Problem Oriented Differential Diagnosis of Tropical Diseases

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Problem oriented differential diagnosis of tropical diseases

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A problem oriented differential diagnosis of tropical diseases was written for primary care physicians. The text describes the basic principles of travel medicine, survival in the tropics, and heat injury. Tables organizing the incubation period and geographic distribution of tropical diseases are provided. The remaining text discusses tropical diseases organized by organ system, signs, and symptoms. The purpose is to assist primary care physicians in arriving at a limited differential diagnosis of tropical diseases before consulting tropical medicine texts and initiating empiric therapy and diagnostic workup.
Table of Contents

Problem-oriented Differential Diagnosis of Tropical Diseases

<table>
<thead>
<tr>
<th>Chapter</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preface</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Chapter 1</td>
<td>Introduction</td>
<td>3</td>
</tr>
<tr>
<td>Chapter 2</td>
<td>Medical advice for the traveler</td>
<td>5</td>
</tr>
<tr>
<td>Chapter 3</td>
<td>Human factors in heat injury, and in jungle and desert survival</td>
<td>40</td>
</tr>
<tr>
<td>Chapter 4</td>
<td>Differential diagnosis of infectious tropical diseases based on travel history</td>
<td>56</td>
</tr>
<tr>
<td>Chapter 5</td>
<td>Fever</td>
<td>91</td>
</tr>
<tr>
<td>Chapter 6</td>
<td>Neurological complications</td>
<td>96</td>
</tr>
<tr>
<td>Chapter 7</td>
<td>Ophthalmological complications</td>
<td>115</td>
</tr>
<tr>
<td>Chapter 8</td>
<td>Ear, nose, and throat complications</td>
<td>125</td>
</tr>
<tr>
<td>Chapter 9</td>
<td>Pulmonary complications</td>
<td>131</td>
</tr>
<tr>
<td>Chapter 10</td>
<td>Cardiovascular complications</td>
<td>140</td>
</tr>
<tr>
<td>Chapter 11</td>
<td>Hepatobiliary complications</td>
<td>148</td>
</tr>
<tr>
<td>Chapter 12</td>
<td>Gastrointestinal complications</td>
<td>159</td>
</tr>
<tr>
<td>Chapter 13</td>
<td>Genitourinary complications</td>
<td>177</td>
</tr>
<tr>
<td>Chapter 14</td>
<td>Musculoskeletal complications</td>
<td>188</td>
</tr>
<tr>
<td>Chapter 15</td>
<td>Dermatological complications</td>
<td>196</td>
</tr>
<tr>
<td>Chapter 16</td>
<td>Hematological complications</td>
<td>220</td>
</tr>
<tr>
<td>References</td>
<td></td>
<td>232</td>
</tr>
</tbody>
</table>


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Preface to military physicians

The military physician is faced with diagnosing and treating tropical diseases on a routine basis.

Some of these diseases are limited by their geographic distribution to the tropics, and therefore, are rarely seen in the United States of America. But traveling servicemembers do bring home tropical diseases to their physicians based in temperate areas. Nearly an entire unit returns from an exercise in Panama with fever and elevated liver function tests. A soldier returns from an assistance visit to Kenya and has a vasovagal syncope episode at the nearby airport as he steps off the plane with fever and chills. An officer returns from a desert exercise in the Middle East with a strange skin lesion.

Some of these diseases have been nearly eradicated from the United States of America by public health measures, but remain quite prevalent in third world countries. Polio, measles, diphtheria, hepatitis, salmonella and many more, are still endemic in many countries. A soldier entering these areas is exposed to these illnesses.

Some of these diseases only become manifest in the turmoils and disruptions of warfare itself. Melioidosis became a frequent cause of pulmonary disease in South Vietnam, thought to be due to the disturbance of the soil by digging trenches and foxholes. A unit rested in a field in the Pacific only to be stricken by scrub typhus at a later date. There was an outbreak of louse-borne typhus as a Russian army encircled a trapped, desperate German army. Cystic acne and fungal skin infections were an unexpected and common cause of severe disability among our troops in South Vietnam, aggravated by the tropical and operational environment.

A military physician was suddenly deployed to a rural area in the tropics, and not only had to be wary of the development of tropical diseases in his unit, but also provided primary care to the local population who were already infected with a variety of tropical diseases.

Many biowarfare agents are exotic or tropical diseases. Though their use is limited by the Geneva Conventions, military physicians may be faced with an attack on their unit by these agents of warfare.
This manual was written by a primary care physician for primary care physicians to solve a dilemma. We are faced with evaluating the initial presentation of tropical diseases, but there is no reference that arranges tropical diseases by geographical distribution, incubation period, and by presenting signs and symptoms. The tropical medicine textbooks arrange tropical diseases by infectious agent. The indexes of these texts do not direct you to the differential diagnosis of nodular skins lesions, hilar adenopathy, hepatitis, etc. Infectious disease consultants may not be readily available or may be inexperienced in tropical diseases. The primary care physician must have a tool to reduce the number of possible tropical diseases from 170 to a handful before ordering tests, initiating empiric therapy, and consulting the tropical medicine textbooks and literature for the finer details of a tropical illness. This handbook is organized to get you give you a head start in diagnosing tropical diseases and diseases of third world countries.

I do not pretend to be an expert in tropical diseases, but have read three major tropical medicine textbooks from front to back and other standard references in order to prepare this text. I have been frustrated by multiple encounters with tropical diseases in my military practice, attempting to use these standard references to form a basic differential diagnosis. My medical consultants were often in the same situation, lacking a solid background in practical, clinical tropical medicine. I conceived this text to help me in my practice of military medicine, never knowing who would walk into my office next or what country I may be in tomorrow. The first draft of this manual was field tested for one year by an internist operating a large travel medicine clinic at Yale University and found the manual very helpful in daily practice.

I would like to express my deep appreciation to the peer reviewers of this manual for all of their support, critiques, and recommendations. Their vast experience in tropical disease from both the military and civilian perspective was invaluable. These peer reviewers are Dr. Wilbur Downs, MD, MPH; Dr. Robert E. Shope, MD; and Dr. Ralph E. Evans, MD; MPH; all professors at the Yale School of Medicine, Department of Epidemiology and Public Health.

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CHAPTER I

Introduction to
Problem-oriented Differential Diagnosis of Tropical Diseases

It is estimated that by 1990, 700-800 million passengers a year will travel by air carriers. By 1995, hypersonic transports will carry passengers from the United States of America to the Orient in a matter of hours. The mobility of the world population is increasing every year. American travelers often visit the tropics. This country accepts a steady flow of immigrants and tourists from the tropics. Immune-compromised patients are increasing in number and can be opportunistically infected with many diseases that are considered to be exotic or tropical. Therefore, it is likely that physicians will be increasingly involved in evaluating patients that may have a tropical disease.

The average physician may not see the need to study tropical diseases. So when confronted with a patient that may have a tropical disease, the physician has difficulty deciding on what diagnostic approach to take. Having the basic information in hand, the travel and illness history, the physical examination and preliminary laboratory data, the physician turns to the literature for direction. But textbooks on tropical medicine are arranged by the etiologic agent of infection and poorly cross-referenced by clinical problems.

This handbook is arranged to assist physicians in forming a complete preliminary differential diagnosis of tropical diseases to complement the usual differential diagnosis of more familiar diseases. This will allow physicians to focus their literature review, resulting in a more timely initiation of empiric therapy pending receipt of specific confirming diagnostic tests.
"Problem-oriented Differential Diagnosis of Tropical Diseases" is arranged into three parts:

Part one

Chapter II Medical advice for the traveler and treating physician

Chapter III Human factors in heat injury, and in desert and jungle survival

Part two

Chapter IV Travel history

An array of tables that utilize information from the travel history to provide the first step in forming a differential diagnosis. The tables are arranged by estimated incubation period, geographical area visited, and maximum historical risk of contraction of infectious tropical diseases by the traveler.

Part three

Chapters V through XVI Clinical Presentation.

Each chapter is arranged by clinical problems to further refine the preliminary differential diagnosis.

With a scratch pad in hand, a physician can flow through Parts TWO and THREE and form a differential of five to fifteen possible tropical diseases. This provides a focal point to make efficient use of time for reading the literature on these diseases and formulating a rational approach to diagnosis and treatment.
Chapter II
Medical advice for the traveler

Introduction
I. Travel in the tropics

Travelers' diarrhea
I. Introduction
II. Etiology
III. Prevention
IV. Treatment

Personal protection measures
I. Accidents
II. Animals
III. Arthropods
IV. Chronic disease
V. Clothing
VI. Culture
VII. Food and drink
VIII. Gynecological problems
IX. Handicrafts
X. Heat
XI. High Altitude
XII. Personal Security
XIII. Pregnancy
XIV. Sexually transmitted diseases
XV. Swimming

Immunization and chemoprophylaxis
I. Recommendations

Medical problems of air travel
I. Introduction
II. Hypoxia
III. Changes in barometric pressure
IV. Neuropsychiatric problems
      (including motion sickness and jet lag)
V. Miscellaneous problems of air travel

Medical problems of sports diving
I. Introduction
II. Problems of inspired air
III. Compression and decompression problems
IV. Other environmental hazards while diving
V. Psychiatric problems

Summary checklist for the traveler and medical staff
I. Pre-trip   II. Trip   III. Post-trip
Introduction

I. Travel in the tropics

The tropical traveler comes from many walks of life. The student, the research scientist, the adventurer, the retiree, the businessman, the visiting relative, and the vacationer all require a complex array of medical needs for tropical travel; and all have varying levels of experience in the planning and execution of a safe, healthy trip. But, it is the first time visitor who especially needs reassurance and counseling to dispel the myths and images of the tropics. Engrained into the traveler's unconsciousness by movies and novels is the notion that the tropics are desolate and primitive, where man is assaulted by broiling heat, diarrhea, swarming insects, hostile natives, and man-eating animals. The reality is that comfortable lodging, good food, air conditioning, and good medical care by English-speaking physicians are available in all major tropical towns and cities. At times, the heat, humidity, and rain can be oppressive, but this is balanced by many pleasant days. The climate in many areas of the tropics is often not that much different from southern Florida, the lower Rio Grande River basin, or southern California.

Depending on the season, location visited, and prior health status, tropical travelers may actually be at higher risk for suffering a relapse of their own chronic medical problems than from acquiring a tropical disease. Most of the tropical diseases of significance are infectious diseases that may be prevented by immunization, chemoprophylaxis, and common sense personal protection with insect repellents, proper clothing, mosquito netting, and good personal hygiene.

This chapter will provide primary care practitioners with information about travelers' diarrhea, personal protection measures for tropical travel, immunizations, and chemoprophylaxis for major infectious tropical diseases, and the medical problems of air travel and sports diving. The chapter is concluded with summary pre-trip, trip, and post-trip checklists.
The specific details of immunizations, chemoprophylaxis, and infectious disease alerts are available from several sources that keep updated information on a weekly or monthly basis as follows:

1. Traveler's clinics, established in university health care systems and large corporations, and are increasing in number and quality each year.

2. State health departments.

3. Centers for Disease Control (CDC), (404)-329-3311. CDC also annually publishes a booklet, Health Information for International Travel, available from:
   U.S. Department of Health and Human Services
   Public Health Service
   Centers for Disease Control
   Center for Prevention Services
   Division of Quarantine
   Atlanta, Georgia, 30333

4. World Health Organization.

Chapter III below, Human Factors in Heat Injury and in Desert and Jungle Survival, will provide the reader with additional information for the tropical traveler at risk for developing heat injury or at risk for being isolated in the tropics in an emergency situation while traveling in rural areas.

Travelers' diarrhea

I. Introduction

A well-known complication of tropical travel among laymen is travelers' diarrhea, but actually the prevalence of this medical problem is quite variable, ranging from infrequent to common. The individual risk of developing travelers' diarrhea is dependent on the traveler's itinerary, where and what foods the traveler consumes, and the level of contamination of consumed fluids.

The tropical traveler is just as likely to develop constipation as diarrhea. The effects of jet lag, dehydration, irregular eating schedules, and altered diet contribute to the development of constipation.
Travelers’ diarrhea is a self-limited and rarely fatal, acute onset of frequent, unformed stools; possibly accompanied by fever, malaise, nausea, vomiting, abdominal distension, abdominal cramps, tenesmus, and dysentery. Traveler’s diarrhea may be mild or force the traveler to bed. The syndrome usually lasts for a few days, but some cases may become chronic. The traveler may have more than one episode during a trip or may have a relapse of signs and symptoms after returning home.

II. Etiology

Multiple infectious agents have been associated with travelers’ diarrhea. The psychosocial stresses and the dietary disruptions of travel, over-indulgence of foods irritating to the bowel, drug side-effects, and exacerbation of chronic intestinal diseases may also contribute to the production of loose stools and abdominal cramps in travelers. The more frequently isolated infectious agents are:

   Enterotoxigenic Escherichia coli, the most common agent isolated.
   Shigellosis.
   Salmonellosis, including typhoid fever.
   Acute diarrhea due to Campylobacter.
   Rotaviral enteritis.
   Unknown.

2. Uncommon.
   Vibrio parahaemolyticus food poisoning, most often from ingestion of raw or poorly cooked seafood.
   Other viral gastroenteropathies, such as Norwalk-like virus.
   Giardiasis.
   Amebiasis.

3. Occasional.
   Aeromonas hydrophilia.
   Yersiniosis.
   Pleisiomonas shigelloides.
   Cholera.
   Vibrio fluvialis.
   Cryptosporidiosis.
   Balantidiasis.
   Strongyloidiasis.
III. Prevention

The academic arena for the prevention of travelers' diarrhea has been alive with controversy as methods to prevent the problem were identified and tested. However, the current consensus of opinion is that on the basis of apparent risk/benefit ratios, prophylactic antimicrobial agents are not recommended for the prevention of travelers' diarrhea and that utilizing personal protection measures by rational dietary precautions remains a reasonable and effective method of prevention. The details of these dietary precautions are outlined in the Personal Protection Measures Section below.

The role for antimicrobial therapy is reserved for the early and efficacious treatment of travelers' diarrhea, should it develop; or for the chemoprophylaxis of selected, high risk patients, for whom the adverse effects of travelers' diarrhea out-weigh the risk of potential antimicrobial drug reactions.

For high risk patients, the two most effective choices for chemoprophylaxis and the potential adverse reactions are:

1. Doxycycline; 100 mg daily for three weeks: should not be used in women who are pregnant or breast feeding, infants, children, and patients sensitive to tetracyclines; important adverse reactions include photosensitivity reaction, gastroenteritis, enterocolitis, glossitis, esophagitis, monilial over-growth, hypersensitivity reactions, hemolytic anemia, and eosinophilia.

2. Trimethoprim-sulfamethoxazole, double-strength; one tablet daily for three weeks: should not be used in women who are near term during pregnancy or breast feeding, infants less than two months of age, patients with megaloblastic anemia due to folate deficiency, and patients sensitive to trimethoprim or sulfonamides; important adverse reactions include blood dyscrasias, gastroenteritis, glossitis, hepatitis, pancreatitis, pseudomembranous colitis, central nervous system reactions, hypersensitivity reactions, Stevens-Johnson syndrome, toxic nephrosis, and hypoglycemia.

Studies in Mexico have shown that bismuth subsalicylate tablets are effective in the reducing travelers' diarrhea attack rate from 40% in controls, taking no tablets, to 14% in subjects taking two bismuth subsalicylate tablets four times a day. Since this protocol may be complicated by salicylate intoxication, patients taking salicylates or who are sensitive to salicylates should not follow this suggested protocol.
The Centers for Disease Control is not recommending bismuth subsalicylate for the prophylaxis of travelers' diarrhea.

All other over-the-counter medications touted for preventing travelers' diarrhea should be avoided by the traveler. For example, iodochlorhydroxyquin and other halogenated hydroxyquinoline compounds (Entero-Vioform, Meyaform, Intestopan, and others) have not been shown to prevent travelers' diarrhea and may be associated with serious neurological adverse reactions.

IV. Treatment

Since the majority of episodes of travelers' diarrhea are self-limited, adequate oral rehydration, resting of the bowel with alterations in diet, and a brief course of antibiotics will usually provide satisfactory treatment. Patients with high fever and chills, bloody diarrhea, or persistent symptoms greater than 48 hours should seek medical care. Infants are easily dehydrated and require more vigorous attention to maintain hydration. Antimotility medications, used for the temporary relief of abdominal cramps, should be used with caution. These drugs may prolong travelers' diarrhea and may intensify the illness; especially if the infective agent is invasive, such as Shigella, Salmonella, Campylobacter, Vibrio parahaemolyticus, and Entamoeba histolytica.

An oral rehydration and dietary regimen recommended by the Centers for Disease Control for the treatment of travelers' diarrhea is:

1. Oral hydration solution:

   Water, carbonated or boiled.............. 8 ounces
   Fruit juice........................................ 8 ounces
   Honey or corn syrup.......................... 1/2 tsp
   Table salt...................................... 1 pinch
   Baking soda................................. 1/4 tsp

2. Supplement with additional carbonated drinks, or boiled water or tea as required. Unweaned infants should continue breast-feeding.

3. Avoid solid foods until the diarrhea abates and then begin with soft bland foods, such as rice, applesauce, crackers, and toast.
Antimicrobial treatment of travelers diarrhea is effective when instituted at the onset of symptoms and will reduce the severity and duration of signs and symptoms:

1. Older than 12 years of age:
   a. Doxycycline 100 mg twice a day for three days; or,
   b. Trimethoprim-sulfamethoxazole, double strength, one tablet twice a day for three days.

2. Children from 8 weeks old to 12 years old:
   a. Trimethoprim-sulfamethoxazole suspension, 5 cc per 10 kg of weight up to 40 kg (20 cc).

Children less than eight weeks of age or children who are dehydrated, should be medically evaluated as soon as possible. All patients with bloody diarrhea or symptoms persisting greater than 48 hours without improvement should also undergo a medical evaluation. Attempts to continue oral rehydration should not be stopped if the patient develops vomiting.

Personal protection measures

I. Accidents

Accidents are a major cause of morbidity and mortality among tropical travelers. Defensive driving, use of automobile safety belts, and avoidance of night driving and driving under the influence of drugs and alcohol are just as important in the tropics as in developed countries. Riding motorcycles without helmets and proper clothing is not recommended. Since the highway design is poor in many areas, pedestrians should be extra cautious while walking near roads or town streets. Casual trips into the rural areas without proper personal and survival equipment should be avoided.

II. Animals

Rabies is endemic in domestic and wild animals in many areas of the tropics. Brucellosis and anthrax are more common in tropical domestic animals. Travelers exposed to rat infested areas are at risk for contracting plague, hemorrhagic fever with renal syndrome, Lassa fever, and leptospirosis.
III. Arthropods

Arthropod-borne illnesses occur more frequently in the tropics. Travelers should consider the protective use of long sleeved shirts, pants with tucked in leg cuffs, and hats to minimize exposed skin and apply insect repellents to decrease chances for arthropod biting. Mosquito netting can provide protection while sleeping. Screened living quarters provide additional protection. Travelers can adjust their itinerary to avoid the habitats and feeding times of known vectors, such as mosquitoes, sandflies, ticks, mites, fleas, and flies.

Scorpions, spiders, ants, bees, and wasps should be avoided. Certain fleas and flies utilize human subcutaneous tissue for larval development (see Dermatological Complications Chapter).

Some of the more important arthropod-borne problems include:

1. African trypanosomiasis: tse-tse fly found in rural Africa.
2. American trypanosomiasis: reduviid bugs that feed nocturnally in poorly constructed buildings in rural South America.
3. Dengue fever: daytime-feeding mosquito, Aedes species, found about human habitations throughout most of the tropics.
4. Far Eastern and Central European tick-borne encephalitis: forest-dwelling ticks found in central and eastern Europe and the Union of Soviet Socialists Republics. May also be transmitted in unpasteurized milk.
5. Filariasis: variety of urban and rural mosquitoes found in many areas across the tropics.
7. Leishmaniasis: indoor- and outdoor-feeding phlebotomine sandflies found across the tropics from the Americas to the Indian subcontinent.
8. Malaria: nocturnal-feeding mosquito, Anopheles species, found about human habitations in many areas across the tropics.
9. Onchocerciasis: blackflies found in many areas of the Americas, Africa, and the Middle East.
10. Plague: rat fleas found in rodent- or rabbit-infested, upland or mountainous rural areas of the Americas, Africa, and Asia.
11. Yellow fever: Urban yellow fever is transmitted by the daytime-feeding mosquito, Aedes aegypti, found about human habitations in the Americas and Africa. Jungle yellow fever is transmitted by a variety of mosquito vectors in tropical forests and banana plantations.

IV. Chronic disease

Tropical travelers with chronic diseases have a higher risk for developing a decompensation of their condition while traveling. Heat, self-imposed stress, psychosocial stress of travel, jet lag, problems with medication compliance, alterations in diet, high altitude, and lower quality of some foreign medications, if purchased by the traveler, are all possible contributing factors. The traveler may obtain references for medical care from their embassy or consulate.

V. Clothing

The tropical traveler should wear light, tan-colored clothes with a broad-rimmed hat, but be prepared for chilly nights. If the traveler anticipates touring rural areas, then long-sleeved shirts, pants, and boots should be worn. Depending on the season and altitude, hypothermia may occur in the tropics in improperly clothed tourists.

VI. Culture

The traveler should make an attempt prior to travel to gain an understanding of the culture of the countries they plan to visit. An understanding of the proper clothing, climate, cultural attitudes, courtesies, dining habits, sanitation, and hygiene may prevent unnecessary anxiety or embarrassment.

VII. Food and drink

Safe food and drinks include:
1. Coffee and tea boiled for at least five minutes.
2. Bottled soft drinks, carbonated water, beer, and wine.
3. Properly boiled and iodinated water.
4. Pasteurized and refrigerated milk and cheese.
5. Well-cooked eggs, meat, fish, and seafood served and eaten while hot.
6. Nuts, fruits, and vegetables that can be shelled, peeled, or skinned at the time of serving by the traveler.
7. Vegetables that have been boiled or immersed in chlorine solution.

Unsafe food and drinks include:
1. Untreated water, tap water, and ice cubes.
2. Unpasteurized dairy products.
3. Raw or poorly cooked eggs, meat, fish, shellfish, and seafood.
4. Soups, salads, sauces, raw vegetable, cold platters, custards, pastries, and desserts.
5. Dishes prepared and left to stand.
6. Note: many shellfish species, warm-water sea fish, and reef fish unpredictably contain toxins and environmental pollutants that are not inactivated by cooking.

Tropical diseases transmitted by contaminated food and drink are:
1. Abdominal angiostrongyliasis: fecal-oral.
2. Angiostrongyliasis: raw snails, slugs, crabs, prawns, fish; contaminated leafy vegetables.
3. Anisakiasis: raw saltwater fish and squid.
4. Ascariasis: uncooked produce with contaminated soil.
7. Cholera: fecal-oral, contaminated food, water, or seafood.
9. Cysticercosis: fecal-oral, contaminated food or water.
12. Dracunculiasis: contaminated water.
13. Echinococcosis: fecal-oral, contaminated food or water.
15. Enteric bacteria (see Travelers' Diarrhea Section above): fecal-oral, contaminated food, water, seafood, shellfish.
19. Giardiasis: contaminated water or food.
22. Hepatitis A, non-A non-B: fecal-oral, contaminated food, water, or shellfish.
24. Hymenolepiasis: fecal-oral, contaminated food or water.
25. Lassa fever: contaminated food.
26. Leptospirosis: contaminated food or water.
27. Melioidosis: contaminated water.
29. Q-fever: unpasteurized milk.
30. Taenia: raw beef or pork.
32. Toxoplasmosis: raw meat, contaminated food or water.
33. Trichinosis: raw meat.
34. Trichuriasis: fecal-soil-oral.
35. Tuberculosis: unpasteurized dairy products.
36. Tularemia: contaminated rabbit or hare meat, and water.

VIII. Gynecological problems

The traveling woman has a higher risk for vaginitis with the increased heat and humidity of the tropics and/or with the use of prophylactic antibiotics. Irregular menses or dysmenorrhea may occur with the disruptions and stresses of travel.

See also Sexually Transmitted Diseases below in section XIV.

IX. Handicrafts

Handicrafts made from goat hides may be contaminated with anthrax spores.

X. Heat

The tropical traveler needs to be reminded of the importance of increasing their daily intake of fluids to prevent heat injury. A more detailed discussion of heat injury can be found below in the Human Factors in Heat Injury Chapter.
XI. High altitudes

Many popular tourist sites are located 8,000 feet or more above sea level, especially in the Andes and Himalayas. The traveler who rapidly ascends to these locations may develop altitude sickness or an exacerbation of chronic cardiopulmonary diseases before they become acclimatized. Even at 10,000 feet above sea level, healthy, unacclimatized travelers may experience exercise intolerance, dyspnea, and headache, and may later develop nausea and vomiting. Acclimatization may be accelerated by taking acetazolamide 250 mg every 8-12 hours, beginning 24-48 hours before ascent, and continuing during the day of ascent.

Tropical travelers must be prepared for a radical cooling of the climate and an increase in solar ultraviolet radiation as they ascend from the lowlands into the mountains.

XII. Personal security

The traveler, especially a solitary woman, is more vulnerable to violations of their personal security while traveling in unfamiliar areas and should take extra precautions to protect that security.

XIII. Pregnancy

Uncomplicated pregnancy is not a contraindication to travel. However, traveling after 32 weeks gestation may be uncomfortable, and after 36 weeks gestation, air travel is restricted by many airlines as a safety regulation. Pregnancy in a tropical traveler may be complicated by congenital or perinatal infections that occur more frequently in the tropics, such as African and American trypanosomiasis, gonococcal conjunctivitis, hepatitis B, malaria, rubella, syphilis, and toxoplasmosis.

Live-attenuated vaccines, such as measles, mumps, rubella, and yellow fever vaccines, are contraindicated in pregnancy. Yellow fever vaccine may be given if the pregnant traveler is unavoidably exposed to an outbreak of the disease. Live-attenuated trivalent polio vaccine may be given if the traveler is at substantial risk for exposure, as is the case in many tropical countries.
Inactivated vaccines and toxoids may be given with a greater degree of safety than live-attenuated vaccines in pregnancy, but may not be recommended unless there is a substantial risk of exposure. Tetanus toxoid booster is recommended if it has not been administered in the last 10 years.

XIV. Sexually transmitted diseases

Sexually transmitted diseases are more prevalent, with a wider variety of infectious agents and a higher number of antibiotic resistant infections, in many areas of the tropics. The tropics are not spared from the current epidemic of acquired immune deficiency syndrome. The traveler should seriously consider not engaging in casual or promiscuous sexual activities.

XV. Swimming

Swimming in other than chlorinated swimming pools and actively circulated salt water along a beach is not recommended. However, beaches may be contaminated by human sewage and feces and salt water is the home of hazardous biting and stinging fish, jelly fish, sea anemones, and corals. Dracunculiasis, enteric pathogens, leeches, leptospirosis, primary amebic encephalitis, and schistosomiasis are associated with exposure to fresh water standing in pools, canals, lakes, and streams. Many vectors for arthropod-borne diseases are more abundant about bodies of water.

Immunizations and chemoprophylaxis

I. Recommendations

Specific immunization and chemoprophylactic requirements, and / or recommendations, for each country of entry are continually changed and updated by Centers for Disease Control and the World Health Organization. These are readily available in periodic publications and by telephone. State health departments may be of assistance as well. Some confusion exists in immunizations regulations where, for example, an immunization may not be required to travel from country "A" to "C" or from country "A" to "B", but may be required to travel from
country "B" to "C". Therefore, travelers must have their itinerary carefully checked and have appropriate vaccinations administered to avoid unexpected detainment or quarantine at some ports of entry.

Infectious tropical diseases responding to immunization or chemoprophylaxis are:

1. Cholera: The inactivated cholera vaccine is not recommended for travelers unless it is a requirement for entry into a country. The risk of transmission to travelers is low, except for travelers living or working in endemic areas with poor sanitation or in patients with compromised gastric defense mechanisms.

2. Diphtheria: Current diphtheria toxoid immunization status is recommended for tropical travelers as diphtheria remains a serious, prevalent disease across the tropics.

3. Japanese encephalitis: Inactivated Japanese encephalitis vaccine is not recommended for travelers, except for travelers who will be living or visiting endemic or epidemic areas for a prolonged period of time. The disease is most common in rural areas of the Far East, Southeast Asia, and Indian subcontinent, especially where rice and pig farming are practiced, during the wet season or early dry season.

4. Haemophilus influenzae, type B: The Haemophilus b polysaccharide vaccine is recommended in all children from age 18 months to 4 years old prior to tropical travel.

5. Hepatitis A: Immune globulin prophylaxis is recommended for tropical travelers who will be visiting rural areas or areas with poor sanitation and unsafe water supplies.

6. Hepatitis B: Inactivated hepatitis B vaccine is recommended for tropical travelers who are health care workers or who plan extended visits to high risk areas, or short term travelers who anticipate exposure to needles (drug use, tattooing, acupuncture, venipuncture), sexual contact with the local population, and medical or dental care in high risk areas.

7. Influenza: Current influenza immunization status is recommended for high risk patients with chronic diseases, such as diabetes, cardiopulmonary or renal disease.
8. Malaria: Malaria chemoprophylaxis and mosquito personal protection measures are recommended for all tropical travelers to areas where malaria is known to occur. If the traveler develops a flu-like illness, the traveler should seek early diagnosis and treatment of a possible malarial infection, as these protective measures do not completely prevent the disease.

9. Measles: Current live-attenuated measles immunization status or significant evidence for natural immunity (especially prevalent in patients born before 1957) is recommended for tropical travelers as measles remains a serious, prevalent disease across the tropics.

10. Meningococcal meningitis: Inactivated meningococcal vaccine is recommended in travelers who anticipate a prolonged exposure to local populations of high risk areas, or exposure to a case of meningococcal meningitis in an epidemic situation.

11. Mumps: Current live-attenuated mumps immunization status or significant evidence of natural immunity is recommended for tropical travelers, especially children and young adult males, as mumps remains a prevalent problem in the tropics.

12. Pertussis: Current pertussis immunization status is recommended for tropical travelers as pertussis remains a serious, prevalent disease across the tropics, especially in children.

13. Plague: Inactivated plague vaccine is recommended only for travelers that are at high risk for exposure in research and other field activities, and for those who anticipate a prolonged exposure to rodent- and rabbit-infested, plague-enzootic rural areas of the Americas, Africa, and Asia.

14. Pneumococcal infections: Pneumococcal polysaccharide vaccine is recommended only in high risk patients with chronic lung disease, heart disease, renal disease, or diabetes mellitus, or who are asplenic or have splenic dysfunction.

15. Poliomyelitis: Current polio immunization status is recommended for travelers as poliomyelitis remains a serious, prevalent disease across the tropics.

16. Rabies: Pre-exposure inactivated rabies vaccine is only recommended in travelers who are rabies research workers, animal handlers, veterinarians, animal control workers, or wildlife workers, especially those visiting and working in rabies-epizootic areas of the tropics.
17. Rubella: Current rubella immunization status or significant evidence of natural immunity is recommended for travelers, especially in women of child-bearing age, as rubella remains a serious, prevalent problem across the tropics.

18. Smallpox: Smallpox has been declared eradicated world-wide and smallpox vaccination should not be given to travelers.

19. Tetanus: Current tetanus toxoid immunization status is recommended for tropical travelers as tetanus remains a serious, prevalent disease across the tropics.

20. Tuberculosis: BCG vaccine is not recommended for travelers. Since tuberculosis is more prevalent in developing areas of the world, the traveler to these areas, anticipating prolonged exposure to the local population, should have a tuberculin skin test before, and if negative, after the trip.

21. Typhoid fever: Inactivated typhoid vaccine is recommended in travelers who anticipate prolonged exposure to potentially contaminated food and water in developing communities or rural areas of the tropics.

22. Typhus fever: Typhus vaccine is not currently recommended in travelers since no cases of typhus have been reported in American travelers since 1950.

23. Yellow fever: Live-attenuated yellow fever vaccine is required for entry into some countries and is recommended for travelers who anticipate travel in areas with active yellow fever transmission. The vaccine is only given at designated vaccination centers established by each state health department.

Medical problems of air travel

I. Introduction

Tropical travelers primarily use commercial air transportation as their means of travel. Most experienced travelers have a casual attitude towards flying, but are unaware that certain medical problems may develop during or may be exacerbated by air travel. It is important to evaluate patients in the pre-trip visit for medical risk factors that are affected by air travel and counsel the patient on the various medical problems that may develop as a consequence of flying.
II. Hypoxia

There are physical limitations of the pressurization systems on aircraft that prevent the cabin oxygen and barometric pressure from being maintained at sea level values in the cabin during all phases of flight. The average business or commercial jet aircraft can maintain sea level conditions to a flight altitude between 16,500 feet to 23,000 feet above sea level, depending on the type of aircraft. However, most of these passenger jet aircraft fly economically at flight altitudes of 32,000 feet to 42,000 feet above sea level, which results in the passengers experiencing an interior cabin altitude of 6,000 to 10,000 feet above sea level. Commercial passenger air carriers, regulated by the Federal Aviation Administration, usually restrict their operational cabin altitudes to 6,000 feet above sea level. But business jets and foreign commercial passenger air carriers may not follow this recommendation and fly with cabin altitudes greater than 6,000 feet above sea level. In a healthy patient at a cabin altitude of 8,000 feet above sea level, the blood oxygen saturation is around 90%, with an arterial partial pressure of oxygen at about 55 mm Hg. The mild hypoxia experienced at these cabin altitudes is tolerated well by the average passenger, but a few unwary patients with certain medical conditions may develop symptomatic hypoxia. Patients who live at altitudes above 5,000 feet above sea level are acclimatized to the effects of mild hypoxia, and can comfortably tolerate higher cabin altitudes than patients living at sea level.

The symptoms of hypoxia include decreased night and day visual acuity, fatigue, headache, unreliable psychomotor function, euphoria, paresthesias, hyperventilation, deterioration of neuromuscular control, syncope, convulsions, myocardial ischemia or infarction, and coma. The hypoxic air traveler may appear to be intoxicated with alcohol, confusing flight attendants about the cause of the traveler's aberrant behavior.

Anemia and hypoxia:

Sickle cell trait: If the patient with sickle cell trait does not have other hemoglobinopathies, then the patient can be reassured that supplemental oxygen will not be required at the usual operational cabin altitudes.
Sickle cell disease: It is not difficult to induce a sickling crisis at cabin altitudes above the altitude of the home of the patient with sickle cell anemia. Therefore, these patients should receive supplemental oxygen on any flight to maintain their inspired FiO2 equal to that of their point of origin.

Anemia: The patient with anemia at sea level actually has a physiologic altitude above sea level and will experience symptomatic hypoxia at cabin altitudes lower than the healthy patient. A patient with a hemoglobinopathy, such as thalassemia, has a reduced oxygen carrying capacity, and often has an accompanying anemia. These patients will also become hypoxic at lower cabin altitudes. Generally, patients without cardiopulmonary disease who have a chronic anemia with a hemoglobin greater than 8.5 gm/100 cc, will tolerate the hypoxia of flight in a commercial passenger air carrier. Patients with a chronic anemia having a hemoglobin less than 8.5 gm/100 cc, may require a preflight blood transfusion and/or inflight supplemental oxygen to prevent symptomatic hypoxia. The table below estimates the approximate preflight physiological altitudes of a nonsmoking, 70 Kg male, with varying degrees of acute and chronic anemia, not complicated by cardiopulmonary disease.

<table>
<thead>
<tr>
<th>Hgb gm/100 cc</th>
<th>Acute Anemia</th>
<th>Chronic Anemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>14</td>
<td>1200</td>
<td>800</td>
</tr>
<tr>
<td>13</td>
<td>2400</td>
<td>1500</td>
</tr>
<tr>
<td>12</td>
<td>3500</td>
<td>2300</td>
</tr>
<tr>
<td>11</td>
<td>4800</td>
<td>3200</td>
</tr>
<tr>
<td>10</td>
<td>*6000</td>
<td>4000</td>
</tr>
<tr>
<td>9</td>
<td>7200</td>
<td>4800</td>
</tr>
<tr>
<td>8</td>
<td>8400</td>
<td>5600</td>
</tr>
<tr>
<td>7</td>
<td>9500</td>
<td>*6300</td>
</tr>
<tr>
<td>6</td>
<td>11000</td>
<td>7200</td>
</tr>
</tbody>
</table>

* The preflight, anemic patient at sea level, with no other cardiopulmonary problems, may require supplemental oxygen for the prevention of mild hypoxia above this physiological altitude.

