formed in air from released acid]	Colorless, yellow, or red, fuming liquid with an acrid, suffocating odor. (3 ppm) ^j (62 ppm throat) ^O	24 ^c	92 ^c	Poor	Pharmaceutical and jewelry manufacturing, engraving, fertilizers, dyes, explosives, reprocessing spent nuclear fuel. ^j
Nitrogen dioxide 10102-44-0	Irritant gas (may be delayed effects up to 72 hrs or more); poss metHB (see asphyxia)	Yellowish-brown liquid or reddish- brown gas (above 70°F) with a pungent, acrid odor. Solid below 15° F. (1 ppm) (10-20 ppm eye) ^O	12 ^c	20 ^c	Poor	Production of nitric acid, lacquers, dyes, other chemicals, rocket fuels, explosives. Released from autos, fuel burning, welding, dynamite blasting, tobacco smoke. ^h

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ¹	Source/Use
			Impairment ^k	Fatality		
Parathion 56-38-2	cholinergic	Pale-yellow to dark-brown liquid with a garlic-like odor. Solid below 45° F. (0.47 mg/m ³) ^j	2 mg/m ³ ^g	10 mg/m ³ ^g	Effective	Insecticide. ^j
Phosgene 75-44-5	Irritant gas	Colorless gas with a suffocating odor like musty hay. (0.4 – 1.5 ppm) ^{h, N} (1-2 ppm eye, 2 ppm nasal, 2-3 ppm throat) ^O	0.3 ^d	0.75 ^d	Effective	Chemical, dye, pesticide, pharmaceutical manufacturing. ^h Historical war gas.
Phosphine 7803-51-2	Irritant gas (may be delayed onset of lung injury); systemic asphyxiant (brain, kidney, cor, liver) [beware: Al or Zn phosphide rodenticides and water!]	Colorless gas. (0.14 ppm GM r, garlic odor) ^M	2 ^c	3.6 ^c	Marginal	Semiconductor and plastics industries, flame retardant production, fumigant/pesticide in stored grain. ^h

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Phosphorus trichloride 7719-12-2	Irritant vapor/corrosive	Colorless to yellow, fuming liquid with an odor like hydrochloric acid. (no data) ^j	2 ^c	5.6 ^c	Effective	Pesticides, surfactants, gasoline additive, plasticizers, dyes, chlorinating agent, textile finishing, chemical and insecticide manufacturing. ^j
Phosphorus oxychloride 10025-87-3	Irritant vapor/corrosive (poss. liver and kidney)	Clear, colorless to yellow, oily liquid with a pungent & musty odor. Solid below 34° F. (no data) ^j	0.5 ^g	0.85 ^c	Marginal	Manufacture of plasticizers, gasoline additives, chlorinating agent and catalyst, and fire retarding agent. ^j
Propylene oxide 75-56-9	Irritant gas/corrosive; neuro/CNS depression (alkylating agent)	Colorless liquid or gas (above 94° F) (45 ppm GM d, 35 ppm GM r, sweet odor) ^M (457 ppm eye, animal) ^O	290 ^c	870 ^c	P	Food additive, fumigant, pesticide, solvent, detergent manufacturing, component in brake fluids. ^j

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Sulfuric acid 7664-93-9	corrosive	Colorless to dark-brown, oily, odorless liquid. Pure compound is solid below 51° F; often used in aqueous solution). (1 mg/mg ³) ^h	8.7 mg/m ³ ^e	160 mg/m ³ ^e	Effective (decreased effectiveness in humid and dry conditions)	Fertilizer production, food additive, petroleum refining, ore processing, explosives/batteries/dye/paper/glue/metal industries. ^h
Sulfur dioxide 7446-09-5	Irritant gas; (poss sulf.Hb)	Colorless gas. (2.7 ppm GM d, 4.4 ppm GM r, metallic taste/sharp irritating) ^M (20 ppm eye, 6-12 ppm nasal & throat) ^O	1 ^c	27 ^c	Effective	Paper/pulp industry, fumigant, preservative, bleach, food processing, petroleum industry, mining. ^h
Sulfur trioxide 7446-11-9	Irritant vapor/corrosive (similar effects to sulfuric acid)	Colorless liquid that can also exist as ice- or fiber-like crystals or as a gas. When exposed to air, it rapidly takes up water and gives off white fumes. ^h (no data) ^h	10 mg/m ³ ^f	30 mg/m ³ ^f	Marginal	Intermediate for sulfuric acid production, explosives manufacturing, solar energy collectors, oxidizing agent. ^h

Summary of Common Acutely Toxic Industrial Chemicals from Inhalation Exposure						
Chemical/ CASRN	Toxidrome Class(es) and Other Prominent Effects	Physical Description ^a , (Odor Threshold), and (Irritation Threshold: eye, nasal, or throat)	1-hour Exposure Guidelines ^b (ppm)		M40 Mask Filter Effectiveness ^l	Source/Use
			Impairment ^k	Fatality		
Toluene-2,4- diisocyanate 584-84-9	Irritant vapor/corrosive; neuro/psych [allergic sensitization – lung (asthma)/skin (eczema)]	Colorless to pale- yellow solid or liquid (above 71°F) with a sharp, pungent odor. (0.4 ppm) ^j	0.083 ^d	0.51 ^d	Effective	Chemical intermediate, polyurethane foams, nylon, hardener in polyurethane adhesives and finishes. ^j

Notes:

CASRN = Chemical Abstract Service Registry Number

^a Source = U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health,

“Pocket Guide to Chemical Hazards”, June 1997.

^b The exposure guidelines are maximum airborne concentrations below which it is believed nearly all individuals could be exposed for up to 1 hour without experiencing impairment or fatality. Guidelines were selected from a hierarchy of sources:

1) U.S. Environmental Protection Agency, Acute Exposure Guideline Levels (AEGL), <http://www.epa.gov/oppt/aegl/>, accessed Feb 2005. 1-hour AEGL-2 and AEGL-3 selected.

2) American Industrial Hygiene Association, Emergency Response Planning Guidelines (ERPG) and Workplace Environmental Exposure Level Guides Handbook, 2005. ERPG-2 and ERPG-3 selected.

3) U.S. Department of Energy, Temporary Emergency Exposure Limits (TEEL), Revision 20, April 2004. TEEL-2 and TEEL-3 selected.

^c Interim AEGL

^d Final AEGL

^e Proposed AEGL

^f ERPG

^g TEEL

^h Source = Agency for Toxic Substances and Disease Registry, ToxFAQs™, Toxicological Profiles, and Medical Management Guidelines, <http://www.atsdr.cdc.gov/>, accessed 8-11 April 2005.

ⁱ Environmental persistence provided as the half-life in air

^j Source = National Library of Medicine, Hazardous Substances Data Bank (HSDB), accessed 11 April 2005.

^k Impairment = irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape (adopted from definition of AEGL-2 in the National Research Council, “Standard Operating Procedures for Developing Acute Exposure Guideline Levels for Hazardous Chemicals”, 2001.

^l Mask effectiveness based on filter assessment using the C2 canister presented in US Army Edgewood Chemical and Biological Center, “Toxic Industrial Chemicals Assessment of NBC Filter Performance”, ECBC-TR-093, Sept 2000. The filter effectiveness against cyanogen chloride and propylene oxide are based on other information. “Poor”: Offers no significant protection or only short-term limited protection. Operations in areas with these TICs should be avoided. If the mask is worn, filter change out is required. “Marginal”: Use as an escape-only mask to leave the TIC contamination or against brief, low concentration challenges; replace filter immediately after chemical exposure ends. “Excellent”: Will filter the TIC effectively; filter change out is required at end of exposure. The only TICs in the table that the M40 was specifically designed for

protection in tactical situations are hydrogen cyanide, cyanogen chloride, and phosgene; for tactical situations involving these chemicals, doctrine should be followed. Only a NIOSH-Approved respirator should be used against TICs in non-military unique settings (e.g., painting, welding, and other industrial-type settings), and spill and emergency response. In military-unique situations and deployed settings, the same restrictions should apply; however, there are some instances in a deployed setting where a soldier only has an M40 and may be in a situation where they could use it to help protect themselves (e.g., find themselves downwind from a plume that periodically drifts from an industrial site to their base camp; etc.). This should be considered on a case-by-case basis, taking into consideration what all of the confounding factors are in a deployed situation.

^M Source = Odor Thresholds for Chemicals with Established Occupational Health Standards, AIHA, 1989.

^N Sense of smell becomes fatigued with time & CANNOT be relied upon to warn of the continuous presence of the chemical (Sources: ACGIH, ATSDR, or Patty's Toxicology, 5th Edition)

^O Warning Properties of Industrial Chemicals, Occupational Health Resource Center, Oregon Lung Association.

ND = no data available

r = recognition threshold

d = detection threshold

GM = geometric mean

PART FIVE: ILLNESSES DUE TO ENVIRONMENTAL STRESSORS**High Altitude**

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ACUTE MOUNTAIN SICKNESS (AMS)

- Self-limited symptom complex.
- Universal susceptibility among the unacclimatized:
 - ◆ Apparently not influenced by physical fitness level.
 - ◆ Some soldiers inherently more susceptible than others.
 - ◆ Same symptoms can occur on repeated exposures.
 - ◆ Prior AMS at similar elevation is a predictor.
- Symptom onset 3-24 hours after rapid (<24 hours) ascent to above 6,000 ft (1,829 m):
 - ◆ Severity peaks at 24-72 hours.
 - ◆ Usually subsides over the course of 3-7 days.
 - ◆ Can occur after acclimatization with rapid ascent to higher altitude.
- Probable cause: hypoxia-induced subclinical cerebral edema.
- Often precedes both high altitude cerebral edema (HACE) and high altitude pulmonary edema (HAPE), but majority of cases do not progress to HACE or HAPE.
- Incidence and severity worsen with vigorous physical activity during ascent or during the first 1-3 days at altitude.

ALTITUDE	INCIDENCE (%)		
	MILD	MODERATE	SEVERE
~ 7,000 ft (2,130 m)	0-40	0-10	0
~10,000 ft (3,050 m)	60-70	0-40	0
~12,000 ft (3,660 m)	10-40	40-80	10-20
~14,000 ft (4,270 m)	20	60	20
~18,000 ft (5,500 m)	0	10	90

Symptoms/Signs (headache and nausea most common):

- Headache
 - ◆ Usually symmetric, nonfocal, throbbing.
 - ◆ Most intense at night and shortly after arising in the morning (increased hypoxemia caused by decreased ventilation during sleep).
 - ◆ Worsened by strenuous exercise, changes in position, valsalva.
 - ◆ Sometimes helped by *mild* exercise (increased ventilation).
 - ◆ Anorexia, nausea, and vomiting.

- ◆ Weakness, lassitude, general malaise.
- ◆ Decreased coordination, dizziness or light-headedness.
- ◆ Oliguria.
- ◆ Sleep disturbances and periodic breathing with recurrent apneic periods (may persist for weeks even after other symptoms have resolved).

Diagnosis:

- Presumptive: headache and at least one other sign or symptom in an individual who ascended from low (<5,000 ft or 1,524 m) to high altitude or from high to higher altitude in the previous 24-48 hours.
- Differential:
 - ◆ Viral gastroenteritis.
 - ◆ Hangover.
 - ◆ Exhaustion.
 - ◆ Dehydration.
 - ◆ CO poisoning.
 - ◆ HACE.

Prevention and Public Health Measures:

- Staging and graded ascent [above 2,500 m sleeping altitude should not be increased more than 2000 ft/day (600 m/day) with an extra day for increases of 2000-4000 ft (600-1200 m).
- Avoid over exertion.
- Acetazolamide 125-250 mg PO BID starting 24 hours pre-ascent, continue 48 hours post-ascent.
 - ◆ Prevents AMS in 50-75%; reduces symptoms in most of the other cases.
 - ◆ Side effects: peripheral paresthesias and polyuria; impairs physical endurance performance.
 - ◆ Contraindications: sulfa sensitivity.
 - ◆ High dose dexamethasone *not* recommended. Prevents symptoms of AMS; symptoms may recur when the drug is stopped.

Treatment:

- Descent to lower elevation.
- Portable hyperbaric chamber.
- Continuous supplemental oxygen.
 - ◆ sufficient quantities not usually available for tactical situations. Low-flow oxygen especially effective during sleep.
- Acetazolamide, up 500 mg PO TID.
- Dexamethasone, 2 to 4 mg PO Q6H (can be combined with acetazolamide treatment).
- Palliative care:
 - ◆ Analgesics (aspirin 325 to 1000 mg PO Q4-6H, acetaminophen 325 mg PO Q4H to 1000 mg PO Q6H, ibuprofen 200 to 800 mg PO Q4-6H, or other NSAIDs).
 - ◆ Opioids may be more successful, but they should *not* be used because of respiratory depression and reduction of cognitive function.
 - ◆ Prochlorperazine 5 to 10 mg PO Q6-8H can be used to treat nausea and vomiting. Respiratory stimulation a potentially beneficial side effect.
 - ◆ Alcohol and other respiratory depressants should be avoided.

HIGH ALTITUDE PULMONARY EDEMA (HAPE)

- Noncardiogenic pulmonary edema occurring in unacclimatized individuals following a rapid ascent to high altitude (>8000 ft; 2400 m).
- Combination of hypoxia-induced pulmonary hypertension and an increase in permeability of the pulmonary capillary endothelium; elevated pulmonary artery pressure, normal left atrial filling pressure and normal ventricular function.
- Untreated, can be rapidly fatal and is the most common cause of death among the altitude illness syndromes. If recognized early and treated appropriately, it usually resolves rapidly and without permanent adverse consequences.
- Often preceded by AMS; frequently seen in individuals with HACE; most cases of HAPE occur without concomitant HACE.
- Incidence relatively low, but military impact, especially in small units, can be significant because of the serious prognosis and need for rapid evacuation.

Incidence:

- Varies widely with geography, population at risk and the specific circumstances of exposure. Incidence estimates: 1/10,000 at 6-10,000 ft (1800-3000 m, Colorado skiers), 1/50 at 20,320 ft (6200 m, Mt. McKinley climbers), 15/100 at 11-18,000 ft (3300-5500 m, in Sino-Indian conflict).
- Increased risk in acclimatized individuals who reascend rapidly following several days to weeks at a lower altitude.
- Prior episodes may increase risk to as high as 60%.
- Subclinical form of HAPE occurs frequently:
 - ◆ Manifested primarily by rales in the right mid-lung field.
 - ◆ Rales found in 1/3 to 1/2 of persons exercising at altitudes higher than 11,500 ft (3500 m).
 - ◆ Clinical significance unclear (most do not progress to frank pulmonary edema).
 - ◆ Usually begins within the first 2-4 days after rapid ascent.

- ◆ Onset during the second night of sleep at altitude very common.
- ◆ Can also occur in acclimatized soldiers who ascend rapidly from a high to a higher elevation.
- ◆ Can progress very rapidly (<12 hours) to coma and death.

Risk Factors:

- Moderate to severe exertion.
- Cold exposure.
- Anxiety.
- Young age.
- Male sex.
- Obesity.
- Low hypoxic ventilatory response.
- Congenital absence of one pulmonary artery.
- Prior episode of HAPE.

Symptoms/Signs:

- Early pulmonary edema: nonproductive cough and a few rales (common at high altitude even without HAPE).
- Early hypoxemia: dyspnea on exertion, fatigue and weakness with decreased tolerance for physical activity, and increased time needed for recovery after physical exertion.
- Resting tachycardia and tachypnea greater than that induced by altitude alone.
- Nail beds and lips more cyanotic than others' at the same altitude.
- Cough may become productive of frothy, pink or blood-streaked sputum.
- Rales become more numerous and widespread, and wheezing may develop.
- Lung sounds may progress to an audible gurgling in the airway that can be heard without a stethoscope, especially when the affected person is supine.
- Orthopnea may occur in some individuals (<20%).
- Progressive hypoxemia causes progressive dyspnea and cyanosis.

- Mental status deteriorates with progressive confusion; sometimes vivid hallucinations. Ultimately obtundation, coma, and death will occur without treatment.
- Slight fever ($\leq 100^{\circ}\text{F}$, 37.8°C) may be present.
- Mild increase in white blood cell count.
- CXR shows multiple patchy interstitial or alveolar pulmonary infiltrates and may be predominant in the right middle lobe. Pulmonary vasculature may be widened, but heart size usually is normal.
- ECG shows right strain pattern with rightward axis, clockwise rotation, T-wave inversion in the precordial leads and an R-wave in leads V_{1-2} and an S-wave in leads V_{5-6} .

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Diagnosis:

- Presumptive:
 - ◆ Two of the following symptoms:
 - Dyspnea at rest
 - Cough
 - Weakness
 - Chest tightness or congestion
 - PLUS
 - ◆ Two of the following signs:
 - Rales or wheezing in at least one lung field
 - Central cyanosis or inappropriate tachypnea or tachycardia is present.
- Differential:
 - ◆ Pneumonia
 - ◆ Congestive heart failure
 - ◆ Pulmonary embolus
 - ◆ In a military setting, possible exposure to chemical warfare agents.

Prevention and Public Health Measures:

- Adequate acclimatization, avoidance of risk factors, and pharmacologic prophylaxis.
- Unacclimatized soldiers should sleep at as low an altitude as possible.
 - ◆ Avoid cold exposure and strenuous exertion until adequately acclimatized.
 - ◆ Acetazolamide may help prevent HAPE.

- Nifedipine 20 mg PO Q8H, starting on day of ascent, continuing thru 3 days at destination is indicated only for those with a history of prior episodes. Because hypotension is a possible side effect of this dose regimen, medical officers should consider administering a test dose or starting the regimen prior to ascent.

Treatment:

- Immediate descent is the definitive treatment, and should never be voluntarily delayed. Descent of even a few thousand feet (300-1,000 m) may be beneficial.
 - ◆ Descent should be by passive means (exertion, cold, and anxiety can increase pulmonary artery pressure).
 - ◆ Keep soldier as warm and comfortable as possible, and administer supplemental oxygen (4-6 L/min until improvement, then 2-4 L/min) during descent.
 - ◆ Soldiers with mild HAPE symptoms and who are ambulatory may walk down slowly.
 - ◆ **Soldiers with any altitude illness should not be unaccompanied.**
- Use portable hyperbaric chamber if descent not possible or oxygen unavailable (may require 4 or more hours of treatment in the chamber to be effective for HAPE).
- Increase beneficial effect and conserve oxygen supply by:
 - ◆ expiratory positive airway pressure mask (EPAP)
 - ◆ pursed-lips breathing
- Nifedipine: Start with a 10 mg immediate release capsule orally then extended release formulation 30 mg PO Q12-24H.