(Adapted from McNeil, EL. Airborne Care of the Ill and Injured.)
Heart disease and hypoxia:

Coronary artery disease: Patients with coronary artery disease are at risk for developing myocardial ischemia or infarction when the cabin altitude, or their combined physiological altitude and cabin altitude, exceeds 6,000 feet above sea level. Patients with coronary artery disease should not attempt air travel without inflight medical attention and monitoring unless they have been free of unstable angina, arrhythmias, and congestive heart failure for a period of six to eight weeks following a myocardial infarction. The patient with coronary artery disease may also present for care for an acute coronary event within several days after an uneventful air trip, sometimes recalling mild prodromal symptoms in flight, such as indigestion. The patient with coronary artery disease may require supplemental oxygen during air travel.

Congestive heart failure: Even though symptoms are stabilized by medical therapy, a patient with congestive heart failure may become symptomatic with the hypoxia occurring in flight. Generally, patients who can walk 100 yards and climb twelve stairs without symptoms can travel without supplemental oxygen at cabin altitudes less than 6,000 feet above sea level.

Pulmonary disease and hypoxia:

Premature infants or term infants less than seven days old have underdeveloped respiratory systems and are at risk for developing symptomatic hypoxia in flight. Infants with pulmonary disease should not be transported by air without proper medical attention and monitoring.

The patient with pulmonary disease may decompensate in the flight environment because of hypoxia and thickening of pulmonary secretions while the patient is inhaling dry cabin air. The process of air compression to pressurize the cabin markedly warms and dehydrates the cabin air. Patients with pulmonary disease may require supplemental, humidified oxygen during air travel. The patient with chronic lung disease has a limited ability to increase ventilation efforts in response to the hypoxia that develops inflight. Mild exercise, such as walking the aisles, and obstructive sleep apnea inflight increase their oxygen requirements further. In general, patients with a vital capacity of less than 50% predicted are at the highest risk, and may require medical attention and monitoring during air travel. If the patient's preflight inspired FiO2 requirements to maintain a partial pressure of arterial oxygen of 100 mm Hg., are known, the table below provides an estimate of the FiO2 requirements at various
altitudes in order to maintain the patient's partial pressure of arterial oxygen at 100 mm Hg.

FiO2 Requirements at various altitudes

<table>
<thead>
<tr>
<th>CABIN ALTITUDE IN FEET</th>
<th>0</th>
<th>2000</th>
<th>4000</th>
<th>6000</th>
<th>8000</th>
<th>10000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Required</td>
<td>21</td>
<td>23</td>
<td>25</td>
<td>27</td>
<td>29</td>
<td>32</td>
</tr>
<tr>
<td>FiO2 at Sea level</td>
<td>30</td>
<td>33</td>
<td>*35</td>
<td>38</td>
<td>42</td>
<td>45</td>
</tr>
<tr>
<td>4000</td>
<td>40</td>
<td>44</td>
<td>47</td>
<td>51</td>
<td>55</td>
<td>60</td>
</tr>
<tr>
<td>6000</td>
<td>50</td>
<td>54</td>
<td>59</td>
<td>64</td>
<td>69</td>
<td>75</td>
</tr>
<tr>
<td>8000</td>
<td>60</td>
<td>65</td>
<td>70</td>
<td>76</td>
<td>83</td>
<td>90</td>
</tr>
<tr>
<td>10000</td>
<td>70</td>
<td>76</td>
<td>82</td>
<td>90</td>
<td>**</td>
<td>**</td>
</tr>
<tr>
<td>12000</td>
<td>80</td>
<td>87</td>
<td>94</td>
<td>**</td>
<td>**</td>
<td>**</td>
</tr>
<tr>
<td>14000</td>
<td>90</td>
<td>**</td>
<td>**</td>
<td>**</td>
<td>**</td>
<td>**</td>
</tr>
</tbody>
</table>

* >33 Patient should not be unattended, more than a nasal cannula will be required.
** Patient will require mechanical ventilation.
(Adapted from McNeil, EL. Airborne Care of the Ill and Injured.)

Another approach to predicting hypoxia inflight was described in a study of thirteen patients with chronic obstructive pulmonary disease by Schwartz, et al. The patients selected for the study had a history of chronic obstructive pulmonary disease with a mean forced expiratory volume in 1 second (FEV1) of 986 (+/- 438) mL, mean FEV1/forced vital capacity of 0.37 (+/-0.10), and mean total lung capacity of 8442 (+/-1115) mL; with no evidence of restrictive pulmonary disease, ischemic heart disease, or cerebrovascular disease; and with resting, room-air arterial partial pressures of oxygen of 55 mm Hg or more. They found that breathing a hypoxic air mixture test (HAMT) of 17.2% oxygen at sea level confidently predicted the patients level of hypoxemia at cabin altitudes of 5400 feet above sea level (1650 meters). They also demonstrated a significant drop in the arterial partial pressure of oxygen during the HAMT when the patient was exercised by walking. The results of this study are tabulated below:

<table>
<thead>
<tr>
<th>13 Patients with COPD</th>
<th>PO2 mm Hg</th>
<th>PCO2 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sea Level</td>
<td>68.0 (+/- 7.3)</td>
<td>40.0 (+/- 5.8)</td>
</tr>
<tr>
<td>HGMT (17.2% O2)</td>
<td>52.5 (+/- 5.6)</td>
<td>37.3 (+/- 8.3)</td>
</tr>
<tr>
<td>HGMT + walking</td>
<td>44.1 (+/- 7.9)</td>
<td>37.3 (+/- 5.8)</td>
</tr>
<tr>
<td>cabin alt. 5,400 feet</td>
<td>51.0 (+/- 9.1)</td>
<td>37.1 (+/- 6.4)</td>
</tr>
<tr>
<td>cabin alt. 7,400 feet</td>
<td>44.7 (+/- 8.7)</td>
<td>36.5 (+/- 5.8)</td>
</tr>
</tbody>
</table>
Seizure disorders and hypoxia:

Hypoxia may precipitate seizures in patients with epilepsy. These patients may require supplemental inflight oxygen and/or sedation.

Ophthalmologic disease and hypoxia:

The diseased retina is sensitive to hypoxia and may require supplemental oxygenation inflight to prevent further retinal compromise.

Uncontrolled intraocular hypertension or glaucoma increase the retina's sensitivity to hypoxia. The hypoxic eye may develop an increase in intraocular tension, creating the potential for pressure-induced retinal compromise.

Self-imposed hypoxia:

Smoking, as an ubiquitous environmental pollutant, should be considered when assessing a patient for the risk of developing hypoxia during air travel. Patients who smoke may have carboxyhemoglobin levels from 2 to 15 percent. For example, a patient who smokes one pack a day of cigarettes may have a carboxyhemoglobin level up to 10 percent. Therefore, while standing at sea level, this patient already has a physiological altitude of about 5,000 feet above sea level. Having concomitant pulmonary dysfunction will push this physiologic altitude even higher. At a cabin altitude of 6,000 feet above sea level, this smoking passenger may easily become hypoxic.

III. Changes in barometric pressure

The cabin barometric pressure during air travel may rapidly change, inducing pressure related problems as gases expand and contract. The volume of gas in a balloon at sea level will expand 1.5 times when taken to a cabin altitude of 10,000 feet above sea level. The pressure changes at a rate equivalent to 200 feet of altitude per minute on ascent and 300 feet of altitude per minute on descent in a typical commercial passenger jet airliner. These changes are accelerated during travel through vertical wind shears or during emergency, explosive decompression of the cabin atmosphere. Changes of barometric pressure encountered in commercial flights are usually of no medical significance, unless the patient has trapped air in a body cavity which cannot ventilate or accommodate the expansion of gases as the altitude increases or
contraction of gases as the altitude decreases. The pilot may have to modify the rate of ascent or descent if a passenger develops a pressure-related medical problem.

**Barotitis media (ear block, barotrauma):**

Barotitis media, the most common medical problem induced by pressure changes, occurs most often on descent as the air in the middle ear contracts and the eustachian tube functionally closes from the negative pressure. The patient develops muffled hearing and middle ear pain, which is sometimes incapacitating, as the tympanic membrane is retracted into the middle ear. The tympanic membrane and middle ear may be traumatized giving the membrane the appearance of an acute infection and resulting in a serous or bloody middle ear effusion. Acute bacterial otitis media may be a delayed, secondary complication. Patients with a concurrent upper respiratory infection are at high risk for developing barotitis media.

Normally as the air in the middle ear expands on ascent, the air escapes through the eustachian tube. Patients with eustachian tube dysfunction, who do not have their middle ears ventilated by patent tympanostomy tubes, may develop a significant traumatic perforation of the tympanic membrane as trapped gases escape. With partial eustachian tube function, perforation may not occur, but the patient may develop barotitis media.

The air traveler should be instructed in the modified Valsalva middle ear ventilation maneuver to relieve barometric problems. Many patients find chewing gum or food helpful during changes in altitude. Infants can be breast or bottle fed. As a prophylactic measure, patients with upper respiratory tract infections, history of eustachian tube dysfunction, or history of barotitis should use a potent nasal spray decongestant one half hour before cabin repressurization and descent begin.

The modified Valsalva maneuver used to relieve barometric problems of the ear on descent is accomplished as follows:

1. Laterally flex the neck away from the affected ear.
2. Pinch the nostrils closed and close the mouth.
3. Accomplish a valsalva maneuver, but allow compressed air to fill the nasopharynx in an attempt to force air into the middle ear, relieving the negative pressure.
If the modified Valsalva maneuver fails, flight attendants are trained in the use of the Plotzer bag, which is part of the basic first aid equipment on many commercial air carriers. The Plotzer bag is connected to the patient's nostril with a rubber tube that has a contoured nasal fitting. As the patient swallows, the flight attendant gently blows air into the nasopharynx in an attempt to introduce air into the middle ear, relieving the negative pressure.

Barosinusitis (sinus block):

Air may also be trapped in the sinuses, especially if the patient has sinusitis, upper respiratory tract infection, chronic vasomotor or allergic rhinitis, or history of recent sinus surgery. Nasal spray decongestants are usually effective in relieving this painful condition.

Aerodontalgia:

Air may be trapped in dental abscesses or improperly prepared fillings resulting in dental or maxillary pain, usually during the ascent phase of flight.

Intestinal gases:

The expansion of intestinal gases may result in indigestion, airsickness, and decrease in respiratory expansion as the diaphragm is displaced upward. Patients with colostomies should wear a larger size of colostomy bag for their journey to accommodate gas expansion. Patients consuming gas-producing foods, with a history of gastrointestinal problems, and with a third trimester pregnancy are at higher risk for developing discomfort from intestinal gas expansion. Rupture of the gastrointestinal tract has occurred in patients with diverticuli, ulcers, or weak surgical anastomoses. Gas trapped in the splenic flexure may cause severe pain and vasovagal syncope, mimicking a myocardial infarction.

Spontaneous pneumothorax:

Patients with pulmonary blebs or emphysema may develop an acute spontaneous pneumothorax with changes in cabin pressure.

Recent surgery or trauma:

Patients with a recent history of surgery or trauma should convalesce at least two weeks before air travel. Those at highest risk are patients with a history of diagnostic
procedures using air or that allow air to leak into a cavity (arthroscopic exam, laparoscopic exam, pneumoencephalogram, thoracentesis, or wound irrigation), surgery introducing air into a body cavity (especially abdominal, neurosurgical, thoracic, or ophthalmologic procedures), trauma introducing air into the body (penetrating eye or thorax injuries, open fractures), or gas producing infections. Orthopedic air splints may expand in flight, compromising the splinted limb.

Recent scuba diving or hyperbaric medical therapy:

Recreational scuba diving or occupational deep-sea diving may allow nitrogen gas to accumulate in the body tissues, especially after prolonged dives. Divers ascending in an aircraft within twenty-four hours of a dive may develop decompression sickness with cardiopulmonary or neurological complications as the decompressing nitrogen gas forms bubbles. Patients who have received hyperbaric medical treatments should be considered as having accomplished a dive.

IV. Neuropsychiatric problems

Hyperventilation:

Hyperventilation is a common anxiety-induced problem of air travel. The symptoms of hyperventilation, which include dizziness, blurred vision, dyspnea, palpitations, chest or epigastric discomfort, muscle spasm, peripheral numbness, and fainting, may mimic pulmonary, cardiac, or neurologic diseases. Hyperventilation may be a symptom of hypoglycemia, flight-induced pulmonary edema, and salicylate overdose. However, hyperventilation may also be a sign of hypoxia that will be exacerbated by the standard treatment for hyperventilation, that is breathing into a paper bag.

Fear of flight / flight phobia:

Some patients develop anxiety during flight or may have a fear of flying. Although the primary care practitioner may be able to reassure the traveling patient with statistics on the safety of air travel relative to other forms of travel, the modern news media system has engrained many images of tragic aviation accidents in the patient's mind. Drugs used in the prophylaxis of airsickness usually provide enough short-term sedation to significantly reduce the anxiety of flight; however, a few patients may require short-acting sedatives.
Major psychiatric diseases:

In general, patients with major psychiatric illnesses, such as schizophrenia and mania, tolerate the flight environment poorly and should not utilize air travel. Psychotic patients have become suddenly violent, creating a hazard to the safety of fellow passengers and the flight.

Patients with chronic neuroses, especially with phobic components, may decompensate during a flight, especially if stressful weather is encountered. The neurotic patient may require preflight medication.

The patient with a major affective disorder usually tolerates flying well, but their disorder is often exacerbated by the psychosocial stresses of the total travel itinerary.

Seizure disorders:

As mentioned above, the mild hypoxia of flight may induce seizures. In addition, the obnoxious odors and unusual sounds and lighting conditions of flight may also induce seizures in certain susceptible patients.

Motion sickness (airsickness):

The primary cause of motion sickness is a multifactorial assault of the senses that occurs with travel. The vestibular system senses new, unusual motions that can accelerate or decelerate in any direction during flight. The visual system is disoriented by the lack of normal references and marked alterations in light intensity. The flicker of lights through rotating propeller blades or helicopter rotor blades, or from rotating beacons may induce nausea and vertigo. The olfactory system is introduced to a multitude of new odors, some obnoxious. Gastric distention and gastric sensing of the complicated vibrations and motions of flight may provoke retrograde peristalsis and increased salivation. A warm cabin environment or lack of airflow may increase the feelings of motion sickness. Patients with a prior history of motion sickness may have a fear of recurrence and insignificant stimuli may provoke an uncontrolled motion sickness response.
Common medications used in the prophylaxis of motion sickness are:

**Dimenhydrinate**

Adult: 50-100 mg every four hours beginning one hour before flight.

Child (age 6-12): 30-60 mg every four hours.

Child (age 2-6): 15-30 mg every four hours.

**Meclizine hydrochloride**

Adult: 25-50 mg every 24 hours beginning one hour before flight.

**Scopolamine**

Adult: 0.5 mg applied once in a transdermal patch behind the ear beginning four hours before flight.

Disruption of the circadian rhythm (jet lag):

A disruption of the air traveler's circadian rhythm occurs when the air traveler is on a flight plan that passes through more than two or three time zones. It is not uncommon to pass through five or ten time zones on a typical international flight, placing the traveler at significant risk for a clinical syndrome commonly called "jet lag". The activities of the normal day at the destination are not synchronized with the traveler's rhythm of life at the place of origin, resulting in a biochemical and psychological imbalance. Traveler's with jet lag find that they are unusually tired, but unable to sleep. They complain of difficulty in thinking quickly and clearly, and that they have a reduced exercise and work tolerance. They note a disruption in digestion and kidney function. Constipation is a common complaint.

The effects of jet lag are more intense and prolonged if the patient travels from west to east, and as the traveler's age advances. If the patient travels from west to east, the rhythm of life at the arrival local time is markedly different from the traveler's circadian rhythm set to their departure local time. This results in a radical change in eating, working, daylight exposure, and sleeping times. If the traveler goes from east to west, the traveler will be following the sun on the trip and will arrive at an arrival local time.
not much different from the departure local time at the beginning of the trip; ameliorating some of the effects of jet lag since the patient does not have to cope with a dramatic change in the rhythm of life at the arrival location. Therefore, the traveler going from east to west can pass through more time zones with less symptoms than the traveler going from west to east. In general, it takes one day per time zone crossed to recover fully from the effects of jet lag. Travelers going from west to east, crossing ten time zones in one day, may find their vacation spoiled by the lingering effects of jet lag.

A time zone map has been placed at the beginning of this chapter to assist in estimating the risk for developing jet lag. The International Civil Aviation Organization has developed a formula to estimate the rest period required after a trip has disrupted the circadian rhythm to allow return of useful cerebral function for safe driving, decision making, meetings, etc.:

\[
\text{Rest period} = \text{travel time} + \text{departure zones} + \text{departure coefficient} + \text{arrival coefficient}
\]

The coefficients for local departing and arrival times are:

<table>
<thead>
<tr>
<th>Local Time</th>
<th>Departure Coefficient</th>
<th>Arrival Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 AM to NOON</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>NOON to 6 PM</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>6 PM to 10 PM</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>10 PM to 1 AM</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>1 AM to 8 AM</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

NOTE The higher values of the departure coefficients compensate for lost sleep, while the higher values of the arrival coefficients compensate for the disruption of early morning travel.

The astute traveler may ameliorate some of the effects of jet lag by modifying their wake/sleep/eat cycle to match the cycle of their destination, beginning a few days prior to travel; but often this is impractical with a busy schedule and job responsibilities. Another method, used primarily in west to east travel, involves the induction of a sleep period inflight with sedative hypnotic medications, if the sleep period can be synchronized with the sleep cycle of the destination.

The traveler with insulin-dependent diabetes should monitor their approximate blood sugar value by finger-stick testing every four hours while awake and adjust their insulin
requirements as needed while traveling and under the disrupting effects of jet lag.

V. Miscellaneous problems of air travel

Pregnancy:

The pregnant traveler can accomplish air travel without any harmful effects to the fetus. But the pregnant traveler in her third trimester may experience the discomforts of gastric distention and dependent edema from prolonged sitting during the flight. Most commercial airlines restrict travel after 36 weeks gestation, primarily to avoid a precipitous inflight delivery.

Wired-jaw:

The patient with a wired jaw should not travel to avoid the chance for motion sickness accompanied by vomiting or aspiration.

Diabetes:

Travelers with insulin-dependent diabetes will have to increase the frequency of their self-monitoring of blood glucose during travel and make appropriate changes in their insulin dosages and diet as required, especially if there has been a disruption of their circadian rhythm.

Dependent edema and thrombophlebitis:

Travelers with dependent edema, or with history of thrombophlebitis or venous insufficiency, may have an exacerbation of their condition during a prolonged flight on a crowded airline. These patients should be reminded of the importance of wearing antilymphedema support hose and walking the aisles of the aircraft periodically.

Dry cabin air:

Patients with sicca syndrome, Bell's palsy, eczema, or pulmonary disease, may find that the extremely dry air of airline cabins will exacerbate their symptoms, requiring increased use of medications and measures to preserve hydration.
Medical problems of sports diving

I. Introduction

Sports diving is an increasingly popular recreational activity among tropical travelers. Scuba diving and snorkeling activities are available in the vicinity of many resorts. The chance to see the pristine beauty of a coral reef is difficult for many travelers to miss. Primary care practitioners should counsel their patients during the pre-trip visit on the medical problems of sports diving if the traveler anticipates participating in these activities.

II. Problems of inspired air

The scuba diver is subjected to an artificial system of inspired gases. Failure of the breathing system; improper preparation of the breathing tank atmosphere, and the effects of gas compression during a dive may result in the following problems:

Nitrogen narcosis: Excess inspired nitrogen from air tanks and compression of the inert gas nitrogen during a dive increase the arterial nitrogen tension that may then be complicated by nitrogen narcosis. Clinically, nitrogen narcosis resembles alcohol intoxication or nitrous anesthesia and may be life-threatening at depth. Nitrogen narcosis is more likely to occur at depths greater than seventy feet.

Oxygen intoxication: With the compression of gases during a dive, also dependent on the inspired oxygen concentration from the breathing tanks, the partial pressure of oxygen may attain toxic levels, resulting in pulmonary or central nervous system impairment. Pulmonary edema with atelectasis and convulsions are common complications. Incoordination, nausea, and muscle fasciculations are possible.

Hypoxia: The inspired gas may be deficient in oxygen.

Carbon dioxide intoxication.

Carbon monoxide poisoning.

Exhaustion of the air supply while still submerged.
III. Compression and recompression problems

During the dive, inert gases, the most important being nitrogen, are compressed and saturate the body tissues and blood stream. During ascent, these gases are decompressed and evolve as bubbles, resulting in decompression sickness, which may clinically develop into multiple presentations:

Skin bends: may result in pitting edema and pruritus.

The "bends": mild to severe myalgias or arthralgias.

The "chokes": life-threatening, multiple pulmonary gas emboli, manifested by dyspnea, dry cough, and chest pain.

Central nervous system decompression sickness: gas embolization of the paraspinal venous plexus and brain may result in ascending paralysis, headache, blurred vision, scotomas, diplopia, cranial nerve paralysis, psychiatric disorders, and other multiple, asymmetric focal motor or sensory neurological impairments. The neurologic changes may be irreversible.

Shock syndrome: secondary to neurologic involvement and circulatory damage.

Aseptic bone necrosis: delayed complication of gas embolism in the bone.

The dive may be complicated by barotitis media and barosinusitis.

The "squeeze" occurs during the compression of diving that increases the intravascular pressure. This may result in spontaneous hemorrhage, such as subarachnoid hemorrhage.

IV. Other environmental hazards while diving

The marine environment expands the number of potential tropical environmental hazards for the traveler.

Spinal or skull fracture with resultant central nervous system damage following a diving accident.

Penetrating injuries resulting in subcutaneous foreign bodies, such as fish spines and coral, and wound
infections; some caused by unusual bacteria found in the marine environment, such as *Vibrio vulnificus* and *Vibrio alginolyticus*.

Enteric bacterial infections caused by swallowing marine water or ingestion of improperly cooked seafood.

- **Otitis externa** (swimmer's ear).
- **Conjunctivitis** or **panophthalmitis**.
- **Endometritis**: rare complication.

Exposure to industrial or radioactive wastes dumped in the marine environment.

Exposure to or ingestion of toxic or harmful plants and animals: sharks, killer whales, manta rays, barracuda, needle fish, giant groupers, stingrays, moray eels, blue-ringed octopus, venomous sea snakes, scorpion fish, zebra fish, stonefish, weever fish, catfish, electric fish and eels, jelly-fish, Portuguese man-of-war, sea wasps, sea anemones, sea urchins, sea nettles, other echinoderms, coral, and marine algae.

Accidental drowning or near-drowning.

V. Psychiatric problems

Hyperventilation, decompensation of phobic neuroses, and panic are dangerous and life-threatening if they occur while diving. Psychiatric symptoms may mask a serious, underlying decompression illness, delaying treatment.
Summary checklist for the traveler and medical staff

I. Pre-trip

Dispel myths and reassure the patient about the enjoyment and safety of tropical travel.

Provide the patient with a copy or summary of important medical records and laboratory findings if the patient has a history of significant medical problems. Provide the patient with important addresses and telephone numbers of treating physicians in case a treating foreign physician might require consultation on the patient's past medical history or need to forward copies of medical treatment records.

Ensure that the patient has been briefed on the personal measures that will reduce their chance for contracting an infectious tropical disease. See Personal Protection Measures Section above.

Counsel the patient on the essentials of preventing heat injury; see Human Factors in Heat Injury and Tropical Survival Chapter below.

Determine if the patient will be traveling to isolated, rural areas; and if so, counsel them on the essentials of surviving in the tropical environment in an emergency situation. See Human Factors in Heat Injury and Tropical Survival Chapter below.

Remind the patient that the use of seat-belts while riding in a vehicle in the tropics is very important, as traffic accidents are a common cause of death and injury among tropical travelers.

Ensure that the patient is adequately immunized; see Immunizations Section above.

Ensure that the patient has received written instructions on the use and side-effects of chemoprophylactic agents that may be recommended for the area of travel. Ensure that the local stateside pharmacies are able to support the order for the prescribed chemoprophylactic agents. See Chemoprophylaxis Section above.
Evaluate the patient for potential medical problems that may develop during or as the result of air travel or recreational diving; see Medical Problems of Air Travel Section and Medical Problems of Sports Diving Sections above.

Ensure that travelers are knowledgeable about the warning signs and symptoms of decompensation of their own chronic medical problems, as travel may precipitate a change in the stability of their health.

Provide the patient with a list of recommended supplies:

1. Extra pair of prescription glasses or contacts, and a pair of sunglasses; plus repair parts and a copy of the traveler's lens prescription.
2. Adequate supply of the patient's medications with a set of emergency refill prescriptions, which the patient should store in a different location than the medications. Patients with insulin-dependent diabetes mellitus should carry hard candy and adequate supplies of syringes, needles, and alcohol preps, and be encouraged to divide these supplies up into smaller units for transportation in the luggage. These patients should also be given prescriptions to purchase fresh, small supplies of insulin for use along the way on their trip, rather than one large supply of insulin that may lose its potency in the tropical climate.
3. Anaphylactic reaction kit if the patient has a significant history of severe allergic reactions.
4. Insect repellent.
5. Mosquito netting.
7. Thermometer.
8. Medical alert bracelet.
10. Aspirin or acetaminophen tablets.
11. Zinc oxide for skin irritation and sun protection.
12. Motion sickness tablets.
13. Iodine water purification tablets and instructions on their use.
15. Antihistamine / decongestant combination.
16. Throat lozengers.
17. Mild laxative.
18. Chewing gum.
19. Soap, handi-wipe pads, and folded toilet paper.
II. Trip

Practice personal protection measures:
1. Chemoprophylaxis: see Chemoprophylaxis Section above.
2. Food and water precautions: see Traveler's Diarrhea Section above.
3. Protection from the sun and heat injury.
4. Use of automobile safety belts; and avoid travel on motorcycles, especially if the traveler is inexperienced in their safe use and is unable to wear protective clothing and safety helmet.
5. Protection from biting arthropods.
6. Avoid animals, hides, wool products, and fresh water, especially if it is standing in pools or canals.
7. Avoid casual sexual intercourse, especially with native inhabitants / prostitutes in the area of travel.

III. Post-trip

The traveler should visit their primary care practitioner on return, especially if they were ill during the trip or within six months after completion of travel.

Primary care practitioners should brief themselves on the historical risk of a traveling patient acquiring a tropical infectious disease and receive a disease alert briefing from the Centers for Disease Control for the area of travel. See also the Travel History Chapter.

Consider the following routine, baseline laboratory tests:
1. Complete blood count and differential count, noting eosinophil count.
2. Thick and thin preparation of fresh blood for parasite examination.
4. Stool for occult heme.
5. Laboratory screening for asymptomatic sexually transmitted diseases.***
CHAPTER III
Human factors in heat injury, and in desert and jungle survival

I. Introduction

Heat injury
I. Introduction
II. Risk factors associated with heat injury
III. Classification of heat injuries
IV. Cooling the heat stroke victim
V. Essentials of heat injury prevention

Essentials of desert survival
I. Introduction
II. Water
III. Temperatures
IV. The sun
V. Sand and wind
VI. Thunderstorms
VII. Food
VIII. Animals
IX. Desert illnesses
X. Additional comments

Essentials of jungle survival
I. Introduction
II. Water
III. Climate
IV. Food
V. Animals
VI. Jungle illnesses
VII. Additional comments
Human factors in heat injury, and in desert and jungle survival

I. Introduction

The traveler to the tropics has an increasing number of opportunities to visit the more remote areas of the tropics. A forced landing in a bush plane or broken-down Land Rover may abruptly place the traveler in an isolated situation, perhaps even injured. The traveler should be prepared to deal with these potentially life-threatening emergencies. The primary health care staff is in a position to provide valuable counseling during the pre-trip visit on heat injury and on the human requirements for survival in the tropical desert, semi-arid lands, and jungles.

Heat injury

I. Introduction

Historically heat injury was not recognized as a major health problem in the tropics, primarily due to the lack of proper diagnosis and under-reporting of cases. But standardization and improvement of the medical criteria defining heat injury and the inclusion of heat casualties in many disease reporting systems has demonstrated the significant health risk imposed by the heat of the tropical climate, especially for the unwary and unprepared traveler.

It is a myth that the indigenous inhabitants of the tropics are less susceptible to heat injury since it is now known that human physiology has only limited adaptations which allow acclimatization to heat stress. Hundreds of heat stroke deaths occur annually in pilgrimages to Mecca. More soldiers died from heat injuries, numbering in the thousands, than from battle wounds in the Six Day War of 1967 and Israeli-Arab War of 1973 in the Middle East, despite the soldiers' previous heat acclimatization. However, the indigenous tropical inhabitant may be more aware of the factors leading to heat injury than the traveler from temperate climates, and take unconscious or deliberate steps towards heat injury prevention.

In contrast, the traveler from a temperate climate may be relying on inaccurate instincts, such as thirst, to estimate his response to heat and may be motivated by previously learned, contraindicated anecdotes to avoid adequate fluid intake or ingest excess salt. Travelers often underestimate
their ability to avoid heat injury and are usually poorly informed about the human factors in heat injury, and therefore, are at high risk for developing a heat injury in their travels to the tropics.

II. Risk factors associated with heat injury

The traveler should be aware of the risk factors that increase their risk for developing a heat injury:
1. Improper hydration: the most important factor associated with heat injury.
2. Ingestion of alcohol beverages.
3. Poor physical conditioning.
4. Obesity.
5. Age: the elderly and newborn are at the greatest risk.
6. History of a recent febrile illness or presence of chronic diseases, especially neurologic, cardiopulmonary, endocrine, and dermatologic diseases.
7. History of recent marked psychosocial stress associated with inattention and carelessness, or the presence of a disabling mental illness.
8. History of a previous heat injury.
9. Inadequate heat acclimatization: Humans can make some psychological and physiological adaptations to heat stress when exposed over four to seven days. Unfortunately, the protection provided by this acclimatization is limited when environmental temperatures are high or travelers are exerting themselves.
10. Use of certain drugs: amphetamines (including over-the-counter diet aids), anticholinergics, antipsychotics, atropine, diuretics, glutethimide, lysergic acid diethylamide, monoamine oxidase inhibitors, phenothiazines (may be used by travelers with gastroenteritis), and scopolamine (available in many motion sickness preparations).

III. Classification of heat injuries

Heat cramps are painful paroxysms of muscle spasm occurring after heavy physical exertion and sweating in warm or hot climates (or when excessively clothed in cold climates). The body core temperature is normal or mildly elevated. The syndrome responds promptly to the replacement of lost fluids and electrolytes, preferably by the oral route.
Heat exhaustion usually occurs in the unacclimatized or elderly traveler who is not consuming adequate fluids on exposure to higher environmental temperatures. The syndrome is characterized by the acute onset of weakness, dizziness, disorientation, headache, nausea, vomiting, abdominal cramps, hypotension, and occasionally syncope. A history of exertion is often not obtained. The core body temperature is normal. If the core body temperature is elevated, then the patient may be in the transition phase to heat stroke, or may be a patient with a heat stroke partially treated by cooling measures that may have been utilized on transport to the medical treatment facility. The syndrome responds quickly to cooling and administration of lost fluids and electrolytes, preferably by the oral route. The weakness and headache may persist after treatment for several hours. Heat exhaustion may be a serious problem in travelers with chronic diseases.

Exertional heat stroke is most often seen in patients exerting themselves without increasing hydration in warm, humid climates or in hot climates. It is associated with the acute onset of weakness, dizziness, ataxia, staggering gait, incoherent speech, disorientation, headache, nausea, vomiting, muscle cramps, hyperventilation, hypotension, sweating, and occasionally unconsciousness. The patient may develop any of the complications discussed in the Classic Heat Stroke Section below. In contrast to classic heat stroke, the core body temperature is usually only moderately elevated. Adequate treatment is usually obtained by prompt cooling and administration of glucose-saline solutions. If hypernatremia is present, the initial saline solutions should be hypotonic rather than physiologic.

Classic heat stroke may occur in the traveler to hot climates, especially those with any of the risk factors outlined above. Those at highest risk are travelers who are not treating themselves with adequate fluid intake, regardless of their degree of physical activity or exposure to the sun. The syndrome is characterized by the acute onset of headache, dizziness, delirium, nausea, vomiting, hyperventilation, and the rapid onset of shock syndrome and coma. The core temperature is markedly elevated and sweating may or may not be present. The course of the illness may be complicated by myocardial necrosis and infarction, permanent central nervous system disabilities, renal failure, hemorrhagic phenomena, hepatocellular necrosis, and delayed secondary pneumonia and sepsis. Mortality may be reduced by vigorous cooling of the core body temperature, intravenous hydration, correction of electrolyte imbalances and coagulation defects, protection of the airway, and standard intensive care treatment of complications as they arise.
IV. Cooling the heat stroke victim

Standard cooling therapy accepted in temperate climates stresses the importance of the use of the ice water bath. It would seem that the reduced heat-exchanging blood flow through cooled, vasoconstricted skin capillaries and the heat-generating shivering seen in patients submerged in an ice water bath is not productive in cooling the patient. But the ice bath is effective in quickly lowering the core body temperature, even though it may easily make the unmonitored patient hypothermic. However, ice is not readily available in many areas of the tropics or its supply can be rapidly exhausted in mass heat casualty situations, so primary health care practitioners must consider other methods of cooling the heat injured patient.

Evaporative cooling methods may be employed in areas where ice is not available. An effective method, utilized by military organizations and along pilgrimage routes to Mecca, has been to remove the patient's clothing, spray them with a fine mist of water from a standard garden hose nozzle or hand-pumped sprayer, and blow air over the patient with a generator-powered electric fan. In the absence of electricity, manual fanning can be utilized, but with reduced efficiency of cooling. This method may cool patients as rapidly as an ice water bath without the risk of hypothermia when fanning is discontinued as shivering is achieved. It is most effective in arid or semi-arid conditions where evaporation occurs rapidly; but it can be utilized in humid climates as well.

V. Essentials of heat injury prevention

The traveler to the tropics should be given counseling on the human factors relevant to the prevention of heat injury, especially if the traveler's itinerary is to areas where they may encounter an emergency survival situation.

1. Understand the risk factors listed in the Risk Factors Associated With Heat Injury Section above.

2. Recognize that heat injury may appear suddenly with few prodromal symptoms, disabling the patient with central nervous system symptoms, vomiting, and muscle cramps.

3. Expect environmental temperatures to be higher in automobiles, airplanes, and around man-made structures, than the temperatures reported at weather stations.
4. Wear light, tan-colored clothing and a broad-rimmed hat to decrease solar radiation exposure while outdoors and cover exposed skin with sun screen lotion.

5. Expect decreased exercise tolerance until acclimatization is achieved after being exposed to the higher temperatures for four to seven days.

6. Do not take prescribed or over-the-counter salt tablets, but rather increase the amount of salty foods eaten or lightly salt foods at the dinner table.

7. Understand that thirst is a poor and unreliable indicator of the degree of dehydration, appearing only after the patient has lost more than one to several liters of fluid by sweating and evaporation.

8. Understand that sweating is a poor indicator of the rate of fluid loss, especially in arid climates where patients perceive that they are not sweating at all as the sweat rapidly evaporates from the skin.

9. The most important factor in the prevention of heat injury is to maintain adequate fluid hydration. It is easier to become dehydrated than water intoxicated in the tropical environment. The table below outlines the approximate additional fluid requirements for each increment in the wet-bulb, globe temperature (WBGT), which adjusts for the effects of humidity and direct solar radiation; and suggests work/exercise and rest periods to minimize exertional heat production.

Table III.1

<table>
<thead>
<tr>
<th>WBGT of</th>
<th>Additional fluid requirements in quarts/hour</th>
<th>Work/rest cycle each hour divided into minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>78-82</td>
<td>0.5</td>
<td>50 / 10</td>
</tr>
<tr>
<td>83-85</td>
<td>1.0</td>
<td>45 / 15</td>
</tr>
<tr>
<td>86-88</td>
<td>1.5</td>
<td>30 / 30</td>
</tr>
<tr>
<td>88+</td>
<td>2.0</td>
<td>20 / 40</td>
</tr>
</tbody>
</table>
Essentials of desert survival

I. Introduction

The discipline of tropical medicine conjures up images of steaming jungles. Actually, a great deal of the area known as the tropics is covered by desert habitat. Therefore, patients who plan trips into the desert or semi-arid regions of the tropics should be given counseling on the human factors relevant to surviving in the desert in an emergency situation.

The desert is a fatiguing, dehydrating environment that can easily drain any human, both emotionally and physically. The body may partially acclimatize to this hostile environment during a two week period, but this adaptation process is limited.

At first glance, the desert is an endless sea of sand, rock, and sparse vegetation. But there are micro-habitats within the total landscape which hold the elements for survival. The chances for survival may be enhanced by understanding the impact of special environmental hazards in the desert on the human factors for survival.

II. Water

Water discipline is essential. Having a source of water is the main key to desert survival. In the hot, dry, windy desert, it is easy to dehydrate without noticeable sweating. The body requires a minimum of one to two gallons of water a day to survive in the desert. It is preferred that the water be consumed in small amounts at regular intervals, but in adequate amounts to prevent dehydration. Becoming dehydrated will impair the survivor's ability to forage for water and to think clearly. The sensation of thirst is a very poor indicator of the degree of dehydration and a significant degree of dehydration usually exists before the sensation of thirst is felt. Therefore it is dangerous to rely on the thirst mechanism as a signal to determine when it is time to drink water.

Finding water in the desert may seem to be a formidable task, however the discussion below will help improve the chances for finding water.
Watch out for game and cattle tracks since they may lead to a source of water as mammals require a regular source of water. Birds may fly directly to water sources in the morning and evening hours. There are many stories from people stranded in the Australian deserts who claim their success in surviving was due solely to following a flight of birds to water. Vegetation may show the depth of the water table. Palm trees usually indicate water three feet below the surface, salt grass suggests water six feet below the surface, and cottonwood or willow trees indicate water at a depth of twelve feet. Plants that seem to have adapted to the dry conditions by having small, waxy leaves or water storage mechanisms, should not be used to indicate a source of nearby water. Dew forming on plants may be an important, though minor, source of water. The desert survivor should also be alert for the sudden appearance of rainstorms as a source for gathering water.

The desert survivor should seek out man-made features since these may lead to aqueducts, dwellings, or water storage devices and wells. Domestication of animals is now universally found in deserts. The support of these animals requires stock ponds, water tanks, and water pumping devices. Water may also be found at cliff bases and in moist, sandy washes. There are steep-walled canyons in deserts that may contain pools of water or springs. These are called arroyos and tinajas in the New World and wadis in the Middle East.

The use of solar stills has been popularized, but the amount of effort and body water utilized in constructing the still often exceeds the water collected. The acrid water that can be obtained in small quantities from water storing plants, such as cacti, is usually not safe to drink and may cause diarrhea, aggravating the survivor's dehydration. Also the ease with which water may be obtained from these plants is over-estimated.

Once a source of water is found, it is then important to determine if the water is potable, that is safe to drink. Animal tracks at the water's edge, green algae, or small invertebrates in the water suggest the water is potable. Avoid water that appears alkaline, lifeless, and edged with white crystals. The ingestion of alkaline water may cause diarrhea. Purify the water with two tablets of iodine per quart of water, and add one quarter teaspoonful of salt per quart of water when possible. Filter the water through cloth-covered straw, if available, to decrease sediment ingestion. Water should be consumed in regular, small amounts to ensure better absorption. Water consumed in irregular, large amounts is often lost through increased sweating and urination resulting from water overload.
Non-potable water should not be discarded as it can be used as radiator water to cool engines. It can also be used to wet personal clothing, enhancing evaporative cooling of the body and decreasing the need for sweating.

III. Temperatures

The desert is not well known for moderate temperatures. Summer temperatures vary from 90-120 degrees Fahrenheit in the day to 40-85 degrees Fahrenheit at night. Winter temperatures vary from 20-60 degrees Fahrenheit in the day to 0-30 degrees Fahrenheit at night. Hyperthermia and hypothermia in such extremes are real threats to survival in the desert, even during a twenty-four hour period. Adequate intake of water, restriction of daytime activities, and proper sheltering from the sun will decrease the chances for heat injury. Cold injury may be decreased by removing wet clothing at night, by not discarding clothing removed in the heat of day, and by seeking shelter from the effects of wind chill. Hypothermia may occur in unprepared desert survivors in temperatures as high as seventy degrees Fahrenheit.

IV. The sun

The sun will affect desert survival. The sun and the radiant heat that it develops may elevate ground temperatures to 200 degrees Fahrenheit. Therefore the desert survivor must not only seek shade from the direct and reflected sun rays, but the survivor must be elevated off the ground by at least one foot to avoid the additive heating effects of ground temperature elevation. Sunburn may cause disability, decrease sweat gland function, and increase the risk for secondary bacterial infection. The intense direct and reflected ultra-violet radiation may injure the retina leading to "sun-blindness". The desert survivor should modify sunglasses or cover the eyes with shades to allow only a slit of light to enter the eyes.

V. Sand and wind

The sand and wind work in unison to dehydrate and abrade the skin and mucous membranes resulting in conjunctivitis, corneal abrasions, and increasing the risk for secondary skin infections. Sand may contaminate food and water with abrasive
materials. Sand storms are particularly dangerous. During these storms, injuries from sand blast dramatically increase and members of a group of desert survivors may become separated. Dust devils, small vortices of wind and sand, have been known to throw humans to the ground resulting in disabling bone fractures and lacerations. The wind may produce wind chill at night or in the winter and increase the chances for cold injury. The desert survivor should seek shelter from these natural elements.

VI. Thunderstorms

Although rain may be welcomed by the desert survivor as a source of potable water, sudden cloud bursts may result in lethal lightning strikes and flash floods. The floods may travel miles away from the storm and suddenly flood desert areas where no rain is evident. The desert survivor should not camp in dry washes.

VII. Food

Desert survivors are more likely to die of dehydration than starvation since their survival situation is usually resolved, for better or for worse, in a short period of time. The large variety of desert plants and complexity of plant identification make the safe consumption of plants unlikely. Many desert plants are poisonous or will cause diarrhea when ingested. Cooked birds, bird eggs, snakes, and mammals are generally the safest food to eat in the desert if the survivor's food supply is exhausted. Eating food without the simultaneous ingestion of water will increase dehydration as the body forces fluid into the digestive tract to aid digestion of dry food.

VIII. Animals

The safest policy is to avoid all animals in the desert since most desert survivors are unlikely to excel at animal identification and many animals carry infectious diseases. It is best to strictly avoid ants, wasps, bees, spiders, centipedes, scorpions, snakes, and reptiles, as a majority of these may cause harm. Rodents and other mammals may harbor disease vectors such as lice, fleas, mites, ticks, and flies. Mammals may be infected with diseases, such as rabies or
plague. The desert survivor should be cautious when stepping or placing hands around logs, cacti, bushes, grass, and rocks, especially at night when most desert animals are more active.

IX. Desert illnesses

The following medical problems are likely to be aggravated by the desert environment:

1. Heat cramps, heat exhaustion, heat stroke, and hypothermia, with associated dehydration and electrolyte imbalance.

2. Sunburn and sun-blindness.

3. Secondary bacterial or fungal skin infections due to abrasion and puncture of the skin by moisture, sunburn, wind, chap, sand, plants, rocks, and insect bites.

4. Psychological stress secondary to fear of death or animal attacks, loneliness, and excessive concern about minor injuries.

5. Renal lithiasis precipitated by dehydration.

The following infectious diseases may develop in the desert environment:


2. Rickettsial: louse-borne typhus (Middle East and Asia) and Q-fever.

3. Bacterial: anthrax, brucellosis, gastroenteritis secondary to enterotoxigenic Escherichia coli, plague, salmonellosis, shigellosis, tetanus, trachoma, tuberculosis, and typhoid / paratyphoid fever.


5. Protozoal: amebiasis, giardiasis, and cutaneous leishmaniasis.

6. Helminthic: ascariasis, echinococcosis, hookworm, schistosomiasis, and taeniasis.
X. Additional comments

The best way to increase the chances for survival is to plan for an emergency survival situation before it occurs. Insure that immunizations are current. Carry more food and water than is required. Learn navigation techniques and carry topographical maps, marking known water sources along the planned route of travel.

Should a survival situation occur, the single most important thing to develop is a positive attitude that survival is possible, no matter the odds or circumstances. Do not let psychological stress overwhelm common sense. Focus a majority of effort on maintaining adequate hydration. Dawn and early morning are the best times to forage and change locations. Travel at night should only be with the aid of a light to prevent injury and snake bites. Messages should be left for rescue teams stating intentions when changing locations and mark the trail used in the move. Signal mirrors during the day, signal lights at night, and signal fires during the day or night are highly effective in the desert for attracting the attention of rescue teams.

Essentials of jungle survival

I. Introduction

Much of the tropics is covered by jungle habitat. Therefore, patients planning trips into jungle habitat should be given counseling on the human factors relevant to surviving in the jungle in an emergency situation.

Movies and television have imparted a dangerous, mystical quality to the jungle. At first glance, the jungle seems to be an impenetrable, hostile tangle of vegetation, stalked by man-eating animals. But these attributes have been magnified out of proportion to the reality that the elements for survival are readily available in the jungle. One can improve the probability for survival by understanding the effects of the jungle environment on the human factors for survival.
II. Water

Finding water is not difficult in the jungle when compared to the efforts the desert survivor must exert in his search for water. Rain water is the safest to drink, and easy to obtain in the monsoon season. The jungle survivor should purify all water for consumption with two tablets of iodine per quart of water, and add one quarter teaspoonful of salt per quart of water when possible. The water should be filtered through cloth-covered straw to decrease sediment ingestion. As with desert survival, ingestion of one to two gallons of water per day, in small amounts at regular intervals, is a major requirement for survival in the jungle.

III. Climate

The hallmarks of the jungle climate are high temperatures, high humidity, and heavy rains. The rains are present throughout the year at the equator and are seasonal in the form of monsoons, alternating with dry seasons, away from the equator. In general, the climate is more moderate and predictable than in the temperate zones. The heat and humidity are often not worse than that encountered in the cities of the southern United States during the summer months, they are just more persistent. Chilly days and nights are common during the winter months in the jungle, putting the unprepared jungle survivor at risk for developing hypothermia. The body may become partially acclimatized to the jungle climate after two weeks of exposure, but even then, the jungle climate can be mentally and physically oppressive.

IV. Food

Food is abundant in the jungle. Bananas, coconuts, oranges, lemons, papaya, and raspberries are recognizable and edible. Navele nuts, breadfruit, nakarika, and mangoes are also edible, but their appearance must be learned by the jungle survivor. Experience will also aid the jungle survivor in identifying and cooking the tubers of taro, yam, and yucca. Palm hearts may be used to make a refreshing salad. Generally, other fruits and flowers eaten by monkeys or birds are safe to eat. Cooked fish, fowl, crawfish, mammals, birds, and bird eggs are safe to eat and can be roasted inside banana leaves. Meat can also be cooked inside hollow bamboo sections. When the ends of the bamboo are sealed after cooking, the meat will not spoil for up to three days, if the seal is not broken.
The jungle survivor should not wash foods with contaminated water to prevent becoming infected with water-borne diseases.

V. Animals

Disease-carrying insects are the most significant health threat to the jungle survivor. Ticks, flies, and midges are present at anytime, while mosquitoes are most prevalent at dusk and dawn.

Sweat bees are bothersome in hot, dry weather, but will not bite. Ants, scorpions, spiders, and centipedes should be avoided. The rice-borer moth of Southeast Asia is attracted to evening lights and may cause burning, slow-healing skin lesions when the small, barbed hairs on their bodies are ground into human skin.

Leeches are common in the tropical Pacific and Southeast Asia. The leech bite commonly becomes secondarily infected and ulcerated, opening a portal for water-borne diseases and increasing the jungle survivor’s disability. The leech will release itself when touched by a burning cigarette, alcohol, or insect repellant. The number of leech bites can be decreased by tucking in the pant cuffs and applying a strap around the lower legs.

The probability of being bitten by a snake is no greater than in the southern United States, but the same precautions should be practiced. The jungle survivor should be careful where he steps or places his hands around bushes, trees, and rocks. The most dangerous snakes in the jungle are cobra, coral snake, bushmaster, fer-de-lance, Maylayan pit viper, rattlesnake, and in the salt water, the sea snake.

The large, meat-eating predators, such as lions, crocodiles, etc., are seldom seen and tend to avoid man. They will not attack unless provoked, wounded, or cornered.
VI. Jungle illnesses

The following medical problems are likely to develop in the jungle environment:

1. Heat cramps, heat exhaustion, heat stroke, and hypothermia, with associated dehydration and electrolyte imbalance.

2. Secondary bacterial or fungal skin infection due to abrasion and puncture of the skin by plants, moisture, insect bites, and leech bites.

3. Contact dermatitis similar to poison ivy.

4. Tree nettle stings.

5. Trench (immersion) foot.

6. Psychological stress secondary to the fear of animal attacks, loneliness, disorientation, and excessive concern about minor injuries.

The following infectious diseases may develop in the jungle environment:

1. Viral: arthropod-borne fevers, hemorrhagic illnesses, and encephalitides; rabies and viral hepatitis.

2. Rickettsial: typhus and Q-fever.

3. Bacterial: anthrax, brucellosis, cholera, gastroenteritis secondary to enterotoxigenic Escherichia coli, leprosy, leptospirosis, relapsing fever, salmonellosis (and paratyphoid fever), shigellosis, tetanus, tuberculosis, typhoid fever, and yersiniosis.


5. Protozoal: amebiasis, cutaneous and visceral leishmaniasis, giardiasis, malaria, and toxoplasmosis.

6. Helminthic: ascariasis, filariasis, hookworm disease, loiasis (Africa), onchocerciasis, schistosomiasis, strongyloidiasis, taeniasis, trichinosis, and trichuriasis.
VII. Additional comments

Jungle topographic maps are often inaccurate as the jungle tends to hide topographic features from aerial surveys, such as small streams and swamps. The jungle survivor should be prepared for these possible deceptions.

The additional comments outlined in the discussion on the essentials of desert survival are also valid for jungle survival and should be reviewed.***
Chapter IV
Differential diagnosis of infectious tropical diseases based on travel history

I. Introduction
II. Incubation period
III. Geographical distribution
IV. Practical example

Tables for differential diagnosis of infectious tropical diseases by estimated incubation period

- Incubation period less than one week
- Incubation period one week to one month
- Incubation period one month to three months
- Incubation period three months to one year
- Incubation period greater than one year
- Incubation period unknown or variable

Tables for geographic areas and countries

- Alphabetical listing of countries
  Prefaced by geographical area code
  Geographical Areas #A1, #A2, #A3, and #A4- tables
  Geographical Areas #B1, #B2, #B3, and #B4- tables
  Geographical Areas #C1, #C2, #C3, and #C4- tables

Quick reference summary table for incubation period and geographical distribution of infectious tropical diseases
Differential diagnosis of infectious tropical diseases based on travel history

I. Introduction

A travel history should be carefully obtained from a patient suspected of having a tropical disease. Gaining a knowledge of the estimated incubation period and the geographical areas visited by the sick traveler will greatly reduce the number of possibilities in the differential diagnosis of infectious tropical diseases. This will then permit health care providers to focus their reading and diagnostic work-up, resulting in a more timely diagnosis and treatment. This chapter is arranged as a series of lists and a summary table to assist the health care providers in developing a differential diagnosis of infectious tropical diseases based on the patient's travel history.

The travel history should include:

1. The dates of travel.
2. The exact locations visited by the tropical traveler.
3. The general ecology and elevation of the visited locations.
4. The patient's recollections of contact with animals or arthropods.
5. The patient's recollections of consumption of raw foods or untreated fluids.
6. The patient's recollections of contact with fresh water, plants, or soils; such as swimming or wading, hiking through abrasive brush, or walking barefoot.
7. The immunization history, including the date special immunizations were received before travel.
8. The chemoprophylaxis history; noting doses, compliance, side-effects, and use of foreign over-the-counter medications.
9. Determine if fellow travelers are also ill.
II. Incubation period

The incubation period can often be estimated from the travel history. The section labeled, "Incubation Period" in this chapter is divided into six subheadings:

1. Less than one week.
2. One week to one month.
3. One month to three months.
4. Three months to one year.
5. Greater than one year.
6. Incubation period unknown, unpredictable, or highly variable.

The diseases under each subheading are grouped by etiologic agent and are accompanied by the range of their incubation period. Many diseases with long incubation periods can be found listed under several subheadings.

III. Geographical distribution

The subtropical, tropical, and northern temperate world has been divided into fifteen geographical areas which follow ecologic and geographical boundaries. The first twelve areas are a modification of the geographical areas outlined in Strickland, Hunter's Tropical Medicine, 6th Edition, 1984. This chapter contains these areas listed under the following headings:

Area #A1: The southern United States, Central America, and northern South America.
Area #A2: Amazon River Basin.
Area #A3: western South America.
Area #A4: southeastern South America.
Area #B1: northern Africa.
Area #B2: western Africa.
Area #B3: east-central Africa.
Area #B4: southeastern Africa.
Area #C1: Middle East and south-central Asia.
Area #C2: Indian subcontinent.
Area #C3: Southeast Asia, South Pacific, Australia.
Area #C4: the Far East.

The Geographical Distribution of Infectious Tropical Diseases is introduced by an alphabetical listing of countries, with each country prefaced by its assigned geographical area code from Area #A1 through Area #C4.
Each geographical area is described. The listing in summary tables for each geographic area is subdivided into three categories of risk for acquiring infectious tropical diseases. The exact risk of acquiring a disease varies within each area depending on the season, degree of sanitation and disease vector control practiced by the visited community, the elevation of the visited location, the health and immunization status of the traveler, and the degree of the traveler's personal hygiene, eating habits, and chemoprophylaxis compliance. Each infectious disease is dynamic and its prevalence and location may vary during seasons and epidemics, with fluctuations in vector and reservoir populations, and changes in man's behaviors in relation to the ecosystem. Therefore, only a notation of historical maximum risk is estimated for each disease. This estimation is based on a compilation of the epidemiologic description for each disease found in: Benenson, Control of Communicable Diseases in Man, 14th Edition, 1985; Strickland, Hunter's Tropical Medicine, 6th Edition, 1984; Warren and Mahmoud, Tropical and Geographic Medicine, 1984; and Manson-Bahr and Bell, Manson's Tropical Diseases, Nineteenth Edition, 1987. The Centers for Disease Control in Atlanta, Georgia, and the Armed Forces Medical Intelligence Center, Fort Detrick, Maryland, can provide the physician with current infectious disease alerts for each tropical and subtropical country to augment the information contained in these listings.

IV. Practical example

A patient traveled to Kenya on a two week vacation to a commercial hotel in Nairobi. This trip also included a five day safari to a tropical savannah with primitive camping for four nights in the final week of this vacation. The patient developed a three day course of traveler's diarrhea, which began on the last day of his safari adventure, but he returned to his job in Pueblo, Colorado in good health. However, four weeks later, the patient was quite ill with a fever and went to his family physician for care.

His physician considers the possibility of a tropical disease and estimates an incubation period of four to five weeks based on the travel history. By referring to the listing of incubation periods for one month to three months, the physician is presented with forty possible tropical diseases. By referring to the summary table for Geographical Area #B4, which includes Kenya, he refines his differential of infectious tropical diseases to twenty diseases:
1. Bacterial:
   - brucellosis
   - nonvenereal endemic syphilis
   - tetanus
   - venereal syphilis
   - yaws

2. Helminthic:
   - cysticercosis
   - diphyllobothriasis
   - filariasis
   - hookworm disease
   - shistosomiasis
   - strongyloidiasis
   - taeniasis
   - toxocariasis
   - trichinosis
   - trichuriasis

3. Mycobacterial:
   - tuberculosis

4. Protozoal:
   - African trypanosomiasis
   - amebiasis
   - malaria
   - visceral leishmaniasis

5. Viral:
   - poliomyelitis
   - viral hepatitis

With this focused list of infectious tropical diseases and a knowledge of the patient's signs and symptoms (see Part III of this text), the clinician can make effective use of his or her time for academic review of these diseases and efficient use of confirming diagnostic tests.***
## Tables for differential diagnosis of infectious tropical diseases by estimated incubation period

### Incubation period less than one week

<table>
<thead>
<tr>
<th>Incubation period in days</th>
<th>Infectious tropical disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>From</td>
<td>To</td>
</tr>
<tr>
<td><strong>Bacterial</strong></td>
<td></td>
</tr>
<tr>
<td>3 YRS</td>
<td>Actinomycosis</td>
</tr>
<tr>
<td>1</td>
<td>Acute diarrhea due to campylobacter</td>
</tr>
<tr>
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<td>Anthrax</td>
</tr>
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<td>5</td>
<td>Brucellosis</td>
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<td>Genitourinary gonococcal disease</td>
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<td>Gonococcal conjunctivitis</td>
</tr>
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<td>Lyme disease</td>
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<td>Relapsing fever</td>
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<td>Salmonellosis</td>
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<td>Shigellosis</td>
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<tr>
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</tr>
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<td><strong>Chlamydial</strong></td>
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</tr>
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<td>5</td>
<td>Inclusion conjunctivitis</td>
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<tr>
<td>3</td>
<td>Lymphogranuloma venereum</td>
</tr>
<tr>
<td>5</td>
<td>Nongonococcal urethritis</td>
</tr>
<tr>
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<td>Psittacosis</td>
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</table>
**Incubation period less than one week**

### Helminthic

<table>
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<tr>
<th>Time (wks)</th>
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</tr>
<tr>
<td>4</td>
<td>60 Ascariasis</td>
</tr>
<tr>
<td>2</td>
<td>3 Cutaneous larva migrans/ cat or dog hookworm</td>
</tr>
<tr>
<td>1 YRS</td>
<td>7 Strongyloidiasis (threadworm)</td>
</tr>
<tr>
<td>5</td>
<td>45 Trichinosis</td>
</tr>
<tr>
<td>1 YRS</td>
<td>30 Trichuriasis (whipworm)</td>
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### Mycoses

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<tr>
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<td>18 Histoplasmosis</td>
</tr>
<tr>
<td>4</td>
<td>10 Tinea corporis</td>
</tr>
<tr>
<td>4</td>
<td>10 Tinea cruris</td>
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### Protozoal

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<tr>
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<td>360 Amebiasis</td>
</tr>
<tr>
<td>5</td>
<td>40 American trypanosomiasis (Chagas)</td>
</tr>
<tr>
<td>5</td>
<td>25 Giardiasis</td>
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<tr>
<td>3</td>
<td>30 Primary amebic meningoencephalitis</td>
</tr>
<tr>
<td>5</td>
<td>23 Toxoplasmosis</td>
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<td>20 Trichomoniasis</td>
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### Rickettsial

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<td>6</td>
<td>21 Scrub typhus</td>
</tr>
<tr>
<td>2</td>
<td>10 Tick-borne rickettsioses of the eastern hemisphere</td>
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(Boutonneuse & N. Asian tick fever, Queensland tick typhus)

### Viral

<table>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>2 Adenoviral and enteroviral hemorrhagic conjunctivitis</td>
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<tr>
<td>2</td>
<td>6 Chikungunya fever</td>
</tr>
<tr>
<td>3</td>
<td>12 Crimean-Congo hemorrhagic fever</td>
</tr>
<tr>
<td>3</td>
<td>15 Dengue fever</td>
</tr>
<tr>
<td>3</td>
<td>21 Ebola and Marburg virus diseases</td>
</tr>
<tr>
<td>0</td>
<td>2 Epidemic viral gastroenteropathy</td>
</tr>
<tr>
<td>3</td>
<td>12 Group C virus fevers</td>
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<tr>
<td>4</td>
<td>14 Japanese encephalitis</td>
</tr>
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<td>3</td>
<td>8 Kyasanur Forest disease</td>
</tr>
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<td>6</td>
<td>21 Lassa fever</td>
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<td>3</td>
<td>35 Poliomyelitis</td>
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<td>3</td>
<td>6 Rift Valley fever</td>
</tr>
<tr>
<td>1</td>
<td>5 Rotaviral enteritis</td>
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<td>6 Venezuelan equine encephalitis</td>
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<td>6 Yellow fever</td>
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### Incubation period from one week to one month

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#### Bacterial

- Actinomycosis
- Acute diarrhea due to campylobacter
- Bartonellosis
- Brucellosis
- Chancroid
- Granuloma inguinale
- Leptospirosis
- Lyme disease
- Melioidosis
- Meningococcal meningitis
- Nocardiosis
- Nonvenereal endemic syphilis
- Paratyphoid fever
- Pertussis
- Pinta
- Relapsing fever
- Tetanus
- Tularemia
- Typhoid fever
- Venereal syphilis
- Yaws
- Yersiniosis

#### Chlamydial

- Inclusion conjunctivitis
- Lymphogranuloma venereum
- Nongonococcal urethritis
- Psittacosis
- Trachoma

#### Helminthic

- Angiostrongylia
- Anisakiasis
- Ascariasis
- Schistosomiasis
- Strongyloidiasis (threadworm)
- Trichinosis
- Trichuriasis (whipworm)
Incubation period from one week to one month

<table>
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Incubation period from one month to three months

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<th>From</th>
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<td>Yaws</td>
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<td>YRS</td>
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65
Incubation period from one month to three months

Viral

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Incubation period: from three months to one year

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Protozoal

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### Incubation period: unknown or variable

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Incubation period: unknown or variable

Helminthic (cont)
UNK Paragonimiasis
UNK Streptocerciasis
UNK Trichostrongyliaasis

Mycobacterial
UNK Mycobacterioses

Mycoses
UNK Chromomycosis
UNK Cryptococcosis
UNK Entomophthoromycosis due to Basidiobolus
UNK Entomophthoromycosis due to Conidiobolus
UNK Lobo's disease
UNK Rhinosporidiosis
UNK Scytalidium and Hendersonula dermatitis
UNK Tinea pedis
UNK Tinea unguium
UNK Tinea versicolor

Protozoal
UNK Balantidiasis
UNK Cryptosporidiosis

Viral
UNK Oropouche fever
UNK Sindbis fever
### Alphabetical Listing of Countries Prefaced by Geographical Area Code

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</tr>
<tr>
<td>C1</td>
<td>Yemen, Democratic</td>
</tr>
<tr>
<td>B2</td>
<td>Zaire</td>
</tr>
<tr>
<td>B4</td>
<td>Zambia</td>
</tr>
<tr>
<td>B4</td>
<td>Zimbabwe (formerly Rhodesia)</td>
</tr>
</tbody>
</table>
Geographical area #A1

Geographical Area #A1, is located in Central America. This area includes the southern deserts of the United States of America, Mexico, the Caribbean Islands, all of the remaining Central American countries, and coastal Colombia, Venezuela, and Guyana.

Countries in geographical area A1

Antigua
Bahamas
Barbados
Barbuda
Belize (formerly British Honduras)
British Honduras (now Belize)
Colombia (northern coastal portion adjacent to Central America)
Costa Rica
Cuba
Dominica
Dominican Republic
El Salvador
Grenada
Grenadines
Guadeloupe
Guatemala
Guyana (coastal, Atlantic Ocean)
Haiti
Honduras
Jamaica
Martinique
Mexico
Montserrat
Netherlands Antilles
Nevis
Nicaragua
Panama
Saint Christopher
Saint Lucia
Saint Vincent
Tobago
United States of America (S.W. deserts and Deep South)
Venezuela (coastal, Atlantic Ocean)
Geographical area #A2

Geographical area #A2, is located in the Amazon River Basin. This area includes French Guiana, Suriname, the northern two thirds of Brazil, southern Guyana and Venezuela, and Colombia, Peru, and Bolivia east of the Andes Mountains.

Countries in geographical area A2

Bolivia (east of the Andes)
Brazil (Amazon Basin)
Colombia (east of the Andes)
French Guiana
Guyana (interior)
Suriname
Trinidad
Venezuela (interior highlands)

Geographical area #A3

Geographical area #A3 is located in western South America. This area includes Colombia, Ecuador, Peru, and Bolivia west of the Andes Mountains to the Pacific Ocean, and the northern half of Chile. It also includes the western mountain region of Argentina.

Countries in geographical area A3

Argentina (west of the Andes)
Bolivia (west of the Andes)
Colombia (west of the Andes)
Ecuador
Peru
**Geographical area #A4**

Geographical area #A4 is located in southeastern South America. This area includes Paraguay, Uruguay, southern Brazil, southeastern Bolivia, all of Argentina east of the Andes Mountains, and the southern half of Chile.

Countries in geographical area A4

Argentina (East of the Andes)
Brazil (Southern highlands)
Paraguay
Uruguay

**Geographical area #B1**

Geographical area #B1 is located in northern Africa. This area includes Egypt, Libya, Tunisia, Algeria, Morocco, West Sahara, Mauritania, and northern Mali, northern Niger, northern Chad, and northern Sudan. The Sahara Desert occupies most of this area.

Countries in geographical area B1

Algeria
Chad (northern portion)
Egypt
Libyan Arab Jamahiriya (formerly Libyan Arab Republic)
Libyan Arab Republic (formerly Libyan Arab Jamahiriya)
Mauritania
Morocco
Niger (northern portion)
Sudan (northern portion)
Tunisia
Geographical area #B2

Geographical Area #B2 is located in western Africa. This area includes the countries along the west African coast from Senegal to Namibia, and in the interior, southern Mali, southern Niger, southern Chad, Central African Empire, and Zaire.

Countries in geographical area B2

Angola
Benin, People's Republic of (formerly Dahomey)
Cameroon, United Republic of
Cape Verde
Central African Empire (now Central African Republic)
Central African Republic (formerly Central African Empire)
Chad (southern portion)
Congo
Dahomey (now Benin)
Equatorial Guinea
Gabon
Gambia
Ghana
Guinea
Guinea-Bissau (formerly Portuguese Guinea)
Ivory Coast
Liberia
Mali (southern portion)
Namibia
Niger (southern portion)
Nigeria
Portuguese Guinea (now Guinea-Bissau)
Sao Tome and Principe
Senegal
Sierra Leone
Togo
Upper Volta
Zaire
Geographical area #B3

Geographical area #B3 is located in east central Africa. This area includes the southern two thirds of Sudan, and all of Ethiopia and Somalia.

Countries in geographical area B3

Afars and the Issas, French Territory of the
(now Djibouti)
Djibouti (formerly Afars and the Issas,
French Territory of the)
Ethiopia
Somalia
Sudan (southern portion)

Geographical area #B4

Geographical area #B4 is located in south-eastern Africa. This area includes Uganda, Kenya, Tanzania, Zambia, Malawi, Zimbabwe, Mozambique, South Africa, Botswana, and Madagascar, as well as other islands off the east African coast.

Countries in geographical area B4

Botswana
Burundi
Comoros
Kenya
Lesotho
Madagascar
Malawi
Mauritius
Mozambique
Reunion
Rhodesia (now Zimbabwe)
Rwanda
South Africa
Swaziland
Tanzania, United Republic of
Uganda
Zambia
Zimbabwe (formerly Rhodesia)
**Geographical area #C1**

Geographical area #C1 is located in the Middle East. This area includes southern Union of Soviet Socialist Republics, Turkey, Syria, Lebanon, Israel, Jordan, Iraq, Iran, Afghanistan, Saudi Arabia, Kuwait, Qatar, United Arab Emirates, P.D.R. of Yemen, Yemen, and Oman.

Countries in geographical area C1

- Afghanistan
- Bahrain
- Cyprus
- Iran (Islamic Republic of)
- Iraq
- Jordan
- Lebanon
- Oman
- Qatar
- Saudi Arabia
- Syrian Arab Republic
- Trucial Sheikhdoms (now United Arab Emirates)
- Turkey
- Union of Soviet Socialist Republics (south-central portion)
- United Arab Emirates (formerly Trucial Sheikhdoms)
- Yemen
- Yemen, Democratic

**Geographical area #C2**

Geographical Area #C2 is located in the Indian subcontinent. This includes southeastern Iran, Pakistan, India, Sri Lanka, the Maldives Islands, southern Nepal, Bangladesh, Butan, and the eastern extension of India north of Burma.

Countries in geographical area C2

- Bangladesh
- Bhutan
- Ceylon (now Sri Lanka)
- India
- Maldives
- Nepal
- Pakistan
- Sri Lanka (formerly Ceylon)
Geographical area #C3

Geographical Area #C3 is located in Southeast Asia and the South Pacific. This includes Burma, Thailand, Kampuchea, Laos, Vietnam, Philippines, Malaysia, Indonesia, Papua-New Guinea, northern Australia, and the South Pacific Islands.

Countries in geographical area C3

American Samoa
Australia (north of the Tropic of Capricorn)
Brunei
Burma
Cambodia (now Democratic Kampuchea)
Christmas Island
Democratic Kampuchea (formerly Cambodia)
Fiji
French Polynesia (Tahiti)
Gilbert Islands (now Kiribati)
Indonesia
Kiribati (formerly Gilbert Islands)
Lao People's Democratic Republic
Laos (now Lao People's Democratic Republic)
Malaysia
Nauru
New Caledonia and Dependencies
New Hebrides (now Vanuatu)
Niue
Papua New Guinea
Philippines
Pitcairn
Samoa (formerly Western Samoa)
Samoa, Western (now Samoa)
Singapore
Solomon Islands
Tahiti (French Polynesia)
Thailand
Tonga
Tuvalu
Vanuatu (formerly New Hebrides)
Viet Nam (formerly Viet Nam, Socialist Republic of)
Geographical area #C4

Geographical Area #C4 is located in eastern Asia. This area includes far eastern Union of Soviet Socialist Republics, Mongolia, China, North Korea, South Korea, Japan, and the Pacific islands north of the Philippines.

Countries in geographical area C4

China
Japan
Korea, North
Korea, Republic of (South)
Mongolia
Taiwan
Union of Soviet Socialist Republics (far eastern portion)
### Incubation period and geographical distribution of infectious tropical diseases

**For geographical areas #A1 TO #A4**

#### Historical maximum risk notation:

- **0** = Disease is rare or not likely to occur in the traveler more frequently than while living in the urban United States.
- **1** = Disease is present but only occasionally transmitted to travelers.
- **2** = Disease is prevalent and is a hazard to travelers.
- **3** = Disease is a serious health hazard to travelers.

#### Incubation period notation:

- **YRS** = Incubation period is in greater than one year.
- **UNK** = Incubation period is unknown, unpredictable, or highly variable.