- After evacuation to lower altitude:
 - ◆ continue to ensure adequate oxygen and reduced pulmonary artery pressure
 - ◆ bed rest, supplemental oxygen and nifedipine. (Invasive procedures such as bronchoscopy or pulmonary artery catheterization are *not* indicated unless the clinical course deteriorates and the diagnosis is in doubt. Endotracheal intubation seldom necessary.)

HIGH ALTITUDE CEREBRAL EDEMA (HACE)

- Clinically apparent edema in the brain associated with a rapid ascent to high altitude:
 - ◆ hypoxia-induced increase in permeability of the blood-brain barrier (vasogenic edema), or
 - ◆ hypoxia-induced alteration of cellular fluid regulation with an intracellular fluid shift (cytotoxic edema), or
 - ◆ some combination of the two mechanisms.
- HACE patients often have HAPE (1/3 of patients), while most HAPE patients may not have concomitant HACE.
- Significant impact on military units operating at high altitude due to serious prognosis and the need for rapid evacuation.

Incidence:

- Occurs in unacclimatized individuals who ascend rapidly from low to high altitude, or from high to higher altitude.
 - ◆ Overall incidence (1% of rapid ascenders) lower than that of AMS or HAPE.
 - ◆ Majority of cases occur above 12,000 ft (3,600 m).
- Risk factors same as for AMS. AMS itself and previous episode of HACE are risk factors.
- Time of onset following high altitude exposure generally occurs later than AMS or HAPE. Mean duration of exposure before onset of HACE symptoms may be 5 days with a range of 1-13 days.
- Progression to death if untreated: 12-72 hours.

Symptoms/Signs:

- Early (AMS signs):
 - ◆ Severe headache, nausea, vomiting and extreme lassitude. None is invariably present.
 - ◆ Cyanosis and general pallor.
 - ◆ Mental status: confusion, disorientation, drowsiness, impaired mentation, withdrawal.
 - ◆ Truncal ataxia and change in mental status help differentiate early HACE from AMS. May progress to ataxic gait.
 - ◆ Coexisting HAPE symptoms.

- Later:
 - ◆ Visual changes, anesthetics, paresthesias, rigidity, hemiparesis, clonus, pathological reflexes, hyperreflexia, bladder and bowel dysfunction, hallucinations, seizures and coma.
 - ◆ Papilledema (up to half of soldiers with HACE).
 - ◆ Lumbar puncture and CT / MRI (not necessary for dx): elevated CSF pressure, cerebral edema.

Diagnosis:

- Presumptive: If a soldier does not have symptoms of AMS, both ataxia and mental status changes should be present for a presumptive diagnosis of HACE especially after the first couple of days of exposure.
- Differential: altitude-related stroke or transient ischemic attack, infection, migraine cephalgia, trauma, hypothermia, substance abuse, psychosis and severe cerebral hypoxia resulting from HAPE.

Prevention and Public Health Measures:

- No proven efficacy of preventive measures (low incidence precludes adequate studies).
- Based on hypothesis that AMS is a subclinical form of HACE, AMS preventive measures may prevent HACE.

Treatment (should be started on the basis of the presumptive diagnosis):

- Definitive treatment of HACE is immediate descent.
- Outcome improves with degree of descent; more than 1000 ft (300 m) may be needed for clinical improvement; descent to an altitude below 8000 ft (2400 m) is optimal.
 - ◆ Ambulatory patients can descend by foot if accompanied.
 - ◆ Portable hyperbaric chamber can be lifesaving when descent is unavoidably delayed (may require at least 6 hours of pressurization).
- Continuous supplemental oxygen at flow rates of 2 - 6 L/min should always be administered if available (but not a substitute for descent). Supplemental oxygen can be added to the air intake of a portable hyperbaric chamber to increase its efficacy.

- Adjunctive therapy: Dexamethasone 4-8 mg PO initially, followed by 4 mg PO, IV, or IM BID.
- Loop diuretics and osmotic diuretic agents such as mannitol, urea and glycerol also have been suggested, but experience is limited. (Careful attention must be paid to volume status when using diuretic agents in the treatment of altitude illness syndromes, because many soldiers will have altitude-induced decrease in intravascular volume concomitant with their edema.)
- Following descent, hospital management of HACE consists of supplemental oxygen, dexamethasone, supportive care, and possibly also diuretic agents. Comatose patients may require intubation with hyperventilation and bladder catheterization.
- Evaluate for concomitant HAPE and treat immediately if present.

HIGH ALTITUDE PERIPHERAL EDEMA

Incidence:

- May occur in up to 1/3 of soldiers who ascend to high altitude.

Symptoms/Signs:

- Most evident in the hands and peripheral areas of the face, and most evident upon awakening.
- Benign, but may cause soldiers enough discomfort to degrade their performance to some degree; more common in females.
- Usually associated with decreased urine output and a weight gain of approximately 6-12 pounds.

Diagnosis:

- Presumptive:
 - ◆ Diagnosis can often be made by history alone because it tends to recur consistently with repeat ascents.
- Differential:
 - ◆ Differential diagnosis includes cardiogenic edema, allergic reactions, and edema of the upper extremities caused by pack straps or binding by tight clothing.

Treatment:

- Can be treated successfully with mild diuretics.
- Definitive treatment is descent to a lower elevation.

HIGH ALTITUDE RETINAL HEMORRHAGE (HARH)

Incidence:

- Areas of bleeding from retinal vessels during altitude exposure.
- Can be found in association with other altitude illness syndromes, but are not directly related to them.
- Usually asymptomatic and affects military operations only in the rare instance in which they affect an individual soldier's vision (i.e., hemorrhage into the macular area).
- Appears not to be related to the state of acclimatization, and multiple incidents of retinal hemorrhage are possible throughout any altitude deployment.

Symptoms/Signs:

- Fundoscopic exam will show hyperemia and engorgement of the disc and increased tortuosity of retinal vessels.
- Retinal hemorrhages appear as “splinter” and “flame” type hemorrhages in the superficial layers of the retina, but hemorrhages in the deeper layers can occur.

Diagnosis:

- Differential diagnosis includes hemorrhage from vascular disease, diabetes mellitus, septic infarcts or from hypoxia caused by cardiac and respiratory disease.

Treatment:

- Self-limited resolve 1-2 weeks after descent; descent is not necessary for hemorrhages outside of the macula.
- When a macular hemorrhage is diagnosed, descent is imperative to promote healing and prevent further hemorrhage.

THROMBOEMBOLIC EVENTS

- Soldiers who ascend to high altitude are at increased risk for thromboembolic events including: thrombophlebitis, deep venous thrombosis, pulmonary embolus, transient ischemic-attacks (TIA) and stroke.
- Unusual below 14,000 ft (4,300 m).
- Treatment follows standard clinical guidelines, including appropriate anticoagulation. In a field setting, low-dose subcutaneous heparin (5,000 units every 8–12 hours) can be used for anticoagulation prior to and during evacuation.

HIGH ALTITUDE PHARYNGITIS AND BRONCHITIS

- Frequent during prolonged stays (>2 wks) at high altitude; common at altitudes over 18,000 ft (5500 m).
- Sore throat, chronic cough and severe cough spasms provoked by exercise are the primary manifestations.
- Although desiccation of mucous membranes can lead to an increased number of upper respiratory infections, pharyngitis and bronchitis at high altitude are seldom due to infection.
- The impact of altitude-related pharyngitis and bronchitis on military operations is related primarily to the discomfort it causes to individual soldiers. Cough spasms could cause soldiers to be easily detected by opposing forces in some tactical situations.
- Treatment involves ample hydration, steam inhalation, hard candies or soothing lozenges and a mild cough suppressant.
- A mask or a porous, breathable silk balaclava as a mouth covering to reduce respiratory heat and moisture loss.
- Decongestant nasal sprays may relieve cold-induced vasomotor rhinitis and lessen mouth breathing.

MEDICATIONS FOR TREATING ALTITUDE ILLNESSES

MEDICATION	INDICATIONS	DOSAGE	COMMENTS
oxygen	Severe AMS headache, cyanosis HAPE HACE	2-6 L/min 2-6 L/min 2-6 L/min	DO NOT DELAY DESCENT DO NOT DELAY DESCENT
acetazolamide	AMS prevention AMS treatment sleep disorders peripheral edema	125 mg PO QID or 250 mg PO BID, Starting 48 H before ascent, continuing for 48 H after ascent. 125 mg PO QID or 500 mg PO TID 250 mg PO TID or 250 mg PO QID 250 mg PO TID for 3 doses	Side effects: parasthesias, fatigue, altered taste. Contraindicated with sulfa sensitivity.
dexamethasone	AMS treatment HACE	2-4 mg, PO QID 4-6 mg Q6H PO, IM or IV	For <u>severe</u> AMS Only DO NOT DELAY DESCENT Few side effects if used only 3-4 days
acetaminophen	AMS headache	325 mg PO Q4H to 1000 mg PO QID	

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MEDICATION	INDICATIONS	DOSAGE	COMMENTS
ibuprofen	AMS headache	200-800 mg PO TID Or QID	Other non-steroidal anti-inflammatories Stomach irritation
aspirin	AMS headache	325-1000 mg PO Q4-6H	Stomach irritation
	superficial thrombophlebitis	325-1000 mg PO Q4-6H	
heparin	thromboembolism deep venous thrombophlebitis	PTT adjusted to 2-3 INR; 5000 U SC, Q8-12H in the field	REQUIRES EVACUATION
triazolam	insomnia	0.125 mg, PO QHS	Short-term use only. Possible short-term memory loss
temazepam	insomnia	30 mg, PO QHS	
nifedipine	HAPE treatment	10 mg sublingually, followed by 30 mg PO QID	
	HAPE prevention	20 mg PO TID started, 24 H before ascent, continuing Q8H until 72 H after ascent	
prochlorperazine	nausea/vomiting	5-10 mg Q6-8H PO or IV; or 25 mg PO BID	Also stimulates respiration

FREEZING INJURY (FROSTBITE)

- Most freezing injuries will be recognized and initially managed by nonphysician medical providers.
- Initially, all frozen tissue has the same appearance: cold, hard, and bloodless.
- Digits, nose, ears, and face are the most commonly affected areas.
- Many feel pain as skin temperature falls but ignore it as a warning sign.

Symptoms/Signs:

- First degree frostbite
 - ◆ minor injury to superficial skin
 - ◆ blanches white, thaws quickly, forms red, painful wheal, no blisters; desquamation of skin in 7-10 days
- Second degree frostbite
 - ◆ whole epidermis involved
 - ◆ limited motion over site of injury
 - ◆ blisters form, clear fluid confirms 2° injury
 - ◆ blood in blister means deep 2° or early 3° injury
 - ◆ tissue under blisters susceptible to infection, leave blister intact
- Third degree frostbite
 - ◆ dermis to reticular layer involved
 - ◆ tissue is white, hard, and immobile
 - ◆ deep tissues cyanotic
 - ◆ skin loss through sloughing and mummification, healing slow
 - ◆ residual cold sensitivity is common
- Fourth degree frostbite
 - ◆ involves skin, underlying tissues, sometimes bone
 - ◆ with rewarming, no blister formation distal but blisters may occur proximal in less damaged tissue
 - ◆ rewarming brings significant pain
 - ◆ permanent anatomic and functional loss
- Corneal frostbite is a rare, but profoundly disabling injury.

The evolution is similar to any deep ocular keratitis. Permanent corneal opacification requiring corneal transplant is a common outcome.

First Aid and Field Management:

- It is important to remember that active warming of frozen tissue should be deferred until there is no risk that injured tissue will be refrozen. Once tissue has thawed, it is absolutely essential that it be protected from re-exposure to cold.
- If refreezing can be prevented during evacuation, then frozen tissue can be immediately warmed by contact with warm skin. The groin or axillae are particularly effective areas for warming frozen tissue.
- Tissue must not be exposed to temperatures in excess of 102-103°F which will aggravate the injury. Do not expose tissue to motor engine manifolds or exhaust, hot water, open flames, stove tops, or incandescent bulbs. Many frostbite injuries have been substantially worsened by exposure to inappropriate warming techniques.
- Frostbitten tissue is vulnerable to trauma and infection and should be carefully protected from physical injury during evacuation.

Hospital Management:

- Warming of still-frozen tissue, treatment of various phases, of the injury as it evolves, and evaluation for coincident injury and illness. If the tissue has already thawed on arrival at the MTF, additional active warming should not be done.
- Digits or entire hands or feet can be warmed in a temperature monitored water bath kept at 102-104°F. The face or ears can be warmed by towels kept wet with water warmed to 102-104°F. Warming should be continued until no further improvement in circulation and mobility is noted. This usually requires 15-45 minutes depending on the initial temperature and size of the injured part.
- After warming, the frostbitten tissue should be carefully and atraumatically dried, completely covered in bulky dry dressings, and kept slightly elevated to decrease swelling.
- Establish IV access (dehydration and hypovolemia are

common with significant cold injuries and cold exposure).

- Tetanus prophylaxis as appropriate (frostbite is a tetanus-prone wound).
- Analgesia should be provided with NSAIDs (may reduce post-injury ischemia) and narcotics as needed.
- Because wound anaerobes and streptococci appear to be early causes of post-injury infection, prophylactic penicillin is recommended (2-4 M units IV OD or 500 mg PO QID).
- In second, third and fourth degree injuries, necrotic tissue is usually removed by whirlpool debridement once or twice daily in skin temperature saline or dilute Betadine.

For complete guidance please see TB MED 508, Prevention and Management of Cold-Weather Injuries (www.usariem.army.mil/download/tbmed508.pdf).

NON-FREEZING COLD INJURY (NFCI, IMMERSION INJURY)

- Results from prolonged (hours/days) exposure to wet-cold; but above freezing conditions.
- The feet are the most common area of injury, but injuries can occur to hands.
- Two principal types: trench foot (combined effects of sustained cold exposure and restricted circulation during ground operations); and immersion foot (continuous immersion in cold water).
- Injured tissue is pale, anesthetic, pulseless and immobile.
- Diagnosis confirmed when above signs and symptoms do not change after warming.
- Skin is frequently macerated and slightly edematous. The degree of injury is not completely apparent early on.
- After several hours (occasionally as long as 24-36 hours), a marked hyperemia develops associated with severe burning pain and reappearance of sensation proximally, but not distally; blanches with elevation; lasts a few days to many weeks depending on the severity of the injury.
- Edema and blisters develop in the injured areas as perfusion increases. Poorly perfused skin will slough.
- Persistence of no pulse in an extremity after 48 hours suggests severe deep injury and high likelihood of substantial tissue loss.
- Boots and socks should not be replaced until feet are warm and have normal feeling.
- Injured extremity must be carefully protected during evacuation.
- Severe pain may develop during evacuation if warming occurs, but extremity should not intentionally be deprived of passive warming (dry covering and protection from cold). Soldiers performing the evacuation should be equipped and trained to provide adequate analgesia.
- Do not massage or actively warm extremity.
- NFCIs should be evacuated to a rear echelon hospital.

- The skin should be protected with dry dressings. Intact blisters should be left intact; ruptured blisters should be sharply debrided and dressed. Open blisters, ulcers, and areas of necrosis should be periodically monitored with surveillance aerobic cultures.
- Infections should be treated immediately with antibiotics.

For complete guidance please see TB MED 508, Prevention and Management of Cold-Weather Injuries (www.usariem.army.mil/download/tbmed508.pdf).

HYPOTHERMIA

- “Core” temperature (clinically usually taken to be the same as rectal temperature) is below 95°F (35°C). Hypothermia is caused by greater heat loss to the environment in excess of heat production by the body.
- The principal manifestations of mild to moderate hypothermia are shivering and mental status change.
- Persistent shivering is evidence of incipient hypothermia and should always be taken seriously. Shivering will diminish as hypothermia worsens.
- Mental status change may be the only clinical evidence of significant hypothermia. Withdrawal and irritability are common. As hypothermia worsens, subtle mental status changes progress to frank confusion, lethargy, withdrawn behavior, and obtundation. The degree of mental status change is not a reliable guide to the degree of hypothermia.

Clinical Manifestations:

- 90-95°F (32-35°C)
 - ◆ Mild hypothermia
 - ◆ Shivering and vasoconstriction present
 - ◆ Impaired fine and gross motor skills
 - ◆ Mental processes slow, errors in judgment
 - ◆ Bradycardia with PVCs possible
- 82.5-90°F (28-32°C)
 - ◆ Moderate hypothermia
 - ◆ Shivering and vasoconstriction attenuated or gone
 - ◆ Lethargy, staggering gait
 - ◆ Atrial arrhythmias
 - ◆ Bradycardia
 - ◆ Hypopnea
- < 82.5°F (< 28°C)
 - ◆ Severe hypothermia
 - ◆ Absent shivering or vasoconstriction
 - ◆ Loss of consciousness
 - ◆ Muscles and joints rigid
 - ◆ Vital signs reduced or absent
 - ◆ Risk of ventricular fibrillation/cardiac arrest
 - ◆ Core temperature below 77°F (25°C) – spontaneous ventricular fibrillation

PATIENT ISN'T DEAD UNTIL WARM & DEAD

Field Management:

- Anyone suspected of hypothermia should be considered to be at risk of sudden death from ventricular fibrillation or hypotension, and steps should be taken to prevent those complications. Handling should be minimal and gentle.
- Copious insulation to prevent heat loss (incompressible material under casualty if possible).
- Protection from wind and wet. Get wet clothes off.
- When indicated, endotracheal intubation is safe as it does not seem to increase the risk of ventricular fibrillation.
- Oxygen supplementation is usually not needed because of the low oxygen requirements in hypothermia; ventilation can be assisted by mask and bag.
- Treat dehydration and hypovolemia (common in hypothermic casualties).
- Victims of severe hypothermia often appear to be in cardiac arrest; they are unconscious and without perceptible signs of life. Initiating CPR in the absence of solid clinical indications may unnecessarily result in conversion of perfusing and quiet bradycardia ventricular fibrillation. Use cardiac monitoring if available. If not, then take extra time to assess pulse, which may be slow and difficult to appreciate.
- Give glucose/sugar if available.