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>INCUBATION (DAYS)</th>
<th>Maximum risk in geographical area</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FROM - TO</td>
<td>#A1 #A2 #A3 #A4</td>
</tr>
<tr>
<td><strong>Bacterial</strong></td>
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<td></td>
</tr>
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<td>Anthrax</td>
<td>2 7</td>
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<tr>
<td>Bartonellosis</td>
<td>16 120</td>
<td>0 0 1 0</td>
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<tr>
<td>Brucellosis</td>
<td>5 30</td>
<td>2 2 2 2</td>
</tr>
<tr>
<td>Acute diarrhea due to Campylobacter</td>
<td>1 10</td>
<td>3 3 3 2</td>
</tr>
<tr>
<td>Chancroid</td>
<td>3 14</td>
<td>1 1 1 1</td>
</tr>
<tr>
<td>Diphtheria</td>
<td>2 5</td>
<td>1 1 1 0</td>
</tr>
<tr>
<td>Enterotoxigenic Escherichia coli</td>
<td>0 3</td>
<td>3 3 3 2</td>
</tr>
<tr>
<td>Genitourinary gonococcal disease</td>
<td>2 7</td>
<td>1 1 1 1</td>
</tr>
<tr>
<td>Gonococcal conjunctivitis</td>
<td>1 5</td>
<td>1 1 1 1</td>
</tr>
<tr>
<td>Granuloma inguinale</td>
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<td>4 19</td>
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<td>Melioidosis</td>
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<td>Meningococcal meningitis</td>
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<td>1 2 2 2</td>
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<tr>
<td>Nonvenereal endemic syphilis</td>
<td>14 90</td>
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<td>Pinta</td>
<td>14 21</td>
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<td>3 3 3 2</td>
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<td>Tularemia</td>
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<tr>
<td>Typhoid fever</td>
<td>7 21</td>
<td>2 3 3 2</td>
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<td>Yaws</td>
<td>14 90</td>
<td>1 0 0 0</td>
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<td>Yersiniosis</td>
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<td>DISEASE</td>
<td>INCUBATION (DAYS)</td>
<td>MAXIMUM RISK IN GEOGRAPHICAL AREA</td>
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<td>Inclusion conjunctivitis</td>
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<td>1  1  0  0</td>
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<td>Anisakiasis</td>
<td>0 - 30</td>
<td>0  0  1  0</td>
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<td>Ascarisis</td>
<td>4 - 60</td>
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<tr>
<td>Cutaneous larva migrans</td>
<td>2 - 3</td>
<td>1  1  0  0</td>
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<td>Cysticercosis</td>
<td>56 - YRS</td>
<td>1  1  1  1</td>
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<td>360 - 360</td>
<td>1  0  0  0</td>
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<td>90 - YRS</td>
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<td>Enterobiasis (Pinworm)</td>
<td>30 - 45</td>
<td>1  1  1  1</td>
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<tr>
<td>Filarial hypereosinophilia</td>
<td>UNK - UNK</td>
<td>1  1  1  1</td>
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<td>Filarisis</td>
<td>30 - 360</td>
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<td>Hookworm disease</td>
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<td>UNK - UNK</td>
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<td>UNK - UNK</td>
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<td>360 - YRS</td>
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<td>UNK - UNK</td>
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<td>Schistoamiasli</td>
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<td>Toxocariasis</td>
<td>21 - 180</td>
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<td>5 - 45</td>
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<td>360 - YRS</td>
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<td>Tuberculosis</td>
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<td>Mycetoma</td>
<td>30 - 360</td>
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<td>Paracoccidiomycosis</td>
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<td>Sporotrichosis</td>
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<td>Tinea capitis</td>
<td>10 - 14</td>
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<tr>
<td>Tinea corporis</td>
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<td>Tinea cruris</td>
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<td>Tinea pedis/unguim/versicolor</td>
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<tr>
<td>DISEASE</td>
<td>Protozoal</td>
<td>Rickettsial</td>
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<td>MAXIMUM RISK IN GEOGRAPHICAL AREA</td>
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<td>Balantidiasias</td>
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<td>Toxoplasmosis</td>
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<td>Visceral leishmaniasis (Kala-azar)</td>
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<td>Rickettsial</td>
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<tr>
<td>Louse-borne typhus fever</td>
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<td>Murine typhus</td>
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<td>Q fever</td>
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<td>Rocky mountain spotted Fever</td>
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<td>Trench fever</td>
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<td>Adeno/Enteroviral hemorrh. conjunctivitis</td>
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<td>Argentine &amp; Bolivian hemorrhagic fever</td>
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<td>Dengue fever</td>
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<td>Group C virus fevers</td>
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<td>Hepatitis, viral</td>
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<td>Mayaro fever</td>
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<td>Oropouche fever</td>
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<td>Poliomyelitis</td>
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<td>Rabies</td>
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<td>Rotaviral enteritis</td>
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<td>3</td>
</tr>
<tr>
<td>Rubeola (measles)</td>
<td>8 13</td>
<td>3</td>
</tr>
<tr>
<td>Sandfly fever</td>
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<tr>
<td>Venezuelan equine encephalitis</td>
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<td>1</td>
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<tr>
<td>Yellow fever</td>
<td>3 6</td>
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Summary: incubation period and geographical distribution
of infectious tropical diseases
For geographical areas #B1 TO #B4

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than while living in the urban United States.
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Incubation period is estimated in days.
YRS=Incubation period is in greater than one year.
UNK=Incubation period is unknown, unpredictable, or highly variable

<table>
<thead>
<tr>
<th>Disease</th>
<th>Incubation (DAYS) FROM - TO</th>
<th>Maximum risk in geographical area #B1 #B2 #B3 #B4</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Bacterial</em></td>
<td></td>
<td></td>
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<tr>
<td>Anthrax</td>
<td>2 7</td>
<td>1 1 2 2</td>
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<td>Chancroid</td>
<td>3 14</td>
<td>1 2 2 2</td>
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<td>Cholera</td>
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<td>Clostridium perfringens food poisoning</td>
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<td>0 0 0 1</td>
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Summary: incubation period and geographical distribution of infectious tropical diseases
For geographical areas #C1 to #C4

Historical maximum risk notation:

0=Disease is rare or not likely to occur in the traveler more frequently than while living in the urban United States.
1=Disease is present but only occasionally transmitted to travelers.
2=Disease is prevalent and is a hazard to travelers.
3=Disease is a serious health hazard to travelers.

Incubation period notation:

Incubation period is estimated in days.
YRS=Incubation period is in greater than one year.
UNK=Incubation period is unknown, unpredictable, or highly variable

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Chapter V
Fever

I. Fever and tropical disease
II. Acute fever / continuous / mild
III. Acute fever / continuous / major finding
IV. Acute fever / hyperpyrexic
V. Acute fever / intermittent
VI. Acute fever / relapsing
VII. Chronic fever
Fever

I. Fever and tropical disease

The febrile patient in the tropics or a febrile traveler returning from the tropics is not only possibly infected with the usual infectious agents of the temperate climates, such as Streptococcus or influenza virus, but may also be infected with a wide variety of tropical infectious agents, such as Plasmodium falciparum or dengue virus. In addition, there are infectious agents that are distributed world-wide, such as Mycobacterium tuberculosis or hepatitis-B virus, but have higher infection rates in tropical or subtropical, developing communities than in temperate, developed communities. Therefore, the differential diagnosis of a febrile illness in the tropics must be expanded to include diseases geographically limited to the tropics and diseases that occur world-wide, but occur more often in the tropics. Additional considerations for infectious tropical diseases associated with fever are listed below in the sections on acute and chronic fever.

Fortunately, a majority of the febrile illnesses acquired in the tropics are acute and self-limited, but there are many that may cause significant morbidity and mortality and require prompt diagnosis and treatment.

The differential diagnosis should also include non-infectious causes of fever as outlined below:

1. Autoimmune reaction.
2. Connective tissue disease.
7. Neoplastic disease.

II. Acute fever / continuous / mild.

Infectious tropical diseases that have an acute onset of continuous fever, but the fever is absent or mild (normal to 102 °F) and is usually not a predominant sign of the illness, include:

1. Amebiasis.
2. Ascariasis.
3. Angiostrongyliasis.
4. Bacterial gastroenteritis.
5. Bartonellosis.
6. Cryptosporidiosis.
7. Diphtheria.
8. Dracunculiasis.
9. Giardiasis.
10. Isosporiasis (other than due to Toxoplasma gondii).
11. Loiasis.
12. Lymphogranuloma venereum.
13. Pertussis.
15. Ross River fever (arthropod-borne arthritis and rash).
17. Strongyloides: during pulmonary stage.
18. Venereal syphilis, secondary, or with Jarisch-Herxheimer reaction after treatment of primary, secondary, or tertiary syphilis.
19. Viral gastroenteritis (other than rotavirus).

III. Acute fever / continuous / major finding

The acute onset of continuous fever with the fever as a major finding, possibly accompanied by chills, rigors, delirium, or febrile seizures, may be associated with:

1. Abdominal angiostrongyliasis.
2. Anthrax.
3. Arthropod-borne viral fevers:
   a. Encephalitides.
   b. Hemorrhagic fevers.
   c. Other arthropod-borne viral fevers producing an influenza-like illness.
5. Brucellosis.
6. Fascioliasis.
7. Leptospirosis.
8. Nocardiosis.
11. Primary amebic meningoencephalitis.
15. Rubeola (measles).
17. Shigellosis.
18. Smallpox: was associated with fever and prostration.
19. Trichinosis.
20. Tularemia.
21. Typhoid fever.
IV. Acute fever / hyperpyrexic

Tropical diseases that may be associated with acute fever exceeding 105 °F include:

1. Bacterial meningitides: with loss of thermoregulatory mechanisms complicating progressive meningitis.
2. Brucellosis.
3. Dengue fever.
5. Leptospirosis.
7. Shigellosis.

V. Acute fever / intermittent

Intermittent fever is characterized by regular paroxysms of fever with intervening afebrile periods.

1. American trypanosomiasis: usually beginning in the evening.
2. Malaria: usually beginning afternoon or early evening.
   a. Plasmodium vivax and Plasmodium ovale: 48 hours (tertian periodicity), beginning after two to seven days of irregular or daily paroxysms.
   b. Plasmodium malariae: 72 hours (quartan periodicity), beginning two to seven days after irregular or daily paroxysms.
   c. Plasmodium falciparum: the periodicity of falciparum malaria is often irregular, but may develop a regular pattern as well.

VI. Acute fever / relapsing

A relapsing pattern of fever is characterized by a period of continuous or intermittent fever, followed by an afebrile period, sometimes with apparent recovery, followed a febrile relapse. The interval of the relapse is usually irregular. The illness may be characterized by one relapse or many. Tropical diseases that may have a pattern of relapsing, acute fever are:

1. African trypanosomiasis.
2. Anthrax, inhalation.
4. Brucellosis.
5. Clonorchiasis: with biliary complications.
6. Filariasis.
7. Malaria.
8. Opisthorchiasis: with biliary complications.
9. Relapsing fever.
10. Trench fever.

VII. Chronic fever

Chronic fever, fever lasting more than two weeks, may be continuous low grade fever or undulating fever, with gradual waxing and waning of fever followed by afebrile periods. Often chronic fever is accompanied by night sweats, malaise, and weight loss. Chronic fever is a possible sign of:

1. Actinomycosis.
2. Aspergillosis.
4. Brucellosis.
5. Coccidiomycosis.
6. Cryptococciosis.
7. Echinococciosis.
8. Giardiasis.
10. Lyme disease.
11. Melioidosis
12. Mycobacterioses, atypical.
15. Paragonimiasis.
16. Primary amebic meningoencephalitis.
17. Psittacosis.
19. Schistosomiasis.
20. Toxocariasis.
21. Toxoplasmosis.
22. Tropical eosinophilia.
23. Tuberculosis.
24. Viral hepatitis.
25. Visceral leishmaniasis/***
Chapter VI

Neurological complications

Introduction

I. Neurology in the tropics
II. Specific tropical neurological complications
   A. Cerebral malaria
   B. Jamaican neuropathy
   C. Kuru
   D. Lathyrism
   E. Spinal cord adhesive arachnoiditis
   F. Tropical ataxic neuropathy
   G. Tropical spastic paraplegia

Mental status

I. Delirium
II. Psychiatric disorders
III. Dementia / encephalopathy
IV. Coma

Central nervous system infections

I. Cerebral malaria
II. Aseptic meningitis
III. Other meningitides
IV. Meningoencephalitis
V. Encephalitis
VI. Encephalomyelitis
VII. Myelitis
VIII. Tabes dorsalis
IX. Brain cysts or abscess

Neuritis / neuropathy

I. Introduction
II. Radiculitis
III. Optic atrophy
IV. Optic neuritis
V. Cranial neuropathy
VI. Peripheral neuropathy

Miscellaneous neurologic findings

I. Ataxia
II. Paralysis
III. Convulsions
IV. Headache
Neurological complications

Introduction

I. Neurology in the tropics

Significant mortality and morbidity, with permanent neurologic sequelae, result from neurological diseases in the tropics. This is primarily due to exposure to an increased number of infectious agents capable of infecting the nervous system, malnutrition, wide-spread alcohol and drug abuse, inadequate primary and tertiary health care, inadequate diagnostic laboratories, inadequate public health policies to promote public safety, poor industrial and occupational hygiene practices, and environmental contamination with heavy metals, hydrocarbons, and organophosphates. Late presentation for health care complicates attempts at preserving neurologic function.

Epilepsy is more common in developing communities than in communities of developed countries. It is a major health problem due to social prejudice and isolation, mythical beliefs, exclusion of the patient from school and the workplace, and inadequate primary health care.

The following neurological complications are less common in the tropics than in temperate climates:

1. Cerebrovascular accidents.
2. Degenerative disk disease.
4. Metastatic malignant central nervous system lesions.
5. Multiple sclerosis.
6. Primary degenerative dementia (presenile or senile dementias).
7. Trigeminal neuralgia.

In contrast, the following neurological problems occur more frequently in the tropics:

2. Bacterial meningitis: incidence increased by untreated otitis media, sinusitis, and other bacterial infections.
4. Brain cysts: secondary to helminthic diseases, especially cysticercosis and echinococcosis.
6. Cerebral malaria.
7. Encephalitis: particularly arthropod-borne encephalitides.
8. Epilepsy.
9. Hysteria: secondary to ritual cults, witchcraft, voodoo, etc.
11. Industrial and occupational biochemical and heavy metal intoxication.
13. Lathyrisn (chickpea) intoxication.
14. Leprosy.
15. Mental retardation due to perinatal trauma and protein malnutrition.
17. Nutritional neuropathies.
18. Pesticide intoxication.
19. Poliomyelitis.
22. Spinal cord adhesive arachnoiditis.
23. Tetanus.
24. Tropical ataxic neuropathy.
25. Tropical spastic paraplegia.

II. Specific tropical neurological complications

Cerebral malaria

Cerebral malaria is a devastating neurological disease that occurs in about two percent of malaria infections and is most commonly caused by Plasmodium falciparum. Travelers are at higher risk for more severe disease as a result of malaria.

The pathology of cerebral malaria results from occlusion of cerebral capillaries by infected red blood cells, which is accompanied by petechial hemorrhages, perivascular inflammation, and microglial cell response. Use of corticosteroids, combined with fluid overload, leads to cerebral edema.

The most common clinical findings are headache, photophobia, retinal hemorrhages, organic psychosis, amnesia, delirium, vertigo, convulsions, meningeal irritation, and coma.
Uncommon clinical findings are intention tremor, chorea, myoclonus, aphasia, cerebellar ataxia, hemianopsia, and hemiparesis. Papilledema and cranial nerve palsies are rare.

The cerebrospinal fluid pressure is normal or elevated. The cerebrospinal fluid glucose is normal, protein may be elevated, and rarely, there is a small to moderate number of lymphocytes.

Cerebral malaria is a medical emergency requiring intensive care support, anticonvulsant therapy, and intramuscular chloroquine plus intravenous quinine. If chloroquine-resistant malaria is suspected, then the patient should be given intravenous quinine, and replace chloroquine with pyrimethamine and sulfonamide. Corticosteroids, fluid overload, and heparin are contraindicated.

The mortality rate ranges from 20% to 80%, but fortunately, full recovery of neurologic function can be expected in survivors.

Jamaican neuropathy (Strachan neuropathy)

Jamaican neuropathy is a degenerative sensory neuropathy that occurs in Jamaican sugar cane workers, and malnourished tropical residents of Africa, the Middle East, Indian subcontinent, and the Far East. The cause is unknown, but it is strongly associated with chronic malnutrition. The disease is also associated with yaws and venereal syphilis. An exogenous intoxication, most likely dietary, has not been ruled out.

The disease has two presentations. The first involves the pyramidal tract with impairment of posterior column sensation, bladder control, girdling lumbar pain. The second, less common than the first, presents with sensory ataxia, numbness and burning of the feet, deafness, visual impairment with optic atrophy, and central scotomas; and in the lower extremities, mild spasticity, muscle wasting, and foot drop.

Kuru

Kuru, although declining in prevalence, occurs in tribesmen of New Guinea highlands who practice burial ritual cannibalism. It remains of interest as it is a classic example of a slow-virus disease.
Lathyrism

Lathyrism is an acute or insidious, permanent, demyelinating spastic paraplegia following consumption of improperly cooked Lathyrus sativus (chickpea). The demyelination involves the lateral spinal cord. This condition appears in times of food shortages when larger than usual amounts of chickpea are consumed. It is geographically found in Algeria, Ethiopia, and India.

Spinal cord adhesive arachnoiditis

Spinal cord adhesion with transverse myelitis or an ascending radiculomyelopathy is a common cause of paralysis in the tropics. The neurologic findings are paralysis, paresthesias and sensory root pain, bladder dysfunction, and muscle atrophy. The etiology is unknown, but the disease is associated with tuberculosis, syphilis, schistosomiasis, and as a sequela to pyogenic meningitis. It is geographically found in South America, West Indies, Africa, India, and Sri Lanka.

Tropical ataxic neuropathy (Nigerian ataxic neuropathy)

Tropical ataxic neuropathy is a demyelinating sensory neuropathy involving the peripheral nerves, posterior and lateral columns of the spinal cord, and spinocerebellar tracts. The neurological findings are a sensory ataxia, hypesthesia, hyporeflexia, optic atrophy, and deafness; which are similar to Jamaican neuropathy. The disease is associated with the intake of staple foods rich in cyanogenic glycosides. The most common source is improperly cooked Manihot esculenta (cassava, manihot, tapioca), and also certain strains of sorghum, millet, and maize. The disease responds poorly to therapy; therefore, prevention is recommended by removing the offending staple food from the diet or providing instructions to reduce the cyanide content of the staple food before cooking. The disease is found in the West Indies, Africa, and Asia.

Tropical spastic paraplegia

An etiology cannot be found for one third of the cases of spastic paraplegia. These cases are collectively given the diagnosis of tropical spastic paraplegia.
Mental status

I. Delirium

Delirium is a common neuropsychiatric finding in many tropical diseases. The patient may have hallucinations, illusions, incoherent speech, insomnia, disorientation, and memory impairment, which appear acutely and tend to fluctuate greatly in intensity.

1. Cerebral edema: most commonly accompanying infections, such as relapsing fever and cerebral malaria; see Central Nervous System Infections Section below.
2. Dehydration: especially in pediatric patients who are not receiving adequate oral rehydration therapy.
3. Fever: a multitude of tropical diseases are associated with fever and delirium is always a possible complication in those with severe disease; see Fever Chapter above.
4. Head trauma.
5. Heat stroke and severe heat exhaustion.
6. Hepatic failure / hepatic encephalopathy: viral hepatitis, alcoholism, electrolyte and fluid imbalance, herbal intoxication, cirrhosis; see also Hepatobiliary Complication Chapter below.
8. Hypoxia.
10. Meningitis / encephalitis: see Central Nervous System Infections Section below.
12. Reye's syndrome: noted to be occurring with greater frequency in the tropics, but this may be due to increased recognition as it is known that this syndrome is often overlooked; associated with vomiting, convulsions, delirium, coma, and decerebrate posturing.
13. Snake bite (venomous): may progress to coma.
14. Thiamine deficiency (beriberi).
II. Psychiatric disorders

Patients with tropical infectious diseases, poisonings, and nutritional deficiencies may present with acute or delayed onset psychiatric findings, confusing the diagnosis. The following should be considered in the differential diagnosis of major psychiatric diseases:

3. Brucellosis: chronic disease is associated with depression and intense psychosomatic complaints leading to misdiagnosis.
4. Crimean-Congo hemorrhagic fever: depression is common in the recovery phase.
6. Cysticercosis: focal brain lesions may produce psychiatric symptoms and signs, organic personality disorder.
10. Illicit drug abuse or abuse of socially accepted drugs:
    a. Alcohol intoxication: amnesia, hallucinations, depression, anxiety, psychomotor impairment, and dementia.
    b. Amphetamine, or other sympathomimetic, intoxication: psychomotor agitation, grandiosity, delusions, delirium, and depression.
    c. Barbiturate, or other sedative-hypnotic, intoxication: mood lability, maladaptive behavior, psychomotor impairment, amnesia, and depression.
    d. Cannabis intoxication: euphoria, altered perceptions of time and space, apathy, delusions, emotional lability, amnesia, and depersonalization.
    e. Cocaine intoxication: psychomotor agitation, grandiosity, maladaptive behavior.
    f. Hallucinogen intoxication: maladaptive behavior, hallucinations, illusions, delusions, depression, depersonalization, and intensified sense perceptions.
    g. Opioid intoxication: euphoria, apathy, maladaptive behavior, psychomotor impairment, dementia.
    h. Phencyclidine intoxication: hallucinations, paranoid ideation, psychomotor agitation, depression, marked anxiety, emotional lability, grandiosity, euphoria, and maladaptive behavior.
15. Plant poisoning: Digitalis purpurea, apathy and psychosis; Solanum species (immature potatoe sprouts or potatoe skin exposed to air during growth), hallucinations; Catha edulis (khat, wild tea), psychosis; and Datura stramonium (jimsonweed), hallucinations.
17. Thiamine deficiency (beriberi): Korsakoff's psychosis.
18. Typhoid fever: psychosis, mania, depression.

III. Dementia / encephalopathy

A major feature of encephalopathy is global organic dementia; but encephalopathy may appear acutely, is accompanied by fluctuating neurologic findings and convulsions, and may progress to coma.

1. Cirrhosis and hepatic encephalopathy.
2. Crimean-Congo hemorrhagic fever.
4. Electrolyte imbalance.
5. Heat stroke.
7. Hypoglycemia.
8. Hypoxia.
9. Lead intoxication.
10. Reye's syndrome.
12. Wernicke's encephalopathy.

Dementia is accompanied by the gradual onset of memory impairment, impairment of abstract thinking, impairment of impulse control and judgement, language construction difficulties, personality changes, anxiety, depression, paranoid ideation, delusions, and psychosocial dysfunction. In addition to primary degenerative dementia, the differential diagnosis of dementia in the tropics should include:

1. Alcoholism.
2. Cyanocobalamin (vitamin B12) deficiency.
3. Cysticercosis: with multiple brain cysts.
4. Hypertension, untreated, associated with arteriosclerosis and multiple brain infarctions.
5. Niacin deficiency (pellagra).
6. Paragonimiasis: with multiple focal granuloma and abscesses.
7. Thiamine deficiency (beriberi).
8. Venereal syphilis, tertiary.

IV. Coma

See also Central Nervous System Infections Section below.

Additional etiologies for coma in the tropics include:

1. Alcohol intoxication.
2. Diabetic ketoacidotic coma: diabetes is frequently untreated in communities with inadequate primary health care.
3. Elasmobranch fish poisoning.
5. Hepatic failure
6. Insecticide poisoning.
7. Mushroom poisoning.
8. Plant poisoning: Dieffenbachia species; Datura stramonium (jimsonweed, thorn apple), Boophone disticha (gifbol), Lantana camara (wild sage), Euphorbia species, Hura polyandra (devil tree), Jatropha curcas (Barbados nut), and Nerium oleander (oleander).
10. Trauma.
11. Uremia.

Central nervous system infections

I. Cerebral malaria

See discussion of cerebral malaria in chapter introductory comments above.

II. Aseptic meningitis

The findings in meningitis are headache, meningeal irritation, fever, and usually photophobia. Additional etiologies of aseptic meningitis in the tropics include:

1. Argentine and Bolivian hemorrhagic fevers.
2. Australian encephalitis (Murray Valley fever).
3. Epstein-Barr virus infection: rare complication.
4. Equine encephalitides.
5. Far Eastern tick-borne encephalitis.
7. Leptospirosis.
8. Lyme disease.
10. Sindbis fever.

III. Other meningitides

See also Meningoencephalitis Section below.

Tuberculous meningitis is more common among infants and young children in developing communities than in developed countries. The differential diagnosis of acute purulent meningitis in the tropics must include:

1. Anthrax.
2. Campylobacter septicemia: rare complication.
3. Candidiasis.
5. Gnathostomiasis: eosinophilic meningitis.
7. Melioidosis.
8. Meningococcal meningitis.
12. Salmonellosis.
15. Tularemia.
16. Typhoid fever.

III. Meningoencephalitis

Tropical infections capable of producing meningitis and/or encephalitis include:

1. African trypanosomiasis.
2. American trypanosomiasis.
3. Angiostrongylia: eosinophilic meningoencephalitis.
4. Australian encephalitis (Murray Valley fever).
5. Bartonellosis.
7. Cryptococcosis: especially in immune-compromised patients.
8. Cysticercosis: may be accompanied by meningeal irritation.
9. Equine encephalitides (St. Louis, Venezuelan, eastern, and western).
10. Far Eastern tick-borne encephalitis.
13. Leptospirosis.
15. Louse-borne typhus fever.
16. Lyme disease.
17. Mucormycosis: especially in immune-compromised patients.
18. Murine typhus.
20. Primary amebic meningoencephalitis.
22. Rift Valley fever.
23. Rocky Mountain spotted fever.
24. Toxoplasmosis.
25. Trichinosis.
26. Variola (smallpox): was a possible complication of smallpox.
27. Venereal syphilis.
28. West Nile fever.

IV. Encephalitis

See also Meningoencephalitis Section above.

See also Encephalomyelitis Section below.

Encephalitis is accompanied by fever, headache, altered state of consciousness, focal neurologic signs, and convulsions. The differential diagnosis of encephalitis in the tropics should include:

1. Poliomyelitis.
2. Rabies.
3. Rubeola (measles).
5. Toxocariasis: rare complication.
6. Yellow fever.

V. Encephalomyelitis

Additional etiologies for encephalomyelitis include:

2. Poliomyelitis.
3. Post-vaccinal encephalomyelitis: following vaccination for measles, smallpox, and rabies (Semple-type vaccine).
4. Rubella (measles).
5. Typhoid fever.
6. Variola (smallpox): was a complication of smallpox.

VI. Myelitis

See also Encephalomyelitis Section above.

Myelitis is accompanied by inflammation of the spinal cord with pain, hyperesthesia, anesthesia, motor disturbances, paralysis, loss of sphincter control, and hypereflexia. In the tropics, myelitis may be caused by:

1. Opiate abuse: transverse myelitis may complicate a return to opiate abuse after a period of abstinence.
2. Poliomyelitis.
4. Schistosomiasis: transverse myelitis and spinal cord granulomas.
5. Trichinosis.

VII. Tabes dorsalis

Tabes dorsalis as a complication of venereal syphilis is more common in developing communities where adequate primary health care is not available. It is accompanied by abnormal pupil reflexes; and in the lower extremities, impaired vibratory and position sensation, hyporeflexia, and muscle wasting. Tabes dorsalis is associated with optic atrophy and dementia.

VIII. Brain cysts or abscess

The differential diagnosis of brain cyst or abscess in the tropics should be expanded to include:

1. Amebiasis: brain abscess is a rare, but usually fatal complication.
2. Cysticercosis: brain cysts.
3. Echinococcosis due to Echinococcus granulosus or Echinococcus multilocularis: less likely due to E. multilocularis; brain cysts.
5. Paragonimiasis: brain abscesses, including cerebral granulomas.
6. Primary amebic encephalitis: brain abscess.
Neuritis / neuropathy

I. Introduction

The medical terms, "neuritis" and "neuropathy", are used interchangably to denote a pathologic change in a nerve leading to functional disturbances; such as, pain, paresthesias, anesthesia, sensory loss, motor loss and muscle atrophy, and hyporeflexia.

II. Radiculitis

Gnathostomiasis is an unusual etiology for radiculitis.

III. Optic atrophy

Additional etiologies for optic atrophy in the tropics include:

1. Adverse drug reaction: chloramphenicol, clioquinol (Entero-Vioform), isoniazid, para-aminosalicylic acid, quinine, and tryparsamide.
4. Methanol intoxication.
5. Niacin deficiency (pellagra).
6. Onchocerciasis.
7. Plant poisoning: Argemone mexicana oil resulting in endemic dropsy.
8. Thiamine deficiency (beriberi).
10. Untreated optic neuritis.
11. Venereal syphilis.

IV. Optic neuritis

Multiple sclerosis is a common cause of optic neuritis in temperate climates, but this disease is rare in the tropics. The diseases listed below are common causes for optic neuritis:

1. Brucellosis.
2. Onchocerciasis.
3. Toxoplasmosis.
4. Tuberculosis.
5. Venereal syphilis.
V. Cranial neuropathy

See also Meningoencephalitis Section and Encephalitis Section above.

The differential diagnosis of central or peripheral cranial neuropathy in the tropics should include:

1. Adenoviral or enteroxviral hemorrhagic conjunctivitis: uncommon complication.
3. Angiostrongyliaisis.
5. Ciguatera fish poisoning.
6. Cryptococcosis.
7. Cysticercosis.
8. Diphtheria.
10. Lyme disease.
11. Nasopharyngeal carcinoma.
12. Poliomyelitis.
15. Schistosomiasis.
17. Tetanus.
18. Thiamine deficiency (beriberi).
19. Venereal syphilis.

VI. Ganglionic neuropathy

American trypanosomiasis is associated with ganglionic neuropathy resulting in megaesophagus and megacolon.

VII. Peripheral neuropathy

Acquired peripheral neuropathy is due to infections, metabolic disorders, nutritional deficiencies, and biochemical intoxication.

1. Adverse drug reaction: arsenics, chloroquine, clioquinol (Enterovioform), dapsone, disulfiram, ethambutol, isoniazid without pyridoxine replacement, metronidazole, nitrofurantoin, para-aminosalicylic acid, pentamidine, perhexilene maleate, phenytoin, chronic overdose of pyridoxine, quinine, and streptomycin.
2. Alcoholism.
5. Cyanocobalamin deficiency (vitamin B12).
7. Diphtheria.
8. Diphyllobothriasis.
10. Environmental poisoning: arsenic, lead (peripheral neuropathy affects adults more frequently than children), and mercury.
11. Giardiasis.
12. Industrial intoxication: acrylamide, carbon disulfide, hexanes, methyl N-butyl ketone, organophosphates, polychlorinated biphenyls (PCB), styrenes, and trichloroethylene.
13. Leprosy.
15. Lyme disease.
17. Malnutrition.
20. Salmonellosis.
21. Thiamine deficiency (beriberi).
22. Trichinosis.
23. Tri-ortho-cresyl phosphate (TCP or ginger-jake): a contaminant of "moonshine" and cooking oils.
24. Typhoid fever.
25. Uremia: untreated renal failure common in the tropics.
26. Venereal syphilis, tertiary.

Miscellaneous neurologic findings

I. Ataxia

Additional considerations for the etiology of ataxia in the tropics include:

1. Alcoholic cerebellar degeneration.
2. American trypanosomiasis.
3. Ciguatera fish poisoning.
5. Cyanocobalamin deficiency (vitamin B12).
6. Cysticercosis.
7. Elasmobranch fish poisoning.
8. Glue-sniffer's ataxia.
12. Lyme disease.
15. Thiamine deficiency (beriberi).
16. Tropical ataxic neuropathy.

II. Paralysis

See also Central Nervous System Infections Section above.
See also Neuritis / Neuropathy Section above.
See also Brain Cyst / Abscess Section above.

Infectious, environmental, and nutritional etiologies for paralysis are more common in the tropics than cerebrovascular accidents.

1. Ascorbic acid deficiency (scurvy): pseudo-paralysis is a possible complication.
2. Ciguatera fish poisoning: secondary to polyneuropathy caused by neurotoxins.
5. Ebola and Marburg virus diseases: hemiplegia is a possible complication.
6. Echinococcosis due to Echinococcus granulosus or Echinococcus multilocularis: secondary to brain cysts.
8. Hypokalemia: secondary to severe diarrhea and malabsorption.
10. Malaria: secondary to cerebritis and cranial neuropathy.
14. Plant poisoning: secondary to neurotoxin; see also Respiratory Failure Section of the Pulmonary Complications Chapter; Melia azedarach (chinaberry or syringa), Phytolacca americana (pokeweed), Cicuta maculata (water hemlock), and Nerium oleander (oleander).
15. Poliomyelitis: secondary to cranial neuropathy.
and encephalomyelitis.
17. Schistosomiasis: secondary to cranial neuropathy, transverse myelitis, and brain granulomas.
20. Tetradon fish poisoning: secondary to neurotoxin.
22. Trauma: untreated peripheral nerve and central nervous system trauma is common in the tropics.
23. Trichinosis: secondary to peripheral neuritis, myelitis, and encephalitis.
25. Venereal syphilis, tertiary: secondary to polyneuropathy and tabes dorsalis.

III. Convulsions

See also Central Nervous System Infections Section above.
See also Brain Cysts / Abscesses Section above.

Febrile convulsions are common in the tropics. Seizures are frequently complicated by burns as the unconscious, seizing patient rolls into floor level heating and cooking fireplaces. Cysticercosis is a leading cause of seizure disorders in the tropics. Additional considerations for the etiology of convulsions in the tropics should include:

1. Drug abuse: cocaine, opiates, phencyclidine, and sedative-hypnotics.
2. Echinococcosis due to Echinococcus granulosus or Echinococcus multilocularis: secondary to brain cysts.
3. Heterophyiasis: secondary to cerebral granulomas.
5. Hypoglycemia.
6. Hypoxia.
9. Mushroom poisoning.
12. Perinatal trauma.
13. Plant poisoning: Dieffenbachia species, Strychnos nux-vomica, Aconitum napellus (monkshood), Hura polyandra (devil tree), Jatropha curcas (Barbados nut), Ricinus communis (castor bean), Laburnum species, and Conium maculatum (poison parsley).
15. Schistosomiasis: secondary to cerebral granulomas.
17. Sickle cell anemia.
18. Snake bite (venomous).
19. Tetraodon fish poisoning.
20. Thiamine deficiency (beriberi).
21. Trichinosis.
23. Vitamin D deficiency: may be complicated by hypocalcemia resulting in convulsions.

IV. Headache

See also Central Nervous System Infections section.

Psychosocial stresses of poverty and the social transition to industrialization and urbanization produce muscle contraction headaches. Migraine headaches occur as frequently in the tropics as in temperate climates. Headache is a frequent, but nonspecific finding, in a majority of tropical infectious diseases. Additional etiologies for headache in the tropics include:

1. Chikungunya fever.
2. Crimean-Congo hemorrhagic fever.
3. Cryptosporidiosis.
4. Dengue fever.
5. Diphyllobothriasis.
7. Elasmobranch fish poisoning.
8. Enterotoxigenic Escherichia coli gastroenteritis.
10. Fascioliasis.
11. Filariasis.
15. Hymenolepiasis.
17. Lassa fever.
18. Mayaro fever.
19. O'nyong nyong fever.
20. Oropouche fever.
22. Plague.
23. Q fever.
24. Relapsing fever.
25. Rickettsial pox.
26. Sandfly fever.
27. Schistosomiasis.
28. Scombroid fish poisoning.
29. Scrub typhus.
30. Taeniasis.
31. Tick-borne rickettsioses of the eastern hemisphere (Boutonneuse fever, Siberian tick typhus, Queensland tick typhus).
32. Trench fever.
33. Vibrio parahaemolyticus food poisoning.
34. Yersiniosis.***
Chapter VII
Ophthalmologic complications

Introduction
I. Ophthalmology in developing communities and the tropics

Photophobia and ocular pain
I. Introduction
II. Photophobia
III. Ocular pain

Conjunctival signs
I. Conjunctival injection
II. Conjunctivitis

Corneal signs
I. Keratoconjunctivitis
II. Keratitis

Anterior chamber findings
I. Anterior uveitis / iritis / iridocyclitis
II. Glaucoma

Ocular lens signs
I. Cataracts

Posterior chamber signs
I. Posterior uveitis / chorioretinitis
II. Other retinal complications
Ophthalmologic complications

Introduction

I. Ophthalmology in developing communities and the tropics

The sharp contrast between ophthalmology in developing communities compared to developed communities is easily demonstrated by listing the major causes of blindness for each.

In the developed communities of the world, blindness occurs more often late in life, usually as the result of an underlying, chronic illness. Some of the causes for blindness, such as glaucoma and cataract, are treatable with medication or surgery. The most common causes for blindness are:

2. Cataract.
4. Optic nerve atrophy: arteriosclerosis and arteritis.
5. Corneal diseases.
7. Temporal arteritis.

But in the developing communities of the world, blindness occurs as much in the young as in the elderly. These communities carry a heavy burden of blinding ocular diseases that are treatable eighty percent of the time, but go untreated due to the inadequacies of their primary health care systems. The patients are more likely to present for care, when it is available, late in the course of their illness when irreversible damage may have been done, magnifying the tragedy. The blindness rate in developing communities is five times as great as in developed communities. The most common causes for blindness are:

1. Trachoma.
2. Untreated cataract.
3. Xerophthalmia due to vitamin A deficiency and rubeola.
4. Onchocerciasis (river blindness): a filarial disease transmitted by blackflies in tropical Africa, Central America, and South America.
5. Corneal scarring due to untreated keratitis, keratoconjunctivitis, corneal ulcers, and corneal trauma. Gonococcal conjunctivitis, which is common in the tropics, may rapidly involve the cornea when untreated.

7. Untreated glaucoma.

8. Methyl alcohol intoxication.


This chapter mainly focuses on the differential diagnosis of the painful red eye in the tropics. Neurologic disorders of the eye, such as central blindness, optic neuritis, and cranial neuropathy, are discussed in the chapter, "Neurologic Complications".

Photophobia and ocular pain

I. Introduction

Photophobia and ocular pain are often present with constitutional symptoms in many rickettsial and viral illnesses. Photophobia and ocular pain may be caused by conjunctivitis, corneal disease, iritis, and glaucoma, as discussed in other sections below.

II. Photophobia

See also conjunctival signs, corneal signs, and anterior chamber signs below.

Photophobia is often present as a non-specific symptom in:

1. Filariasis.
2. Group C virus fevers.
3. Louse-borne typhus fever.
5. O'nyong Nyong fever.
6. Psittacosis.
7. Rickettsial pox: rare disease of urban areas.
8. Rift Valley fever.
9. Rocky Mountain spotted fever.
10. Rubeola (measles).
11. Sandfly fever.
12. Venezuelan equine encephalitis.
III. Ocular pain

Intense ocular or retro-orbital pain, often accompanied by fever, headache, and myalgia, is present as a non-specific symptom in:

1. Dengue fever (break-bone fever).
2. Sandfly fever.
3. Trench fever.
4. West Nile fever.

Conjunctival signs

I. Conjunctival injection

As a non-specific clinical finding of many spirochetal, rickettsial, and viral diseases, conjunctival injection, a noticeable suffusion of the conjunctival blood vessels, is often seen in tropical diseases. Conjunctival injection occurs with a minimal amount of discharge, but when present, is watery. Conjunctivitis may have conjunctival injection as an early sign. The following diseases frequently have conjunctival injection as a non-specific clinical finding:

1. Argentine and Bolivian hemorrhagic fever.
2. Chikungunya fever.
3. Dengue fever.
4. Environmental exposure.
5. Group C virus fevers.
6. Hemorrhagic fever with renal syndrome: may be accompanied by subconjunctival hemorrhage.
8. Leptospirosis.
11. Oropouche fever.
12. Relapsing fever.
13. Rift Valley fever.
15. Scrub typhus.
16. Tick-borne rickettsioses of the eastern hemisphere (Boutronasse fever, North Asian tick fever, and Queensland fever).
17. Venezuelan equine encephalitis.
18. Vitamin A deficiency: secondary to xerosis (dryness) of the conjunctival mucosa.
19. West Nile fever.
20. Yellow fever.
II. Conjunctivitis

Conjunctivitis is a serious disease in the tropics since it often goes untreated, placing the cornea at risk for secondary infection and irreversible damage. Poor sanitary conditions, crowding, malnutrition, and underdeveloped primary health care systems promote the perpetuation of conjunctivitis in a community, often in epidemics.

The clinical differentiation of conjunctivitis from other common causes of the "red or pink eye" begins with noting that the visual acuity is normal. There is painful, irritating inflammation of the conjunctiva with a purulent ocular discharge. The sticky discharge may seal the eye lids together, especially while sleeping. The infection is usually bilateral and the redness of the eye is more peripheral, away from the cornea. The cornea appears clear and bright. The patient may complain of photophobia.

Trachoma is a chlamydial conjunctivitis that is common in the tropics and is a major cause of preventable blindness in the world. Chronic and repeated infections may result in xerosis and keratoconjunctivitis, which is then complicated by blindness. Untreated neonatal gonococcal conjunctivitis may be rapidly complicated by corneal involvement and blindness.

Conjunctivitis may be caused by:

1. Adenoviral or enteroviral epidemic hemorrhagic conjunctivitis: associated with prominent chemosis, periorbital edema, and preauricular lymphadenopathy.
2. American trypanosomiasis (Chagas' disease): occasionally this protozoal infection is introduced into the eye resulting in unilateral conjunctivitis and periorbital swelling.
4. Diphtheria: may be accompanied by purulent rhinitis or exudative pharyngitis.
5. Ebola and Marburg virus diseases.
6. Environmental exposure to dry wind and sand.
7. Gonococcal conjunctivitis.
8. Inclusion conjunctivitis due to Chlamydia trachomatis: a follicular conjunctivitis that infrequently involves the cornea, seen in newborns and in association with chlamydial genitourinary infections.
9. Lassa fever.
10. Leptospirosis.
11. Loiasis: conjunctival swelling and inflammation due to the subconjunctival migration of an adult filarial worm.

12. Lymphogranuloma venereum due to Chlamydia trachomatis: rare complication.

13. Miasis (maggot infestation): botfly larvae may invade the conjunctiva and cornea stimulating a catarrhal conjunctivitis and secondary bacterial infection.

14. Nongonococcal urethritis: often due to Chlamydia may be complicated by conjunctivitis; Reiter's syndrome may be confused with this presentation as well.

15. Onchocerciasis (river blindness).


17. Plant poisoning: when rubbed into the eye, the latex of Euphorbia (pointsettia) and Hippomane (manchineel) and the sap of Hura polyandra (devil tree) may cause a burning, vesiculating conjunctivitis and periorbital contact dermatitis; also contact with the toxic hairs, spines, and fluids of various insects when these are rubbed into the eye.

18. Rocky Mountain spotted fever.


20. Toxoplasmosis.

21. Trachoma: follicular and papillary conjunctivitis with tarsal scarring, lid entropion, trichiasis, xerosis, corneal pannus, corneal ulcers, secondary infection, and eventual blindness with repeated infections.

22. Trichinosis: chemosis.

23. Tularemia.

24. Variola (smallpox).

Corneal signs

I. Keratoconjunctivitis

Some episodes of conjunctivitis are complicated by corneal inflammation. This is more common in:

1. Adenoviral or enteroviral epidemic hemorrhagic conjunctivitis.

2. Herpetic conjunctivitis: a serious infection in developing communities when proper diagnosis and treatment are not available.


4. Plant poisoning: see conjunctivitis section above.
5. Trachoma.
6. Tuberculosis.
7. Vitamin A deficiency: as a complication of xerophthalmia.

II. Keratitis

Keratitis may cause a painful, red eye with photophobia. However, in contrast to conjunctivitis, there is usually no purulent ocular discharge and the redness is localized to the perilimbus. The patient often complains of blurred vision. The pain may be dramatically relieved by the application of a local anesthetic. Keratitis may be complicated by corneal ulceration. Severe, untreated keratitis may result in a permanent decrease in visual acuity or blindness. Corneal disease is associated with the following diseases:

1. Adenoviral or enteroviral epidemic hemorrhagic conjunctivitis: may be complicated by mild keratitis or punctate keratitis.
2. Aspergillosis.
3. Gonococcal conjunctivitis: may be rapidly complicated by corneal ulcerations and blindness without prompt recognition and treatment.
4. Inclusion conjunctivitis due to Chlamydia trachomatis: may be complicated by corneal pannus, obstructing vision.
5. Leprosy: corneal scarring may be a complication of leprosy when lid palsies lead to corneal xerosis and environmental exposure; corneal pannus and ulcerations.
8. Onchocerciasis (river blindness): may be complicated by punctate and sclerosing keratitis, a major cause of blindness in endemic areas.
9. Rubeola (measles): may be complicated by xerophthalmia and keratomalacia.
10. Salmonellosis: may be complicated by corneal ulcerations.
11. Trachoma: may be complicated by corneal pannus and punctate keratitis, especially with repeated infections.
12. Tularemia: may be complicated by corneal ulcerations.
13. Variola (smallpox): keratitis was a common complication of variola when lesions involved the ocular area.
14. Vitamin A deficiency: may be complicated by corneal ulcerations and xerophthalmia.
Anterior chamber findings

I. Anterior uveitis / iritis / iridocyclitis

Anterior uveitis is a general term for inflammation involving the posterior cornea, iris, or ciliary body. Iritis refers to inflammation of the iris. Iridocyclitis refers to inflammation of the iris and ciliary body. However, both have similar signs and symptoms.

Anterior uveitis may cause a painful, red eye, but the redness is perilimbal. The patient complains of intense photophobia. A sticky ocular discharge is usually not present unless the anterior uveitis is an extension of conjunctivitis or corneal ulceration. The patient complains of a decrease in visual acuity. The pupil is constricted. The ocular globe is tender and there is no relief of pain with the application of local anesthetics.

Anterior uveitis may be a complication of the following diseases:

1. Adenoviral or enteroviral epidemic hemorrhagic conjunctivitis.
2. Brucellosis.
3. Congenital rubella.
4. Cysticercosis.
5. Gnathostomiasis.
7. Lassa fever.
8. Leprosy.
10. Loiasis.
12. Onchocerciasis.
13. Rubeola.
15. Toxoplasmosis.
16. Tuberculosis.
17. Variola (smallpox).
18. Venereal syphilis.

II. Glaucoma

Acute angle-closure glaucoma is another cause of a painful, red eye. The patient complains of blurred vision due to edema and "steaming" of the cornea. The pupil is usually dilated. Acute angle-closure glaucoma is a serious problem leading to blindness in developing communities when prompt medical care is
not available. Acute angle-closure glaucoma is more common among natives of Vietnam, Burma, Thailand, and Java, but is quite rare among Melanesians, Polynesians, Mongolians, and Bedouins.

Other causes for glaucoma include:
1. Argemone mexicana poisoning (Mexican poppy): ingestion of this plant's seeds may be complicated by "epidemic dropsy" and glaucoma.
2. Congenital rubella.
3. Onchocerciasis (river blindness).

Ocular lens signs

I. Cataracts

Cataracts may be associated with:
1. Congenital rubella.
2. Congenital syphilis.
3. Cysticercosis.
4. Leprosy.
5. Onchocerciasis.
6. Takayasu's arteritis.
7. Toxoplasmosis.

Posterior chamber signs

I. Posterior uveitis / chorioretinitis

Posterior uveitis is an inflammation of the posterior chamber of the eye, which includes the choroid and retina. Chorioretinitis is an alternate term for posterior uveitis. Although most cases of chorioretinitis are idiopathic, one must also consider the following diseases in the tropics:
1. Brucellosis: choroiditis.
2. Candidiasis: may be a complication in the immune-compromised host.
3. Congenital rubella.
5. Cysticercosis.
7. Onchocerciasis (river blindness).
II. Other retinal complications

Other retinal complications include:

1. Angiostrongyliasis: retinal hemorrhage and detachment.
2. Cysticercosis: rarely complicated by retinal detachment.
3. Echinococcosis due to Echinococcus granulosus: retinal cysts.
5. Hypertensive retinopathy: hypertension and its complications often go unrecognized and untreated where primary health care is not readily available.
8. Rift Valley fever: retinal vasculitis, edema, and hemorrhage, and macular exudates.
10. Toxocariasis: retinal granulomas and macular scarring.
11. Vitamin C deficiency: retinal hemorrhage is a possible complication of severe vitamin C deficiency.***
Chapter VIII
Ear, nose, and throat complications

Face and parotid gland
I. Facial swelling
II. Parotid gland

Ear
I. Otitis externa
II. Otitis media

Nasopharyngeal complications
I. Nasal integument
II. Nasopharyngeal carcinoma
III. Rhinitis
IV. Epistaxis
V. Sinusitis

Mouth and throat
I. Lips
II. Stomatitis
III. Glossitis
IV. Pharyngitis
V. Laryngeal complications
Ear, nose, and throat complications

Face and parotid gland

I. Facial swelling

Additional considerations for facial swelling in the tropics include:

1. Actinomycosis: suppurative granulomas with sinus tracts containing pus and "sulfur" granules.
2. Allergic reactions.
3. Burkitt's lymphoma: mass lesion, usually located in jaw or nasopharynx.
4. Entomophthoromycosis due to Conidiobolus: deep mycosis with nasofacial swelling.
5. Rubeola (measles): preceding progression to cancrum oris, a complication of measles in the tropics.

II. Parotid gland

Parotitis may complicate:

1. American trypanosomiasis (Chagas' disease): parotid swelling may lead to parotid duct obstruction and secondary parotitis.
2. Rocky Mountain spotted fever.
3. Typhoid fever.
4. Yellow fever.

Ear

I. Otitis externa

Otitis externa due to fungal infections, primarily Aspergillus species, is more common in the warm, humid tropics; especially after swimming or splashing water into the ear. This is generally a benign condition, unless there is secondary bacterial infection, which may be initiated at breaks in the skin caused by the patient scratching the ear canal.
II. Otitis media

The usual etiologies for otitis media in the tropics are not much different than those for temperate climates. However, otic complications do occur in diseases that have been eliminated from the developed world by immunization. The differential diagnosis must include:

1. Barotrauma: after high altitude flight, barotrauma may cause signs in the middle ear that may be confused with or later complicated by otitis media.
2. Burkitt's lymphoma: as a complication of a nasopharyngeal mass lesion.
3. Chlamydia trachomatis: infections by this organism may infrequently cause otitis media.
4. Diphtheria.
5. Pertussis.
6. Rocky Mountain spotted fever.
7. Rubeola (measles): deafness as a complication.

Nasopharyngeal complications

I. Nasal integument

See also Mucocutaneous Lesions section of the Dermatologic Complications chapter.

The following nasal integument complications are possible:

2. Entomophthoromycosis due to Conidiobolus: nasal swelling and obstruction of nasal airflow.
3. Leprosy: perforation of the nasal septum.
4. Mucormycosis: necrosis of the nasal mucosa, cartilage, and bone, with involvement of the palate; black nasal turbinates are a usual finding.
5. Myiasis (maggot infestation).
7. Pentastomiasis (tongue worm infestation).
II. Nasopharyngeal carcinoma

Nasopharyngeal carcinoma is a poorly differentiated squamous cell carcinoma that is more common in eastern Africa, the Far East, and Malaysia. The patient may complain of nasal airflow obstruction, but usually notes cervical swelling first. Cranial neuropathies are a common complication. This tumor has been associated with Epstein-Barr virus.

III. Rhinitis

Rhinitis may be present in:

1. Allergic rhinitis.
2. Chlamydial pneumonitis of the newborn.
4. Diphtheria: a purulent nasal discharge may be the only clinical sign.
5. Entomophthoromycosis due to Conidiobolus.
6. Inclusion conjunctivitis.
7. Mucormycosis: often the discharge is bloody.
8. Nasopharyngeal carcinoma.
10. Rotaviral enteritis.

IV. Epistaxis

See also Hemorrhagic Complications section.

Epistaxis may complicate:

1. Dengue fever.
2. Leprosy.
3. Leptospirosis.
4. Mucocutaneous fungal infections.
5. Mucocutaneous leishmaniasis.
7. Myiasis (maggot infestation).
8. Nasopharyngeal carcinoma.
10. Relapsing fever.
11. Tularemia.
12. Typhoid fever.
13. Uncontrolled hypertension.
V. Sinusitis

Mycotic sinusitis occurs more frequently in the tropics and sub-tropics. In the Sudan, Aspergillus species are the most common etiology for sinusitis. The differential diagnosis should include:

1. Aspergilloid.
2. Entomophthoromycosis due to Conidiobolus.
3. Mucormycosis: especially in debilitated, malnourished, or immune-compromised patients.
4. Salmonellosis.

Mouth and throat

I. Lips

Cancrum oris is a rapidly developing, necrotic ulcer of the face, primarily the lips, that is caused by several bacteria and spirochetes. This condition is most often seen in malnourished children with other systemic illnesses, especially measles.

II. Stomatitis

Stomatitis occurs in developing communities as a complication of malnourishment.

1. Ariboflavinosis.
2. Follicular hyperkeratosis: caused by a deficiency of essential fatty acids and B-complex vitamins.
3. Pyridoxine deficiency.

III. Glossitis

In the tropics and sub-tropics, glossitis primarily occurs as a complication of malnutrition or malabsorption.

1. Ariboflavinosis.
2. Cyanocobalamin deficiency (vitamin B12).
3. Diphyllobothriasis: an intestinal helminthic infection that may be complicated by malabsorption.
4. Folate deficiency.
5. Follicular hyperkeratosis.
6. Giardiasis: this intestinal protozoal infection may be complicated by malabsorption.
8. Niacin deficiency (pellagra).
IV. Pharyngitis

Pharyngitis may accompany:

1. Chikungunya fever.
2. Dengue fever.
3. Diphtheria.
5. Epstein-Barr virus disease.
7. Inclusion conjunctivitis.
8. Lassa fever.
9. Malaria: may be a prodromal symptom.
10. Melioidosis.
11. Meningococcal meningitis.
12. Paratyphoid fever.
13. Plague.
15. Sindbis fever.
16. Toxoplasmosis.
17. Tularemia: ulcerative.
18. Venezuelan equine encephalitis.
19. West Nile fever.
20. Yersiniosis.

V. Laryngeal complications

Laryngeal complications may occur in:

1. Ascariasis: obstruction with migration of the parasite worm into the airway.
2. Diphtheria: obstructive, exudative membranes adhere to laryngeal wall and epiglottis.
3. Leeches: leeches have been found in the upper respiratory tract, sometimes resulting in fatal airway obstruction.
5. Rhinosporidiosis: verrucoid lesions of the larynx.
6. Rubeola (measles): may be complicated by an obstructive laryngeal swelling.
7. Tetanus: laryngospasm.
8. Tuberculosis: laryngitis.
Chapter IX
Pulmonary complications

Introduction
I. Pulmonary disease in the tropics

Pneumonitis
I. Pneumonitis with allergic symptoms
II. Pneumonitis

Pneumonia
I. Pneumonia with consolidation only
II. Pneumonia complicated by cavitation

Hemoptysis
I. Hemoptysis in the tropics

Pleural effusion
I. Eosinophilic pleural effusion
II. Pleural effusion

Pulmonary abscess or empyema
I. Pulmonary abscess
II. Empyema

Hilar adenopathy
I. Hilar adenopathy as a clinical finding

Respiratory failure
I. Neurotoxic respiratory failure
II. Rabies

Miscellaneous pulmonary complications
I. Bronchospasm
II. Chronic bronchitis
III. Cough as the only pulmonary symptom
IV. Pulmonary cysts
V. Nodular infiltrates
VI. Other miscellaneous pulmonary complications
Pulmonary complications

Introduction

I. Pulmonary disease in the tropics

Pulmonary problems cause significant morbidity and mortality in the tropics, comparable to that caused by gastrointestinal problems. A leading cause of death among children in the tropics is acute febrile respiratory disease caused by parainfluenza virus, respiratory syncytial virus, or as a complication of measles. Chronic pulmonary infections and residuals of acute infections only partially treated, if at all, are a frequent cause of disability among adults in the tropics.

Pneumococcal pneumonia and aspiration pneumonia are more common in the tropics. Bronchiectasis, as a complication of pneumonia, is more common in the tropics, although this is probably due to inadequate antibiotic treatment rather than a predisposition based on geographic location.

Antibiotic resistance is more prevalent in the tropics. For example, in South Africa, some pneumococcal serotypes are resistant to penicillins, erythromycin, tetracyclines, trimethoprim-sulfamethoxazole, chloramphenicol, and aminoglycosides. The pneumococci are still sensitive to rifampin, but for how long? This is quite a contrast to the nearly uniform sensitivity of pneumococci to penicillin in the United States of America.

Pneumonia caused by Pneumocystis carinii and other unusual opportunistic pathogens is a common complication of malnourishment in children, especially those with marasmus, due to a decay in immune competency. The growing number of patients with acquired immunodeficiency syndrome are at risk for developing opportunistic pulmonary complications.

Regardless of a patient's immune status, protozoal and helminthic infections with pulmonary involvement occur more frequently in the tropics and should be additional considerations for the physician's differential diagnosis.

Certain tropical pulmonary infections are difficult to diagnose or may have a confusing clinical presentation. For example, melioidosis may mimic tuberculosis. Paragonimiasis may co-exist with, and actually reactivate, dormant tuberculosis.
Pneumonitis

I. Pneumonitis with allergic symptoms

Certain helminthic infections may cause pneumonitis with allergic symptoms as the result of a hypersensitivity reaction, thought to be due to the passage of the parasite through the lung tissues or perhaps an allergic reaction to by-products of the parasite. Fungal infections may induce a hypersensitivity pulmonary reaction. This reaction is usually associated with peripheral eosinophilia, urticaria or other maculopapular rashes, and wheezing.

1. Ascariasis: a potent parasitic allergen.
2. Aspergillosis.
4. Cutaneous larva migrans due to Ancylostoma braziliense and caninum (dog and cat hookworm).
5. Gnathostomiasis.
6. Hookworm disease.
7. Schistosomiasis.
8. Strongyloidiasis (threadworm).
10. Trichinosis.

II. Pneumonitis

Other infections may be associated with pneumonitis, although some of these may progress to pneumonia with consolidation or necrosis.

1. Actinomycosis.
2. Anthrax.
4. Chlamydial pneumonitis of the newborn.
5. Coccidiomycosis.
7. Enterotoxigenic Escherichia coli: pneumonitis as a result of aspiration.
8. Epstein-Barr virus disease: as a complication of acute primary infection.
10. Melioidosis.
11. Psittacosis.
12. Q-fever.
13. Rocky Mountain spotted fever.
15. Toxoplasmosis.
16. Tuberculosis.
17. Tularemia.
18. Typhoid fever.

Pneumonia

I. Pneumonia with consolidation only

Pneumonia with consolidation may be caused by a variety of bacteria and viruses, as in the temperate climates; however in the tropics, the diseases listed below must also be considered:

1. Amebiasis.
2. Blastomycosis.
3. Heat stroke: pneumonia is a common delayed complication of heat stroke as the result of septicemia.
4. Paracoccidiomycosis.
5. Paragonimiasis.
6. Plague.
7. Rocky Mountain spotted fever: with severe infections.
8. Tetanus: the neuromuscular complications of tetanus may result in aspiration pneumonia, laryngospasm, and pulmonary emboli.
10. Variola (smallpox): secondary bacterial pulmonary infection was a common complication of variola.

II. Pneumonia complicated by cavitation

Bacterial and mycotic pneumonia may be complicated by necrosis and cavitation, especially with inadequate therapy. The diseases listed below, occur more frequently in the tropics. They are mostly chronic infections with fever, productive cough, and usually nocturnal sweats, and when active, induce a prolonged debility. Melioidosis is an exception, as it may be complicated by a fulminant course of cavitary pneumonia, septicemia, and death.

1. Actinomycosis.
2. Coccidiomyosisis.
3. Cryptococcosis.
4. Histoplasmosis.
5. Melioidosis.
7. Nocardiosis.
8. Paragonimiasis.
10. Tuberculosis.
Hemoptysis

I. Hemoptysis in the tropics

See also Hemorrhagic Complications Section in the Hematologic Complications Chapter.

Hemoptysis may occur as the result of hemorrhagic phenomena, bronchitis, bronchiectasis, pneumonia, trauma, or pulmonary vasculitis, abscess, thromboembolism, or carcinoma. After ruling out bleeding from the nasopharynx or gastrointestinal tract as the source of blood in the sputum, the following diseases must be considered in the differential diagnosis:

5. Echinococcosis due to Echinococcus granulosus: complicating metastatic cystic pulmonary lesions.
7. Murine typhus.
8. Nocardiosis.
11. Strongyloidiasis: accompanying the organism's migration through the lung.
12. Trichinosis: as the result of pulmonary vasculitis and passage of newborn larvae through the lungs.
13. Tuberculosis: may be the only presenting sign.

Pleural effusion

I. Eosinophilic pleural effusion

Certain tropical diseases may be complicated by an eosinophilic pleural effusion, as well as peripheral eosinophilia.

1. Amebiasis.
2. Ascariasis.
3. Coccidiomycosis.
4. Filarial hypereosinophilia: when pulmonary complications are present, there are usually only allergic signs and symptoms, such as wheezing, and diffuse, miliary infiltrates on chest X-ray, but pleural effusions have been reported.

5. Hookworm disease: when complicated by anasarca.

6. Paragonimiasis.

7. Strongyloidiasis: a rare complication.

II. Pleural effusion

The following infections have been complicated by pleural effusion, but pleural fluid eosinophilia is usually absent:

1. Anthrax.
2. Brucellosis: a rare complication.
3. Capillariasis due to Capillaria philippinensis: may be complicated by a transudative pleural effusion associated with ascites.

5. Filarialiasis: may be complicated by a transudative pleural effusion associated with ascites.
6. Lassa fever: may be complicated by pleural effusion, sometimes associated with ascites.
7. Melioidosis.
8. Psittacosis.
10. Tularemia.

Pulmonary abscess or empyema

I. Pulmonary abscess

Pulmonary abscess is a possible complication of the following infections:

1. Amebiasis.
2. Blastomycosis.
3. Candidiasis: as an opportunistic infection, especially in the immune-compromised or malnourished patient, causing pulmonary abscesses that appear as nodular pulmonary infiltrates.
4. Fascioliasis: as an occasional complication.
5. Melioidosis.
6. Paragonimiasis.
7. Strongyloidiasis: in the immune-compromised patient, this disease may have a fulminant course with gram-negative septicemia, paralytic ileus, meningitis, and pneumonia with abscess formation.

II. Empyema

Empyema is usually a complication of common bacterial infections, such as staphylococcal, pneumococcal, or gram-negative pneumonias. This complication is more common in the tropics as these infections are often inadequately treated. Empyema is accompanied by fever, nocturnal sweats, pleuritic pain, chronic cough, and weight loss. In the tropics, these additional infections may be complicated by empyema:

1. Actinomycosis.
2. Blastomycosis.
4. Paragonimiasis.
5. Tuberculosis.

Respiratory failure

I. Neurotoxic respiratory failure

Neurotoxic paralysis of the respiratory muscles may cause respiratory failure. This complication is usually accompanied by paresthesias, gastrointestinal upset, ataxia, convulsions, and shock syndrome.

1. Botulism.
2. Ciguatera fish poisoning: cranial neuropathy.
3. Diphtheria: peripheral neuritis, cranial neuropathy, encephalitis, obstructive respiratory failure with bronchopulmonary exudates, and croup.
4. Elasmobranch fish poisoning: visual disturbances and coma.
5. Mushroom poisoning: psychosis, and coma.
6. Organophosphate contamination: acute and chronic organophosphate poisoning are more common in the underdeveloped countries of the tropics.
7. Paralytic shellfish poisoning: incoherent speech, aphonia, motor weakness, and chest tightness.
8. Plant poisoning: Boophone disticha (bulb of gifbol), nicotiana species (tobacco), Gelsemium sempervirens (yellow jessamine), Strychnos nux-vomica, Melia azedarach (chinaberry or syringa), Aconitum napellus (monkshood), laburnum species, Colchicum autumnale
(autumn crocus), Phytolacca americana (pokeweed), umbellifera species (water hemlock and poison parsley), oleander species, and Manihot esculenta (bitter cassava, manihot, and tapioca).

10. Tetraodon fish poisoning.

II. Rabies

Respiratory failure associated with meningoencephalitis is the usual fatal complication of rabies.

Miscellaneous pulmonary complications

I. Bronchospasm

The following infections have been confused clinically with asthma.

1. Ascariasis.
2. Echinococcosis due to Echinococcus granulosus: due to anaphylactoid reaction to fluid escaping from an echinococcal cyst.
3. Filarial hypereosinophilia.
5. Strongyloidiasis.
6. Toxocariasis.

II. Chronic bronchitis

Chronic bronchitis may be a predominant clinical finding in:

1. Melioidosis.
2. Paragonimiasis.
3. Tuberculosis.

III. Cough as the only pulmonary symptom

Cough without other pulmonary findings may be present in:

1. Anisakiasis.
2. Relapsing fever.
3. Louse-borne typhus.
4. Viral illnesses.
IV. Pulmonary cysts

The following diseases may be complicated by pulmonary cysts:

1. Echinococcosis due to Echinococcus granulosus and occasionally Echinococcus multiforme.
2. Paragonimiasis.

V. Nodular infiltrates

Nodular infiltrates are a common finding in the following infections, leading to confusion with malignant lesions:

1. Blastomycosis.
2. Coccidiomycosis.
3. Cryptococcosis.
4. Histoplasmosis.
5. Toxocariasis.

VI. Other miscellaneous pulmonary complications

The following infections have additional pulmonary complications:

2. Anthrax: mediastinitis and severe respiratory distress.
4. Diphtheria: airway obstruction due to exudative bronchopulmonary involvement.
7. Malaria due to Plasmodium falciparum: pulmonary edema associated with fluid overload and possibly use of corticosteroids.
10. Rocky Mountain spotted fever: pulmonary edema with severe disease and fluid overload.
12. Tuberculosis: pulmonary fibrosis and bronchopulmonary fistulas.***
Chapter X
Cardiovascular complications

Introduction
I. Cardiovascular disease in the tropics

Shock syndrome
I. Shock syndrome as a complication of tropical disease

Cardiac inflammation
I. Pericarditis
II. Myocarditis
III. Endocarditis

Congestive heart failure
I. Congestive heart failure in the tropics

Cardiac arrhythmias
I. Cardiac arrhythmias

Miscellaneous cardiovascular problems
I. Myocardial infarction
II. Cor pulmonale
III. Myocardial fibrosis
Cardiovascular complications

Introduction

I. Cardiovascular disease in the tropics

The broad spectrum of the availability of medical care in the tropics and subtropics may alter one's perception of the prevalence of cardiovascular disease. In the focal areas that are experiencing development and have successful public health programs, coronary artery disease appears as a common cause for admission to the hospital. But in the more underdeveloped and isolated areas of the tropics, untreated hypertension may be the most frequent cardiovascular disease afflicting the local population.

But in general, the following cardiovascular problems are more common in the tropics:

1. Untreated hypertension and its complications.
2. Cardiovascular complications of syphilis.
3. Takayasu's disease.
4. Endomyocardial fibrosis.
5. Untreated congenital heart disease.
6. Untreated rheumatic heart disease.
7. Pericarditis.
8. Endocarditis.
9. Parasitic infestations of the heart.

Although the incidence of hypertension in the tropics is no greater than in temperate countries, untreated hypertension is a major problem in populations that have underdeveloped primary health care systems. Therefore, the physician visiting these areas or the physician treating an immigrant from the tropics is more likely to find the complications of untreated hypertension during patient evaluation. The most prevalent complications are hypertensive cardiovascular disease, cerebral vascular accident, renal failure, and peripartum cardiac failure.

Venereal syphilis occurs commonly in many areas of the tropics. Social and cultural attitudes towards promiscuity, prostitution, and polygamy, and the decreased availability of primary health care are factors molding the predominance of secondary and tertiary venereal syphilis within a given community. As many as ten percent of the deaths from cardiovascular disease in developing areas of the tropics may be the result of the cardiovascular complications of untreated
venereal syphilis. These cardiac complications include: aortitis with secondary hypertension, aortic aneurysm, aortic insufficiency, and coronary ostitis with angina. Other treponematoses, such as nonvenereal endemic syphilis, pinta, and yaws, also occur commonly in the tropics; however, these rarely present with cardiovascular complications.

Takayasu's disease is an obstructive arteritis that is most often found in young adult females from the orient. The disease is also found in Latin America and Africa. The recent substantial increase in oriental immigrants into this country may make the disease appear more prevalent here as well.

The precise etiology is unknown, but it is probably an autoimmune disease. Hypertension, artery obstruction, and artery pain are the major clinical findings along with constitutional symptoms of fever, arthralgias, weight loss, and fatigue. Neurologic complications of artery obstruction, pulmonary hypertension, and intermittent claudication occur as the disease progresses. The aortic arch and its branch vessels are the arteries most often involved. The prognosis for recovery is poor, as this disease is usually relentless and progressive until death occurs from cerebral compromise or cardiac failure, usually within five years of diagnosis.

Endomyocardial fibrosis is a restrictive cardiomyopathy of unknown etiology that is more common in central Africa, India, Malaya, Japan, and northern and eastern South America. Up to twenty-five percent of the deaths from cardiovascular disease in central Africa may be due to this disease. Children and young adults are most often affected.

The endocardium is usually damaged in the apex and outflow tracts of the ventricles. This may extend into the heart valves leading to valvular insufficiency. Congestive heart failure is common with left-sided involvement. The disease may be clinically confused with constrictive pericarditis or rheumatic heart disease. The prognosis is poor, and palliative cardiovalvular surgery is often not an available option for treatment in developing communities, though it may effectively improve selected patients.

Congenital heart disease occurs with the same frequency in the tropics as in the temperate climates, but it is more likely to go undiagnosed and untreated in areas with inadequate medical care. Early deaths from congenital heart disease contribute to the higher infant mortality rates in developing countries. Those patients that survive may not be diagnosed as having congenital heart disease until they present for medical care as an adult with an unrelated problem. With inadequate
antibiotic chemoprophylaxis, these patients are at higher risk for developing endocarditis.

Given the global distribution and high frequency of streptococcal infections, communities with inadequate primary care in the tropics have a higher incidence of rheumatic fever and rheumatic heart disease. These complications of untreated streptococcal infections are more likely to occur early in life, and without antibiotic chemoprophylaxis, recurrent infections are common. Rheumatic heart disease is the most common cause of congestive heart failure in the children of developing countries. It should be considered in the differential diagnosis of failure-to-thrive in these pediatric populations.

**Shock syndrome**

I. Shock syndrome as a complication of tropical disease

Shock syndrome is a possible complication in the acute stages of intoxications, bacterial, rickettsial, or viral infections as outlined below:

1. Anthrax.
2. Argentine and Bolivian hemorrhagic fevers.
5. Ciguatera fish poisoning.
6. Clostridium perfringens food poisoning.
7. Crimean-Congo hemorrhagic fever.
8. Dengue hemorrhagic fever.
11. Hemorrhagic fever with renal syndrome.
12. Lassa fever.
13. Louse-borne typhus fever.
14. Malaria due to Plasmodium falciparum.
15. Meningococcal meningitis.
17. Paralytic shellfish poisoning.
18. Plant poisoning: some plant poisonings are complicated by hypotension, but the primary fatal complications of plant poisoning are respiratory paralysis and status epilepticus.
19. Rift Valley fever.
20. Rocky Mountain spotted fever.
23. Tetraodon fish poisoning.
24. Yellow fever.

Cardiac inflammation

I. Pericarditis

Bacterial and tuberculous pericarditis are more common in the tropics. It may complicate:

1. Amebiasis: most often an extension of an amebic abscess in the left lobe of the liver.
2. American trypanosomiasis.
4. Echinococcosis due to Echinococcus granulosus: complicating a cardiac cyst.
5. Histoplasmosis: usually as an extension of mediastinal lymph node involvement.
7. Psittacosis.
8. Q-fever.
10. Toxoplasmosis.
11. Tuberculosis.

II. Myocarditis

Myocarditis is more often due to bacterial and parasitic infections in the tropics and subtropics than in the temperate climates where viral infections predominate as the etiologic agent. American trypanosomiasis (Chagas' disease) is the classic example of parasitic myocarditis. As many as six percent of the rural inhabitants of Central and South America may have myocardial damage caused by the protozoan, Trypanosoma cruzi. Other diseases complicated by myocarditis include:

2. Coxsackie virus infection.
3. Diphtheria: common early or late complication in patients requiring hospitalization.
4. Ebola and Marburg virus diseases: with severe disease.
5. Leptospirosis.
7. Lyme disease.
9. Primary amebic encephalitis.
11. Relapsing fever.
12. Rocky Mountain spotted fever.
14. Tetanus.
15. Toxocariasis: rare complication.
16. Toxoplasmosis.
17. Trichinosis.
18. Typhoid fever.
19. West Nile fever.
20. Yellow fever.

III. Endocarditis

Subacute bacterial endocarditis is more common in the tropics due to inadequate medical care of bacterial infections and septicemia, higher prevalence of rheumatic heart disease and untreated congenital heart disease, untreated skin infections, and the common use of unsterilized needles in remote clinics, tattooing, and illicit drug use. Patients who are malnourished, immune-suppressed, or receiving intravenous therapy, are at higher risk as well. Etiologies for endocarditis in the tropics are:

1. American trypanosomiasis (Chagas' disease).
2. Brucellosis.
4. Cryptococcosis: most often in immune-compromised patients.
5. Diphtheria: rare complication.
6. Histoplasmosis.
7. Psittacosis.
8. Q fever.
10. Tularemia.
11. Typhoid fever.

Congestive heart failure

I. Congestive heart failure in the tropics

In addition to congestive heart failure due to congenital heart disease and rheumatic heart disease, the following causes for congestive heart failure occur more frequently in the tropics:

1. African trypanosomiasis.
4. Heterophyiasis: rare complication due to myocardial fibrosis.
5. Iron deficiency.
7. Plant poisoning: certain plants are cardiotoxic, such as, Argemone mexicana (Mexican poppy) containing a glycoside sanguinarine, and Cannabis sativa containing cannabinol.
8. Rocky Mountain spotted fever: with severe infection.

Cardiac arrhythmias

I. Cardiac arrhythmias

Patients with myocarditis may have cardiac arrhythmias, and in addition, the following should be considered:

1. Diphtheria: fatal, sudden arrhythmias may occur weeks after an apparent recovery from diphtheria myocarditis.
2. Echinococcosis due to Echinococcus granulosus: conduction delays are possible if the parasite strategically encysts itself along the cardiac conduction pathways.
3. Plant poisoning: nicotine, oleander, and digitalis are the most dangerous plant cardiotoxins.

Miscellaneous cardiovascular problems

I. Myocardial infarction

As an underdeveloped community transforms itself into a developed community and adopts the lifestyle of industrialized communities, coronary artery disease and myocardial infarction are likely to become common health problems. Myocardial infarction is also a common complication of heat stroke.