Hospital Management:

- Casualties who have stable circulation and only mild to moderate degrees of hypothermia can be given the chance to rewarm spontaneously. If they fail to rewarm spontaneously, then active rewarming should be started. They should be admitted to an intensive care unit be given warmed humidified oxygen and gradually hydrated.
- Although virtually every cavity in the body has been lavaged with warm fluid for the treatment of hypothermia (stomach, urinary bladder, colon, abdomen and chest),

the two most common, use gastric lavage and intraperitoneal lavage using peritoneal dialysis equipment. These techniques are effective. They raise core temperature 0.5 to 1.5°F per hour.

- Use heated systems if available.
- The following steps are suggested for the management of hypothermic cardiac arrest. Endotracheal intubation, if not already done in the field, should be done, and assisted ventilation begun with heated humidified oxygen. Since hypothermic resuscitation tends to be prolonged, early institution of mechanical compression and ventilation is appropriate. In the hypothermic casualty, oxygen requirements and carbon dioxide production will be low. Ventilation needs to be guided by blood gas measurements to avoid excessive respiratory alkalosis. Blood gas measurements can be interpreted as reported by the laboratory; temperature “correction” is no longer considered appropriate.
- As a general rule, antiarrhythmic and vasoactive drugs are not useful during resuscitation from hypothermia until core temperatures exceed 90°F. Below that temperature, drug effects are absent or unpredictable. Also, since drug metabolism is markedly slowed below 90°F, applying American Heart Association Advanced Cardiac Life Support (ACLS) drug protocols in hypothermia causes the accumulation of drugs which have no manifest effect when administered, but which suddenly and dramatically express themselves as they regain activity at higher core temperatures. If drug therapy of ventricular fibrillation is required, bretylium at its usual doses would be the drug of choice.

For complete guidance please see TB MED 508, Prevention and Management of Cold-Weather Injuries
(www.usariem.army.mil/download/tbmed508.pdf).

HEAT CRAMPS

- Patients with heat cramps present with painful tonic contractions of skeletal muscle.
 - ◆ can occur during work or many hours after work
 - ◆ usually preceded by palpable or visible fasciculation and lasts 2-3 minutes
 - ◆ recurrent and may be precipitated by manipulation of muscle
 - ◆ involve the voluntary muscles of the trunk and extremities
 - ◆ no systemic manifestations except those attributable to pain
 - ◆ despite the salt depletion associated with heat cramps, frank signs and symptoms of heat exhaustion are unusual
 - ◆ no significant complications have been reported from heat cramps except muscle soreness
- Differential diagnosis:
 - ◆ tetany due to alkalosis (hyperventilation, severe gastroenteritis, cholera) or hypocalcemia
 - ◆ compartment syndrome
 - ◆ strychnine poisoning
 - ◆ black widow spider envenomation
 - ◆ abdominal colic

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Management:

- Replenish salt orally or parenterally.
 - ◆ Response to therapy is sufficiently dramatic to be valuable in the differential diagnosis.
 - ◆ Route of administration determined by the urgency of symptom relief.
 - ◆ See Preparation of 0.1 Percent Salt Solution: Two Methods.
 - ◆ Salt tablets should not be used as an oral salt source.

HEAT EXHAUSTION

- Presenting complaints: weakness, thirst, syncope, fatigue, nausea, vomiting, hyperventilation, dyspnea, muscle cramps, confusion, anxiety and agitation, mood change, orthostatic dizziness, ataxia, hyperthermia and frontal headache.
- Frequently superimposed on other conditions that increase circulatory load, such as febrile illness, or produce fluid-electrolyte losses, such as gastroenteritis.
- Rectal temperature should be frequently monitored to ensure that core temperature is falling to normothermic levels.
- The management of heat exhaustion is directed to correcting the two pathogenic components of the illness: excessive cardiovascular demand and water-electrolyte depletion. The load on the heart is reduced by rest and cooling. Water-electrolyte depletion is corrected by administering oral or parenteral fluids.

Heat exhaustion casualties retain the ability to cool spontaneously if removed from the stressful circumstances. However, spontaneous cooling is necessarily observed only **AFTER** cooling has occurred. Casualties with incipient heat stroke and heat exhaustion are hard to distinguish initially. **THEREFORE, ACTIVE COOLING SHOULD BE PROVIDED FOR ALL CASUALTIES WHO ARE AT RISK FOR HEAT STROKE.**

- Any loss of consciousness and mental status changes need to be documented. Such patients need to be evaluated as potential heat stroke cases.
- Normal saline should initially be given in 200-250 mL boluses to an amount sufficient to restore normal circulatory function. No more than 2 liters of NS should be administered without laboratory surveillance if laboratory support is available. Subsequent parenteral fluid replacement should be D5/O.5 NS or D5/O.2 NS.
- A single episode of heat exhaustion does not imply any predisposition to heat injury. Repeated episodes of heat exhaustion require thorough evaluation.

For complete guidance see TB MED 507/AFPAM 48-152(I), Heat Stress Control and Heat Casualty Management (www.usariem.army.mil/download/tbmed507.pdf).

HEAT STROKE

At presentation, the distinction between heat exhaustion and incipient heat stroke is frequently impossible. Individuals who do not respond dramatically to rest and fluid-electrolyte repletion should be observed for 24 hours with laboratory surveillance for the delayed complications of heat stroke. Coagulopathy, persistent encephalopathy or persistent elevation of body temperature suggest the probability of severe heat stroke. Immediate institution of active cooling and evacuation to a rear echelon hospital is required. Active cooling should be continued throughout evacuation.

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Incidence:

- Heat stroke occurs in two settings sufficiently different to produce different clinical pictures and management. The primary clinical difference between the two is that exertional heat stroke is complicated by acute rhabdomyolysis with consequent renal failure.
 - ◆ “Classical” heat stroke occurs in individuals, frequently with impaired thermoregulation due to illness or medication, exposed passively to heat and dehydration. It is principally an epidemic affliction of young children and elderly occurring during urban heat waves.
 - ◆ “Exertional” heat stroke occurs in physically active individuals experiencing substantial endogenous heat loads.
- Encephalopathy is a sine-qua-non of heat stroke. Its presentation ranges from loss of consciousness and confusion to seizures or coma with decerebrate rigidity. The etiology of encephalopathy is not known.
- Coagulopathy due to DIC is common. The principal causes of DIC seem to be thermal damage to endothelium, rhabdomyolysis, and direct thermal platelet activation causing intravascular microthrombi. Fibrinolysis is secondarily activated. Hepatic dysfunction and thermal injury to megakaryocytes slows the repletion of clotting factors.
- Hepatic injury is common. Transaminase enzyme elevation, clotting factor deficiencies, and jaundice can be seen in the course of heat stroke.

- Renal failure following heat stroke can be caused by several factors: myoglobinuria from rhabdomyolysis in exertional heat stroke, acute tubular necrosis due to hypoperfusion, glomerulopathy due to DIC, direct thermal injury and hyperuricemia.
- Rhabdomyolysis is a frequent acute complication of exertional heat stroke. Acute muscular necrosis releases large quantities of potassium, myoglobin, phosphate, and uric acid, and sequesters calcium in the exposed contractile proteins.

If heat stroke is suspected and temperature is elevated, cooling should not be delayed to accomplish a diagnostic evaluation. Cooling and evaluation should proceed simultaneously.

Diagnosis:

- Heat stroke presents as collapse with variably severe encephalopathy and hyperthermia. There may be clinical evidence of dehydration, coagulopathy or shock.
- Laboratory evaluation should be directed by the differential diagnosis appropriate for the clinical circumstances. Patients with heat stroke require serial monitoring of platelets and plasma clotting factors, renal and hepatic function, and electrolyte and acid-base status.
- Differential includes: infection (particularly meningococemia and *P. falciparum* malaria), pontine or hypothalamic hemorrhage, drug intoxication (cocaine, amphetamines, phencyclidine, theophylline, tricyclic antidepressants), alcohol or sedative withdrawal, severe hypertonic dehydration, and thyroid storm.

The patient with heat stroke requires early evacuation to medical facilities with intensive care capabilities. Active cooling should be started immediately and continued during evacuation.

Treatment:

- Clinical outcome of patients with heat stroke is primarily a function of magnitude and duration of temperature elevation.
 - ◆ Most important therapeutic measure is rapid reduction of body temperature.

- ◆ Any effective means of cooling is acceptable.
- Immersion in cool or iced water with skin massage is a classic technique for cooling heat stroke patients. Both have demonstrated effectiveness in lowering body temperature. Ice water probably produces the most rapid rate of cooling, but is uncomfortable and often difficult to obtain.
 - ◆ In hot dry environments, construct field expedient immersion baths by:
 - digging plastic-lined shaded pits (The water is cooled by contact with cool subsurface sand and surface evaporation.), or
 - rigging shallow canvas tubs in elevated frames in ventilated shade (The water is cooled by evaporation from the wetted canvas surface. In the case of canvas tubs, the water can cool to nearly the atmospheric dew point temperature, often as low as 50°F in deserts.)
 - ◆ If immersion devices not prepared in advance, cool water can be kept in Lyster bags.
- Cooling can also be accomplished by wetting the body surface and accelerating evaporation by fanning. The water can be applied by spraying or by application of thin conforming cloth wraps (sheets, cotton underwear). (Circulating cooling blankets—unlikely to be available in the field situation—will also lower body temperature. Although cooling blankets have the advantage of maintaining a dry working environment, their limited contact surface provides slower cooling than immersion or surface wetting techniques. Their best use is probably maintaining normal body temperature in the period after resuscitation and rapid cooling where temperature instability is characteristic.)
- Invasive cooling techniques (e.g., ice water lavage or enemas, peritoneal lavage with cool fluids) are NOT recommended because they do not provide faster cooling, and they do have associated complications.
- Rectal temperature should be closely monitored during active cooling; discontinue cooling at 38.3°C to avoid hypothermia.
- Heat stroke patients usually do not require aggressive fluid resuscitation.
 - ◆ Fluid requirements of 1 to 1.5 liters in the first few hours are typical. FLUID OVERLOAD MUST BE AVOIDED.

- ◆ Since heat stroke patients are frequently hypoglycemic, the initial fluid should include dextrose.
- Airway control is essential. Vomiting is common.
 - ◆ Endotracheal intubation should be used in patients who cannot adequately protect their airways.
 - ◆ Supplemental oxygen should be provided when available.
 - ◆ Nasogastric intubation to control vomiting should be done as soon as practicable.
- Patients are frequently agitated, combative or seizing. Valium is effective for control and can be administered intravenously, endotracheally or rectally.
- Hyperkalemia is the most life threatening early clinical problem.
 - ◆ Measurement of plasma $[K^+]$ is an early priority when available. Tall T-waves on the surface ECG are consistent with hyperkalemia but not definitive. The interpretation of plasma $[K^+]$ early in the clinical course of heat stroke is difficult due to confounding electrolyte and acid-base disturbances.
 - ◆ Clinically significant hyperkalemia is manifested by electrocardiographic changes including increased T-wave amplitude, slowed A-V conduction with widening of the P-R interval, diminishing P-wave amplitude and "sine wave" ventricular rhythms.
 - ◆ Hyperkalemia greater than 6.5 meq/L or with electrocardiographic changes should be treated. Glucose (50 gm slow IV), insulin (20 units of regular insulin IV) and sodium bicarbonate (1-2 amps IV) will lower plasma $[K^+]$ within minutes. Serious ventricular dysrhythmia should be treated with IV calcium chloride (1-2 amps).
 - ◆ Cardiac monitoring and electrocardiography can be used to supplement laboratory monitoring for changes in plasma potassium (T-wave amplitude) and calcium (QT interval).
- Acute renal injury is common in exertional heat stroke.
 - ◆ Urinary catheterization to monitor urine output and obtain urine for $[Na^+]$ should be done early.

- ◆ Early management of suspected acute renal failure should include assuring adequate renal perfusion and mannitol (12.5-25 gm IV).
- After cooling and hemodynamic stabilization, continuing care is supportive and is directed at the complications of heat stroke as they appear.
 - ◆ Patients with heat stroke frequently have impaired temperature regulation for several days with alternate periods of hyperthermia and hypothermia. Constant monitoring is essential and clinically significant deviations in temperature may require either cooling or warming measures.
- The effects of rhabdomyolysis that require management are renal injury due to myoglobinuria and hyperuricemia, hyperkalemia, hypocalcemia and compartment syndromes due to muscle swelling. Assurance of adequate renal perfusion and urine flow will moderate the nephrotoxic effects of myoglobin and uric acid.
 - ◆ Hyperkalemia can be managed by Kayexalate or dialysis.
 - ◆ Hypocalcemia does not usually require treatment.
 - ◆ Increasing tenderness or tension in a muscle compartment may represent increasing intracompartmental pressures. Direct measurement of intramuscular pressure or fasciotomy should be considered at this point. Pain and paresthesias may not signal the compartment syndrome until permanent damage has occurred.
- Prognosis is worse in patients with more severe degrees of encephalopathy. Permanent neurologic sequelae can develop after heat stroke including cerebellar ataxia, paresis, seizure disorder, and cognitive dysfunction.
 - ◆ Management of encephalopathy is supportive, directed at minimizing cerebral edema by avoiding fluid overreplacement and assuring hemodynamic, thermal and metabolic stability. Intravenous mannitol has been used to treat life threatening cerebral edema if renal function is adequate. The efficacy of dexamethasone for treating heat stroke induced cerebral edema is not known.

- ◆ Neurologic deterioration after initial recovery may represent intracranial hemorrhage related to DIC or hematoma related to trauma unrecognized at the time of initial presentation.
- Subclinical coagulopathy does not require active management, but clinically significant bleeding is an ominous sign. Treatment is directed at reducing the rate of coagulation and replacement of depleted clotting factors.
- Other complications include gastrointestinal bleeding, jaundice due to hepatic injury, aspiration pneumonia, noncardiogenic pulmonary edema, and myocardial infarction. Immuno-incompetence and infection are late complications, particularly in patients with severe renal failure.

MEDICATIONS REPORTED TO INCREASE HEAT ILLNESS RISK

Anticholinergics Atropine Scopolamine	Antihistamines Diuretics Tricyclic antidepressants Major tranquilizers	Amphetamines Cocaine Ergogenic aids (especially those containing ephedrine) Alcohol Beta-blockers

For complete guidance see TB MED 507/AFPAM 48-152(I), Heat Stress Control and Heat Casualty Management (www.usariem.army.mil/download/tbmed507.pdf).

PREPARATION OF 0.1 PERCENT SALT SOLUTION: TWO METHODS

- Add table salt directly to drinking water using any of the following proportions:

1 1/2 level mess kit spoons dissolved in 5-gallon can

9 level mess kit spoons dissolved in Lyster bag

1 level canteen cup dissolved in 250-gallon water trailer

- Prepare a saturated salt solution (approximately 26%) and add specific quantities of this 26% saturated salt solution to drinking water to make a 0.1% salt solution:

Saturated salt solution is made by adding 9 level tea-spoons of table salt to 2/3 of a canteen cup of water. Saturated salt solutions are NOT safe to drink. Be sure saturated salt solutions are properly diluted.

0.1% salt solution can be made using saturated salt solution added to plain, potable water in any of the following proportions:

1/8 canteen cap (1 qt size) added to 1 quart canteen of water

1/4 canteen cap (2 qt size) added to 2 quart canteen of water

1 mess kit spoonful added to 1-gallon can of water

5 mess kit spoonfuls added to 5-gallon can of water

1/2 canteen cup added to Lyster bag

4 canteen cups to 250 gallon water trailer

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PART SIX: DEPLOYMENT DERMATOLOGY
(see also appendices G thru I)

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TERMINOLOGY

- Macule: small (<1 cm) flat colored lesion (e.g., purpura, ecchymoses, lentigenes).
- Patch: large (>1 cm) flat lesion (e.g., tinea versicolor).
- Papules: small (<1 cm) raised lesion (e.g., acne "pimples").
- Maculopapular: an eruption with both macules and papules (e.g., many viral exanthems).
- Plaques: large (>1 cm) raised lesion (e.g., psoriasis). Has a large surface area in compared to its height.
- Nodules: large (>1 cm), deepset, palpable, solid, round or ellipsoidal lesion (e.g., deep tumors).
- Vesicles: small (<1 cm), circumscribed, elevated lesion that contains fluid. Walls are thin (translucent).
- Bullae: large (>1 cm) same as vesicle.
- Pustules: like a papule (raised, <1 cm) lesion that contains pus.
- Erosions: moist, circumscribed, usually depressed lesion that results from loss of epidermis.
- Ulcer: lesion which there has been destruction of the epidermis and at least the upper dermis.
- Cysts: a sac that contains liquid or semisolid material (e.g., epidermal inclusion cyst).
- Atrophy: diminution in the size of a cell, tissue, organ, or part of the body (dermal or epidermal).
- Sclerotic: circumscribed or diffuse hardening or induration of the skin.
- Scale: abnormal shedding or accumulation of stratum corneum in perceptible flakes.
- Crusts: results when serum, blood or purulent exudate dries on the skin surface.
- Necrotic: dead tissue, usually sharply demarcated, black.
- Excoriations: superficial excavations of the epidermis resulting in linear or punctate abrasions/ulcerations.
- Fissures: linear cleavages or cracks in the skin that are deep and usually painful.