II. Cor pulmonale

In the tropics and subtropics, schistosomiasis, complicated by liver disease and portal hypertension, must be added to the differential diagnosis of cor pulmonale.
III. Myocardial fibrosis

The helminthic fluke infection, heterophyiasis, has been a rare cause of myocardial fibrosis in the Philippines.***
Chapter XI
Hepatobiliary complications

Introduction
I. Hepatobiliary disease in the tropics
II. Bantu siderosis
III. Hepatitis B and hepatocellular carcinoma
IV. Idiopathic cirrhosis
V. Indian childhood cirrhosis
VI. Portal fibrosis

Jaundice
I. Jaundice in the tropics

Ascites
I. Ascites

Hepatitis
I. Hepatitis

Hepatic granulomas and abscesses
I. Hepatic granulomas
II. Hepatic abscesses

Hepatic failure
I. Acute hepatic failure
II. Chronic liver failure / cirrhosis

Hepatomegaly
I. Hepatomegaly without significant splenomegaly
II. Hepatomegaly that may be associated with splenomegaly

Biliary tract and pancreatic complications
I. Cholelithiasis
II. Cholecystitis
III. Cholangitis
IV. Cholangiocarcinoma
V. Pancreatitis
Hepatobiliary complications

Introduction

I. Hepatobiliary disease in the tropics

Hepatobiliary disease is thought to be more prevalent in the tropics. Certainly, inhabitants of the tropics and subtropics are exposed to many more agents, particularly parasites, that are capable of infecting the hepatobiliary system than are inhabitants of temperate climates. For example, the liver fluke, Opisthorchis viverrini, infects perhaps 5.4 million inhabitants of southeast Asia, but rarely infects inhabitants of North America. Hepatitis B infection occurs world-wide, but is much more prevalent in China, Indonesia, the Philippines, Africa, and South America than in North America, northern Europe, and Australia.

There are many reasons for the increased prevalence of hepatobiliary diseases in the tropics and subtropics. Poor human waste disposal, poor sanitary conditions during food preparation, unclean water supplies, and over-crowding increase the chance for transmitting water-borne or fecal-borne hepatobiliary diseases. The cultural practice of consuming raw foods, such as fish and crustaceans, increases the chance for transmission of hepatobiliary diseases that cycle through the food chain. The growing number of immune-compromised patients and the millions of malnourished inhabitants of developing communities have an impaired ability to resist hepatobiliary infections and are at increased risk for developing unusual opportunistic infections.

The following hepatobiliary problems are more common in the tropics and subtropics:

1. Bantu siderosis.
2. Hepatitis B and hepatocellular carcinoma.
3. Idiopathic cirrhosis.
4. Indian childhood cirrhosis.
5. Infectious diseases with hepatobiliary complications, especially helminthic, protozoal, and viral diseases.
6. Portal fibrosis of the liver.
7. Veno-occlusive disease of the intrahepatic venules due to pyrrolyzidine alkaloid poisoning; these alkaloids are found in herbal teas or contaminated grains.
II. Bantu siderosis

Bantu siderosis is an acquired hemochromatosis resulting from cultural practices. The Bantu people of southern Africa consume beer brewed in cast-iron pots. Cast-iron pots are used in their general cooking as well. This results in an unusual dietary elemental iron burden, leading to an acquired hemochromatosis. The excess consumption of hepatotoxic alcohol products contributes to the damaging effects of iron deposition in the liver. Cirrhosis and its complications occur in the final stage of this disease.

III. Hepatitis B and hepatocellular carcinoma

Although hepatitis B is found worldwide, in even the most remote of populations, there is a difference in its epidemiology when comparing populations. In developed communities with a temperate climate, hepatitis B is primarily transmitted by blood products, contaminated needles, and exposure to contaminated blood. The disease reaches its peak prevalence in young adults and the chronic carrier rate is low. However, in developing communities, especially those located in southeastern Asia, Pacific islands, China, and sub-Saharan Africa, hepatitis B is primarily transmitted from mothers, who are chronic carriers of the hepatitis B antigen, to newborns at the time of birth. The disease reaches its peak prevalence in children and the chronic carrier rate is high, being greater than fifty percent in some communities. The prevalence of the chronic carrier state may be reduced when an inexpensive, efficacious hepatitis B vaccine is made widely available.

Hepatocellular carcinoma is a leading cause of premature death in communities with a high carrier rate of hepatitis B antigen, especially in sub-Saharan Africa, Asia, and the Far East. Those patients who develop chronic persistent or chronic active hepatitis B, especially if acquired in infancy, are at highest risk for developing hepatocellular carcinoma. The hepatitis B genome has been detected in the tumor cells of some cases. Although hepatocellular carcinoma has been associated with chronic hepatitis B infection, epidemiologic investigations are still in progress to determine if other factors might be present.

There may also be an association between hepatocellular carcinoma and aflatoxin poisoning. Aflatoxins are carcinogenic and may contaminate food, especially grain products, stored in a humid climate. Communities exposed to high levels of aflatoxin contamination coincidentally may have a higher incidence of hepatocellular carcinoma.
IV. Idiopathic cirrhosis

Based on autopsy studies, it has been determined that cirrhosis of the liver is more common in tropical climates than temperate climates, and many of these cases are considered "idiopathic". The development of hepatitis B identification techniques has led to the conclusion that most of the cases of idiopathic cirrhosis are the result of chronic hepatitis B infection.

V. Indian childhood cirrhosis

Indian childhood cirrhosis is possibly a familial disease that is most prevalent in the Indian subcontinent, notably in Sri Lanka and Burma. But it is also found in Malaysia, the Middle East, western Africa, and Central America. The highest incidence is found in the age group one to five years old. The disease has an insidious onset with the development of silent hepatomegaly. The affected child then develops an acute hepatitis with focal necrosis which evolves into a micronodular cirrhosis. The child may die within a year from liver failure. Corticosteroids and supportive measures are helpful in prolonging life and may prevent progression of the disease, but it is known that the disease may spontaneously arrest at any stage.

VI. Portal fibrosis

Portal fibrosis, without parenchymal liver disease or clinical manifestations, is found as an incidental finding on liver biopsy and autopsy in the tropics, especially in Africa. Its cause is unknown, but seems to be acquired locally rather than inherited. Since it has not been associated with hepatic insufficiency, no therapy is recommended.

Akee fruit, Blighia sapida, poisoning may be complicated by portal fibrosis, with a "pipestem" appearance of the portal veins noted on histologic sections; if the patient survives the acute poisoning that may be accompanied by vomiting, convulsions, shock syndrome, and coma.

VII. Veno-occlusive disease of the intrahepatic venules

The seeds of the ragwort plant, Senecio burchelli, may be found as a contaminant in wheat in Jamaica, Israel, Egypt, South Africa, and India. These seeds, as well as other parts of the plant, contain hepatotoxic pyrrolizidine alkaloids. The alkaloid has also been found in herbal teas. Ingestion of this alkaloid leads to a marked proliferation of the intima of small intrahepatic venules which results in the acute onset of
hepatomegaly and ascites. As the intoxication progresses, there is centrilobular thrombosis of the hepatic veins, necrosis of nearby parenchymal cells, and nonportal fibrosis of the liver leading to portal hypertension and hepatic failure. The intoxication is more prevalent in children and is usually fatal, but spontaneous recovery has been observed. The treatment is limited to supportive measures.

Seeds of the legume, Crotalaria fulva, may also cause veno-occlusive disease of the liver.

Jaundice

I. Jaundice in the tropics

See also hepatitis, cirrhosis, and cholangitis sections below.

In the tropics, one must consider hepatotoxic liver disease, hemolytic anemia, and physical obstruction of the biliary tract, usually by helminthic parasites, as etiologies for jaundice. Clinically, jaundice may be difficult to detect in patients with darkly pigmented skin, especially if they are exposed regularly to the sun and wind. These patients may also have "muddy" pigmentation of the sclera which interferes with the ability to detect icteric sclera.

Common hepatotoxins include:

1. Heavy metal poisoning: antimony and arsenic.
2. Mushroom poisoning.
3. Plant poisoning: seeds of Senecio burchelli (ragwort), Blighia sapida (akee), and Crotalaria fulva.

Jaundice may accompany hemolysis due to:

1. Babesiosis.
2. Favism: due to ingestion of Vicia faba (fava bean) by patients who are G-6-PD deficient.
3. G-6-PD deficiency: with a severe hemolytic crisis or during the neonatal period, associated with the use of 8-aminoquinolones, sulfones, sulfonamides, nitrofurans, analgesics, quinine, chloramphenicol, probenecid, vitamin K, and other drugs.
4. Malaria: with heavy parasite burdens.
5. Rift Valley fever.
7. Snake bite: venomous.
8. Thalassemia: with the complications of beta
   thalassemia major, such as severe hypochromic
   microcytic anemia, cholecystitis, and cirrhosis.
9. Yellow fever.

The differential diagnosis of obstructive jaundice
includes:

1. Amebiasis: rarely, jaundice may be a sign of a large
   amebic liver abscess.
2. Ascariasis.
3. Clonorchiasis.
4. Diphyllobothriasis.
5. Echinococcosis due to Echinococcus granulosus and
   Echinococcus multilocularis.
6. Fascioliasis.
7. Hepatocellular carcinoma.
8. Opisthorchiasis.

Ascites

I. Ascites

Ascites may complicate:

1. Capillariasis due to Capillaria philippinensis.
2. Chronic hepatitis B.
3. Clonorchiasis.
4. Fasciolopsiasis.
5. Filariasis.
6. Hepatocellular carcinoma.
8. Lassa fever.

Hepatitis

I. Hepatitis

See also Acute Hepatic Failure Section below

A variety of infections may be complicated by hepatitis:
1. Ascariasis.
2. Brucellosis: jaundice is uncommon.
3. Candidiasis: may be an opportunistic infection in the immune-compromised.
5. Cryptococcosis.
6. Epstein-Barr virus disease.
7. Genitourinary gonococcal disease: may be rarely complicated by perihepatitis.
10. Lymphogranuloma venereum.
11. Nongonococcal urethritis due to Chlamydia trachomatis: may be rarely complicated by perihepatitis.
12. Opisthorchiasis: may be complicated by allergic hepatitis.
13. Paratyphoid fever.
15. Q fever.
16. Relapsing fever.
17. Toxoplasmosis.
18. Typhoid fever.
19. Venereal syphilis.
20. Viral hepatitis (A and B).

**Hepatic granulomas and abscesses**

I. Hepatic granulomas

A granulomatous reaction may accompany:

1. Enterobiasis (pinworm): may be a rare complication.
2. Histoplasmosis.
3. Q fever.
4. Schistosomiasis.

II. Hepatic abscesses

Hepatic abscesses may complicate:

1. Amebiasis.
2. Ascariasis.
3. Echinococcosis due to Echinococcus granulosus.
Hepatic failure

I. Acute hepatic failure

Hepatotoxins and viral infections may cause acute hepatic failure.

1. Carbon tetrachloride poisoning.
2. Ebola and Marburg virus disease.
4. Malaria due to Plasmodium falciparum.
5. Mushroom poisoning.
6. Rift Valley fever.
7. Rocky Mountain spotted fever.
8. Senecio burchelli (ragwort) poisoning.
9. Yellow fever.

II. Chronic liver failure/cirrhosis

Chronic liver failure and cirrhosis may complicate:

1. Bantu siderosis: see introduction to hepatobiliary complications section above.
2. Beta thalassemia major.
3. Chronic active hepatitis B.
5. Schistosomiasis: especially when the disease co-exists with hepatitis B infection.
6. Visceral leishmaniasis (Kala-azar).

Hepatomegaly

I. Hepatomegaly without significant splenomegaly

Hepatomegaly without significant splenomegaly may be present in a variety of diseases. When the hepatomegaly is acute in onset, as with rickettsial and viral infections, the liver margin is usually tender and the patient may complain of right shoulder pain. When the hepatomegaly is insidious in onset, as with most helminthic infections and malignancy, then the liver margin is often nontender and the patient might complain only of fullness or heaviness in the right upper quadrant.

1. Amebiasis.
2. Ascariasis.
5. Carbon tetrachloride poisoning.
6. Clonorchiasis.
7. Dengue hemorrhagic fever.
8. Echinococcasis due to Echinococcus granulosus and Echinococcus multilocularis.
10. Filarial hypereosinophilia: hepatomegaly primarily noted in the pediatric population.
13. Hepatocellular carcinoma.
15. Louse-borne typhus fever (epidemic typhus).
17. Murine typhus.
18. Opisthorchiasis.
19. Plant poisoning: Senecio burchelli (ragwort) and fava bean; Argemone mexicana (Mexican poppy), which grows in India, Mauritius, Fiji, and South Africa, and may contaminate mustard oil.
20. Rocky Mountain spotted fever.
22. Sickle cell anemia: in an advanced stage.
23. Toxocariasis.
24. Viral hepatitis.

II. Hepatomegaly that may be associated with splenomegaly

The following diseases may be complicated by hepatomegaly with splenomegaly:

1. American trypanosomiasis (Chagas' disease).
2. Babesiosis.
4. Histoplasmosis.
5. Kyasanur Forest disease.
7. Malaria.
8. Melioidosis.
11. Plague.
13. Relapsing fever.
16. Thalassemia: beta thalassemia major with severe disease usually by age two.
17. Toxoplasmosis.
18. Tularemia.
19. Typhoid fever.
20. Visceral leishmaniasis (Kala-azar).
21. West Nile fever.
22. Zinc deficiency.

**Biliary and pancreatic tract complications**

I. Cholelithiasis

Cholelithiasis may be associated with:

1. Clonorchiasis.
2. Opisthorchiasis.
3. Sickle cell anemia.
4. Thalassemia: beta thalassemia major.

II. Cholecystitis

Cholecystitis may complicate the following diseases:

1. Acute diarrhea due to Campylobacter.
2. Ascariasis.
4. Echinococcosis due to Echinococcus granulosus.
5. Sickle cell anemia.
6. Thalassemia: beta thalassemia major.

III. Cholangitis

Cholangitis may complicate the following diseases:

1. Ascariasis.
2. Clonorchiasis.
3. Opisthorchiasis.
4. Sickle cell anemia.
5. Taeniasis.

IV. Cholangiocarcinoma

Clonorchiasis and opisthorchiasis have been associated with cholangiocarcinoma, probably as the result of recurrent or chronic cholangitis.
V. Pancreatitis

Pancreatitis may be a complication of the following diseases:

1. Ascariasis.
2. Clonorchiasis.
4. Scorpion sting: seen in children as a complication of the sting of certain species of scorpions found in Trinidad.***
Chapter XII
Gastrointestinal complications

Introduction
I. Contrast between tropical and temperate zones

Dysphagia
I. Dysphagia in the tropics

Vomiting
I. Introduction
II. Vomiting and flu-like symptoms
III. Vomiting and respiratory tract symptoms
IV. Vomiting and acute abdomen
V. Vomiting and hepatobiliary symptoms
VI. Vomiting and neurologic symptoms

Gastroenteritis
I. Introduction
II. Vomiting and diarrhea predominate
III. Gastroenteritis and symptoms of systemic toxicity
IV. Gastroenteritis and flu-like symptoms
V. Gastroenteritis and multiple system symptoms

Diarrhea
I. Introduction
II. Dysentery
III. Bloody diarrhea
IV. Diarrhea predominates
V. Diarrhea and flu-like symptoms
VI. Diarrhea and acute abdomen
VII. Diarrhea and hepatobiliary symptoms
VIII. Diarrhea and pulmonary symptoms
IX. Diarrhea and neurologic symptoms
X. Diarrhea and rheumatoid symptoms

Acute abdomen
I. Introduction
II. Appendicitis
III. Peritonitis
IV. Intestinal obstruction
V. Megacolon

(continued next page)
Malabsorption
I. Alpha heavy chain disease
II. Calcific chronic pancreatitis
III. Hypolactasia and lactose intolerance
IV. Malnutrition and malabsorption
V. Parasitic infections and malabsorption
VI. Protein-losing enteropathy
VII. Tropical enteropathy
VIII. Tropical sprue

Proctologic problems
I. Pruritus ani
II. Proctitis
III. Rectal prolapse
Gastrointestinal complications

Introduction

I. Contrast between tropical and temperate zones

There are many gastrointestinal diseases that occur more frequently in the tropics and subtropics than in the temperate zones of the world. Most of these have infectious etiologies. Malnutrition, poor sanitary conditions, untreated water supplies, and substandard housing found in many developing communities contribute to the higher prevalence of gastrointestinal diseases. Travelers are frequently infected as well.

Often an inhabitant of the tropics will present with a gastrointestinal problem and is found to have multiple infections, and possibly, malnutrition. The treating physician must be aware of this common presentation and realize that the diagnosis and treatment of gastrointestinal problems in the tropics may be quite complicated. The patient may be asymptomatic and tolerant of parasitic infestations of the gastrointestinal tract, and his current symptoms are not caused by the presence of the parasite.

The following gastrointestinal problems are more common in the tropics:

1. Diarrhea: primarily due to infections by bacteria, rotavirus, and parasites.
2. Duodenal ulcer and pyloric stenosis as a complication: mostly in west and east Africa, India, and Afghanistan.
4. Intestinal obstruction.
5. Malabsorption:
   a. Alpha-chain disease.
   b. Calcific chronic pancreatitis.
   c. Hypolactasia.
   d. Malnutrition.
   e. Parasitic infections.
   f. Tropical sprue.
   g. Tuberculosis.
Dysphagia

I. Dysphagia in the tropics

Dysphagia in the tropics is more frequently caused by infectious diseases and by carcinoma (in eastern Asia) than in the temperate zones. Infectious diseases that should be included in a differential diagnosis are:

1. Actinomycosis.
2. American trypanosomiasis (Chagas' disease).
3. Entomophthoromycosis due to Conidiobolus.
4. Tetanus.
5. Trichinosis.

Dysphagia as a symptom of esophageal stricture due to ingestion of caustics is more common in the tropics.

Vomiting

I. Introduction

Nausea and vomiting are common symptoms in developing countries as a result of infectious diseases, some not normally encountered in temperate, developed countries. Vomiting is rarely an isolated finding, but may be a symptom of many bacterial, helminthic, and viral tropical diseases. Vomiting associated with diarrhea will be discussed below under the headings of gastroenteritis and diarrhea.

II. Vomiting and flu-like symptoms

See also Gastroenteritis section.

See also Meningoencephalitis and Encephalitis sections.

Vomiting associated with a flu-like illness is usually due to a viral infection. Fever, headache, and myalgia are common symptoms.

1. Arthralgia is also present in:
   a. Chikungunya fever.
   b. Dengue fever.
   c. Group C virus fever.
   d. O'nyong nyong fever.
   e. Rift Valley fever.
   f. Viral hepatitis.
2. Hemorrhagic phenomena are often present in:
   a. Crimean-Congo hemorrhagic fever.
   b. Dengue hemorrhagic fever and dengue shock syndrome.
   c. Ebola and Marburg virus disease.
   d. Hemorrhagic fever with renal syndrome.
   e. Lassa fever.
   f. Rift Valley fever.
   g. Yellow fever.

3. Neurologic findings are prominent in:
   a. Australian encephalitis (Murray Valley encephalitis).
   c. Poliomyelitis: severe muscle spasms, stiffness in neck and back, aseptic meningitis, and flaccid paralysis.
   d. Rift Valley fever: meningoencephalitis.
   e. Venezuelan equine encephalitis.
   f. West Nile fever: meningoencephalitis.

4. Non-specific viral syndrome:
   a. Sandfly fever.

III. Vomiting and respiratory tract symptoms

Vomiting may be present in bacterial or viral illnesses irritating the pharynx or lower respiratory tract.

1. Associated with pharyngitis:
   a. Chikungunya fever.
   b. Diphtheria: marked pharyngeal exudate, purulent rhinitis, laryngitis, and airway obstruction.
   c. Lassa fever.
   d. Venezuelan equine encephalitis.
   e. Yersiniosis.

2. Associated with pulmonary symptoms:
   c. Pertussis: rhinitis, paroxysmal cough with inspiratory whoop, petechial facial rash and conjunctival hemorrhage due to straining, aspiration pneumonia, and possible apnea in infants.

163
IV. Vomiting and acute abdomen

Acute abdomen is often accompanied by vomiting. There are tropical diseases, primarily helminthic, that may cause or mimic acute abdomen as a result of inflammation, obstruction, invasion, or perforation of the intestines.

1. Abdominal angiostrongyliasis: appendicitis with right lower quadrant mass, intestinal perforation, and peritonitis.
2. Anisakiasis: hematemesis, appendicitis, regional enteritis, and peritonitis with abdominal abscess formation.
3. Ascariasis: small bowel obstruction, appendicitis, cholecystitis with obstructive jaundice, pancreatitis, and peritonitis.
5. Fasciolopsiasis: intestinal obstruction.
6. Hemorrhagic fever with renal syndrome: may mimic acute abdomen.
7. Malaria: may mimic appendicitis.

V. Vomiting and hepatobiliary symptoms

Hepatobiliary disease may cause nausea and vomiting. Tropical diseases that have hepatobiliary involvement accompanied by vomiting are:

1. Ascariasis: cholecystitis with obstructive jaundice.
2. Babesiosis: hepatosplenomegaly, jaundice, and hemolytic anemia.
5. Fascioliasis: hepatomegaly and jaundice.

VI. Vomiting and neurologic symptoms

Vomiting may be associated with neurologic diseases that cause meningeal irritation, cerebral edema, or increased intracranial pressure.

1. Angiostrongylia: blindness, cranial neuropathy, and meningoencephalitis.
2. Australian encephalitis (Murray Valley encephalitis).
Gastroenteritis

I. Introduction

Gastroenteritis is a very common medical problem in the tropics and subtropics. Gastroenteritis is usually acute in onset and associated with fever, abdominal cramps, diarrhea, nausea, and vomiting. Travelers are quite prone to developing gastroenteritis or traveler's diarrhea. Due to poor sanitary conditions and lack of clean water found in many parts of the developing world, the indigenous population is frequently affected as well. Since proper oral rehydration is often not initiated and primary health care may not be available, dehydration is a serious complication in the pediatric population of the tropics. Dehydration, and its metabolic complications, is a major cause of death among children of underdeveloped communities.

II. Vomiting and diarrhea predominate

Illnesses in which vomiting and diarrhea are the predominant symptoms are usually caused by bacteria and viruses, or provoked by gastrointestinal toxins.

1. Acute diarrhea due to Campylobacter.
2. Cholera.
3. Clostridium perfringens food poisoning.
4. Epidemic viral gastroenteropathy (Norwalk-like virus).
5. Gastroenteritis caused by enterotoxigenic Escherichia coli.
6. Heavy metal food poisoning: cadmium or zinc may leach into food from metal cooking vessels or...
food storage containers.
7. Rotaviral enteritis: rapid development of dehydration is common in children.
8. Salmonellosis.
10. Shigellosis.
11. Staphylococcal food poisoning: dehydration is a common complication in children.

III. Gastroenteritis and symptoms of systemic toxicity

Gastroenteritis that presents with sudden, severe systemic toxicity, accompanied by neurologic findings, respiratory failure, and shock syndrome, is usually due to ingestion of toxins, either naturally occurring or man-made, such as pesticides. Gastroenteritis with marked systemic toxicity may develop after the ingestion of the following natural reservoirs for toxins:

2. Elasmobranch fish poisoning (sharks and rays): paresthesia, ataxia, visual disturbances, and coma.
3. Mushroom poisoning: convulsions, muscle spasms, psychiatric disturbances, ataxia, and coma.
4. Plant poisoning: by mistaken identification, food contamination, excessive ingestion of certain plants during droughts, and overdose of plants used for medicinal or illicit purposes. Examples include- Nicotiana species (tobacco), solanum species (potatoe skins not covered by earth and potatoe sprouts), Melia azedarach (alkaloids of chinaberry and syringa), Aconitum napellus (roots and seeds of monkshood), Lantana camara (wild sage), rhododendron species, euphorbia species (latex euphorbon, manchineel, devil tree, Barbados nut, and castor bean), legumin species (rosary pea, laburnum, and wisteria), Colchicum autumnale (autumn crocus), Phytolacca americana (pokeweed), umbellifera species (water hemlock and poison parsley), and oleander plants.
5. Tetraodon fish poisoning (blowfish, puffers, ocean sunfish, porcupine fish): paresthesia, flaccid paralysis, and convulsions.
IV. Gastroenteritis and flu-like symptoms

Gastroenteritis that is accompanied by flu-like symptoms, such as fever, chills, headache, and myalgia, is usually due to viral infections.

1. Neurologic symptoms are also present in:
   a. Argentine and Bolivian hemorrhagic fever: encephalopathy.
   c. Kyasanur Forest disease: meningoencephalitis.
   d. Lassa fever: encephalopathy.
   e. Oropouche fever: meningoencephalitis.
   f. West Nile fever: meningoencephalitis.

2. Hemorrhagic phenomena are also found with:
   a. Argentine and Bolivian hemorrhagic fever.
   b. Ebola and Marburg virus disease.
   c. Kyasanur Forest fever.
   d. Lassa fever.

3. Arthralgia accompanies:
   a. Kyasanur Forest fever.
   b. Mayaro fever: arthritis.
   c. Oropouche fever.
   d. West Nile fever.

4. Pulmonary involvement is common in:
   a. Kyasanur Forest fever.
   b. Tularemia: hilar adenopathy, pleuritic pain, pleural effusion, and pneumonia.

V. Gastroenteritis and multiple system symptoms

Gastroenteritis may be associated with other diseases, which are not listed above, that affect multiple systems. These diseases are primarily bacterial, helminthic, and protozoal.

1. Anthrax: edematous papules that develop into pruritic vesicles and finally painless ulcers at the site of infection, bloody diarrhea, shock syndrome, and metastatic bacteremia resulting in meningitis and pneumonia.

2. Echinococciosis due to Echinococcus granulosus: hepatomegaly, urticaria, asthma, convulsions, paralysis, and metastatic cystic lesions and abscesses.

3. Heterophylasis: intestinal granulomas, myocardial fibrosis, convulsions, and eosinophilia.
4. Malaria: severe headache and myalgia, urticaria, hepatosplenomegaly, hemolytic anemia, renal failure, and coma.

5. Meningococcal meningitis: pharyngitis, hemorrhagic phenomena, shock syndrome, and metastatic bacteremia with septic arthritis.

6. Schistosomiasis: pruritus and urticaria, maculopapular rash, vesicular rash, headache, hepatosplenomegaly and portal hypertension, generalized lymphadenopathy, eosinophilia, bloody diarrhea, protein-losing enteropathy, intestinal polyposis, arthritis, secondary salmonellosis infection, and renal, urologic, pulmonary, and neurologic complications.

7. Trichinosis: myalgia, eosinophilia, conjunctivitis, photophobia, headache, petechiae and purpura, maculopapular rash, myocarditis, dysphagia, myositis, myelitis, meningoencephalitis, paralysis, delirium, psychosis, coma, hemoptysis, and pneumonitis.

**Diarrhea**

I. Introduction

Diarrhea is a major cause of morbidity and mortality in developing communities. The mortality rate from dehydration is high in the pediatric population, especially where hospitalization is not available and proper oral rehydration is not widely practiced.

Diarrhea contributes to the development of malnutrition, which in turn, makes the patient more likely to have a prolonged illness or recurrent infections. Diarrhea is also a sign of advanced malnutrition. There are no ideal prophylactic medications or vaccinations available to prevent diarrhea, in part, due to our current level of pharmaceutical technology, and in part, due to the diverse number of pathogens that may cause diarrhea. Pathogens causing diarrhea are increasingly resistant to antibiotics, complicating treatment plans once a diagnosis is made.

Bacterial and viral diarrheas generally last less than two weeks. Diarrhea lasting more than two weeks is usually due to helminthic or protozoal infections, malabsorption, inflammatory bowel disease, severe malnutrition, or post-infection colonic dysfunction.
Diarrhea complicated by hemorrhage with anemia, or bowel perforation with peritonitis, is more common in the tropics.

If the electrolyte balance, glucose content, and osmolality of a solution are ideal, diarrhea can be treated by oral rehydration. Oral rehydration is an effective treatment for diarrhea, even if the quantity of stool production is high, as in cholera. This is because the co-transport of sodium and glucose, which take water with them across the gut wall, is preserved, even in severe infections. The oral rehydration solution with glucose and electrolytes recommended by the World Health Organization is:

1. One liter of water.
2. Glucose, 20 gm.
3. Sodium chloride, 3.5 gm (90 mEq/L Na+).
4. Potassium chloride, 1.5 gm (20 mEq/L K+).
5. Sodium bicarbonate, 2.5 gm (30 mEq/L HCO3-).
6. Osmolarity, 330 mOsm/L.

II. Dysentery

Fever, general toxicity, cramping abdominal pain, and diarrhea with blood, mucus, and pus are present in dysentery. All of the organisms capable of producing dysentery in the tropics may also cause milder illnesses with watery diarrhea or loose stools.

1. Acute diarrhea due to Campylobacter.
2. Amebiasis.
3. Balantidiasis: mild diarrhea possible or ulcerative colitis-like illness.
4. Diarrhea caused by enterotoxigenic Escherichia coli: watery diarrhea and marked dehydration possible.
5. Enterobiasis: rare complication.
7. Shigellosis: watery diarrhea possible.

III. Bloody diarrhea

See also Hemorrhagic Phenomena section.

In addition to the organisms causing dysentery, the following diseases may present with bloody diarrhea, but without mucus or pus.
1. Anthrax.
2. Clostridium perfringens food poisoning.
5. Hookworm disease.
6. Intussusception: more common in the tropics, associated with intestinal obstruction and vomiting.
8. Niacin deficiency (pellagra).
9. Plant poisoning: Hura polyandra (devil tree), Jatropha curcas (Barbados nut), Ricinus communis (castor bean), Colchicum autumnalis (autumn crocus), and oleander plants.
10. Schistosomiasis.

IV. Diarrhea predominates

See also Gastroenteritis section.

Diarrhea is a predominant symptom in the following:

1. Acute diarrhea due to Campylobacter.
2. Balantidiasis.
3. Capillariasis due to Capillaria philippinensis: vomiting, marked borborygmus, pleural transudate, ascites, protein-losing enteropathy, and malabsorption syndrome.
6. Diarrhea caused by enterotoxigenic Escherichia coli.
7. Echinostomiasis: diarrhea possible.
10. Kwashiorkor.
12. Rotaviral enteritis: vomiting and rapid onset of dehydration in children.
15. Shigellosis.
V. Diarrhea and flu-like symptoms

Diarrhea may be associated with influenza-like symptoms, such as fever, headache, myalgia, and pharyngitis.

2. Paratyphoid fever.
3. Schistosomiasis.
4. Typhoid fever: often the patient complains of constipation and may have ileus secondary to inflammation of the bowel.
5. Yersiniosis.

VI. Diarrhea and acute abdomen

Acute abdomen may be the predominant presentation complicating diarrhea in the following:

1. Amebiasis: peritonitis, ulcerative colitis-like illness, and liver abscesses.
2. Diphyllobothriasis: intestinal and biliary obstruction.
3. Fasciolopsiasis: intestinal obstruction.
4. Shigellosis: appendicitis and peritonitis.
5. Taeniasis: intestinal obstruction and appendicitis.
6. Typhoid fever: intestinal perforation as late complication.
7. Yersiniosis: enterocolitis, mesenteric lymphadenitis, and may mimic appendicitis.

VII. Diarrhea and hepatobiliary symptoms

Many diarrheal diseases may be complicated by hepatobiliary symptoms.

1. Amebiasis: liver abscesses.
2. Clonorchiasis: ascites, hepatomegaly, cirrhosis, pancreatitis, cholangitis, and a higher risk for cholangiocarcinoma.
3. Fasciolopsiasis: ascites.
5. Opisthorchiasis: hepatomegaly, jaundice, cholangitis, and increased risk for cholangiocarcinoma.
7. Visceral leishmaniasis (Kala-azar): hepatosplenomegaly.
VIII. Diarrhea and pulmonary symptoms

Diarrhea and pulmonary symptoms may be present in:

1. Amebiasis: pleuritic pain, productive cough, hemoptysis, dyspnea, right pleural effusion, and right-sided pulmonary parenchymal densities.
2. Anthrax: nonproductive cough, stridor, pneumonia, pleural effusion, and hilar adenopathy.
4. Paragonimiasis: productive cough, pleuritic pain, dyspnea, chronic bronchitis, bronchiectasis, pleural effusions, and pulmonary infiltrates, granulomas, cysts, and fibrosis.
5. Schistosomiasis: pneumonitis.
7. Tuberculosis: hemoptysis, productive cough, and pulmonary infiltrates and cavitary lesions.

IX. Diarrhea and neurologic symptoms

Neurologic symptoms may complicate:

1. Acute diarrhea due to Campylobacter: convulsions and meningitis.
4. Niacin deficiency: peripheral neuritis, dementia, and psychosis.
5. Paragonimiasis: convulsions and paralysis.
7. Salmonellosis: meningitis.
8. Schistosomiasis: convulsions, scotomas, cranial neuropathy, transverse myelitis, and paralysis.
10. Strongyloidiasis: meningitis in immune-compromised patients.
11. Tuberculosis: meningitis.
12. Typhoid fever: delirium, coma, convulsions, meningitis, encephalomyelitis, transverse myelitis, spastic paralysis, peripheral neuritis, cranial neuropathy, and Guillain-Barre' syndrome.

X. Diarrhea and rheumatoid symptoms

Several pathogens causing diarrhea are associated with post-infection onset of rheumatoid symptoms or metastatic bacteremia resulting in arthritis.

1. Acute diarrhea due to Campylobacter: arthritis.
2. Salmonellosis: arthritis.
5. Tuberculosis: arthritis.

**Acute abdomen**

I. Cross references

Vomiting and Acute Abdomen Section.
Diarrhea and Acute Abdomen Section.

II. Appendicitis

Helminthpiel intestinal infections, shigella, and malaria may cause or mimic appendicitis.

1. Abdominal angiostrongyliasis: mimic.
3. Ascariasis.
4. Enterobiasis (pinworm).
5. Malaria: mimic.
7. Taeniasis.

III. Peritonitis

Several tropical infectious diseases may present as a generalized peritonitis.

1. Abdominal angiostrongyliasis.
3. Ascariasis: intestinal perforation.
5. Tuberculosis: abdominal tuberculosis is more common in the tropics and is associated with fever, nocturnal sweating, malabsorption syndrome, ascites, intestinal obstruction, pulmonary involvement, lymphadenitis, genitourinary involvement, osteomyelitis, arthritis, pericarditis, meningitis, and adrenocortical insufficiency.
6. Strongyloidiasis (threadworm): intestinal perforation and ulcerative colitis-like illness; more likely to occur in immune-compromised patients, associated with ileus, pneumonia, meningitis, and shock syndrome.
7. Typhoid fever: intestinal hemorrhage and intestinal perforation.
IV. Intestinal obstruction

The following causes of intestinal obstruction are more common in the tropics:

1. American trypanosomiasis (Chagas' disease): may be complicated by volvulus.
2. Ascariasis: may be complicated by volvulus or intussusception.
3. Diphyllobothriasis.
4. Fasciolopsiasis.
5. Intussusception: more common in the tropics with vomiting, abdominal pain, and hematochizia.
6. Taeniasis.
7. Tuberculosis.
8. Volvulus: more common in the tropics with vomiting, obstipation, abdominal pain and distention, and peritonitis with gangrenous changes in the bowel.

V. Megacolon

Toxic megacolon is always a possible complication of severe peritonitis and ulcerative colitis. But in the tropics, American trypanosomiasis (Chagas' disease) may cause megaviscera possibly by inducing an autoimmune degeneration of the intramural autonomic ganglia in the smooth muscle of the esophagus, stomach, and colon. Infrequently, shigellosis may be complicated by toxic megacolon.

Malabsorption

I. Alpha heavy chain disease

Alpha heavy chain disease is a malignant production of excessive alpha heavy chain fragments of gamma globulin. This disease primarily affects young adults in north Africa, the Middle East, and Pakistan. Immunoelectrophoresis is required to detect this monoclonal gammopathy. The disease has nonspecific signs and symptoms of nausea, abdominal pain, malabsorption, and edema.

II. Calcific chronic pancreatitis

Calcific chronic pancreatitis reduces the production of pancreatic enzymes, and is more common in Africa and the Indian subcontinent than in temperate zones. This disease is associated with abdominal pain, steatorrhea, weight loss, and secondary diabetes mellitus.
III. Hypolactasia and lactose intolerance

Diarrhea developing as a result of lactose intolerance is more common in South America, Africa, India, and Asia. In these populations, there is a genetically-controlled decrease in the production of the enzyme, lactase. Nausea, abdominal pain and distention, and diarrhea occur after the ingestion of dairy products.

IV. Malnutrition and malabsorption

Malnutrition is accompanied by infections, decreased production of stomach acid and digestive enzymes, altered gut microflora, and dysfunctional gut motility. These contribute to the development of malabsorption.

V. Parasitic infections and malabsorption

A few tropical parasitic diseases are associated with malabsorption syndrome.