DERMATOLOGIC EXAMINATION

■ History

- ◆ Chief Complaint: Onset (when and where), Evolution (of individual lesions, spread), Duration, Associated Symptoms (pruritus, pain, paresthesia), Aggravate/alleviate (seasonality, menses, temperature, sunlight), Treatment
- ◆ Prior history: PMH, Allergies (medicines and other), Exposures (occupational, hobbies, soaps, travel), ROS
- ◆ Meds: Rx, OTC, topical, oral
- ◆ Family: hereditary conditions, atopy

■ Examination

- ◆ Patient's general appearance and temperature
- ◆ Number of lesions - single, few, abundant, innumerable
- ◆ Primary lesion
 - Type - macule, papule-plaque, patch, nodule, vesicle-bulla, pustule, telangiectasia, purpura, cyst
 - Shape - annular, polygonal, round, oval, arciform, linear, iris/targetoid, umbilicated, reticulate
 - Borders - serpiginous, discrete, confluent, demarcated
 - Color - uniform, variable, circumscribed, blanching
 - Palpation - consistency, temperature, mobility, tenderness, depth
- ◆ Distribution
 - Number of lesions - single, local, regional, generalized, universal, grouped
 - Pattern (symmetric, random (scattered) or focal (e.g., sites of pressure or exposure, intertriginous, extensor surfaces, follicular, acral, palms and soles, truncal, extremities)
- ◆ Secondary lesions - crust, weeping, scale, pustule, erosion, fissure, atrophy, excoriation, ulcer, lichenified, scarred, sclerosis
- ◆ Comment when appropriate on mucosa, hair, nails, teeth, eyes, joints, lymph nodes, sensation, ability to sweat

■ Laboratory examinations

- ◆ Microbiology - Wood's lamp, KOH, Tzanck, Gram stain, culture, darkfield, scabies scraping
- ◆ Blood – rapid plasma reagin (RPR), CBC with differential, immune serologies, chemistries, TFTs
- ◆ Patch testing
- ◆ Biopsy
- ◆ Other - radiographic, stool, urinalysis

ECZEMAS AND DERMATITIS (two names for the same thing)

Four components: red, scaly, itchy, vesicular. Vesicles may be clinically obvious (as in poison ivy), oozing (as in dyshidrotic eczema), or microscopic (as in nummular dermatitis).

- **Atopic Dermatitis** - common, chronic, dry, scaly, itchy eruption on the scalp, face, flexor areas of the extremities, that can also be generalized. This condition is usually worsened by overbathing with drying soaps; or exposure to allergens, chemicals or wool; and possibly stress.

- ◆ **Topical Management**

- Limit bathing to brief bath or shower (less than 5 minutes with cool water) once daily or every other day. Explain that the more water that one is exposed to - or the hotter the water - the worse the skin will become.
- Use a mild soap (e.g., Dove, Tone, Basis, Cetaphil). If someone is prone to recurrent impetiginization (superficial bacterial infection) of their skin, an antibacterial soap (e.g., Lever 2000) can be used.
- Lubricate the skin often. Apply ointments such as Vaseline, Aquaphor, Eucerin, or even Crisco shortening. Ointments are better than creams which are better than lotions. The simpler (and often cheaper) the moisturizer the better, as the addition of fragrances, colorizers, preservatives may irritate the skin.
- For oozing lesions, first dry out the eruption using aluminum acetate astringents (e.g., Domeboro's soaks) TID. Use two tablets in a quart of warm water and apply with a clean washcloth. Be sure to rinse the skin with plain water afterward to avoid overdrying. Stop using the soaks when the oozing stops. Steroid creams can be applied to oozing areas and ointments may be resumed once the oozing stops.

- ◆ Facial Eczema and Intertriginous Areas
 - Hydrocortisone 1% (hydrocortisone 2.5% or desonide if eczema is severe) cream or ointment BID.
 - For sensitive facial skin, you can try pramasone lotion 1% or 2.5% (doesn't contain some of the potentially irritating substances usually found in creams and lotions).
- ◆ Body Eczema (except intertriginous areas)
 - Triamcinolone 0.1% cream or ointment (low-mid potency steroid) QD – BID or Hydrocortisone valerate 0.2% (this is stronger than plain hydrocortisone, even though it is a lower percentage – a fact that often confuses patients).
 - Fluocinonide cream or ointment (mid-high potency steroid) QD to BID for stubborn plaques.
- ◆ Antihistamine Therapy
 - Sedating antihistamines probably just makes patient too sleepy to know that he or she is itchy: hydroxyzine (Atarax) 10-25 mg PO QHS; diphenhydramine (Benadryl) 25-50 mg PO QHS; chlorpheniramine (Chlortrimaton) 4-8 mg PO QHS.
 - Nonsedating forms do not help significantly.
- ◆ Antibiotics - Only if 2° infection
 - Systemic - dicloxacillin, cloxacillin, cephalexin for staph and strep coverage.
 - Mupirocin (Bactroban) ointment TID.
- ◆ Other topical antipruritic preparations one can use prn
 - Camphor/menthol preparations (Sarna, Sarnol, Vick's Vaporub); pramasone (Prax).
 - Avoid Caladryl because topical Benadryl portion may cause allergic sensitization.
 - Avoid topical benzocaine because this also induces frequent allergic contact dermatitis.
- ◆ Pediatric Eczema
 - Hydrocortisone 1% cream (hydrocortisone 2.5% or desonide if eczema is severe). May use short courses of higher strength topical steroid but do

not use on the face, groin, or armpits for more than one week.

- Frequent moisturizing is essential.
- Antihistamine therapy: hydroxyzine, diphenhydramine or chlorpheniramine in pediatric doses.
- Antibiotics (as above) if secondarily infected.

◆ **Tips**

- Many patients use medications properly but don't improve due to excessive bathing with hot water and strong soaps that irritate and dry the partly-healed rash. Skin may worsen because patients overbathe faster than the medicines can help. Have your patients shower once daily with lukewarm water, using no scrubber or washcloth, use mild cleansers or soaps briefly, pat (rather than rub) dry the skin with a soft towel, and apply the medicine or moisturizer right away to seal moisture into the damp skin.
- This is a chronic condition; a realistic goal is to obtain reasonable control, not cure. The patients need to realize that this condition cannot be cured. They must take responsibility for their treatment (i.e., as long as they continue to vigorously scratch and if they do not use their medications as often as they should, their condition will not improve). Stress and anxiety may have significant roles in the persistence and exacerbation of eczema.

■ **Acute Allergic Contact Dermatitis**

This form of contact dermatitis is extremely itchy and often appears in linear streaks. Common causes include plants, such as poison ivy; rubber and elastic products; nickel and metals with nickel alloys; topical preparations such as fragrances, sunscreens, neomycin/ Neosporin products. Begin the treatment by identifying the offending agent, if possible, and removing it. Treatment:

- ◆ Mild - erythema without vesicles
 - Triamcinolone cream 0.1% BID for 7-14 days.
- ◆ Moderate - erythema, vesicles, exudate
 - Cool tap water or Domeboro compresses TID.
 - Apply moderate-to-potent steroid.

- ◆ Severe - widespread with marked edema and bullae
 - Topical therapy as above.
 - Oral prednisone (1 mg/kg/day) tapered over 14-21 days or IM triamcinolone (up to 1 mg/kg x 1 dose).
 - This can debilitate a soldier for days. Consider admitting severely affected soldiers to your field hospital and dress them in loose clothing (e.g., a loincloth made of towels or chux).

- **Seborrheic Dermatitis** - very common, chronic, scaly eruption on oily areas of the scalp, hairline, ears, forehead, sides of the nose; rarely also the midchest, midback and pubic areas. Commonly confused with lupus erythematosus and rosacea (if on face), and psoriasis (if on scalp).

Start with OTC Shampoos. They can be used to wash all the seborrheic areas (scalp, face, etc). With all medicated shampoos, keep shampoo on the hair for 15 minutes before rinsing. Use daily for best results. Shampoos can be rotated every 2-3 months to prevent tolerance.

- ◆ Tar containing (e.g., T-Gel, T-Sal, Ionil-T, Pentrax Gold, Polytar)
- ◆ Zinc containing (e.g., DHS zinc, Head and Shoulders)
- ◆ Sulfur and salicylate/salicylic acid (e.g., Ionil Plus)
- ◆ Selenium sulfide 1% (e.g., Selsun Blue)

Prescription Shampoos - use only if the OTC shampoos fail.

- ◆ Selenium sulfide 2.5%
- ◆ Ketoconazole (e.g., Nizoral - expensive)

Topical Steroids

- ◆ For scalp: liquid mid-to-high strength cortisones such as fluocinonide BID for 7 days then QHS.
- ◆ Hydrocortisone 1%-2.5% cream or ointment bid for mild face and body involvement. Moderate strength steroids may be used for 2 weeks.

Topical Antifungals (e.g., clotrimazole but not Nystatin) may be helpful to control *Pityrosporum* (an organism which may exacerbate seborrheic dermatitis in some individuals).

- **Prurigo Nodularis** – not a true eczema. This is an imprecise term referring to a chronic condition consisting of 3-9 mm, very itchy, red-to-brown-to-skin-colored papules, of no known cause. They are usually firm, with a rough or verrucose surface. Treatment is with emollients and topical steroids (as for eczemas). Individual, resistant lesions can be injected with a tiny amount (0.1 cc) intralesional triamcinolone 10 to 40 mg/cc.

ACNE AND RELATED DISORDERS

- **Acne** - therapy is based largely on the type of primary lesions. Treatments require weeks to months to work. Acne is often worse in tropical conditions. Darker-skinned patients may also suffer from post-inflammatory hyperpigmentation; tretinoin or azelaic acid (see below) will often help this also.
- ◆ Mild Acne (mostly comedones – blackheads or whiteheads – few pustules)
 - Benzoyl Peroxide (BP) products QD or BID – may bleach clothing.
 - Tretinoin (e.g., Retin-A) cream (0.025%, 0.05%, 0.1%) or gel (0.01%, 0.025%). This agent is drying, so patients need to build up a tolerance: start every 4th night for a week, then every 3rd night for a week, then every 2nd night for a week, then every night if tolerated.
 - Tretinoin and BP inactivate each other. Use BP in the AM and tretinoin at night.
 - Azelaic acid (e.g., Azelex) QD-BID is also useful.
 - Some practitioners add topical erythromycin or clindamycin to the BP. There are commercial pre-mixed preparations, but these are rarely on formulary because of cost. Having the patient apply the antibiotic and BP at the same time is almost as effective, and a lot cheaper.
 - Some patients will find it too drying to use more than one topical (e.g., tretinoin and BP).
 - ◆ Moderate Acne (mostly papules and pustules with few comedones or cysts). Combine a topical regimen (as above), with *one* of the following oral antibiotics:
 - Tetracycline 250-500 mg PO BID (photosensitivity; not in kids or in pregnancy).
 - Doxycycline 100 mg PO QD (photosensitivity; nausea; take with food).
 - Minocycline 100 mg PO QD (more expensive; take with food; common side effects less common, but severe side effects somewhat more common – although still very rare – compared to plain tetracycline).

- Erythromycin 250-500 mg PO BID (safe in pregnancy; drug interactions; nausea; take with food).
- Septra DS QD (if nothing else works; many side-effects including rashes and rare fatalities).
- ◆ Severe Acne (cystic or scarring)
 - Start with the regimen for moderate acne.
 - Isotretinoin (e.g., Accutane) is the treatment of choice but is expensive, has many side effects (birth defects to dryness), and requires monthly clinical and lab monitoring; usually a 20-week course; it should really be used only by dermatologists, and only in a garrison setting. Considering the risks of moving patients in a war zone; if patient is not responsive to 4-6 months of conventional therapy, consult Dermatology via derm.consult@us.army.mil (see Referrals and Teledermatology section below). Likely isotretinoin therapy should wait until patient rotates home.
- ◆ Acne during pregnancy
 - Can safely use erythromycin (oral or topical) or topical benzoyl peroxide preparations. Tetracyclines and Accutane are contraindicated. Retin-A has not been proven safe in pregnancy.
- ◆ Other points about acne therapy
 - Tretinoin can cause photosensitivity. Light-skinned patients should use sunscreen daily.
 - Tretinoin users need to apply moisturizers as often as necessary for facial dryness. Use simple, inexpensive moisturizers.
 - Tretinoin should only be applied to dry skin (20-30 minutes after washing). Use only a pea-sized amount to cover the face and forehead. Some redness, drying, and peeling may be expected in the first few weeks of use. Tretinoin can be used every other night or stopped for a few days to lessen redness or dryness. Creams are less drying than gels. In arid climates, start with 0.025% cream or even 0.01% cream.

- Requires patience and compliance with treatment to get acne under control. It may take up to 3 months before there is significant improvement. During the first few weeks of Retin-A therapy, acne often seems worse.
- Use mild soaps (e.g., Dove) along with moisturizers to reduce the drying effects of the acne medications.
- Avoid scrubbing face with harsh cleansers (e.g., abrasive granules or Buff-Puff).
- Tetracyclines (particularly minocycline) can cause vertigo – may be a problem for aviators.

■ **Rosacea** – four types: telangiectatic, erythematous, papulopustular; and hyperglandular. May overlap/co-exist. Treatments:

- ◆ Metronidazole 0.75-1.0% cream (e.g., Noritate, Metrocream, Metrogel) BID, sulfa-based lotions (e.g., Sulfacet-R) QD, or azelaic (e.g., Azelex) acid, or tetracycline 250 mg PO QD-BID.
- ◆ Mixed reports on triggering substances (e.g., alcohol, spicy foods, coffee). Sunscreen may be very important, especially in a sunny climate.

■ **Pseudofolliculitis barbae (PFB)**

The treatment for PFB in the field is to avoid shaving. Keep the beard trimmed closely (1/4-inch) with scissors or clippers per regulations. A P2 profile is permitted. There is no benefit in expecting the service member to repeatedly try to shave to see if the PFB has resolved spontaneously – it doesn't. Treatment of choice is laser hair removal, but until patient rotates home, can use topical treatments like for acne (BP wash plus tretinoin QHS), plus hydrocortisone 1% cream prn to soothe irritation.

■ **Acne keloidalis nuchae (AKN)** is a form of scarring alopecia most common in young black men. It consists of firm folliculocentric keloidal papules on the occipital-nuchal region. The papules may coalesce into giant scarring plaques. Etiology is unclear.

- ◆ Avoid close haircuts on the nuchal/occipital region. Remind your gung-ho soldiers that 1/4 -1/2-inch hair is perfectly within regulations. Further remind them that the creation of a permanently disfigured scalp is not worth the military glamour of a high-and-tight.

- ◆ Oral antibiotics (as for moderate acne) should be given for 1-2 months for new onset or actively inflamed AKN. Also add a strong topical steroid (e.g., clobetasol or fluocinolone) scalp solution, gel, or foam QD or BID.
- ◆ Hair growth cannot be restored to scarred papules and plaques. Consider intralesional triamcinolone 10-40 mg/cc IL q 4-6 weeks to soften and reduce these scarred areas.
- ◆ Refer patients to dermatology or plastic surgery for possible resection of large plaques.
- ◆ Uncommonly a patient is prevented from wearing a Kevlar helmet because of pain associated with pressure on the AKN. This occasionally leads to a medical retention board.

PAPULOSQUAMOUS DISORDERS

■ Psoriasis

Psoriasis is a common, chronic, often pruritic, scaly, erythematous eruption on the scalp, ears, palms, soles, and extensor surfaces of the extremities. This condition varies considerably in its extent and may be exacerbated by some medications (lithium, beta blockers, antimalarials, and systemic steroids). When uncertain about the diagnosis, check the scalp, behind the ears, fingernails (pitting, yellow spots, nail separating, and subungual debris).

◆ Topical Steroids

- Face - hydrocortisone 1%-2.5% cream or desonide BID.
- Scalp - same as for seborrheic dermatitis.
- Trunk, feet, and hands - triamcinolone 0.1% cream BID (mild involvement) to betamethasone or clobetasol ointments for more severe cases.
- Intralesional triamcinolone (5 mg/mL) can be effective for small plaques.
- For those patients who do not respond to topical steroids, try applying any mid-potency topical steroid to the trunk or extremity lesions (preferably when they are moist following a shower), then cover it with clear plastic wrap. This occlusive therapy potentiates the topical medicine and if used overnight for up to 2 weeks, it may help to improve or clear a problem area.
- DO NOT USE SYSTEMIC STEROIDS.

◆ Tar Products

- Scalp - see seborrheic dermatitis. For thicker plaques on the scalp, applying Baker's P&S solution overnight, especially under a plastic shower cap, may loosen scale.
- Trunk and extremities - products such as T-Derm or Fototar QHS (warn patients that tars can cause photosensitivity and can stain clothing).

- ◆ Emollients/Lubricants - use BID to decrease scale on plaques. (Remember: ointments are more effective than creams and creams are more effective than lotions.) Lactic Acid 12% (e.g., LacHydrin) can help clear silvery scale.
- ◆ For thick plaques, salicylate gel (Keralyt) QD may be helpful.

■ Pityriasis Rosea (PR)

This benign sometimes itchy eruption is commonly seen in young adults. This typically produces a slightly red or reddish-brown scaly 2-6 cm diameter herald patch followed 2-15 days later by a widespread eruption on the trunk and proximal extremities. Patients with PR remain afebrile and are otherwise healthy. PR often occurs in the spring and fall. Because the condition is harmless and its mode of transmission is not clear, we recommend that no precautions are needed.

Treat with topical antipruritic preparations, emollients, or topical steroids. Brief sunlight exposure (but without getting sunburned) is often helpful.

Differentiation from secondary syphilis is important – the two eruptions can resemble each other. Syphilitics often feel poorly, have malaise, and low grade fevers. Also check for presence (or recent history) of genital lesions or other STDs. Examine for diffuse adenopathy, pharyngitis, palmoplantar lesions, or oral/genital lesions. Remember that travel/deployment is a risk factor for syphilis. Don't hesitate to order an RPR.

■ Lichen Planus

Although there are many variants, classically, lichen planus shows the "7 P's": polygonal (multi-angulated rather than round), planar (flat-topped), pruritic, purple (not red) papules, with a predilection for the periphery (wrist, ankle, and penis). If you look closely, you can see lacy white lines (Wickham's striae) on the papules. In addition, a lacy white pattern is often seen on the genital mucosa and inside the cheeks (careful! there are

normally white lines where the teeth meet called “physiological bite marks”). Mucosal lesions can be erosive and/or painful. If untreated, it will last for 9 months to 2 years. Rarely, lichen planus is triggered by medications or hepatitis C.

Treat with topical steroids as for psoriasis. For oral lesions, steroid gels can be put on a cotton ball and held against the lesions for 10 minutes BID. Sunlight may either improve the condition or may make it worse (very individual).

FUNGAL INFECTIONS

Fungal infections are frequent problems in deployed soldiers, especially in hot humid climates or when daily hygiene is curtailed.

■ Dermatophytes

- ◆ Tinea pedis (“athlete’s foot”), tinea cruris (“jock itch”) and tinea corporis (“ring worm”) are all common, chronic, pruritic and scaly fungal infections. If the diagnosis is in doubt, perform KOH scraping at the first visit. Localized, uncomplicated infections are best treated with twice daily application of a topical antifungal cream (start with miconazole or clotrimazole). Treat affected areas for 1 week *beyond* the point of clinical resolution to prevent relapse. If widespread or very inflammatory, the patient may need griseofulvin 10-15 mg/kg/day (250-750 mg PO for adults) QD with food or milk for 4-6 weeks. Failures occur when fungus is resistant to the medicine, if the fungus has penetrated deep into the hair follicles, or if areas of rash are missed when applying the topical medicine. Fungus loves moist areas, so keep the groin and intergluteal areas as clean and dry as possible. Use of a mild steroid such as hydrocortisone 1% cream BID for ONE WEEK ONLY may help control the itching while the infection is clearing with the topical antifungal. Drying powders (e.g., Zeasorb) can help to keep the wetness under control (decrease the frequency of application or discontinue use of this powder once the area has become dry).
- ◆ Tinea unguium (= “onychomycosis” = “nail fungus”) is best left untreated in the field environment. Topical preparations rarely cure nail infections.
- ◆ Tinea capitis has two common forms. “**Black-dot ringworm**” is a scaly plaque with alopecia and shows abundant hairs broken off at skin level creating “black dots.” A **kerion** is a boggy, inflamed, poten-

tially-scarring plaque usually in kids. Treat both with griseofulvin (taken with meals) for approximately 6-8 weeks. Topicals will not work. Separate comb and face towel from those of other soldiers/family members. Boggy kerions may require a short course of oral prednisone to decrease inflammation and reduce risk of scarring alopecia. Antibacterial antibiotics are generally not needed. Monitor for relapses. Consider referring children with kerions to Dermatology.

■ Other

- ◆ Tinea versicolor appears as light to dark brown, fine powdery scaly discrete and coalescent macules on the back, neck, chest, shoulders, and upper arms. This condition is due to a yeast that is a part of the normal skin flora and can recur in hot, humid conditions. It is harmless. Remind patient that, even if treated perfectly, TV will leave abnormal pigmentation for up to several months beyond cure. Relapses are near-inevitable. Treatment options:
 - Apply Selsun-type shampoo from the neck down to the waist and also to the arms 15-20 minutes prior to daily showering for 1 week, then repeat weekly for the next month.
 - Clotrimazole or spectazole cream BID for 3 weeks.
 - Oral ketoconazole 200 mg PO QD for 1 week is very effective for resistant cases.
- ◆ Macerated tinea pedis is a painful inflammatory disorder usually caused by a polymicrobial infection (mixed dermatophyte and bacterial). It appears as white, gooey cakes in the webspaces of toes.
 - First gently remove as much of the macerated, inflamed tissue as possible using hydrogen peroxide or saline on a gauze pad. Then treat the underlying fungal infection. For 1-2 weeks, use an anti-infective astringent (such as colorless Castellani's paint or gentian violet, if these agents are available) TID. Other drying solutions include teabag soaks (two teabags steeped in 6 oz of water and liquid allowed to

cool), dilute vinegar, or aluminum acetate (e.g., Domeboro's) soaks. After the astringent has dried, apply Vioform HC (a cream that combines hydrocortisone 1% and a mild antibiotic).

- On the subsequent visit, start the directed antifungal therapy.
- Prevention: allow feet to dry completely when the situation permits. Put on dry socks at mid-day and change shoes or boots daily. Wear sandals if conditions permit. Consider antifungal foot powders .

VIRAL INFECTIONS

■ Warts (HPV)

◆ Common Warts

- Topical acid preparations include salicylate and lactic acid preparations (e.g., Occlusol HP, Duofilm, Transversal, or Mediplast). Patients must be very patient and conscientious about applying the medication daily.
- Repeated freezings every 2 weeks with liquid nitrogen (warts can be very resistant to treatment if located on the palms, soles, and in periungual and subungual locations). Freeze time (amount of time you see white frost on the wart) needs to be 15-30 seconds with two separate freeze-thaw cycles (freeze, let thaw by itself, then freeze again). Patients should be informed of possible blister formation (even blood blisters) following the treatments and care of the treatment site (keeping it clean, applying antibiotic ointment if needed, and sterile puncturing the blister if necessary).
- Occasionally you may choose to pare down a wart with a scalpel blade (particularly painful, hyperkeratotic plantar warts) but avoid surgery and electrocautery of warts as this is painful, rarely effective, and may lead to scars that are worse than the warts.

- ◆ Flat Warts - Common in children and young adults. Hard to treat. Lesions appear as small, flat-topped, skin-colored papules and are often found on the face, hands, arms and legs. Treatments include liquid nitrogen (caution in dark skinned patients) and tretinoin (e.g., Retin-A) 0.1% cream QHS. In children, tretinoin can work quite well and leave little scarring; however, it is slow.

- ◆ Plantar Warts - Similar approach as for common warts. Avoid long freezes as this may impede walking and running.

◆ Condyloma (venereal warts)

- Podophyllin - Most effective on moist, mucosal lesions. Apply podophyllin 25%-40% (usually mixed in tincture of benzoin) directly to condyloma with a cotton-tipped applicator. Instruct patient to wash off well in 8 hours. Patient should follow routine hygiene afterwards, refrain from intercourse for several days, and use condoms to diminish risk of spreading the wart.
- Liquid nitrogen – freeze to ice ball with mm margin, thaw, repeat once.
- Soldiers who are unable to return for regular follow-up may use podofilox (e.g., Condylox), a purified dispensable version of podophyllin. Apply per directions.
- Imiquimod (Aldara) can be applied overnight 3 times/week for up to 16 weeks.
- **Note:** Ask the patient to inform the partner(s) so that they may seek care. Gynecologists are better able than dermatologists to provide care for genital warts in women. Remind the patient that genital warts take a number of treatments before clearing and still, many patients have genital warts for a lifetime. Test for other STDs. Advise the use of condoms.

- **Molluscum Contagiosum** - Appears as dome-shaped, umbilicated papules. Common in children and immunocompromised individuals. In healthy adults is often sexually transmitted. Any procedure that gently removes/expresses core of the lesions can be effective. Such treatments include light liquid nitrogen application, topical salicylate/lactic acid preparations (e.g., Keralyt, Duofilm), or podophyllin. In some areas, such as the eyelids in small children, observation only may be prudent since lesions may be self-limited. Cantharidin (e.g., Cantharone) can be very carefully placed on individual lesions, allowed to dry, and then covered with tape for 12-24 hours. The treated sites will blister, so discuss blister care with the parents (keep the treated sites clean, apply antibiotic ointment twice a day, etc.). Often need to repeat once/month for 1-3 months. Most

children experience little or no pain from the treatment but may be annoyed by the abundant small blisters. Do not place this medication on areas of normal skin (apply only to the lesions) and NEVER dispense the medication for home use.

- **Herpes Simplex** - Recurrent painful vesicles on erythematous border, usually on genitals or lips. Treatment is only effective if started within 48-72 hours of outbreak. Initial episode is more severe. Treatment:
 - ◆ Initial outbreak: Acyclovir (Zovirax) 400 mg PO TID or Valacyclovir (Valtrex) 1 gram PO TID for 7-10 days.
 - ◆ Recurrent outbreaks: Acyclovir (Zovirax) 400 mg PO TID or Valacyclovir (Valtrex) 1 gram PO TID for 5 days.
 - ◆ If frequent recurrences (every 1-2 months) can use Acyclovir (Zovirax) 400 mg PO TID or Valacyclovir (Valtrex) 500 mg – 1 gram/day as chronic suppression.

BACTERIAL SKIN INFECTIONS

Streptococcal and staphylococcal infections are briefly addressed in this TG (Streptococcal Infections section in Endemic Infectious Diseases and CA-MRSA section of Resistant Bacteria/In-Theater Nosocomial Infections appendix, respectively). Such infections are not unique to the CENTCOM AOR, and they should be managed as they would in CONUS. Of relevance to dermatology, these bacteria may cause a spectrum of diseases including:

- **Superficial pustular folliculitis/sycosis barbae** – Signs: folliculo-centric pustules and inflammatory papules, may be spread by shaving.
- **Furunculosis** – **Note:** a furuncle is a boil: a deepset, acute, round, tender, perifollicular abscess which ends in suppuration. A carbuncle is two-or-more confluent furuncles.
- **Pyogenic paronychia** – pain, erythema, swelling, and possible purulent drainage around nail fold.
- **Staphylococcal impetigo** – similar to bullous form of streptococcal impetigo.
- **Staphylococcal toxic shock syndrome** – essentially same as streptococcal toxic shock.

GROWTHS (CANCEROUS AND NON-CANCEROUS)

- **Actinic keratoses** are very common in older Caucasians. Chronic, small, indistinct, scaly or raspy, pink to red-brown, easily irritated patches or papules located on sun exposed areas. Liquid nitrogen applied for approximately 3-10 seconds (longer application times are used for the thicker lesions). The treated sites usual heal in about 2-3 weeks.
- **Squamous cell carcinomas (SCCs)** are indurated papules, plaques, with thick keratotic scale and are often crusted, eroded, or ulcerated and are more apt to bleed. SCCs occur most commonly on ears, lips, and dorsal hands. They don't respond well to liquid nitrogen. These require biopsy and surgical excision.
- **Basal cell carcinomas** are the most common skin cancers. The most common appearance is pink to reddish, pearly, somewhat translucent appearing papules and nodules, with rolled borders and fine telangiectasias that frequently ulcerate and bleed. These also require surgical treatment.
- **Seborrheic Keratoses** rarely appear before age 30 and then slowly appear in increasing numbers. They range in size from 0.5-3 cm, range in color from tan-to-dark brown-to-black, are velvety or verrucose-surfaced, and usually appear to be "stuck on" to the skin. They are rarely pruritic, sometimes inflamed if caught on clothing, but are usually asymptomatic.
- **Nevi** are more common in lighter-skinned people and those with a history of sun exposure. May be congenital. Small (< 0.6 cm if acquired, may be any size if congenital), tan to dark brown, flat or raised, regular borders.
- **Melanoma** - Use the ABCDs to recognize suspicious lesions. These guidelines are good but not perfect. The diagnosis is suspected on a clinical basis but made on histopathology. A biopsy is necessary to establish the diagnosis.
 - ◆ Asymmetry (there is no exact limit, but symmetry is a good sign, asymmetry is a bad sign).
 - ◆ Border irregularity (again, there is no exact limit, but regular is a good sign, irregular is a bad sign).

- ◆ Color variety (1-2 colors, evenly distributed, is normal. More colors, or uneven distribution of colors is a bad sign. "Color" can be black, dark brown, medium brown, light brown/tan, red, blue, or gray).
- ◆ Diameter (greater than 6 mm). When normal moles mature (grow and change) they can undergo some changes that are not signs of malignancy. Congenital moles are often much larger than 6 mm.

MISCELLANEOUS

■ Burns and scalds

- ◆ First degree burns are red, second degree are blistered, and third degree are charred. Burns are caused by dry heat, scalds by wet heat (e.g., boiling water). Acutely, all “burns” look alike, whether the cause is dry heat, wet heat, sun, chemicals, or ionizing radiation. Pain should be managed as needed.
- ◆ Will only discuss minor, common burns here, except for a few reminders about more severe types of burn. Severe thermal burns require surgical intervention and ICU-level care for fluid management. Electrical injury may require monitoring for cardiac dysfunction and rhabdomyolysis. Chemical warfare agents are discussed elsewhere.
- ◆ Ordinary chemical burns (i.e., not chemical warfare agents) require flushing with saline (or any available fluid) to neutralize the agent. Alkalines require much more copious irrigation (several liters, even in a minor exposure) than neutral or acid agents.
- ◆ Third degree burns need to be debrided to remove devitalized tissue.
- ◆ Second degree burns – if the blisters can be protected, they should be left intact, as they are a perfect biological dressing. If they cannot be protected, they should be ruptured under sterile (or at least clean) conditions. In either case, the wound should first be cleaned (with saline or peroxide) and covered with a sterile dressing. The wound can be protected with Silver sulfadiazine (e.g., Silvadene - if the patient is not allergic to Sulfas, polysporin, or even just Vaseline). Contrary to what we were all taught in Boy/Girl Scouts, the wound should *not* be allowed to dry out and scab. The wound should be cleaned, dressed, and inspected for signs of infection QD-BID. If any evidence of infection, culture if possible, and treat with cloxacillin or cephalixin.

- ◆ First degree burns are treated like second degree, only less vigorously. Patients should be reminded not to “pick” as the old dead skin peels off; they should moisturize instead.

■ **Arthropod (insect) bites** itching, urticarial-looking papules.

- ◆ Often grouped in 2-4 lesions (“breakfast, lunch, dinner”).
- ◆ Often see a minute punctum (the actual bite) in the center. All arthropod bites look alike clinically.
- ◆ Treat with mid-potency-to-strong topical steroid or pramox for itch. Diphenhydramine (e.g., Benadryl) or hydroxyzine (Atarax) if pruritus is severe.
- ◆ Control insects in area.

Note: even when people are sharing housing, it is common that only one or a few will have arthropod bites, even when the others do not.

■ **Lice (pediculosis and phthiriasis)**

- ◆ Lice infestation causes severe itching. They are highly communicable, especially in crowded conditions. Body lice (*Pediculus humanus corporis*) are especially significant in carrying louse-borne diseases. Head lice (*P. h. capitis*) live in the hair, including sometimes eyebrows and eyelashes; they are transmitted by direct contact or via fomites. Body lice live in clothing, especially in the seams of clothing. Crab lice (*Phthiriasis pubis*) generally live in the pubic hair, although in heavy infestations can be found on facial hair; they are usually transmitted sexually, although can be transmitted by other close contact or via fomites.
- ◆ Lice can be transmitted as long as lice or ova remain alive on the body or fomites. Adult lice have a life span of about 1 month. Head and body lice can survive for up to 1 week without food (i.e., off the human body); crab lice only 2 days. However, the ova (“nits”) remain viable for up to 1 month on fomites.
- ◆ Treatment:
 - IN ALL CASES:
 - Need to treat fomites (clothing and bedding): wash in hot water 140°F (55°C) for 20 minutes, or dry clean, or iron very hot, or wrap in plastic for a month.

- Educate patients not to share clothing, headgear, or bedding.
- May need to do mass inspection of personnel or populace.
- Soak brushes and combs in very hot water or treatment shampoo.
- Standard issue personnel insect repellents will help decrease transmission.
- Pesticide treatment of a building (such as a barracks or family housing) for louse control is not needed and should not be recommended because lice die soon after leaving the host.
- HEAD AND PUBIC LICE (need to comb out nits after treatment)
 - Permethrin 1% cream rinse (e.g., Nix) – rubbed into *dry* hair, and allowed to remain on for 10 minutes (maybe 30-60 minutes if resistance is suspected), then toweled. Do not re-wash for at least 24 hours.
 - Pyrethrin with piperonyl butoxide (e.g., Rid) – shampoos, need to be left in for 10 minutes. Less effective than permethrins. Must be repeated 7-10 days later.
 - Malathion lotions (e.g., Ovid) – rubbed into dry scalp and hair; leave on for 8-12 hours, then wash out with non-medicated shampoo.
 - Lindane (gamma-benzene hexachloride) shampoo, need to be left in for 4 minutes. NOT to be used in children and pregnant women; banned in California because of fears of neurotoxicity.
- BODY LICE
 - Mostly live in the clothing, so questionable if need to treat patient. If so, use single application of permethrin 5% cream (not cream rinse!) left on overnight. Treating the fomites (clothing, bedding, etc) is the key.
- FOR MASS OUTBREAKS (Preventive medicine personnel need to get involved)
 - Consider mass delousing

- Initiate a public awareness program (briefings, fliers for bulletin boards, articles for newspapers)
 - (a) Explain that a louse infestation: (i) Is not a sign of being dirty, (ii) Can happen to anybody anytime, (iii) Can be completely eliminated with proper treatment, (iv) Must be treated promptly because it will spread quickly.
 - (b) Describe signs of louse infestation: (i) Persistent itch, often with infected scratches or rash, (ii) Small silvery egg cases ("nits") attached to individual hairs, (iii) Swollen lymph glands in neck or under arms.
 - (c) Discourage personnel from sharing personal articles (combs, hats, towels, bedding and clothing).
 - (d) Address all family members or personnel in the barracks.

■ Scabies

◆ General

- *Sarcoptes scabiei* is a minute mite that infects only humans. It is transmitted by close contact. Adults may have co-existing STDs. There is a 3-6 week period before itching begins, and the itching continues 3-4 weeks after successful treatment (although starts to gradually get better in 1-2 weeks).
- Scabies lesions are polymorphous and can have papules, vesicles, crusts, excoriations, and secondary pyodermas in addition to the classic burrows. The distribution is a symmetric eruption of pruritic lesions, most often on finger webspaces and volar wrists. Penis, nipples, and axillae are commonly involved. It generally spares the face and scalp in adults but may in infants.

Note: It does *not* look like arthropod bites.
- Nodular scabies occurs on male genitalia and appears as a usually itchy, indurated, 4-6 mm diameter papule without an obvious burrow. This

lesion, in combination with the hand lesions, is virtually diagnostic of scabies.

- The diagnosis is from classic presentation or with a positive scraping that shows a mite or egg. Highest yield for scrapings are from either the web spaces of the fingers, wrists, or genitalia.

◆ **Treatment options**

- First line: permethrin 5% cream (e.g., Elimite) applied to all areas of the body from neck down and washed off after 8-14 hours.
- Lindane: 1% lotion or cream (e.g., Kwell) applied thinly to all areas of body from neck down and washed off thoroughly after 8 hours.
- Ivermectin orally at 0.2-0.4 mg/kg as a single dose. May repeat in 1 week. Most useful in situations where multiple individuals need to be treated or with treatment failure from permethrin.

◆ **Tips about treatment**

- Total body application requires 25-30 gm (1 oz).
- Apply medication to all surfaces, not just affected areas. Rub it under fingernails too.
- Permethrin is now the drug of choice because it is virtually free of side effects and because there is resistance to the other two medications. Permethrin is safe in children and pregnant women. Lindane should not be used in children because of neurotoxicity, or in pregnant woman because of fetal risk of neurotoxicity and aplastic anemia.
- Bedding and clothing must be washed and dried on a hot-cycle or removed from body contact for at least 72 hours.
- Retreatment is probably not necessary when using permethrin. Nevertheless, usually recommend retreatment after 1 week. It may be overkill but it gives patients peace-of-mind.
- Pruritus will persist for about 2-3 weeks because the dead mites remain in the stratum corneum for that long and continue to elicit an itch response. Consider adding antihistamines, or topical Sarna, or a topical corticosteroid to help control itching.