1. Ascariasis: malabsorption may occur with heavy infestation.
2. American trypanosomiasis (Chagas' disease):
   Malabsorption is a possible complication of the altered gut motility associated with megacolon.
4. Cryptosporidiosis: may cause malabsorption in immune-compromised patients.
5. Giardiasis: chronic diarrhea and steatorrhea.

VI. Protein-losing enteropathy

The following infectious diseases may produce protein-losing enteropathy:

1. Capillarisis due to Capillaria philippinensis.
2. Filariasis.
3. Hookworm disease.
4. Schistosomiasis.
5. Tuberculosis.

VII. Tropical enteropathy

Tropical enteropathy is a self-limited persistence of gastrointestinal dysfunction and malabsorption following recovery from an intestinal infection, as the gut restores its normal microflora and mucosa. It is a common cause of chronic diarrhea following an episode of traveler's diarrhea. Chronic
giardiasis may mimic tropical enteropathy.

VIII. Tropical sprue

Tropical sprue has no known etiology, but an infectious organism or toxin is suspected since epidemics are known to occur and tropical residents and visitors are equally affected. This notion is supported by the fact that antibiotics, such as sulfonamides and tetracycline, are effective in inducing a remission of symptoms. Jejunal biopsy is nonspecific, but shows mucosal atrophy. Tropical sprue is associated with: anorexia, abdominal distention, diarrhea, and megaloblastic anemia and other sequelae of nutritional deficiencies.

Proctologic problems

I. Pruritus ani

The differential diagnosis of pruritus ani in the tropics includes:

1. Enterobiasis.
2. Hymenolepiasis.
4. Taeniasis: occurs with passage of proglottids in the stool.

II. Proctitis

Several venereal diseases may be complicated by proctitis:

1. Genitourinary gonococcal disease.
2. Lymphogranuloma venereum.
3. Nongonococcal urethritis.

III. Rectal prolapse

Rectal prolapse is a possible complication of trichuriasis (whipworm) infestation and shigellosis, especially in infants.
Chapter XIII
Genitourinary complications

Introduction
I. Genitourinary complications in the tropics

Genitourinary infections
I. Urethritis
II. Cystitis
III. Pyelonephritis

Glomerulonephropathy
I. Acute glomerulonephritis
II. Nephrotic syndrome
III. Diseases of the renal vasculature

Renal failure
I. Acute renal failure
II. Chronic renal failure

External female and male genitalia
I. External genitalia

Female reproductive system
I. Vaginitis
II. Cervicitis
III. Pelvic inflammatory disease
IV. Toxemia of pregnancy

Male reproductive system
I. Prostatitis
II. Epididymitis
III. Hydrocele
IV. Orchitis
V. Other testicular complications
Genitourinary complications

Introduction

I. Genitourinary complications in the tropics

One recurring theme is that there is a general sparcity of primary health care services and diagnostic facilities in many areas of the tropics and subtropics. This increases the chance for easily treated diseases going untreated or there being a delay in the initiation of treatment. Therefore, the clinician is more likely to see complications of untreated genitourinary tract diseases. There are difficulties in providing long term care for patients with chronic renal failure. Renal dialysis is simply not available in most communities to salvage patients with acute renal failure.

Sexually transmitted diseases, their complications and congenital transmission, occur more frequently in the tropics. This is partly due to the promiscuous attitudes of some cultures and the psychosocial disruption caused by the transition of agrarian, rural communities into industrialized, suburbanized communities. The other reason for a higher prevalence of sexually transmitted diseases is that the demographic constitution of many tropical communities is oriented to a younger, more sexually active population, which often has a lower standard of living, higher unemployment, and poorer health care. The indiscriminant use of antibiotics, purchased over-the-counter, has increased antibiotic resistance of organisms, such as gonorrhea, complicating treatment and intensifying the prevalence of the inadequately treated disease.

Certain sexually transmitted diseases, namely chancroid, lymphogranuloma venereum, and granuloma inguinale, are limited to the tropics and subtropics, except when seen in travelers returning from these areas. Gonorrhea occurs more frequently and herpes genitalis less frequently in developing communities than in developed communities. Acquired immune deficiency syndrome is becoming a growing problem in the tropics, spread through poorly screened, contaminated blood supplies, contaminated needles, homosexual practices, sexual promiscuity and prostitution, vertical congenital transmission, and other as yet undetermined means of transmission.

Acute poststreptococcal glomerulonephritis occurs in epidemics in the Caribbean islands and Africa. The route of entry for "nephritogenic" strains of streptococci is more often
through the skin secondary to untreated impetigo in the tropics than through the pharynx. Additional differences in the patterns of renal disease will be discussed below in the Glomerulonephropathy Section and Renal Failure Section.

Squamous cell carcinoma of the bladder is a very common tumor in areas of the tropics where schistosomiasis due to Schistosoma haematobium is prevalent. Middle-aged men working in Schistosoma infested waters, such as irrigation canals in Egypt, are at highest risk. No direct carcinogenic effect by Schistosoma has been demonstrated, but it is postulated that the chronic bladder irritation, urinary stasis, and recurrent bacterial infections associated with schistosomiasis may facilitate the transition of bladder metaplasia to squamous cell carcinoma in the presence of carcinogens.

Bladder stones are more common among the malnourished children of the tropics. Travelers to the tropics who fail to maintain proper hydration during travel and in the tropical climate are at higher risk for developing renal lithiasis.

Clinicians must be prepared to expand their differential diagnosis of genitourinary diseases to include mycobacterial, helminthic, and protozoal infections, which are all more prevalent in the tropics.

Genitourinary infections

I. Urethritis

The differential diagnosis should include:

1. Genitourinary gonococcal disease.
2. Inclusion conjunctivitis due to Chlamydia trachomatis: may be associated with Reiter's syndrome.
3. Lymphogranuloma venereum: may present initially as a nonspecific urethritis.
4. Nonspecific urethritis due to Chlamydia: may be associated with Reiter's syndrome.
5. Salmonellosis: may be followed by Reiter's syndrome.
7. Shigellosis: may be followed by Reiter's syndrome.
8. Trichomoniasis.
9. Yersiniosis: may be followed by Reiter's syndrome.
II. Cystitis

Additional considerations for the etiology of cystitis include:

1. Acute diarrhea due to Campylobacter.
2. Brucellosis: may be complicated by granulomatous cystitis.
3. Candidiasis: bladder thrush as an extension of vaginitis is more common in the humid, warm tropics and in immune-compromised or malnourished patients.
5. Schistosomiasis: may be complicated by ulcerative cystitis and superinfection by Salmonella; vesiculo-intestinal fistula may develop; may be associated with an increased risk for developing squamous cell carcinoma of the bladder with chronic infection.
6. Tuberculosis: tuberculous cystitis is more frequently encountered in developing communities.
7. Vitamin A deficiency: may mimic cystitis when complicated by pyuria.

III. Pyelonephritis

Pyelonephritis may be a complication of:

1. Brucellosis: may be complicated by granulomatous nephritis which may be confused with renal tuberculosis.
2. Candidiasis: with renal abscess formation possible in immune-compromised or malnourished patients.
3. Melioidosis: renal abscess formation is possible with septicemia.
5. Typhoid fever.

Glomerulonephropathy

I. Acute glomerulonephritis

See also Genitourinary Infections Section above and the Hemorrhagic Phenomena Section of the Hematologic Complications Chapter for additional differential diagnosis of hematuria.
The differential diagnosis of acute glomerulonephritis in the tropics should include the following diseases:

1. Adenoviral disease.
2. Enteroviral disease: including echovirus and coxsackievirus.
3. Epstein-Barr virus disease.
4. Hepatitis-B: rare complication.
5. Malaria: rare complication of Plasmodium falciparum infection.
6. Meningococcal septicemia: more common across central Africa.
7. Mumps: may appear in the recovery phase of a mumps infection.
8. Pneumococcal septicemia: more common where adequate primary care is not available.
11. Tuberculosis: may be complicated by caseating granulomatous nephritis with cyst formation and calcifications.
12. Typhoid fever: rarely complicated by immune complex mediated glomerulonephritis.

II. Nephrotic syndrome

A distinctive lesion associated with nephrotic syndrome in West African children has been called "tropical nephropathy." In contrast to the minimal change glomerular lesions most often noted in renal biopsies of children from temperate climates, the children with tropical nephropathy have structural glomerular lesions with an irregular thickening of the glomerular capillary walls that appear twisted and double-outlined. The basement membrane of these glomeruli are studded with lacunae. Although corticosteroids are prescribed, there is no effective treatment known to date. Chronic renal failure is a common complication of tropical nephropathy.

Nephrotic syndrome is more common among young adults in many areas of the tropics compared to temperate climates, however these reports have been regional and do not reflect the prevalence in all areas. Renal biopsy reveals that the majority of these cases are the result of proliferative forms of glomerulonephritis and immune complex deposition. The diseases listed below have been associated with immune complex disease and nephrotic syndrome:
1. Hepatitis-B.
2. Leprosy.
3. Loiasis: chronic glomerulonephritis or membranous glomerulonephropathy are rare complications.
4. Malaria due to Plasmodium malariae.
5. Schistosomiasis: more likely with heavy worm burdens and with the development of hepatobiliary complications.
8. Visceral leishmaniasis (kala-azar).

III. Disease of the renal vasculature

Hemoglobinopathies have been implicated in causing disease of the renal vasculature. Sickling disorders and thalassemias are more common in many parts of the tropics. These diseases may be complicated by pseudo-filling defects of the renal pelvis, painless hematuria and proteinuria, difficulties in concentrating urine, nephrotic syndrome, distal tubular renal acidosis, and chronic renal failure. The pathophysiologic findings are abnormalities of the renal microvasculature and alterations in perfusion of the renal medulla secondary to the sickling process and microinfarctions.

Renal failure

I. Acute renal failure

See also Shock Syndrome Section of the Cardiovascular Complication Section since shock syndrome may be complicated by acute renal failure.

Acute renal failure is a significant health problem in the tropics due to the late presentation for treatment of patients with severe trauma, severe dehydration, and sepsis, the higher incidence of protozoal and viral diseases that may be complicated by renal failure, and the relative lack of tertiary health care facilities to provide renal dialysis for medical support of these patients. The following diseases may be complicated by acute renal failure:

2. Babesiosis: associated with hemoglobinuria and severe hemolytic anemia.
3. Burkitt's lymphoma: following uric acid nephropathy complicating chemotherapy for this malignancy.
5. Ebola and Marburg virus diseases.
8. Hemorrhagic fever with renal syndrome.
9. Lassa fever: usually associated with the administration of immune plasma for treatment of severe cases.
10. Leptospirosis.
11. Louse-borne typhus fever.
12. Malaria due to Plasmodium falciparum: may be complicated by hemolytic-uremic syndrome; or following severe hemolysis associated with the administration of 8-aminoquinolones in G6PD-deficient patients.
14. Plant poisoning: Abrus precatorius (rosary pea, black-eyed Susan) and Colchicum autumnale (autumn crocus).
15. Pyomyositis: more common in the tropics where adequate medical care is not available.
16. Rocky Mountain spotted fever.
17. Scrub typhus.
18. Septic abortion: more common where adequate medical care is not available.
19. Shigellosis: may be complicated by hemolytic-uremic syndrome.
20. Snake bite.
21. Typhoid fever: rarely may be complicated by hemolytic-uremic syndrome.
22. Yellow fever.

II. Chronic renal failure

The differential diagnosis of chronic renal failure in the tropics should include:

1. Chronic glomerulonephritis.
2. Chronic obstructive uropathy due to untreated benign prostatic hypertrophy and advanced pelvic malignancies.
3. Chronic untreated pyelonephritis.
4. Leprosy: secondary to amyloidosis and erythema nodosum leprosum following an immune-complex reaction to the treatment of lepromatous or borderline forms of leprosy.
5. Schistosomiasis due to Schistosoma haematobium: may be complicated by obstructive uropathy and chronic pyelonephritis.
External female and male genitalia

I. External genitalia

Complications involving the external genitalia may include:

1. Chancroid: multiple, painful, suppurative ulcers most often located on the penis or vaginal introitus, associated with unilateral inguinal lymphadenitis and bubo formation; most common cause of genital ulcers in many areas of the tropics and subtropics.

2. Cutaneous diphtheria: clinically may mimic impetigo or syphilitic ulcers when the lesions involve the penis or vulva.

3. Granuloma inguinale: beginning as a papule on the external genitalia, the lesion becomes a painless, granulomatous, necrotic ulcer that gradually enlarges involving the vulva, scrotum, and groin; pseudo-buboes may develop in the inguinal area; complicated by genital necrosis, severe scarring, and orifice stenosis.

4. Filariasis: lymphatic varicosities and hydroceles may significantly enlarge the scrotum.

5. Herpes genitalis: painful, multiple, vesiculopapular lesions that eventually ulcerate; an uncommon cause of genital ulcers in the tropics.

6. Onchocerciasis: adenolymphoceles of the inguinal and femoral lymph nodes cause "hanging groin"; the groin appears to redundantly hang down over the anterior thigh.

7. Trichomoniasis: may be complicated by labial ulcerations.

8. Venereal syphilis: primary venereal syphilitic lesions may be single, painless, clean ulcers or multiple, painful, purulent ulcers; may co-exist with chancroid, cutaneous diphtheria, and herpes genitalis.

Female reproductive system

I. Vaginitis

Possible etiologies for vaginitis in the tropics include:

1. Amebiasis: ulcerative vaginitis.

2. Candidiasis: more common in humid, warm climates.
3. Cutaneous diphtheria: may be complicated by vaginal lesions that are pustular papules or punched-out ulcers resembling syphilitic ulcers.
4. Enterobiasis (pinworms): the vulvovaginitis caused by this parasite is seen in the pediatric population.
5. Gardnerella vaginitis.
6. Herpetic vaginitis: may be asymptomatic; less frequently seen in the tropics.
7. Myiasis (maggot infestation).
8. Trichomoniasis: may mimic herpetic vaginal lesions in appearance.

II. Cervicitis

Cervicitis may be caused by:

2. Cervicitis due to Chlamydia trachomatis.
4. Herpetic cervicitis.
5. Lymphogranuloma venereum.
6. Trichomoniasis.

III. Pelvic inflammatory disease

In many areas of the tropics, pelvic inflammatory disease is a common cause for infertility and chronic pelvic pain. Nearly one quarter of the gynecologic admissions to hospitals in Asia and Africa are for pelvic inflammatory disease. The differential diagnosis should include:

1. Campylobacter salpingitis: increasingly recognized as laboratory techniques for the isolation of this organism improve.
2. Enterobiasis (pinworms): rarely may migrate into the female reproductive tract causing pelvic inflammation and peritoneal granulomas.
3. Filariasis: may mimic pelvic inflammatory disease by causing pelvic lymphadenitis or pelvic thrombophlebitis.
5. Pelvic inflammatory disease due to Chlamydia trachomatis.
7. Tuberculosis.
8. Vaginal flora: multiple vaginal anaerobes and facultative bacteria have been implicated in causing pelvic inflammatory disease.
IV. Toxemia of pregnancy

Pre-eclampsia and toxemia of pregnancy are more common where adequate prenatal primary care is not available. Prevalence rates as high as thirty percent have been described in some populations in the tropics.

Male reproductive system

I. Prostatitis

The differential diagnosis of prostatitis in the tropics should include:

1. Blastomycosis.
2. Brucellosis.
3. Cryptococcosis.
5. Gram-negative urinary tract bacterial pathogens.
6. Prostatitis due to Chlamydia trachomatis.
7. Prostatitis due to Staphylococcus aureus.
8. Trichomoniasis.

II. Epididymitis

Epididymitis is a common complication of sexually transmitted diseases where primary health care is not adequate. Etiologies would include:

1. Blastomycosis.
2. Brucellosis.
3. Epididymitis due to Chlamydia trachomatis.
4. Filariasis.
5. Genitourinary gonococcal disease.
6. Trichomoniasis.
7. Tuberculosis.

III. Hydrocele

Hydrocele is an occasional complication of filariasis and onchocerciasis.
IV. Orchitis

Orchitis may be a complication of:

1. Brucellosis.
2. Ebola and Marburg virus diseases.
3. Filariasis.
4. Lassa fever.
5. Mumps.
6. Orchitis due to Chlamydia trachomatis.
7. Variola: orchitis was an occasional complication of smallpox.

V. Other testicular complications

Other testicular complications found in the tropics would include:

1. Abdominal angiostrongyliasis: testicular necrosis associated with spermatic cord compression by a right lower quadrant mass.
2. Leprosy: may be complicated by testicular scarring and atrophy, and by gynecomastia.
Chapter XIV
Musculoskeletal complications

Introduction
I. Musculoskeletal complications

Muscular complications
I. Myalgia
II. Muscle spasm

Articular complications
I. Arthralgia
II. Arthritis

Complications involving the bones
I. Osteomyelitis

Miscellaneous musculoskeletal complications
I. Other musculoskeletal complications
Musculoskeletal complications

Introduction

I. Musculoskeletal complications

The therapeutic approach to musculoskeletal problems is more conservative in developing communities than in developed communities. This is primarily due to the lack of adequate medical facilities, difficulties and expense in administering antibiotics for a prolonged period of time, the general poor physical condition of many patients, and the delay in seeking and receiving primary health care. The lack of satisfactory prosthetic devices, rehabilitation facilities, and adequate, prolonged follow-up care in many areas magnifies the impact of disability caused by arthritis, deformities, or amputation. The cultural and social prejudice against the disabled is often more intense in many areas of the tropics and subtropics, increasing the psychosocial stress experienced by the disabled.

Where proper medical care is not available, attempts to internally fix fractures may lead to disastrous results and amputations. Even though the chance for fracture malunion is greater and angular deformities occur more often, external splinting and traction often achieves functional results for the patient, avoiding the risks of internal fixation.

Chronic osteomyelitis is more common in the tropics. Tuberculosis and untreated bacteremia are the leading causes for this complication.

Occupational musculoskeletal trauma is more common in areas of the tropics that are gradually becoming industrialized. But many of these low wage, blue collar workers cannot afford disability insurance and may receive little help from their employers or governments if injured on the job.

Arthritis and degenerative joint disease are less frequently encountered in the tropics. The reasons for this are that the demographic constitution of many tropical communities is skewed towards a younger population and there may be less medical surveillance for non-life threatening conditions, such as arthritis.
Muscular complications

I. Myalgia

Myalgia is a very common, but nonspecific, symptom of many tropical diseases. The onset of myalgia is usually sudden and it is often accompanied by fever and headache. In certain diseases, such as dengue fever and leptospirosis, the myalgias may be severe and disabling, becoming the chief patient complaint. Myalgia is associated with the myriad of diseases listed below:

1. Argentine and Bolivian hemorrhagic fevers.
2. Ascorbic acid deficiency.
4. Bartonellosis.
5. Brucellosis.
6. Chikungunya fever.
7. Ciguatera fish poisoning.
8. Coccidiomycosis.
10. Dengue fever.
12. Filariasis.
15. Hemorrhagic fever with renal syndrome.
17. Lassa fever.
18. Leptospirosis.
20. Lyme disease.
21. Malaria.
22. Mayaro fever.
23. Melioidosis.
24. Murine typhus.
25. O'nyong nyong fever.
27. Oropouche fever.
28. Paratyphoid fever.
29. Psittacosis.
30. Q fever.
31. Rabies.
32. Relapsing fever.
33. Rickettsial pox.
34. Rift Valley fever.
35. Rocky Mountain spotted fever.
36. Sandfly fever.
37. Schistosomiasis.
38. Scombroid fish poisoning.
39. Scrub typhus.
40. Thiamine deficiency (beri-beri).
41. Tick-borne rickettsioses of the Eastern Hemisphere.
42. Toxoplasmosis.
43. Trench fever.
44. Tularemia.
45. Typhoid fever.
46. Varicella (smallpox).
47. Våriola (smallpox).
48. Venezuelan equine encephalitis.
49. Viral hepatitis (A and B).
50. West Nile fever.
51. Yellow fever.

II. Muscle spasm

Muscle spasm may accompany:

1. African trypanosomiasis.
2. Poliomyelitis.
3. Psittacosis.
4. Rabies.
5. Tetanus.

Articular complications

I. Arthralgia

See also Arthritis Section below:

Arthralgia is a common, but nonspecific, symptom of the diseases listed below:

1. African trypanosomiasis.
2. Bartonellosis.
4. Dengue fever.
5. Fascioliasis.
6. Filariasis.
7. Group C virus diseases.
10. O'nyong nyong fever.
11. Opisthorchiasis.
12. Oropouche fever.
13. Paratyphoid fever.

191
15. Rickettsial pox.
16. Rift Valley fever.
17. Rocky Mountain spotted fever.
18. Sandfly fever.
19. Toxoplasmosis.
20. Trench fever.
21. Trichinosis.
22. Viral hepatitis.
23. West Nile fever.

II. Arthritis

See also Arthralgia Section above.

Arthritis may complicate tropical diseases with a variety of presentations. Arthritis may appear suddenly with viral infections or as a complication of septicemia in bacterial and chlamydial infections. With chronic mycobacterial or mycotic infections, the arthritis may gradually appear and last for months or years. Arthritis may be a delayed complication following an acute infection, most often as the result of immune complex disease. Reiter's syndrome, manifested by arthritis, conjunctivitis, and urethritis, is another possible complication. Arthritis may be associated with:

1. Acute diarrhea due to Campylobacter: postdysenteric reactive arthritis occurs in males with the HLA-B27 histocompatibility antigen.
2. Arthropod-borne viral arthritis and rash (Ross River fever): usually involving the small joints of the hand, wrist, elbows, knees, ankles, and toes.
4. Brucellosis: suppurrative arthritis is a rare complication.
5. Candidiasis: occurring in immune-compromised or malnourished patients.
6. Chikungunya fever.
7. Coccidiomycosis: acute transient polyarthritis, but may develop into a chronic, destructive, monarticular arthritis.
9. Dracunculiasis (Guinea worm infection): may be complicated by monarticular arthritis and ankylosis.
10. Inclusion conjunctivitis: Reiter's syndrome.
11. Lyme disease: often clinically confused with early rheumatoid arthritis, presents as an acute, monarticular or asymmetric oligoarticular arthritis of large joints and temporomandibular joints; often
migratory and recurrent; may become chronic with
damage to cartilage and subchondral bone.
12. Lymphogranuloma venereum.
14. Mayaro fever: usually involving fingers, wrists, ankles, and toes; may be disabling.
15. Melioidosis: acute septicemic or chronic suppurative arthritis.
16. Meningococcal meningitis: acute septic arthritis.
17. Nongonococcal urethritis due to Chlamydia trachomatis: Reiter's syndrome.
18. Psittacosis.
19. Rheumatic fever: more common in developing communities where primary health care is not available.
20. Salmonellosis.
21. Schistosomiasis: associated with Schistosoma mansoni infections complicated by severe intestinal polyposis and hypertrophic osteoarthropathy.
23. Sindbis fever: most often involving the hands and feet.
24. Sporotrichosis: chronic, indolent mono- or polyarticular arthritis involving the joints of the extremities; sinus tracts may drain from the joint or bone through the skin.
25. Tuberculosis and nontuberculous mycobacterial infections: chronic, destructive septic monarticular or oligoarticular arthritis involving large joints, especially spine, hips, and knees.
26. Variola (smallpox): joint effusions with inflammation was a complication.
27. Venereal syphilis: a destructive arthritis in congenital infections; transient polyarthritis with secondary syphilis; gummatous involvement of the synovium and Charcot's joint are possible complications of tertiary syphilis.

Complications involving the bones

I. Osteomyelitis

The differential diagnosis of osteomyelitis in the tropics and subtropics should include:

1. Blastomycosis: common complication presenting as cold abscesses and osteolytic lesions.
2. Brucellosis: rare complication.
3. Candidiasis: usually occurring in immune-compromised or malnourished patients.
4. Coccidiomycosis: osteolytic lesions usually seen in the skull, spine, ribs, metacarpals, femur, tibia, and metatarsals.
5. Cryptococcosis: presenting as painful osteolytic lesions.
6. Melioidosis: one of the most common complications of the chronic suppurative form of this disease.
7. Mycetoma: usually as the direct extension of an obvious subcutaneous infection.
9. Salmonellosis: most often involving the ends of long bones or at the site of a recent fracture.
10. Sporotrichosis: osteolytic lesions as an extension of articular involvement.
11. Tuberculosis: most often involving the long bones and vertebrae, trauma may reactivate dormant lesions.
13. Variola (smallpox): was a complication of bacterial superinfection.

**Miscellaneous musculoskeletal complications**

**I. Other musculoskeletal complications**

An assortment of miscellaneous complications may be caused by:

2. Cysticercosis: myositis and muscular cysts.
4. Echinococcosis due to Echinococcus granulosus: bone cysts.
7. Malignant metastatic lesions of Burkitt's lymphoma and nasopharyngeal carcinoma.
11. Schistosomiasis: hypertrophic osteoarthropathy without pulmonary findings.
15. Vitamin D deficiency: osteomalacia.
16. Yaws: polydactylitis, painful osteoperiostitis of the long bones, hypertrophic osteitis of the nasal processes of the maxilla, and perforation of the palate.***
Chapter XV
Dermatological complications

Part I: Dermatologic differential diagnosis by lesion type
I. Introduction

Macules
I. Red macules
II. Hypopigmented macules
III. Petechiae and purpura

Maculopapular rashes
I. Acute maculopapular rash associated with lymphadenopathy
II. Acute maculopapular rash not associated with significant lymphadenopathy
III. Gradual onset maculopapular rash

Papules
I. Red papules
II. Urticaria
III. Creeping eruption
IV. Flesh-colored papules
V. Papules (or plaques) associated with scaling

Nodules
I. Painful nodules
II. Painless nodules

Vesicles
I. Vesicles associated with red papules
II. Vesicles / blisters / bullae

Pustules / abscesses
I. Pustules
II. Abscesses

Ulcers
I. Purulent ulcers without marked lymphadenopathy
II. Purulent ulcer with significant lymphadenopathy
III. Ulcerating nodules
IV. Painless ulcers

Sinus tracts
I. Sinus tracts

196
Miscellaneous dermatologic problems
I. Atrophy
II. Erythema multiforme
III. Erythema nodosum
IV. Genital lesions
V. Lesions of the palms or soles
VI. Mucocutaneous lesions
VII. Nail lesions
VIII. Pruritus

Part II: Descriptive dermatology for tropical diseases
I. Introduction
II. Descriptive tropical dermatology
   for 103 tropical diseases
Dermatologic complications

Part I: Dermatologic differential diagnosis by lesion type

I. Introduction

The differential diagnosis of dermatologic complications of tropical diseases is summarized below, arranged by lesion type. A complete description of the dermatologic findings for each disease is alphabetically arranged in Part II below.

Macules

I. Red macules

Red macules are flat, circumscribed lesions that are red in color and blanch with applied pressure. May be difficult to see on dark or tanned skin, often appearing as hyperpigmented macules rather than red in color.

1. African trypanosomiasis.
2. American trypanosomiasis (Chagas' disease).
3. Argentine and Bolivian hemorrhagic fevers.
5. Dengue fever.
7. Rocky Mountain spotted fever.
8. Typhoid fever: "rose spots".

II. Hypopigmented macules

Hypopigmented macules are flat, circumscribed lesions that lack melanin or adequate blood supply, and appear lighter in color than the surrounding skin.

1. Leprosy.
2. Onchocerciasis.
3. Pinta.
4. Streptocerciasis.
5. Tinea versicolor.
II. Petechiae and purpura

Petechiae and purpura are red or bluish red macules that do not blanch with applied pressure. These lesions may be complicated by vasculitis and become papular. The vasculitic lesions may develop infarction ulcers and gangrene.

1. Argentine and Bolivian hemorrhagic fevers.
2. Ascorbic acid deficiency.
3. Chikungunya fever.
5. Dengue fever.
8. Louse-borne typhus fever.
9. Meningococcal disease.
11. Plague.
12. Relapsing fever.
13. Rift Valley fever.
14. Rocky Mountain spotted fever.
15. Tick-borne rickettsioses of the eastern hemisphere (Boutonneuse fever, Siberian tick typhus, Queensland tick typhus).
17. Yellow fever.

Maculopapular rashes

I. Acute maculopapular rash associated with lymphadenopathy

These diseases may be associated with the acute onset of multiple, flat or raised, erythematous lesions, systemic symptoms, and significant lymphadenopathy.

1. Arthropod-borne viral arthritis/rash (Ross River fever).
2. Brucellosis.
3. Chikungunya fever.
4. Dengue fever.
5. Ebola and Marburg virus diseases.
6. O'nyong nyong fever.
7. Rickettsial pox.
8. Scrub typhus.
9. Tick-borne rickettsioses of the eastern hemisphere (Boutonneuse fever, Siberian tick typhus, Queensland tick typhus).
10. Toxoplasmosis.
11. West Nile fever.
II. Acute maculopapular rash not associated with significant lymphadenopathy

These diseases may be associated with the acute onset of multiple, flat or raised, erythematous lesions and systemic symptoms, but without significant lymphadenopathy.

1. Lassa fever.
2. Louse-borne typhus fever.
3. Mayaro fever.
5. Rocky Mountain spotted fever.
6. Rubeola (measles).
7. Sindbis fever.
8. Typhoid fever.

III. Gradual onset maculopapular rash

1. Coccidiomycosis.
2. Cutaneous leishmaniasis.
3. Leptosy.
4. Lyme disease.
5. Niacin deficiency (pellagra).
7. Yaws.

Papules

I. Red papules

Red papules are erythematous, raised lesions.

1. Acariasis (scabies infestation).
2. Bartonellosis.
3. Chancroid.
5. Mite bite, including chiggers.
7. Onchocerciasis.
8. Tularemia.
10. Yaws.

II. Urticaria

See also Erythema Multiforme Section below.
Urticaria is manifested by transient, pruritic, well-defined, red papules.

1. Allergic shellfish poisoning.
2. Dracunculiasis.
3. Echinococcosis due to Echinococcus granulosus.
4. Fascioliasis.
5. Filariasis.
6. Giardiasis.
8. Loiasis.
9. Mite bite, including chiggers.
11. Scombroid fish poisoning.
12. Strongyloidiasis.
13. Taeniasis.
14. Toxoplasmosis.
15. Trichinosis.

III. Creeping eruption

Creeping eruption is manifested by persistent, pruritic, migratory red papules

1. Cutaneous larvæ migrans due to dog and cat hookworm.
2. Myiasis: horse or cattle botfly larvae.
3. Paragonimiasis.
4. Strongyloidiasis.

IV. Flesh-colored papules

1. Follicular hyperkeratosis.
2. Nonvenereal endemic syphilis.
3. Yaws.

V. Papules (or plaques) associated with scaling

1. Ascorbic acid deficiency.
2. Candidiasis.
3. Chromomycosis (chromoblastomycosis).
4. Diphtheria.
5. Pinta.
7. Tinea corporis and cruris.
8. Tinea pedis.
9. Tinea versicolor.
10. Venereal syphilis.
11. Visceral leishmaniasis.
12. Yaws.
Nodules

I. Painful nodules

Nodules are elevated lesions that are in the deep dermis or subcutaneous tissue. The overlying skin may be moved over the lesion.

1. African trypanosomiasis.
2. American trypanosomiasis (Chagas' disease).
3. Chikungunya fever.
4. Loiasis.
5. Melioidosis.
7. Sporotrichosis.
8. Tinea capitis.

II. Painless nodules

1. Actinomycosis or nocardiosis.
2. Chromomycosis (chromoblastomycosis).
3. Cryptococcosis.
5. Cysticercosis.
7. Entomophthoromycosis due to Conidiobolus.
8. Filariasis.
9. Leprosy.
10. Lobo's disease.
11. Loiasis.
12. Mycetoma.
15. Venereal syphilis.

Vesicles

I. Vesicles associated with red papules

See also Erythema Multiforme Section below.
Vesicles are sharply demarcated, elevated lesions that contain clear fluid.

1. Acariasis (scabies infestation).
2. Candidiasis.
3. Cutaneous larva migrans.
5. Mite bite, including chiggers.
7. Niacin deficiency (pellagra).
8. Plant poisoning.
10. Rickettsial pox.
11. Sindbis fever.
12. Schistosomiasis.
13. Tinea pedis.

II. Vesicles / blisters / bullae

1. Dracunculiasis.
2. Pemphigus foliaceus: chronic flaccid blisters, accompanied by a burning sensation; uncommon dermatologic disease among tropical residents; generalized distribution.
3. Tinea pedis.

Pustules / abscesses

I. Pustules

Pustules are focal, inflammatory lesions that contain purulent material.

1. Blastomycosis.
2. Candidiasis.
3. Chancroid.
4. Gonococcal disease.
5. Meningococcal disease.
7. Tinea capitis.
8. Tinea pedis.
II. Abscesses

1. Anthrax.
2. Coccidiomycosis.
3. Dracunculiasis.
4. Loiasis.
5. Lymphogranuloma venereum.
7. Mycobacteria, atypical.

Ulcers

I. Purulent ulcers without marked lymphadenopathy

Ulcers are depressed lesions with erosion of the overlying epidermis or dermis.

1. Amebiasis.
2. Anthrax.
4. Cancrum oris.
5. Chromomycosis (chromoblastomycosis).
6. Coccidiomycosis.
7. Cryptococcosis.
8. Diphtheria.
10. Tropical ulcer: an acute, painful ulcers appearing after minor trauma on the lower extremities of malnourished children; may be complicated by secondary bacterial infection or squamous cell carcinoma.
11. Venereal syphilis.

II. Purulent ulcer with significant lymphadenopathy

1. Chancroid.
2. Cutaneous leishmaniasis.
3. Filariasis.
4. Lymphogranuloma venereum.
5. Nonvenereal endemic syphilis.
6. Paracoccidiomycosis.
7. Plague.
8. Sporotrichosis.
10. Venereal syphilis: onset of lymphadenopathy delayed one week after appearance of the ulcer in primary syphilis.
III. Ulcerating nodules

1. African trypanosomiasis.
2. Chromomycosis (chromoblastomycosis).
3. Cutaneous leishmaniasis.
4. Entomophthoromycosis due to Basidiobolus.
5. Mycobacteria, atypical.
7. Tinea capitis.
8. Venereal syphilis.

IV. Painless ulcers

Many ulcers may be relatively painless despite their large size.

1. Granuloma inguinale.
2. Leprosy.
3. Lymphogranuloma venereum.
4. Venereal syphilis.
5. Yaws.

**Sinus tracts**

I. Sinus tracts

1. Actinomycosis or nocardiosis.
2. Amebiasis.

**Miscellaneous dermatologic problems**

I. Atrophy

1. Onchocerciasis.

II. Erythema multiforme

Erythema multiforme is an acute onset of discrete, target, hive-like papules with a generalized, symmetrical distribution especially involving the extensor surfaces of the extremities, palms, and soles. The rash may be complicated by tense vesicles and blisters distributed in the lesions or mucosal surfaces. The rash may be associated with drugs, streptococcal
infections, viral and deep fungal infections, neoplasms, and systemic lupus erythematosus. Erythema multiforme is a common dermatologic lesion found in:

1. Coccidiomycosis.
2. Histoplasmosis.

III. Erythema nodosum

Erythema nodosum is the acute onset of crops of red or blue, painful nodules that are 1 to 5 centimeters in size and symmetrically distributed on the anterior shins. Erythema nodosum may be accompanied by purpura.

1. Coccidiomycosis.
2. Drug eruption.
3. Histoplasmosis.
4. Leprosy.
5. Tuberculosis.

IV. Genital lesions

1. Candidiasis.
2. Chancroid.
3. Cutaneous leishmaniasis.
4. Diphtheria.
5. Gonococcal disease.
7. Lymphogranuloma venereum.
8. Mite bite, including chiggers.
10. Tularemia.
11. Venereal syphilis.

V. Lesions of the palms or soles

1. Chikungunya fever.
2. Cutaneous larva migrans.
3. Erythema multiforme.
4. Gonococcal disease.
5. Hookworm disease.
7. Rocky Mountain spotted fever.
8. Scytalidium or Hendersonula dermatitis.
10. Tick-borne rickettsioses of the eastern hemisphere (Boutonneuse fever, Siberian tick typhus, Queensland tick typhus).
11. Tinea pedis.
12. Tularemia.
13. Tungiasis (chigoe infestation).
15. Venereal syphilis.

VI. Mucocutaneous lesions

Mucocutaneous lesions occur on the mucosal surfaces of the eyes, nose, mouth, or vagina; or at the mucocutaneous junctions of these areas, such as the lips and vaginal introitus.