- Treat secondary bacterial infections with oral dicloxacillin, topical mupirocin, or other anti-Staph/Strep antibiotic.
 - Notify sexual partners, close household contacts, and daycare centers as appropriate.
- **Keloids** are firm scars that extend beyond the margins of the original trauma or incision. May rarely interfere with wearing Kevlar or other gear. Try intralesional triamcinolone 10 to 40 mg/cc to decrease itching, tenderness, and to soften the scars (usually every 4-6 weeks for 4-6 months). Can also try strong topical steroids. Monitor for skin atrophy. **DO NOT EXCISE**, it will reform as an even larger keloid.
- **Melasma (or chloasma)** is a symmetric, patchy, brownish, macular facial hyperpigmentation. It occurs most commonly in women with a medium skin color, such as women with an Asian or Hispanic background. Melasma often arises during pregnancy or from female hormone therapy (e.g., OCPs or supplemental estrogen).
- ◆ Patients need to be very conscientious about daily sun protection. Treatment will fail without UVA blockers in their sunscreens (see sunscreens).
 - ◆ First line treatment is bleaching agents containing 2-4% hydroquinone (e.g., Esoterica, Porcelana, Melanex, Eldopaque, Viquin Forte, Solaquine) BID for 3 months or longer.
 - ◆ "Kligman formula" combines hydroquinone, tretinoin cream and a cortisone. Try Eldopaque 4%, Retin-A 0.1% cream, and hydrocortisone 1% cream. Apply pea-sized tabs of all three to one's palm, mix together, and apply to affected areas.
 - ◆ If making limited progress, can try azelaic acid (Azelex) in addition to the above.
 - ◆ Not all patients can be helped, especially if the pigment is in the dermis.
 - ◆ **WARNING:** some people experience a paradoxical darkening (pseudo-ochronosis) from the hydroquinone so advise the patient to test the hydroquinone alone on the inner biceps QHS for 2 weeks as a test site before using it on the face.

- **Alopecia areata** is a common circumscribed area of hair loss on the scalp or face. It is uninflamed, not scaly, and non-scarring. Occasionally linked with autoimmune conditions, especially thyroid disorders. Can be progressive. If the posterior hairline is involved, this suggests a poor prognosis. Can use topical Lidex solution or gel BID or intralesional steroids (Triamcinolone 5 mg/mL) in small amounts injected every 4-6 weeks. If there is erythema, scale, folliculitis, boggy, purulence, or scarring, refer the patient to dermatology, since these suggest another disease process.
- **Urticaria (“hives”)** are very pruritic, red, papules-to-plaques. Sometimes the center is a paler color. The key finding is that individual lesions do not stay for more than 24 hours. Be specific when trying to get this history, because the overall attack of urticaria can last any length of time, but each lesion will last for less than 24 hours.

Urticaria is not a disease, but a reaction to something. That “something” can be an allergy, an infection, or almost anything else. Often, the cause was a subclinical infection a few days or weeks before the eruption started; more often the cause is not found. Patients (and many doctors) will focus on a new exposure that coincides with the eruption, but more likely the inciting cause was a few weeks earlier. Ask about recent illnesses, environmental exposures, medications (including OTC, herbal, etc), and systemic symptoms. As a rule of thumb, the cause will either be easy to find or it will never be found. An elaborate work-up is not indicated in the field – acute urticaria resolves within 6 weeks, chronic urticaria requires a very extensive work-up that will wait until the patient rotates home.

The goal in the short run is to make the patient comfortable. Start with non-sedating anti-histamines such as Allegra, Claritin, or Zyrtec. If these are not adequate, continue using them in the morning, and add a sedating antihistamine at night such as diphenhydramine (Benadryl) or hydroxyzine (Atarax). If need be, increase to the maximum doses. Remember that “non-sedating” is relative; individual

reactions vary greatly and should be assessed before patients on antihistamines try to drive, operate heavy machinery, etc.

■ **Sunscreens and sun protection**

- ◆ Ultraviolet (UV) light is divided into two bands: UVB and UVA. UVB is more associated with sunburns and skin cancer. UVA is more associated with skin aging, photodermatitis, and also skin cancer. UVB levels peak at mid-day; UVA levels are high all day, and UVA penetrates ordinary window glass.
- ◆ Sunscreens are divided into physical (titanium dioxide and zinc oxide) and chemical (all the others). Physical agents leave a visible residue, but have fewer irritant and allergic reactions, last longer, and block both UVB and UVA. Chemical sunscreens do not leave a residue, but cause more irritant and allergic reactions, and have to be reapplied every 2-3 hours to be effective. (They breakdown as they intercept ultraviolet photons, which is how they work.) Recommend that Caucasians use a UVA/UVB sunscreen that is PABA-free. Most sunscreen agents can cause some irritant or allergic reactions but PABA causes the most. (Most U.S.-manufactured sunscreens today are PABA-free; local purchase agent may not be.) Until recently, most chemical sunscreens only blocked UVB; newer ones containing avobenzone (Parsol 1789) also block UVA and are usually free of side effects.
- ◆ How much sun is too much? If you get sunburned, you got too much sun. If you get tanned, you probably got too much sun, although some people tan more easily than others. Anyone with a photosensitive disorder, or anyone who finds that the sun worsens their skin, needs to take precautions all day. Everyone should, if possible, avoid sun exposure or use sunscreen in the middle of the day (1000h-1500h).

REFERRALS AND TELEDERMATOLOGY: WHEN AND HOW

- On the consultation request form, please write legibly and include the following information:
 - ◆ Extent, duration, description (size, color, shape, etc.) and location of the lesion/ problem.
 - ◆ Your differential diagnosis.
 - ◆ Pertinent related history or physical findings.
 - ◆ Pertinent medications and diseases.
 - ◆ Current and previous therapeutic attempts (e.g., acne medications, topical steroids for eczema, Occlusol solution for warts, etc).
- **Prompt Referrals** – Referrals should be made promptly for biopsy of suspected melanoma or for evaluation of wide-spread blistering disorders; petechiae or purpura with hypotension, fever, or renal failure; ulcers with fever; buboes.
- **Routine Referrals** – Unless you are co-located with a Dermatologist, suggest using Teledermatology (below), rather than transporting patient across a high-risk area.
- **Teledermatology** – Now available for deployed providers (not for self-referrals or non-providers). Email derm.consult@us.army.mil. You will get a response within 24 hours. In the body of the email, include:
 - ◆ Generic demographic data: age, race, gender, duty position, branch of service and your unclassified unit/ location. DO NOT include patient identifying information (patient's name or SSN).
 - ◆ History including past and current treatment, medications, and their outcome.
 - List environmental factors affecting the patient (heat, cold, arthropod, etc.)
 - Where did condition start? Has the patient been treated previously?
 - What does it look like? What is its' distribution?
 - Is it improving? Not responding to treatment?
 - Comes and goes? Worsening?
 - What laboratory tests have been done?
 - ◆ Your primary and/or differential diagnosis, if known and any specific questions you may have.

- ◆ Inform the teledermatologist of your capabilities or limitations, such as: no lab; can(not) perform shave/punch biopsies; no pathology support; full pharmacy or basic set only.
- ◆ Attach an appropriate number of digital images. Generally 3 to 5 images in jpeg format.
 - 1st image set: distal view showing the entire anatomic unit(s)
 - Example: if rash or lesion on rt chest, take picture of entire chest and back
 - Example: if rash is on arms/legs, take image of both, front and back
 - 2nd image set: close-up.
 - 12-24 inches from the patient.
 - Set the camera in the close-up mode (flower icon on camera).
 - Use indirect or ambient lighting, if possible, avoiding shadows and glaring.
 - Take images perpendicular to the surface of interest.
 - Check the images to ensure they are in focus and accurately show the condition. Retake any images that are not properly focused.
- ◆ If you have any questions on the teledermatology program please contact derm.consult@us.army.mil.

TIPS

- Oozing lesions should be dried with an astringent such as aluminum acetate soaks (e.g., Domeboro's). Gentian violet and Castellani's paint are excellent astringents and biocidal. Application of a dab of gentian violet is the simplest way to treat masses of kids with infected insect bites on the legs. Inform the parent that gentian violet can stain when it is wet (but once dry, it will be a memento of the kid's visit to the doc).
- Open sores, ulcers, and infected insect bites can be disinfected with Castellani's paint, gentian violet, potassium permanganate, or topical bacitracin.
- Use creams rather than gels or ointments on open oozing lesions such as an acute contact dermatitis.
- Neomycin should be avoided; it is a good broad-spectrum antibiotic but it induces a contact allergy in 10% of patients. Topical Benadryl (Caladryl) also causes frequent contact allergy. Avoid both of these. It should be noted that bacitracin sensitivity is also becoming common. Caladryl can be replaced by calamine.
- Vaseline and Crisco shortening are the cheapest effective moisturizers.
- Refer to Sanford's antibiotic guide.
- When confronting the typical round red itchy scaling lesion in the field, most cases will be due to fungal infections. A course of clotrimazole TID x 2 weeks is a reasonable first course of action. A skin scraping and KOH preparation is an easy method for confirming diagnosis. Avoid steroid/anti-fungal combinations.
- Notes about antifungals:
 - ◆ Nystatin will treat only candida infections (but not dermatophyte or tinea versicolor).
 - ◆ Griseofulvin will treat only dermatophytes (such as tinea corporis but NOT tinea versicolor).
 - ◆ Azole creams will treat candida, dermatophytes, and tinea versicolor.
 - Ketoconazole (oral) is more expensive; rare cases of fatal drug-induced hepatitis; and interacts adversely with several medications. Recommend not using it routinely for tinea versicolor and mild dermatophytoses.

- Topical ketoconazole is also more expensive. Use clotrimazole or miconazole instead. Remember that in tinea versicolor the skin coloration will not improve spontaneously after treatment; you need to expose hypopigmented areas to sunlight (in moderation) to normalize pigment.
- ◆ Griseofulvin can be taken in large doses taken once daily. It is best absorbed with fatty food (whole milk, cheese, or meat — not necessarily fried chicken and potato chips).
- Corticosteroids - Table of relative strengths and trade names

CLASS 1 (Super potent)

clobetasol propionate 0.05%	cream, lotion, ointment	Temovate
betamethasone dipropionate 0.05%	cream, lotion, ointment	Diprolene, Diprosalic

CLASS 2

amcinonide 0.1%	cream, lotion, ointment	Cyclocort
fluocinonide 0.05%	gel	Lidex
halcinonide 0.1%	cream, ointment	Halog
fluocinolone acetoneide 0.2%	cream	Synalar HP
betamethasone dipropionate 0.05%	ointment	Diprosone

CLASS 3

triamcinolone 0.5%	cream, ointment	TAC, Kenalog, Aristocort
betamethasone dipropionate 0.05%	cream	Diprosone

CLASS 4

triamcinolone 0.25%	cream, ointment	TAC, Kenalog, Aristocort
hydrocortisone valerate 0.2%	ointment	Westcort
mometasone furoate 0.1%	cream, lotion, ointment	Elocon

CLASS 5

triamcinolone 0.1%	cream, ointment	TAC, Kenalog, Aristocort
hydrocortisone valerate 0.2%	cream	Westcort
betamethasone valerate 0.1%	lotion, cream, ointment	Valisone
fluocinolone acetoneide 0.05%	cream	Synalar
halcinonide 0.025%	cream, lotion	Halog
fluticasone propionate 0.005%	cream	Cutivate

CLASS 6

aclometasone 0.05%	cream, ointment	Aclovate
desonide 0.05%	cream, ointment	Tridesilon, Desowen
fluocinonide acetone 0.01%	cream	Synalar
hydrocortisone 2.5%	cream, lotion, ointment	Hytone

CLASS 7 (the weakest – best for face and intertriginous areas)

hydrocortisone 1%	cream, ointment	many different names
other topicals with dexamethasone, flumethalone, methylprednisolone, and prednisolone		

6

■ Corticosteroids, cont:

- ◆ For face, axillae, and groin, only use class 6 or 7 without dermatology advice. These areas are very sensitive to steroids and prone to side effects such as skin atrophy, telangiectasias, hypopigmentation, and stretch marks.
- ◆ For body, use class 5, 4, or 3. The same side effects can occur here too.
- ◆ For hands and feet, use class 2 or 1. The same side effects can occur here too; also a risk that large amounts of topical steroids can be equivalent to taking oral steroids and long-term use can suppress HPA axis.
- ◆ For severe contact dermatitis (e.g., severe poison ivy exposure etc.), consider oral steroids for 2-3 weeks. Do not use oral steroids routinely for unknown itchy disorders, and do not use for psoriasis.

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APPENDIX A

STANDARD PRECAUTIONS AND ISOLATION PROCEDURES

To aid in the fight against nosocomial transmission of infectious agents, a combination of standard precautions and isolation procedures has been established. The following is a summarization of the basic guidelines advocated by the CDC as an effective strategy to reduce the incidence of nosocomial infections in health care settings. A basic premise of these guidelines is that all patients, regardless of their diagnosis, are to be treated so as to minimize the transmission of microorganisms from patient to health care worker (HCW), HCW to patient, and patient to HCW to another patient. These guidelines describe the use of a two-tiered approach:

- 1) Standard precautions must be used when caring for all patients. Standard precautions entail a list of basic hygiene procedures that are designed to prevent contact with moist body surfaces and thereby reduce the risk of transmission of bloodborne pathogens.
- 2) When caring for patients with highly transmissible or epidemiologically important pathogens or with poor hygiene, extra barrier or isolation precautions may be necessary. These precautions will help in interrupting contact, droplet, or airborne transmission of these pathogens. There may be instances when more than one extra barrier precaution will need to be implemented.

A

Standard Precautions

These precautions apply when contact is possible with ruptured skin or mucous membranes, blood, all body fluids, secretions, or excretions except sweat. Hand hygiene among HCWs is the most effective means of preventing nosocomially transmitted infections. Hand hygiene consists of washing hands when soiled or disinfecting hands when possibly contaminated, irrespective of whether gloves were worn. These actions should take place immediately after gloves are removed, before and between patient contacts, and any time secretions, excretions, blood, or body fluids or contaminated items or equipment are handled. Gloves should be worn if touching mucous membranes, broken skin, contaminated objects, blood, body fluids, secretions, or excretions. These gloves must be changed between patients and before touching clean sites on the same patient. During procedures that are likely to result in splashing

A of blood, body fluids, secretions or excretions, the HCW should wear a mask, eye protection and a gown to protect mucous membranes, skin and clothing. All visitors, patients and HCWs must not be exposed to contaminated materials or equipment. This means that all reusable equipment must be cleaned and sterilized before reuse. Soiled linen should be transported in a double bag. HCWs must be careful when handling sharp instruments such as needles. This will minimize the risk of bloodborne contamination. Never recap needles. If recapping is unavoidable, use a mechanical device or the one-handed technique. HCWs must ensure that puncture-resistant containers are designated and used for all used sharps instruments. Patient and treatment rooms, cubicles, and bedside equipment should be appropriately cleaned. The hot water and detergents in hospitals are sufficient to decontaminate food service materials therefore no special precautions are needed.

The tables below list examples of conditions necessitating isolation precautions and summarize the measures to be taken for the different isolation precautions.

Indications for Standard and Isolation Precautions	
Precaution category	Condition
Standard	All patients
Airborne	Pulmonary or laryngeal (suspected) tuberculosis, measles, varicella; disseminated zoster
Contact	Hemorrhagic fever such as Ebola, Lassa, and Marburg, (risk for) colonization or infection with multiresistant bacteria, <i>Clostridium difficile</i> infection, acute diarrhea in incontinent patient, RSV infection, croup or bronchiolitis in young infants, skin infections like impetigo, major abscess, cellulitis or decubiti, staphylococcal furunculosis, pediculosis, scabies or cutaneous infections with <i>Corynebacterium diphtheriae</i> , Herpes simplex virus, zoster
Droplet	Meningitis, (suspected) invasive infection with <i>Haemophilus influenzae</i> type B or <i>Neisseria meningitidis</i> , diphtheria, <i>M. pneumoniae</i> , pertussis, influenza, adenovirus, mumps, Parvovirus B19, rubella, streptococcal pharyngitis, pneumonia, scarlet fever in young children

A

Summary of Transmission-based Precautions			
Precaution	Contact	Droplet	Airborne
Patient room	Private	Private	Private with specific ventilation requirements
Gloves	Before entering room		Standard precautions
Hand hygiene	Standard precautions, with hand antisepsis		
Gown	If direct contact with patient or environment	Standard precautions	
Masks	Standard precautions	Within 1 meter of patient	Before entering room special requirements
Other	Limit patient transport		

Excerpted from: Wenzel, Richard P., Timothy F. Brewer and Jean-Paul Butzler, (editors).

A Guide to Infection Control in the Hospital, 3rd Edition. International Society for Infectious Diseases. Boston, Massachusetts. 2004. pp. 38-42.

APPENDIX B

MANAGEMENT OF BATTLEFIELD WOUNDS

Modern warfare's use of body armor and rapid evacuation has allowed a larger percentage of trauma casualties to survive until they reach medical care. A greater emphasis is now required on avoiding complications of severe trauma, including infections. The environment of war leads to grossly contaminated battlefield wounds due to numerous factors, including foreign bodies (projectile fragments, clothing, dirt), high-energy projectiles (devitalized tissue, tissue ischemia, hematoma), and delays in casualty evacuation. Wounds will likely become infected unless rapid, appropriate treatment is initiated.

Common microorganisms causing battlefield infections.

Gram-positive cocci - *Staphylococcus* species, *Streptococcus* species.

Gram-negative rods (bacilli) - *Escherichia coli*, *Proteus*, *Klebsiella* species.

Anaerobic gram-positive and gram-negative rods- *Clostridium*, *Bacteroides*, *Prevotella* species.

Nosocomial transmission - *Pseudomonas*, *Enterobacter*, *Acinetobacter* species.

Patterns of infection are dictated by area of injury.

Skin, soft tissue, muscle and bone - *Staphylococcus*, *Streptococcus*, *Clostridium* species.

Intracranial injuries - *Staphylococcus* and gram-negative rods.

Orofacial and neck injuries - gram-positive cocci and mouth anaerobes.

Thoracic cavity - staphylococcal empyemas and pneumonias due to *Staphylococcus*, *Streptococcus* and *Pseudomonas* species especially with prolonged intubation.

Intrabdominal injuries - *Enterococcus*, gram-negative rods, and anaerobic bacilli.

General principles of treatment focus on:

Treatment within 6 hours of injury.

Surgical debridement.

Copious irrigation.

Tetanus prevention:

Administer 0.5 mL IM of tetanus toxoid if prior tetanus immunization is uncertain, less than 3 doses, or more than 5 years since last dose.

If the immunization status is uncertain or less than 3 doses were received, 250 units IM of tetanus immune globulin in a separate syringe and at a separate site from the toxoid is given.

Recommended antibiotics:

General principles-

Simplicity of use, including ease of transport, reconstitution, and administration; safety; duration of activity; and spectrum of activity all impact drug selection especially in tactical combat casualty care or those undergoing frequent evacuations to higher echelons of care.

Further tailoring of antimicrobial therapy should be guided by the results of bacterial culture and susceptibility data, if available.

For echelon III facilities without this capability, consider batch-testing cultures at a facility with that capability to establish local susceptibilities and resistance trends.

Tactical antibiotics - field administration, echelon I or echelon II without surgical support (see table). For simplicity in a tactical environment, consider gatifloxacin 400 mg orally daily for all wounds except penetrating abdominal injuries, patients in shock, or those unable to tolerate oral antibiotics. For those patients, consider cefotetan 2 gm IV/IM every 12 hours.

Medical treatment facility - echelon II with surgical support and echelon III facility (see table).

Avoidance of broad spectrum antibiotics (imipenem, meropenem, piperacillin/tazobactam, etc.) for therapy of drug resistant bacteria (*Klebsiella*, *Pseudomonas* and *Acinetobacter* species), as these bacteria are not frequently found in fresh wounds and drug pressure may increase the development of resistance at a later date.

Infection Control

It appears most resistant bacteria are acquired by patients through horizontal transfer from other patients, through HCW or device contact, and thus every attempt should be made to adhere to infection control practices.

Minimal interventions include frequent hand washing, frequent use of alcohol hand gel, and/or wearing surgical gloves with changing them between patients.

Equipment that comes into direct contact with a patient should be cleaned between patients.

Consider cohorting those patients undergoing long-term in-hospital care.

Special battlefield wounds that may not require evacuation for surgical debridement.

Soft tissue injuries meeting all of the following criteria:

- Non-mine injuries (due to potential contamination).

- Absence of fracture.

- Entrance and exit wounds measuring less than two centimeters in maximum dimension.

- Pleura or peritoneum not breached.

- No major vascular injury.

- No evidence of frank infection.

Treat with copious irrigation along with a dose of intravenous antibiotic (cefazolin) or oral fluoroquinolone followed by 4 days of oral therapy or monitoring without oral antibiotics.

Table. Antimicrobial prophylaxis of battlefield wounds. These recommendations are for empiric therapy of those with acute battlefield wounds and do not apply to wound infections that develop in personnel receiving long term medical care at echelon III facilities.

Injury	Potential pathogens	Tactical Antibiotic	MTF Antibiotic	MTF Alternatives
Abdominal, pelvic, upper thigh, gluteal region, HEENT wound or chest wound crossing mouth/esophageal/sinus mucosa	• Gram positive (<i>Streptococcus</i> spp, <i>Staphylococcus</i> spp) • Gram negative (<i>E. coli</i>, Enterobacteriaceae) • Anaerobes (<i>Bacteroides</i> species, <i>Clostridium</i> spp)	• Cefotetan*^ 1-2 gm IM/IV every 12 hours or • Cefoxitin *^ 1-2 gm IM/IV every 6-8 hours or • Gatifloxacin 400 mg PO every 24 hours • Ertapenem 1 gm IV/IM Q 24 hours	• Cefotetan^ 1-2 gm IM/IV every 12 hours or • Cefoxitin *^ 1-2 gm IM/IV every 6-8 hours or • Ampicillin/sulbactam*^ 1-2 gm IV every 6 hours or • Ertapenem*^ 1 gm IM/IV every 24 hours	• Fluoroquinolone† + metronidazole infusion 500 mg IV or PO every 6 hours or 1 gm every 12 hours*
Extremity wound††, or HEENT wound or chest wound not crossing mouth/esophageal mucosa, penetrating brain injury	• Gram positive (<i>Streptococcus</i> spp, <i>Staphylococcus</i> spp, Group A and anaerobic streptococcus) • Gram positive anaerobes (<i>Clostridium perfringens</i>, <i>Clostridium tetani</i>) • If water exposure, consider <i>Pseudomonas</i> spp, <i>Aeromonas</i> spp	• Cefotetan*^ 1-2 gm IM/IV every 12 hours or • Cefoxitin *^ 1-2 gm IM/IV every 6-8 hours or • Gatifloxacin 400 mg PO every 24 hours • Ertapenem 1 gm IV/IM Q 24 hours	• Cefazolin*^ 1-2 gm IV push†† followed by oral fluoroquinolone, or • Oral or IV fluoroquinolone† • Ertapenem 1 gm IV/IM Q 24 hours	• Clindamycin* 300-600 mg orally every 6 hours or 450-900 mg IV every 8 hours

IM- intramuscular; IV- intravenous

*Does not cover *Pseudomonas* spp. or *Acinetobacter* spp.

Imipenem/cilastatin or meropenem are required to cover these organisms. Ciprofloxacin may be effective against *Pseudomonas*.

^Would not use in a casualty known to have a severe β -lactam allergy due to potential cross-reaction.

†Gatifloxacin 400 mg orally once a day, moxifloxacin 400 mg orally once a day, or levofloxacin 500 mg orally once a day.

†† Consider addition of enhanced gram-negative coverage (fluoroquinolone or aminoglycoside) for grade III open fractures due to possible excess infection.

Duration of therapy: intracranial involvement- 5 days; open fracture- 72 hours after the time of injury or not more than 24 hours after coverage of wound; perforated GI tract- continue for only 24 hours after surgery in the presence of injury to any hollow viscus, if no hollow viscus involvement single preoperative dose is adequate; oral cavity- 5 days.

APPENDIX C

RESISTANT BACTERIA / IN-THEATER NOSOCOMIAL INFECTIONS

Increasing bacteria resistance is noted throughout the world. Multi-drug resistant bacteria (*Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*) have been recognized among casualties returning from OIF/OEF. Although resistance is often associated with nosocomial infections, the recognition of bacteria from the community with innate resistance is increasingly being reported. Community-acquired methicillin resistant *Staphylococcus aureus* (CA-MRSA) is a well known example.

CA-MRSA

Colonizes approximately 3% of healthy soldiers.

Often carries a gene that is associated with more aggressive and invasive infections.

Rarely recognized in cultures of battlefield wounds near the time of injury.

Typically remains susceptible to trimethoprim-sulfamethoxazole, clindamycin, tetracycline, rifampin, and fluoroquinolones.

Prevention

Routine personal hygiene.

Consider as the infectious etiology if skin and soft tissue infections do not respond to traditional therapy (cefazolin, cephalothin, nafcillin, dicloxacillin, amoxicillin/clavulanate).

If infection likely, recommend therapy with trimethoprim-sulfamethoxazole or clindamycin.

Consider decontamination

Nasal mupirocin ointment (2%) BID for 5-7 days.

Chlorhexidine-based soap shower for 5-7 days.

Multi-drug resistant bacteria

Recognized among casualties injured in OIF/OEF.

Etiology unknown but includes

Skin colonization - colonization with these bacteria has been described but resistance to multi-drugs is not usually present.

Inoculation at the time of injury - possible colonization of wound occurs at the time of the injury but preliminary work from wound cultures and evaluation of infections upon return to CONUS medical facilities implies this less likely.

Horizontal transmission - increasing data supports this as the likely source of infection.

Prevention

Minimize empiric broad spectrum antibiotics (such as carbapenems) in the empiric therapy of battlefield wounds or other infections that occur in theater.

Infection Control

Minimal interventions include frequent hand washing, frequent use of alcohol hand gel, and/or wearing surgical gloves with changing them between patients.

Equipment that comes into direct contact with a patient should be cleaned between patients.

Consider cohorting those patients undergoing long-term in-hospital care.

In-theater nosocomial infections

Personnel receiving prolonged medical care in echelon III U.S. MTFs are at risk of developing nosocomial infections. A nosocomial infection is defined as a new infectious syndrome, such as pneumonia, bacteremia, or urinary tract infection, after 72 hours of hospitalization. These infections often involve multi-drug resistant bacteria (*Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, MRSA).

Management

Initial empirical institution of broad spectrum antimicrobial agents.

Vancomycin when gram-positive pathogens are likely.

Carbapenems (meropenem or imipenem/cilastatin) and amikacin when gram-negative pathogens are likely.

Tailoring of antimicrobial therapy should be guided by the results of bacterial culture and susceptibility data, if available.

For echelon III facilities without this real-time capability, consider batch-testing cultures at another facility that does have that capability, in order to establish local susceptibilities and resistance trends.

Emphasize infection control measures as mentioned under Multi-drug resistant bacteria.

APPENDIX D

DNBI AND HEALTH SURVEILLANCE

- I. Overview.
 - a. Health surveillance consists of any type of information that can be used to inform medical leaders and commanders on the status of the health of the command.
 - b. Typically much effort is focused on “sick call” DNBI (Disease and Non-Battle Injury) surveillance. Although this information is indeed useful in determining the presence of an outbreak of respiratory or gastrointestinal diseases, other forms of health surveillance are also important to report and track. These include:
 - i. Reportable Medical Events (RMEs). These are the vital signs of any public health or preventive medicine activity. These are tracked because of their importance in preventing disease, even in small numbers, usually because of communicability or disease severity.
 - ii. Medical evacuation (MEDEVAC/CASEVAC). Commanders want to know where their soldiers are and what is happening to them.
 - iii. Occupational and Environmental Health Surveillance (OEHS) data from deployed locations.
 - iv. Lost or modified duty. Lost functionality/mission support due to illness or injury.
 - c. These forms of surveillance are outlined in greater detail below, with applicable references and points of contact for access.

- II. Traditional (“Sick Call”) DNBI Surveillance.—**SECURE**
 - a. This information is of great use to the front-line medical providers in detecting outbreaks or clustering of injuries/illnesses that represent opportunities for prevention/intervention and assessing response to any control measures.

- b. Although there have been several ways to report sick call DNBI, CENTCOM theater policy now requires entry into JMeWS. This is discussed below.
- c. References on the procedural aspects and definitions of sick call DNBI are available at: http://amsa.army.mil/AMSA/amsa_home.htm under “Documents,” in the 2002 JCS Memorandum.
- d. A typical sick-call format is shown below, taken from the 2002 JCS Memorandum.



WEEKLY DNBI REPORT



Unit/Command: _____ Troop Strength: _____
Dates Covered: _____ (Sunday 0001) Through _____ (Saturday 2359)
Individual Preparing Report: _____
Phone: _____ E-Mail: _____

CATEGORY	INITIAL VISITS	RATE	SUGGESTED REFERENCE RATE	DAYS OF LIGHT DUTY	LOST WORK DAYS	ADMITS
Combat/Operational Stress Reactions			0.1%			
Dermatologic			0.5%			
GI, Infectious			0.5%			
Gynecologic			0.5%			
Heat/Cold			0.5%			
Injury, Recreational/Sports			1.0%			
Injury, MVA			1.0%			
Injury, Work/Training			1.0%			
Injury, Other			1.0%			
Ophthalmologic			0.1%			
Psychiatric, Mental Disorders			0.1%			
Respiratory			0.4%			
STDs			0.5%			
Fever, Unexplained			0.0%			
All Other, Medical/Surgical						
TOTAL DNBI			4.0%			
Dental		XXXXXX				
Misc/Admin/Follow-up		XXXXXX				
Definable						
Definable						

Problems Identified: _____ Corrective Actions: _____

- e. JMeWS. Current CENTCOM policy is to report all sick call DNBI via JMeWS. This data is considered classified in the U.S. Army, and thus requires security clearance and access to SIPR terminals while deployed. The need for discussing these numbers on the secure side is based on the reporting of population troop strength in calculating these rates.
 - i. Units are responsible for adding themselves to JMeWS via the “Annex Q” input application, although higher headquarters may, and are expected to, monitor these numbers.
 - ii. The JMeWS reporting mechanism may be accessed through the Force Health Protection (FHP) website, at <http://www.fhp.smil.mil> (secure).
 - iii. The JMeWS training website and user’s manual are available at: <https://jmewstraining.saic.com/watchboard/> (unsecure).
 - iv. The FHP site not only allows access to reporting tools, but also contains links to other valuable surveillance activities discussed here, as well as DNBI analysis from AFIOH.
- III. Reportable Medical Events Surveillance (RMES)—**UNSECURE**
- a. RMES is the vital signs of any public health activity, serving as a sentinel for communicable diseases and other preventable diseases of great importance.
 - b. Reporting of RMEs has historically been poor in a deployed environment, since the RMES system was designed for the garrison environment. However, using the procedures outlined below will improve this important activity.
 - c. RMES should be collected by medical providers and sent up the medical chain of command to the Division level. The Division PM officer or designee should then forward up the medical chain of command, **with a copy furnished** by either of the two following procedures:
 - i. The JMeWS II server has RME reporting capability (JMeWS I does not). It will be on-line in late spring or early summer 2005. Report all RME via the Annex Q reporting tool.
 - ii. Forward RME reports to the JMeWS Help Desk at jmews@deploymenthealth.osd.mil or helpdesk@fhp.smil.mil. This will then be forwarded on to AMSA.



DISEASE AND INJURY REPORT FORM

Europe Regional Medical Command
(ERMC REG 40-11)



PATIENT DATA:

Last Name										First Name									
FMP		Social Security Number								Grade		Date of Birth							
Permanent Residence - City or Location										Gender:		☐ MALE ☐ FEMALE							
Country*		APO				Category*				Race:		☐ WHITE ☐ BLACK ☐ HISPANIC							
												☐ ASIAN ☐ AM INDIAN ☐ OTHER							
Unit										UIC									
Unit Location - (e.g. Camp Bondsteel)										Duty Phone									

DISEASE DATA:

Diagnosis Code*		Diagnosis - Refer to Tri-Service Case Definitions		Onset of Symptoms	
Confirmed:		Method of Confirmation:		Admitted:	
☐ YES ☐ NO ☐ PENDING		☐ CLINICAL ☐ CULTURE ☐ SLIDE ☐ BIOPSY ☐ SEROLOGY ☐ OTHER		☐ YES ☐ NO	
				Date of Admission	

Additional information on reverse required for Malaria & Heat/Cold Injuries

REPORTING SOURCE:

Health Care Provider: _____		Comments/Additional Information: (See Tri-Service Reportable Events Guidelines)	
PM POC: _____			
Phone #: _____			
Reporting MTF: _____			
Reported to: Chief, PM for Health Service Area [†] ...		YES	NO
(Landstuhl Regional Medical Center)		☐	☐
Local PM Consultant.....		☐	☐
(Non-STD cases only)			

[†] LRMCM Phone: DSN 486-8111, FAX: DSN 486-8488, Mail: Commander, LRMCM Att: MCEUL-PM, CMR 402, APO AE 09180

FOR PROGRAM MANAGER ONLY:

Case Entered in RMES Database	YES ☐	NO ☐	Date Transmitted to AMSA _____

*SEE REVERSE SIDE FOR CODES

REPORTABLE MEDICAL EVENTS REPORTING FORM, Pg. 1

HEAT OR COLD INJURIES ONLY

Ambient temperature					°C / °F	WBGT					Previous Heat or Cold injury:	☐ YES	☐ NO
Wind Speed					MPH	Body Part or Organ System Affected:							
Rectal temperature					°C / °F		Multi-system involvement: ☐ YES ☐ NO						

MALARIA CASES ONLY

Pertinent Travel:	☐ YES	Country #1	
	☐ NO	Country #2	
Malaria Chemoprophylaxis:	☐ YES	Prophylaxis #1	
	☐ NO	Prophylaxis #2	

DISEASE CODES

006 Anemias	992 Heat stroke	071 Rabies, human
022 Anthrax	065 Hemorrhagic fever	087 Relapsing fever
E997.1 Biological warfare agent exposure	070.1 Hepatitis A, Acute	390 Rheumatic fever, Acute
005.1 Botulism	070.3 Hepatitis B, Acute	066.3 Rift Valley fever
023 Brucellosis	070.51 Hepatitis C, Acute	082 Rocky Mountain Spotted fever
		056 Rubella
008.43 Campylobacter	487 Influenza	
986 Carbon monoxide poisoning		003 Salmonellosis
989 Chemical agent exposure	984 Lead poisoning	120 Schistosomiasis
099.41 Chlamydia	482.8 Legionellosis	004 Shigellosis
001 Cholera	085.4 Leishmaniasis, cutaneous	050 Smallpox
114 Coccioidiomyces	085.5 Leishmaniasis, mucocutaneous	038 Streptococcus, Grp. A, invasive
991.3 Cold injury, frostbite	085.9 Leishmaniasis, unspecified	090 Syphilis, congenital
991.6 Cold injury, hypothermia	085 Leishmaniasis, visceral	096 Syphilis, latent
991.4 Cold injury, immersion type	030 Leprosy	095 Syphilis, late (tertiary)
991.9 Cold weather injury, unspecified	100 Leptospirosis	091 Syphilis, primary/secondary
136.8 Cryptosporidiosis	027 Listeria	097.9 Syphilis, unspecified
007.8 Cyclospora	088.81 Lyme disease	
		037 Tetanus
061 Dengue fever	084 Malaria, falciparum	785.59 Toxic shock syndrome
032 Diphtheria	084.2 Malaria, malariae	124 Trichinosis
	084.3 Malaria, ovale	086 Trypanosomiasis
008.04 E. coli O154:H7	084.6 Malaria, unspecified	011 Tuberculosis, pulmonary
083.8 Elicthiosis	084.1 Malaria, vivax	021 Tularemia
062 Encephalitis	055 Measles	002 Typhoid fever
	036 Meningococcal dis., Meningitis	080 Typhus fever
125 Filariasis	036.2 Meningococcal dis., Septicemia	
	072 Mumps	099.4 Urethritis, non-gonococcal
007.1 Giardiasis	033 Pertussis	979.9 Vaccine, adverse event
098 Gonorrhea	020 Plague	052.9 Varicella, active duty only
	481 Pneumococcal pneumonia	
038.41 Hemophilus influenza, invasive	045 Poliomyelitis	060 Yellow Fever
079.81 Hantavirus infection		
992.3 Heat exhaustion	083 Q fever	

CATEGORY CODES

A11 Army active duty	F41 DEP Air Force active duty	N11 Navy active duty
A31 Army retired	F43 DEP Air Force retired	N31 Navy retired
A41 DEP Army active duty	M11 Marine active duty	N41 DEP Navy active duty
A43 DEP Army retired	M31 Marine retired	N43 DEP Navy retired
F11 Air Force active duty	M41 DEP Marine active duty	K59 Civilian DEP Civilian
F31 Air Force retired	M43 DEP Marine retired	K79 Local National

COUNTRY CODES

BS Bosnia-Herzegovina	HU Hungary	SP Spain
CR Croatia	IT Italy	TU Turkey
CZ Czech Republic	KV Kosovo	UK United Kingdom
GE Germany	MC Macedonia	US United States

PRIVACY ACT INFORMATION

Authority: Section 133, Title 10, United States Code (10 USC 133)

Purpose: The purpose of this form is to compile relevant patient information concerning communicable diseases and injuries occurring among Department of Defense personnel and family members stationed or operating in Europe.

Routine Use: Used to monitor for the emergence of specific communicable diseases or outbreaks which pose a public health threat and to prepare data for inclusion in the U.S. Army Medical Surveillance System.

Disclosure: The requested information is mandatory for compliance with U.S., Host Nation and Army disease reporting laws and regulations. Failure to provide the requested information will prevent effective public health action and contribute to higher disease and injury rates.

- IV. Medical Evacuation Surveillance—**UNSECURE**. This information can be of use in tracking individual patients and what their status is in the evacuation stream.
- a. USTRANSCOM TRAC2ES system shows MEDEVAC information. Available at: <https://www.trac2es.transcom.mil/tdbs/SilverStream/Pages/pgLogin.html>
POC: C, GPMRC Systems, DSN 312-779-7161
 - b. Landstuhl Regional Medical Center (LRMC) Joint Patient Tracking Application (JPTA) shows patient information at LRMC. Available at: <https://dwmmc.lrmc.amedd.army.mil/PatientInformation/secured/login.aspx>
POC: DWMMC, LRMC, DSN 314-486-8127
- V. Occupational and Environmental Health Surveillance (OEHS).—**SECURE**
- a. This information is useful in determining occupational or environmental exposures in deployed settings. Soldiers often return to home station reporting exposures related to deployment, such as asbestos in buildings or drinking water contamination. The OEHS database is the appropriate first resource for determining the basis of these claims.
 - b. The OEHS database is available via the USACHPPM secure website, at www.usachppm1.smil.mil, or via link from the FHP secure website.

APPENDIX E

TRI-SERVICE REPORTABLE MEDICAL EVENTS

Amebiasis	Listeriosis
Anthrax	Lyme disease
Biological warfare agent exposure	Malaria
Botulism	Measles
Brucellosis	Meningococcal disease
Campylobacteriosis	Mumps
Carbon monoxide intoxication	Pertussis
Chemical agent exposure	Plague
Chlamydia	Pneumococcal pneumonia
Cholera	Poliomyelitis
Coccidioidomycosis	Q fever
Cold weather injury	Rabies, human
Cryptosporidiosis	Relapsing fever
Cyclospora	Rheumatic fever
Dengue fever	Rift Valley fever
Diphtheria	Rocky Mountain spotted fever
E. coli 0157:H7	Rubella
Ehrlichiosis	Salmonellosis
Encephalitis	Schistosomiasis
Filariasis	Shigellosis
Giardiasis	Smallpox
Gonorrhea	Streptococcal infection, grp A
Haemophilus influenzae, invasive	Syphilis
Hantavirus disease	Tetanus
Heat exhaustion	Toxic shock syndrome
Heat stroke	Trichinosis
Hemorrhagic fever	Trypanosomiasis
Hepatitis A	Tuberculosis
Hepatitis B	Tularemia
Hepatitis C	Typhoid fever
Influenza	Typhus fever
Lead poisoning	Urethritis, non-gonococcal
Legionellosis	Vaccine adverse event report
Leishmaniasis, unspecified	Varicella, active duty only
Leprosy	Yellow fever
Leptospirosis	

APPENDIX F

LEISHMANIA SCRAPING AND BIOPSY PROCEDURES

1) Criteria for scraping or biopsy:

- Any patient who has had a non-healing lesion (does not have to be an open, weeping ulcer) for greater than 3-4 weeks needs to be suspected of having leishmaniasis.
- These patients should initially be placed on a course of antibiotic therapy for 7-10 days with an antibiotic that has proven activity in Iraq and Afghanistan (recommendation is Augmentin 875 mg BID for 7-10 days).
- At the conclusion of therapy, the patient should be seen by the same practitioner and a decision needs to be made if there was any efficacy to the course of antibiotics. If the lesion has persisted or worsened, a scraping or biopsy should be performed.
- Photos of the lesion prior to scraping or biopsy being done should be taken if the practitioner has the capability. E-mail these photos to WRAIR since this may help in the diagnosis (peter.weina@us.army.mil) and to either derm.consult@us.army.mil or id.consult@us.army.mil

2) Scraping procedure:

- Clean area with alcohol pads and allow to dry.
- Anesthetize with lidocaine 1% or 2% with epinephrine 1:100,000 (unless the epinephrine is contraindicated due to anatomic site).
- Two tissue smears are performed by horizontally scraping (lightly enough to elicit an exudate, but not vigorously enough to cause bleeding) the base of the underlying ulceration with a #15 blade (this often requires removal of the overlying crusted debris). The dermal tissue is then thinly applied in a circular fashion to a dime- to nickel-sized area in the center of the slide. Minimize blood, epithelium (keratinocytes), and purulence on the specimen.
- Additionally, material from the scrapings (and even the overlying crusted debris) should be inserted into a small vial of 95-100% ethanol for PCR analysis.
- Ensure slides are labeled per the format of your

affiliated pathology department and submit per their protocol. If pathology services unavailable locally, ship per address below. Work closely with pathologists to verify adequacy of tissue smear samples.

3) Biopsy/touch prep-impression smear procedure:

- An area of the lesion needs to be cleaned thoroughly with alcohol pads and dried.
- The anticipated area of biopsy should be anesthetized as described above.
- A 4 mm sterile disposable punch or sterile scalpel (#15, #11, or #10) should be used to remove a piece of tissue approximately 3 to 4 mm in circumference and approximately 3 mm deep from the edge of the lesion (see photo for preferred area of biopsy). Lesions on the face, anterior of the neck, and near larger vessels and/or nerves need to be biopsied with extreme caution and a simple surface scraping (described above) may be preferred to a true biopsy.
- The biopsy specimen should be placed on sterile, clean, 2X2 dry gauze briefly to absorb excess blood on the tissue that may interfere with the reading of the touch preparations.
- The tissue should be grasped with forceps and impression smears made on clean slides (four for each biopsy) by rubbing the tissue gently across the surface of the slide in a circular motion.
- Dry thoroughly. Fix with methanol if available.
- The tissue biopsy specimen (after the impression smears are made) should then be placed in a very small amount of ethyl alcohol (just enough to cover the specimen) in a leakproof vial (such as a Nunc™ transport tube).
- The slides and the vial with the tissue should be shipped per your local pathology section protocol or via DHL or Federal Express to the address below. The container should be labeled as diagnostic specimens and no shipping permit is required (all MTFs have personnel and resources to ship diagnostic specimens correctly).
- Complete the patient information sheet provided below and include it with the specimen for each patient biopsied.

- Procedural inquiries should be made to LTC Peter Weina at (301) 319-9956.

SHIPPING ADDRESS

LTC Peter Weina
Director, Leishmania Diagnostic Laboratory
Division of Experimental Therapeutics
503 Robert Grant Avenue
Walter Reed Army Institute of Research
Silver Spring, Maryland 20910-7500

Preferred biopsy area:



Leishmaniasis Patient Information Sheet

Soldier completes Part A; Clinical provider completes Part B

PART A – SOLDIER

Patient Name: _____ SSN: _____ Rank/ Service: _____

Blood type _____ Weight _____ Med Allergies _____ Age _____ DOB: _____

Unit: Company _____ BN _____ BDE/BCT _____ DIV _____

Date soldier arrived in Theater: _____ in Iraq: _____

Places/dates lived in Iraq: (e.g., *FOB Murphy, 10 Jun – 15 Jul 03*) _____

Were rodents present around bivouac area? Y / N Were dogs in the area? Y / N

Places You Slept	# Weeks or N/A	Screens Or Windows? (Y/N)	A/C (Y/N)	Use Bednet (Always/ Sometimes/Never)	Use Repellent (Always/ Sometimes/Never)	Insect Bites Per Night? (<5, 5-20, >20)
Vehicle or Ground						
Tent						
Building						

Your Use of Insect Repellents	Product Was Not Available to Soldier	Product was Available to Soldier			
		Did Not Use	Used Only After Insect Bites – After how many bites? (<5, 5-20, >20)	Used Every Night	Used Other Times <i>Describe When</i>
Bed Net, Treated w/ Permethrin					
Bed Net w/o Permethrin					
Permethrin Treated DCUs					
DEET (green tube) on Skin					
Commercial Insect Repellent					
If Yes, List in Box					

PART B – CLINICAL PROVIDER (*Send form with slides and biopsy*)

Lesion Location & #: _____ Duration? _____

Antibiotic Treatment (type/dose/length): _____

Photos Taken? **N / Y** If Yes, sent to WRAIR? **N / Y**Procedures Done: Scrape Biopsy: **N / Y** Punch Biopsy: **N / Y**Culture: **N / Y** Preserved Tissue: **N / Y** Touch Prep: **N / Y**PCR: **N / Y**

Date Eval: _____ MTF: _____ POC: _____ Phone: _____

E-mail(POC): _____

Clinician Name _____ E-mail (Provider): _____

(stamp) =====

Results: (**POS/NEG**) _____

Notes: _____

APPENDIX G
SKIN DISEASES SEEN IN THE DEVELOPING WORLD

Note: This table lists the most commonly seen skin diseases in rural areas of tropical developing nations. Urbanization, prosperity, and higher latitude will alter the mix.

Condition	Comment
Eczema and dermatitis	Often secondarily infected
Infestations of scabies and head lice;	Often secondarily infected
Tinea (pityriasis) versicolor	Nearly universal in some populations
Pyoderma	Primary infections or secondary infected sites
Dermatophytosis	
Acne vulgaris	
Pigmentary disorders	Often pityriasis alba, melasma, and vitiligo

APPENDIX H

TREATMENT GUIDELINES FOR SELECTED SKIN CONDITIONS

Simple guidance for the training of primary health care workers using the basic unit. (Adapted from World Health Organization: The New Emergency Health Kit. WHO/DAP/90.1. Geneva, 1990.)

Wounds, limited and superficial	Clean with clean soap and water with diluted chlorhexidine solution. Apply gentian violet daily
Burns, mild or moderate	Immerse immediately in cold water or use a cool compress. Continue until pain eases then treat the wounds.
Bacterial infection, mild	Clean with clean soap and water or with diluted chlorhexidine solution. Apply gentian violet twice daily.
Fungal infection	Apply gentian violet daily for 5 days.
Scabies, noninfected	Apply benzyl benzoate.
Scabies, infected	Treat mild bacterial infection as above. When infection is cured, apply benzyl benzoate.

APPENDIX I
EMERGENCY DERMATOLOGY HEALTH KIT

Basic unit

- Benzyl benzoate lotion 25%
- Chlorhexidine 5%
- Gentian violet, powder (needs reconstitution)
- Sulfamethoxazole-trimethoprim 400/80 mg
- Tetracycline eye ointment 1% in 5 gm tubes
- Mebendazole, aspirin, paracetamol acetaminophen

Supplementary unit

<u>Antibiotics</u>	<u>Corticosteroids</u>	<u>Topicals and miscellaneous</u>
Ampicillin, 250-mg tablets Ampicillin, 500-mg vials Penicillin benzathine, 2.4-mU vials Penicillin procaine, 3.4-mU vials Chloramphenicol, 250-mg capsules Chloramphenicol, 1-g vials Nystatin, 100,000 IU tablet Tetracycline, 250-mg capsules	Dexamethasone (injectable) Prednisolone (oral)	Povidone iodine 10% solution Zinc oxide 10% ointment Benzoate 6% /salicyate 3%(Whitfield's) ointment Lidocaine

GLOSSARY

ACLS

Advanced Cardiac Life Support

AEP

acute eosinophilic pneumonia

AFB

acid fast bacilli

AFMIC

Armed Forces Medical Intelligence Center

AFIP

Armed Forces Institute of Pathology

ALT

alanine transaminase (SGPT)

AMS

acute mountain sickness

AOR

area of responsibility

APG

Aberdeen Proving Ground

AKN

acne keloidalis nuchae

APHA

American Public Health Association

ARDS

adult respiratory distress syndrome

ASAP

as soon as possible

AST

aspartate transaminase (SGOT)

ATP

adenosine triphosphate

BAL

bronchio alveolar lavage

BDO

battle dress overgarment

BID

twice per day

BP

blood pressure

BW

biological warfare

CA-MRSA

community-acquired methicillin resistant staphylococcus aureus

CAP

community-acquired pneumonia

CBC

complete blood count

CCHF

Crimean-Congo Hemorrhagic Fever

CDC

Center for Disease Control

CINC

Commander in Chief

CNS

central nervous system

CONUS

continental United States

CPAP

continuous positive airway pressure

CPK

creatinine phosphokinase

CPR

cardiopulmonary resuscitation

CSF

cerebrospinal fluid

CSH

Combat Support Hospital

CXR

chest x-ray

DF

Dengue fever

DIC

disseminated intravascular coagulation

DNA

deoxyribonucleic acid

DNBI

Disease and Non-Battle Injury

DOD

Department of Defense

DOT

directly observed therapy

ECG

electrocardiogram

EDTA

ethylenediamine tetra-acetic acid

ELISA

Enzyme-linked Immunosorbent Assay

EPAP

expiratory positive airway pressure

EPICON

Epidemiologic Consultative Team

ESR

erythrocyte sedimentation rate

EVAC

evacuation (medical)

FAC

free available chlorine

FDA

Federal Drug Administration

FHP

Force Health Protection

FM

field manual

GAS

Group A Streptococcus

GCSF

granulocyte colony stimulating factor

GI

gastrointestinal

HACE

high altitude cerebral edema

HAPE

high altitude pulmonary edema

HARH

high altitude retinal hemorrhage

HBIG

hepatitis B immune globulin

HC

hydrocarbon

HCP

health care practitioner

HCT

hematocrit

HCW

health care worker

HDCV

human diploid cell vaccine

HEPA

high efficiency particulate air

HF

hydrogen fluoride

HFRS

hemorrhagic fever with renal syndrome

HGB

hemoglobin

H-HC

halogenated hydrocarbon

HI

hemagglutination-inhibition

HRIG

human rabies immune globulin

HTH

high test hypochlorite

ICU

intensive care unit

IFA

indirect fluorescent antibody (test)

IgG

immunoglobulin G

IgM

immunoglobulin M

IM

intramuscular

IND

investigational new drug

INH

isoniazid

IU/kg

international units per kilogram

IV

intravenous

LDH

lactate dehydrogenase

LP

lumbar puncture

LRMC

Landstuhl Regional Medical Center

MAT

microscopic agglutination testing

MDI

methylene diisocyanate

MTE

military treatment facility

MTWTF

miosis, tachycardia, weakness, hypertension, and fasciculations

MW

molecular weight

NATO

North Atlantic Treaty Organization

NBC

nuclear biological chemical

NFCI

non-freezing cold injury

NNMC

National Naval Medical Center

NSAID

nonsteroidal antiinflammatory drug

NTE

neuropathic target esterase

OCONUS

outside continental United States

OD

once per day

OEF

Operation Enduring Freedom

OEHS

Occupational and Environmental Health Surveillance

OIF

Operation Iraqi Freedom

OP

organophosphates

OSHA

Occupational Safety and Health Administration

312 GLOSSARY

OTC

over the counter

PCE

perchlorethylene

PCR

polymerase chain reaction

PEEP

positive end expiratory pressure

PFB

pseudofolliculitis barbae

PNS

peripheral nervous system

PO

by mouth

PPD

purified protein derivative

PPT

prolonged prothrombin time

PR

Petyriasis Rosea

PT

prothrombin time

PTT

partial thromboplastin time

PVC

premature ventricular contractions

QD

per day

QID

four times per day

RADS

reactive airways dysfunction syndrome

RBC

red blood cell

RMES

reportable medical events surveillance

RNA

ribonucleic acid

RPR

rapid plasma reagin

SCC

squamous cell carcinomas

SEB

staphylococcal enterotoxin B

SLUDGE

salivation, lacrimation, urination, defecation, gastroenteritis, emesis

SMX

sulfamethoxazole

STD

sexually transmitted disease

TB

tuberculosis

TCA

trichloroethane

TCE

trichloroethylene

TG

technical guide

TIA

transient ischemic attacks

TIC

toxic industrial chemical

TID

three times per day

TMP

trimethoprim

UAE

United Arab Emirates

USACHPPM

U.S. Army Center for Health Promotion and Preventive Medicine

USAID

U.S. Agency for International Development

USAMRICD

U.S. Army Medical Research Institute of Chemical Defense

USAMRIID

U.S. Army Medical Research Institute of Infectious Diseases

USARIEM

U.S. Army Research Institute of Environmental Medicine

USCENTCOM

U.S. Central Command

URI

upper respiratory infection

UV

ultraviolet

VEE

Venezuelan equine encephalitis

VF

ventricular fibrillation

VHF

viral hemorrhagic fever

VT

ventricular tachycardia

WBC

white blood cell count

WNL

within normal limits

WP

white phosphorus

WRAIR

Walter Reed Army Institute of Research

WRAMC

Walter Reed Army Medical Center

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PART ONE:	ENDEMIC INFECTIOUS DISEASES
PART TWO:	EXPOSURE TO BIOLOGICAL WARFARE AGENTS
PART THREE:	EXPOSURE TO CHEMICAL WARFARE AGENTS
PART FOUR:	TOXIC INDUSTRIAL CHEMICAL EXPOSURES
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