1. Amebiasis.
2. Blastomycosis.
3. Candidiasis.
5. Erythema multiforme.
6. Histoplasmosis.
7. Loiasis: conjunctiva.
8. Mucormycosis.
10. Niacin deficiency (pellagra).
11. Nonvenereal endemic syphilis.
13. Rickettsial pox.
15. Trichomoniasis.

VII. Nail lesions

1. Candidiasis.
2. Iron deficiency.
3. Scytalidium or Hendersonula dermatitis.
4. Tinea unguium.
5. Tungiasis (chigoe infestation).

VIII. Pruritus

See also Urticaria Section above.

See also Jaundice Section of the Hepatobiliary Complications Chapter.

Pruritus may accompany:

1. Acarasis (scabies infestation).
5. Echinococcosis due to Echinococcus granulosus.
7. Gnathostomiasis.
8. Mansonella ozzardi infection.

Part II: Descriptive dermatology for tropical diseases

I. Introduction

Refer to Part I of this chapter above for quick reference
differential diagnosis of dermatologic complications, grouped
by lesion type. Part II of this chapter is an alphabetical
listing of those tropical diseases associated with dermatologic
complications, accompanied by a description of the possible
lesions.

II. Descriptive tropical dermatology

Acariasis (scabies infestation): excoriated, red to
purple, pruritic papulovesicular lesions that are a few
millimeters in size and sometimes associated with thread-like
burrows; distributed on flexor surfaces of extremities, finger
webs, axillary folds, breasts, umbilicus, genitalia, and toe
webs.

Actinomycosis or nocardiosis: painless, indurated,
erythematous, nodular plaques that develop multiple sinus
tracts draining pus and "sulfur" granules (actinomycosis);
distributed on the face, neck, thorax, and abdominal wall.

African trypanosomiasis: large, nonpruritic,
evanescent, erythematous, circinate macules with central fading
that last for several weeks; appear two months after an
infesting tse-tse fly bite; distributed on the chest and back;
also indurated, painful, erythematous nodular lesion that
sometimes forms a chancre; lesion appears suddenly and resolves
within two weeks; distributed at tse-tse fly bite sites on
exposed skin, usually on the legs and head.

Allergic shellfish poisoning: urticaria.

Amebiasis: painful, inflammatory, necrotic, deep
ulcers, sometimes with sinus tracts; rare complication;
distributed on abdominal wall, external genitalia, vagina,
cervix, and perineum.
American trypanosomiasis (Chagas' disease): initial lesion is a painful, erythematous, indurated nodule that is 1 to 3 centimeters in size and associated with regional lymphadenopathy; lesion persists for weeks; distributed on exposed skin surfaces at Triatomid bug bite site; later, the parasitemic phase of the acute disease may be accompanied by a nonspecific, generalized, macular erythema.

Anthrax: inflammatory papule that develops in several days into a painless, necrotic abscess or ulcer with annular, brawny, gelatinous edema and a central, black eschar; satellite lesions are possible; located on hands, arms, face, or neck.

Argentine and Bolivian hemorrhagic fevers: macular erythema; distributed on the face, neck, and chest; later, may be complicated by generalized petechiae and clinical hemorrhage.

Arthropod-borne viral arthritis and rash (Ross River fever): nonpruritic, fine, erythematous maculopapular rash appearing on the third or fourth day of the illness; generalized distribution.

Ascorbic acid deficiency: follicular hyperkeratosis with severe deficiency; generalized distribution; may be accompanied by petechiae and purpura.

Bartonellosis: cranberry-like, red, papulonodular, verrucoid lesions that are from 1 mm to 3 cm in size and bleed with minor trauma; distributed on extensor surfaces of extremities, face, and sometimes on scalp and genitals.

Blastomycosis: chronic, usually solitary, lesion that begins as a pustule or verrucoid nodule and grows into an indurated, boggy, erythematous plaque containing multiple pustules, and may heal centrally; may ulcerate; distributed on exposed skin surfaces or mucous membranes, but may be found anywhere on the body.

Brucellosis: intensely pruritic, erythematous maculopapular rash appearing within one week to one month after the initial infection; usually distributed on exposed skin of the upper extremities.

Cancrum oris: painful, necrotic ulceration occurring in ill, malnourished children; distributed on the face and mouth.
Candidiasis: scaly or shiny, erythematous plaques with satellite pustules or discrete vesiculopustules progressing to erosions; distributed over the moist, warm skin folds of the axilla, under breasts, groin, perineum, proximal interdigital skin, and redundant adipose skin (intertrigo), or under the prepuce (balanitis); also thick, cheese-like mucocutaneous lesions that may be rubbed off revealing an underlying, red mucosa; distributed in the mouth (thrush) or the vagina (vaginitis); also paronychia and non-scarring subungal involvement with onycholysis.

Chancroid: beginning as painful, red papules, then developing into painful, multiple, pustular, under-mined ulcers which are associated with satellite pustules and painful, unilateral, inguinal lymphadenopathy or inguinal buboes; complicated by paraphimosis or genital necrosis; distributed on the genitals and perineum.

Chikungunya fever: nonpruritic, erythematous maculopapular rash appearing between the third or tenth day of illness, clearing without desquamation; may be complicated by periarticular nodules; rarely may be accompanied by petechiae and purpura; distributed on the trunk, extensor extremities, and occasionally on the palms and soles.

Coccidiomycosis: a fine, erythematous maculopapular rash occurs with primary infection; diffusely distributed on the trunk and extremities; may be accompanied by erythema multiforme or erythema nodosum; verrucoid, granulomatous ulcers or noninflammatory subcutaneous abscesses occur with disseminated disease.

Crimean-Congo hemorrhagic fever: petechiae and large ecchymoses appear with a hemorrhagic diathesis as a possible complication between the third and fifth day of the illness; generalized distribution.

Chromomycosis (chromoblastomycosis): beginning as small, erythematous, scaly papules; later developing into chronic plaques of cicatrical (scarring), verrucoid nodules or purulent ulcers with satellite lesions; usually distributed on the feet or legs, but may be anywhere; associated with prolonged or occupational contact with tropical soils.

Cryptococcosis: granulomatous nodules; purulent, ulcerative plaque, sometimes with bullae and hemorrhage; generalized distribution.
Cutaneous larva migrans: pruritic, serpentine, erythematous macular lines that wander and progress daily (creeping eruption); may become papular with fine vesicles and crusting; distributed on lower extremities, buttocks, and trunk, but may appear anywhere.

Cutaneous leishmaniasis: gradual onset of red maculopapular or papulonodular lesions that develop into chronic, dry or moist, painless ulcers, sometimes with satellite lesions; often becoming secondarily infected with bacteria; may be associated with a nontender, nodular lymphadenitis, mimicking sporotrichosis; metastatic, granulomatous, mucocutaneous ulcers with facial deformities may occur, especially in the New World; ulcers heal with scarring; distributed on the nasopharynx, face, ears, and extremities, rarely on conjunctiva, larynx, and genitals; may be clinically confused with sporotrichosis and yaws.

Cysticercosis: asymptomatic, subcutaneous nodules; generalized distribution.

Dengue fever: erythematous macular rash occurs within the first 24 hours; maculopapular rash appears on the second or third day of the illness; accompanied by desquamation during resolution; generalized distribution; dengue hemorrhagic fever is accompanied by petechiae and ecchymoses on the forehead and extremities.

Diphtheria: chronic, dry, scaly papules that may resemble insect bites or impetigo; may progress to deep, punched-out ulcers, with or without an adherent eschar, and may be accompanied by local hypesthesia; distributed around wounds or on the face, extremities, or genitals.

Dracunculiasis: small vesicle develops into a bulla over a few days, which finally bursts; followed by the emergence of a worm from the overlying eschar, which requires weeks to months for expulsion; may be complicated by secondary bacterial infection and purulent ulcers along the subcutaneous tract of the worm, or by abscess formation; usually associated with urticaria; distributed on the legs, but the worm may emerge from any location.

Ebola and Marburg virus diseases: erythematous maculopapular rash accompanied by desquamation on resolution; distributed on trunk and back; petechiae with hemorrhagic complications.

Echinococcosis due to Echinococcus granulosus: urticaria.
Entomophthoromycosis due to Basidiobolus: chronic, progressive, diffuse, nodular subcutaneous swelling that rarely ulcerates; distribution spares face, palms, and soles.

Entomophthoromycosis due to Conidiobolus: chronic, progressive, diffuse nodular subcutaneous swelling; distribution limited to the central face.

Fascioliasis: urticaria.

Filarialiasis: ulcerations may develop over involved lymphatics that heal with scarring; chronic nodular papillomatous hyperplastic changes with lichenification occur in the legs with elephantiasis; may be associated with urticaria.

Follicular hyperkeratosis: chronic, flesh-colored, follicular papules appearing in childhood or peripartum; distributed on the extensor surfaces.

Giardiasis: urticaria.

Gonococcal disease: acute appearance of acral pustules or pustular vesicles that quickly erode; distributed primarily on the extremities, may appear on palms, soles, or genitals.

Granuloma inguinale: initially a papule that gradually erodes into painless, granulomatous ulcers with well-defined raised borders and that heal with scarring; distributed on the genitals and inguinal areas.

Hemorrhagic fever with renal syndrome: complicated by bleeding and petechiae; distributed on palate, axillary folds, upper thorax, and head.

Histoplasmosis: disseminated disease may be complicated by oropharyngeal ulcers; associated with erythema multiforme and erythema nodosum.

Hookworm: pruritic, erythematous papules lasting 7 to 10 days that may vesiculate or develop secondary bacterial infection; distributed where skin contacts soil, especially the feet.

Hymenolepiasis: urticaria.

Iron deficiency: may be associated with koilonychia (spooning secondary to thinning of the nail plate).
Lassa fever: faint maculopapular rash appears during the second week of the illness; generalized distribution.

Leprosy:

a. Indeterminate leprosy: single or a few, poorly defined, hypopigmented or slightly erythematous macules without neurologic changes; may resolve spontaneously, persist chronically unchanged, or progress to tuberculoid or lepromatous leprosy; generalized distribution.

b. Tuberculoid leprosy: single or a few, hypopigmented or erythematous, finely maculopapular lesions with well defined borders; complicated by hypesthesia, hair loss, decreased sweating, and neuritis; may resolve spontaneously or progress with central healing; generalized distribution.

c. Borderline leprosy: neuropathy is a predominant finding with skin lesions sharing features of tuberculoid or lepromatous leprosy.

d. Lepromatous leprosy: initially large macular lesions that coalesce, followed by significant nodular infiltration; complicated by loss of body hair, sparing scalp, neuropathies, and obstructive vasculitis (Lucio leprosy), which results in dermal infarcts and ulcers; generalized distribution, but more intense about ears, face, extensor extremities, and buttocks.

e. Erythema nodosum leprosum: treatment of borderline or lepromatous leprosy may be complicated by erythema nodosum.

Lobo's disease: chronic, mobile nodular lesions that enlarge to form granulomatous, verrucoid, nodular plaques accompanied by satellite lesions; generalized distribution, but more often seen on extremities.

Loiasis (loa loa): recurrent, pruritic, erythematous swelling of subcutaneous tissues, which may later be followed by chronic nodules and chronic granulomatous abscesses; may be associated with urticaria; generalized distribution, but the migrating subcutaneous helminths are usually found in the conjunctiva, face, and upper extremities.

Louse-borne typhus fever: erythematous macules appear between the fourth and seventh day of the illness; may progress to erythematous maculopapular rash; may be complicated by petechiae and purpura with skin infarction and gangrene; distributed on trunk, axillary folds, extremities, usually sparing face, palms, and soles.
Lyme disease: erythema chronicum migrans associated with this disease is an expanding, erythematous, annular maculopapular rash, with single or multiple lesions, that resolves spontaneously in a few weeks; distributed at tick bite sites usually on thigh, trunk, or buttocks.

Lymphogranuloma venereum: transient, painless vesicle that erodes into an ulcer, may progress to multiple abscesses; distributed on the external genitalia.

Mayaro fever: erythematous maculopapular rash appears on the fifth day of the illness and resolves in several days without desquamation; distributed on the trunk and extremities, and sometimes faintly on the face and hands.

Melioidosis: painful nodule may appear at the site of skin inoculation; may develop metastatic chronic abscesses.

Meningococcal disease: petechiae and purpura common; may be complicated by vasculitis resulting in acral pustules and gangrene; generalized distribution, but vasculitis more intense on extremities.

Mite bite, including chiggers: pruritic, erythematous papules that become vesiculated; may be associated with urticaria; distributed in groups on ankles, thighs, buttocks, external genitalia, waist, axillary and breast folds.

Mucormycosis: cellulitis of the face accompanied by black, necrotic lesion of the nasal and palate mucosa.

Murine typhus: maculopapular rash appears on the fifth or sixth day of illness; distributed on the trunk.

Mycetoma: small subcutaneous nodule gradually develops into multiple, chronic, painless, granulomatous nodules that are accompanied by sinus tracts discharging pustular grains; generalized distribution, but mostly occurs on the foot and may involve the sole.

Mycobacteria, atypical: chronic, progressive nodules develop into granulomatous ulcers; distributed at site of skin inoculation: skin abscesses may develop gradually.
Myiasis: horse, cattle, rodent, rabbit, or human botfly, or tumbu fly may produce painful, pruritic, erythematous papules that develop into furuncles containing fly larvae; horse or cattle botfly larvae may cause a migratory creeping eruption; generalized distribution; screwworms, green fly, bottle fly, house fly, and black blow fly may infest wounds, mucocutaneous tissue, and eye; patient complains of movement in the lesion.

Niacin deficiency (pellagra): symmetrical, red or red-brown or red-purple, maculopapular plaques that may vesiculate or blister; distributed face, neck, upper chest, arms, and shins, more intense on areas exposed to sun, heat, or friction; mucous membranes of mouth and genitalia may develop ulcers.

Nonvenereal endemic syphilis: papular mucosal patches appear in the mouth followed by disseminated, flesh-colored, papillomatous or papulosquamous lesions, which may become ulcerated resulting in deforming gangosa; generalized distribution.

Onchocerciasis: initially pruritic, erythematous papules or nodules that gradually progress to chronic, nonpruritic, atrophic, hypopigmented lesions; distributed on lower abdomen, groin, or legs.

O'nyong nyong fever: intensely pruritic maculopapular rash that appears on the fourth to seventh day of the illness, which resolves within a week without desquamation; distributed on the face and neck and spreads downward onto the trunk.

Paracoccidiomycosis: painful, granulomatous ulcers with lymphadenitis; generalized distribution; may have painful, mucocutaneous ulcers.

Paragonimiasis: may be complicated by urticaria or creeping eruption; parasite may form nodules.

Pertussis: may develop petechiae secondary to straining during cough paroxysms.

Pinta: hypopigmented, scaly papules or plaques; distributed on the face, neck, chest, extremities.

Plague: acute onset of pustular ulcers, lymphadenitis, and sepsis; distributed on the trunk; may develop generalized purpura that may progress to necrosis and gangrene in the extremities.
Plant poisoning: Anacardium occidentale (cashew nut oil); Mangifera indica (mango); Toxicodendron radicans (poison ivy); Dieffenbachia species; Alocasia species (elephant ear); Euphorbia pulcherrima (poinsettia latex); Hippomane mancinella (manchineel); Hura polyandra (devil tree); and Ricinus communis (castor bean); all may produce pruritic, linear, erythematous vesiculopapular lesions with skin contact to irritating plant products.

Prickly heat: erythematous, vesiculopapular lesions; secondary bacterial infection is common; distributed in warm, moist areas of the body.

Pyridoxine deficiency: seborrheic dermatitis; facial eczema.

Relapsing fever: petechiae may appear; truncal distribution.

Rickettsial pox: vesicles simultaneously arise from discrete maculopapular lesions during the first to fourth day of the illness, then erode into dark crusts; eschar covering shallow ulcer may be found at the site of inoculation by mite bite; distributed over trunk and extremities; vesicles may be found on mucous membranes.

Rift Valley fever: petechiae and purpura occasionally appear on the second to fourth day of the illness; generalized distribution.

Rocky Mountain spotted fever: erythematous macular or maculopapular rash appears on the second to sixth day of illness, then becomes petechial; may be complicated by vasculitis resulting in necrosis and gangrene of the extremities; initially distributed on forearms, ankles, palms, and soles, then becoming generalized.

Rubeola (measles): erythematous maculopapular rash appears during the first 48 hours of the illness, then fades over three to five days; may become confluent over the face; distributed on the face and trunk.

Schistosomiasis: pruritic maculopapular rash may develop at the site of skin penetration by human schistosomes; pruritic maculopapular rash followed by vesiculation may persist for a few days following skin penetration by bird schistosomes; distribution limited to areas of skin exposed to fresh water containing schistosomal cercariae.

Scombroid fish poisoning: urticaria.
Scrub typhus: about the time of onset of the illness, eschar is present at site of inoculating mite bite, accompanied by regional lymphadenopathy; erythematous maculopapular rash appears about the fifth to seventh day of the illness; distribution is first on the trunk and axillae, then buttocks and proximal extremities.

Scytalidium or Hendersonula dermatitis: these plant fungi may produce a dry, diffuse, hyperkeratotic, scaly rash; distributed on the hands, soles, and nails.

Sindbis fever: erythematous maculopapular rash appears on the first or second day of the illness; pruritic vesicles may erupt in crops during an additional ten days; may develop oral vesicles that erode into ulcers; generalized distribution, but more intense over buttocks and legs.

Sporotrichosis: painful, purulent, ulcerating nodules associated with lymphadenopathy; lesions follow course of lymphatics; distributed on extremities.

Streptocerciasis: chronic, pruritic, hypopigmented papules without neurologic findings; may be confused with leprosy; distributed on thorax and shoulders.

Strongyloidiasis: chronic urticaria, or migrating, serpentine urticarial lesions (creeping eruption); distribution most commonly around the waist and buttocks.

Taeniasis: urticaria.

Tick-borne rickettsioses of the eastern hemisphere (Boutonneuse fever, Siberian tick typhus, Queensland tick typhus): by the first day of the illness, an eschar may be found at the site of skin inoculation by the tick bite, associated with regional lymphadenopathy; maculopapular rash appears around the fourth day of illness; petechiae may appear; generalized distribution and may involve soles, palms, and face.

Tinea capitis: one or multiple, painful nodules that may develop into pustular ulcers; heal with scarring; distributed on the scalp.

Tinea corporis and cruris: one or multiple, polycyclic, arciform, or annular erythematous papules with scaling and central healing; generalized distribution, but more commonly found on trunk, crural folds, and thighs.
Tinea pedis: asymmetric or circular, hyperkeratotic, skin-colored or erythematous, scaly papules or plaques that may vesiculate or become pustular; distributed on soles, between toes, and hands; may be associated with tinea unguium.

Tinea unguium: dry, brittle, opaque, thickened nails with onycholysis; debris collects under the nail; may be associated with tinea pedis.

Tinea versicolor: chronic, faintly erythematous or brownish, scaly, hypopigmented macules or papules; lesions often confluent; distributed over thorax and shoulders.

Toxoplasmosis: urticaria; may be complicated by erythematous maculopapular rash associated with lymphadenopathy; generalized distribution, but sparing palms and soles.

Trichinosis: urticaria, polyarteritis nodosum.

Trichomoniasis: mucocutaneous ulcerations, may mimic herpes genitalis at introitus; distributed in the vagina.

Tuberculosis: erythema nodosum.

Tularemia: painful, erythematous papule that develops into a pustular ulcer with raised borders, associated with suppurative lymphadenitis; distributed on extremities, hands, genitals, groin, and axillae.

Tungiasis (chigoe infestation): painful, pruritic nodules with central black spot; distributed on feet, hands, and beneath nails, occasionally on skin exposed to the ground during sleeping.

Typhoid fever: erythematous maculopapular lesions not associated with lymphadenopathy; distributed on abdomen, chest, back, and extremities.

Variola (smallpox): smallpox was accompanied by a maculopapular eruption on the second or fourth day of the illness that developed into umbilicated, pustular vesicles lasting 8 to 15 days, finally the vesicles eroded into crusted papules followed by gradual healing and scarring; lesions commonly developed secondary bacterial infection; distribution began on the face and arms then became generalized, but more intensely involved the periphery; palms and soles could also be affected; could be confused with chickenpox, monkeypox, drug reactions, insect bites, and secondary syphilis.
Venereal syphilis:

a. Primary venereal syphilis: may be a single, painless, well-defined ulcer or multiple, painful, purulent ulcers followed by inguinal lymphadenopathy; usually heal within six weeks without scarring; genital or anal distribution.

b. Secondary venereal syphilis: symmetrical, circular or ring-shaped, non-coalescent, scaly, erythematous papules; may appear as moist verrucoid papules in intertriginous areas; white, coalescing papules may be seen on the oral and genital mucosa; generalized distribution, including palms and soles.

c. Tertiary venereal syphilis: chronic, painless, single or multiple, nodular lesions that may ulcerate (gumma); may be complicated by necrosis and heal with scarring; generalized distribution with grouping of lesions.

Visceral leishmaniasis: skin may be scaly have a diffuse, gray tone; may have petechiae and purpura; generalized distribution, prominent on the hands and face.

West Nile fever: nonpruritic, acute maculopapular rash that is associated with lymphadenopathy and clears without desquamation; generalized distribution.

Yaws:

a. Primary yaws: initially a small, erythematous papule that enlarges to become a superficially ulcerated, raspberry-like, papillomatous papule; may be several centimeters in size; distributed usually on the lower extremities or buttocks.

b. Secondary yaws: the papillomatous lesion becomes disseminated and these may develop into chronic, shallow gummatous ulcers; secondary lesions may also be macular or papulosquamous; hyperkeratosis with fissuring and ulcerations may develop on the palms and soles, resulting in disability; generalized distribution, but more intense in moist areas of the body and at mucocutaneous junctions; multilating facial gangosa may develop from mucocutaneous lesions as a late complication.

Yellow fever: petechiae and purpura; generalized distribution.***
Chapter XVI
Hematologic complications

Hemorrhagic complications
I. Hemorrhagic phenomena complicating tropical disease

Blood cell disorders
I. Anemia
   A. Introduction
   B. Hypochromic microcytic anemia
   C. Normochromic normocytic anemia
      (excluding hemolytic anemia)
   D. Megaloblastic anemia
   E. Hemolytic anemia
   F. Anemia secondary to hypersplenism
II. Eosinophilia
III. Suppression of cell-mediated and humoral immunity

Lymphatic complications
I. Generalized lymphadenopathy
II. Preauricular lymphadenopathy
III. Cervical lymphadenopathy
IV. Hilar lymphadenopathy
V. Inguinal lymphadenopathy
VI. Regional lymphadenopathy

Splenomegaly
I. Tropical splenomegaly syndrome
II. Splenomegaly
III. Splenomegaly that may be associated with hepatomegaly
Hematologic complications

Hemorrhagic complications

I. Hemorrhagic phenomena complicating tropical disease

See also Petechiae and Purpura Section of the Dermatological Complications Chapter.

See also Bloody Diarrhea Section of the Gastrointestinal Complications Chapter.

See also Hemoptysis Section of the Pulmonary Complications Chapter.

See also Epistaxis Section of the Ear, Nose, and Throat Complications Chapter.

See also Shock Syndrome Section of the Cardiovascular Complications Chapter as sepsis and shock may be complicated by disseminated intravascular coagulation.

Perhaps among laymen, hemorrhage is the most widely known and feared complication of tropical diseases, such as yellow fever. And despite modern medical technology and the development of an effective vaccine, epidemics of yellow fever still terrorize populations which have inadequate primary care, killing thousands.

Often hemorrhage is the initial sign of a fatal course to an overwhelming tropical infection, but the bleeding itself is usually not severe enough to produce anemia and hypovolemic shock. In tropical diseases, meningoencephalitis, respiratory arrest, cardiac arrhythmias, and septic shock are more important accompanying complications resulting in death than hemorrhagic phenomena.

The following diseases may be complicated by hemorrhagic phenomena:

1. Argentine and Bolivian hemorrhagic fevers: a frequent complication of both diseases with petechiae, gingival bleeding, epistaxis, hematemesis, melena, metrorrhagia, and mucocutaneous bleeding; the amount of bleeding is usually not life-threatening, but the diseases are often complicated by shock syndrome.
2. Ascorbic acid deficiency: gingival bleeding, bloody diarrhea, hematuria, and subcutaneous hematomas.
3. Bartonellosis: generalized hemorrhage is an occasional complication.
5. Crimean–Congo hemorrhagic fever: petechiae, extensive purpura, thrombocytopenia, and severe generalized hemorrhage.
6. Dengue hemorrhagic fever: petechiae, purpura, thrombocytopenia, and oozing of blood from venipuncture sites.
10. Kyasanur Forest disease: epistaxis, gingival bleeding, hemoptysis, hematemesis, melena, and metrorrhagia.
11. Lassa fever: uncommon complication with gingival bleeding, melena, and metrorrhagia.
12. Leptospirosis: rare complication with epistaxis, hemoptysis, hematemesis, melena, petechiae, and thrombocytopenia; a severe vasculitis may also cause hemorrhagic pneumonitis, adrenal hemorrhage, and subarachnoid hemorrhage.
14. Plague: thrombocytopenia or disseminated intravascular coagulation; severe vasculitis is complicated by purpura and gangrene resulting in the name, "The Black Death."
16. Rickettsial diseases: severe course may be complicated by petechiae, purpura, thrombocytopenia, or disseminated intravascular coagulation.
17. Rift Valley fever: uncommon complication with petechiae, purpura, gingival bleeding, hematemesis, and melena.
19. Tuberculosis: septic miliary tuberculosis may be complicated by disseminated intravascular coagulation.
20. Variola (smallpox): was uncommonly complicated by a fatal hemorrhagic diathesis before the classic rash appeared.
21. Visceral leishmaniasis: disseminated intravascular coagulation (rare) and gastrointestinal hemorrhage complicating portal hypertension.

22. Yellow fever: petechiae, purpura, gingival bleeding, oozing from mucous membranes, hematemesis, melena, and metrorrhagia; bleeding itself rarely life-threatening.

Blood cell disorders

I. Anemia

Introduction

Anemia is a common primary health care problem in the tropics affecting a majority of members in many communities. A distinct feature of tropical anemia is that it is often due to concurrent, multiple etiologies, the most important being anemias due to hemoglobinopathies, malnutrition, malabsorption, and chronic infection. The multifactorial nature of tropical anemia complicates diagnosis and treatment, especially in developing communities where there is a sparsity of sophisticated diagnostic facilities or health care personnel adequately trained in hematology.

The prevalence of anemia in the tropics is often seasonal, varying with the wet seasons when malaria transmission is at its highest. The prevalence of malaria also varies with the altitude as anemia is more common in the lowlands.

The signs and symptoms of anemia include headache, vertigo, dizziness, orthostatic syncope, tinnitus, retinal hemorrhage, dyspnea, tachycardia, palpitations, heart murmur, anorexia, oligomenorrhea, urinary frequency, malaise, and low-grade fever. Anemia may be complicated by weight loss, and high output cardiac failure as the hemoglobin falls below 7 gm/dl.

Hypochromic Microcytic Anemia:

Beta thalassemia and other thalassemia variants: with normal iron stores in the bone marrow, and increased hemoglobin A2 and hemoglobin F.

Iron deficiency: with absent bone marrow iron stores, and decreased serum iron but increased total iron binding capacity.
a. Dietary deficiency: malabsorption syndromes and tropical sprue, iron-poor diet, breast feeding, and failure to meet increased dietary requirements of pregnancy and growth.

b. Chronic blood loss: recurring trauma or peripartum, chronic bleeding skin lesions, hemorrhagic disorders, amebiasis (complicated by bloody diarrhea), ascariasis (in pediatric patients), fasciolopsiasis, hookworm disease, menorrhagia, schistosomiasis (chronic hematuria with bladder involvement), trichostrongyliasis, and trichuriasis.

Lead poisoning: basophilic stippling.

Normochromic Normocytic Anemia (excluding hemolytic anemia):

1. Acute blood loss: trauma or peripartum, ascorbic acid deficiency with hemorrhagic complications, and other hemorrhagic complications (see Hemorrhagic Complications Section above).
2. Amphotericin-B therapy.
3. Anemia of chronic infection: amebiasis, histoplasmosis (rare complication with disseminated disease), leprosy, melioidosis (with chronic suppurative or disseminated disease), schistosomiasis, and tuberculosis.
4. Anemia of chronic liver disease.
5. Anemia of chronic renal failure.
6. Anemia of connective tissue disease.
7. Anemia of hypothyroidism: hypothyroidism is often not treated in developing communities with inadequate primary health care.
8. Aplastic anemia.
10. Myelophthisic: with infiltration of the bone marrow by malignant cells or fibrosis.
11. Severe protein malnutrition.

Megaloblastic Anemia:

1. Cyanocobalamin (vitamin B12) deficiency: especially when the increased dietary requirements in pregnancy or when infants are breast fed by cyanocobalamin deficient mothers (most often dependent on vegetarian diet), chronic pancreatitis, pernicious anemia (uncommon in the tropics), and in malabsorption syndromes.
2. Diphyllobothriasis.
3. Folate deficiency: when increased dietary requirements in pregnancy are not met, alcoholism, in malabsorption syndromes, chronic hemolytic anemia, and sickle cell anemia.

4. Giardiasis.
5. Kwashiorkor.
6. Tropical sprue.

Hemolytic Anemia:

1. African trypanosomiasis.
2. Babesiosis.
3. Bacterial septicemia.
4. Bartonellosis.
6. Disseminated intravascular coagulation.
8. Eclampsia.
9. G-6-PD deficiency: after use of certain drugs.
10. Leptospirosis.
11. Malaria: especially when G-6-PD deficient patients are treated with 8-aminoquinolones.
12. Malignant hypertension.
14. Red cell antibodies: Rh and ABO incompatibilities, and other immunohemolytic anemias.
15. Shigellosis.
16. Sickle cell anemia, including hemoglobin C, D, and E disorders.
17. Snake bite (venomous).
18. Thalassemia.
19. Thrombotic thrombocytopenic purpura.
20. Typhoid fever.

Anemia Secondary To Hypersplenism:

1. Splenomegaly may result in sequestration and destruction of red blood cells resulting in anemia. The differential diagnosis of splenomegaly is addressed in the Splenomegaly Section below and in the Hepatomegaly That May Be Associated With Splenomegaly Section of the Hepatobiliary Complications Chapter. The most common tropical infectious diseases associated with splenomegaly and anemia are malaria, schistosomiasis, and visceral leishmaniasis.
2. Tropical splenomegaly syndrome (see Splenomegaly Section below).

II. Eosinophilia

Eosinophilia is a common clinical finding in the tropics and is primarily associated with allergic, helminthiic, and mycotic diseases.

1. Abdominal angiostrongyliasis.
2. Allergic diseases: allergic rhinitis, anaphylaxis, asthma, chronic urticaria, dermatitis herpetiformis, and drug reactions.
3. Angiostrongyliasis.
4. Anisakiasis.
5. Ascariasis.
6. Aspergillosis.
7. Chlamydial pneumonitis of the newborn.
8. Clonorchiasis.
10. Collagen vascular disorders: allergic angitis, allergic fascitis, and polyarteritis nodosum.
11. Cryptosporidiosis.
12. Cutaneous larva migrans due to dog or cat hookworm.
13. Cysticercosis.
15. Dracunculiasis.
16. Echinococcosis due to Echinococcus granulosus.
17. Entomophthoromycosis due to Basidiobolus.
18. Fascioliasis.
19. Fasciolopsiasis.
20. Filarial hypereosinophilia.
22. Heterophyiasis.
24. Hymenolepiasis.
25. Isosporiasis (other than due to Toxoplasma gondii): rare disease.
26. Loiasis.
27. Malignancy.
29. Opisthorchiasis.
30. Paragonimiasis.
31. Schistosomiasis.
32. Strongyloidiasis (threadworm).
33. Taeniasis.
34. Toxocariasis.
35. Trichinosis.
36. Trichostrongyliaiasis.
III. Suppression of cell-mediated and humoral immunity

Malnutrition, chronic infections, and acquired immune deficiency syndrome are frequent etiologies for suppression of cell-mediated and humoral immunity in the tropics.

Tropical infections implicated in inducing immune suppression are:

1. Acquired immune deficiency syndrome.
3. Filariasis due to Brugia malayi.
4. Leishmaniasis.
5. Leprosy.
6. Malaria.
7. Schistosomiasis.
8. Tuberculosis.

Opportunistic infections associated with immune suppression are:

1. Amebiasis.
2. Aspergillosis.
3. Atypical mycobacterioses.
5. Coccidiomycosis.
6. Cryptococcosis.
7. Cryptosporidiosis.
8. Cytomegalovirus infections.
10. Giardiasis.
12. Herpes simplex (disseminated).
13. Histoplasmosis.
15. Listeriosis.
17. Nocardiosis.
18. Pneumococcal infections and other streptococcal infections: in splenectomized patients or patients with sickle cell anemia.
19. Pneumocystosis.
20. Salmonellosis: in patients with sickle cell anemia.
21. Toxoplasmosis.
22. Tuberculosis.
23. Strongyloidiasis.
24. Varicella-zoster infection.
Lymphatic complications

I. Generalized lymphadenopathy

Diseases that may be complicated by a symmetric, generalized lymphadenopathy are:

1. American Trypanosomiasis.
2. Arthropod-borne viral arthritis and rash (Ross River fever).
3. Argentine and Bolivian hemorrhagic fevers.
4. Bartonellosis.
5. Brucellosis.
6. Dengue fever.
8. Epstein-Barr virus disease.
12. Leptospirosis.
13. Lyme disease.
14. Lymphoma.
17. Rickettsial pox.
18. Rubeola (measles).
19. Schistosomiasis.
20. Visceral leishmaniasis.
21. West Nile fever.

II. Preauricular lymphadenopathy

Adenoviral or enteroviral hemorrhagic conjunctivitis or inclusion conjunctivitis may be complicated by preauricular lymphadenopathy.

III. Cervical lymphadenopathy

Cervical lymphadenopathy is associated with:

1. African trypanosomiasis.
3. Chikungunya fever.
4. Diphtheria.
5. Epstein-Barr virus infection.
7. O'nyong nyong fever.
8. Psittacosis.
10. Toxoplasmosis.
11. Tuberculosis: suppurative lymphadenitis.

IV. Hilar lymphadenopathy

Several infections may present with hilar adenopathy as a clinical finding on chest X-ray.

1. Anthrax.
3. Coccidiomycosis.
5. Filarial hypereosinophilia: an uncommon clinical finding.
6. Histoplasmosis: a rare clinical finding, may be accompanied by mediastinitis.
7. Toxoplasmosis.
8. Tuberculosis.

V. Inguinal lymphadenopathy

Inguinal lymphadenopathy often progresses to lymphadenitis or suppurative, matted buboes. Sexually transmitted diseases are the most common cause for inguinal lymphadenopathy. The differential diagnosis includes:

2. Filariasis: acute infection is associated with epididymitis and orchitis; chronic and recurrent infections are associated with elephantiasis of the lower extremities.
3. Granuloma inguinale: often with overlying granulomatous ulcers in the inguinal area.
4. Lymphogranuloma venereum: often with multiple, bilateral buboes.
5. Mayaro fever.
6. Onchocerciasis: lymphadenitis, may involve the femoral nodes as well.
7. Streptocerciasis.
8. Trichomoniasis.
VI. Regional lymphadenopathy

The following infections may induce lymphadenopathy in any lymph node region. The lymphadenopathy is usually asymmetric and adjacent to an obvious skin lesion or arthropod bite lesion. The infection may progress to suppurative lymphadenitis.

1. African trypanosomiasis.
2. Anthrax.
3. Cutaneous leishmaniasis.
5. Leprosy.
8. Pinta.
10. Scrub typhus: lymphadenitis.
12. Tick-borne rickettsioses of the eastern hemisphere (Boutonneuse fever, Siberian tick typhus, Queensland tick typhus): lymphadenitis.
15. Yersiniosis: may be complicated by mesenteric lymphadenitis, which may be confused with acute appendicitis.

Splenomegaly

I. Tropical splenomegaly syndrome

Tropical splenomegaly syndrome is associated with chronic malaria infection and is distributed in Africa, the Indian subcontinent, and Southeast Asia. The syndrome is characterized by significant splenomegaly, cellular infiltration of the liver, and high concentrations of immunoglobulin-M, other specific malarial antibodies, rheumatoid factor, cold agglutinins, autoantibodies, and circulating immune complexes. Massive sequestration of blood in the spleen leads to anemia. There is progressive disability, an increased susceptibility to infection, and a high mortality rate. Prolonged administration of antimalarial drugs and supportive measures may reduce mortality, but this effect is variable. Splenectomy would significantly increase the mortality rate and is not indicated. The current theory is that this syndrome is due to an unregulated immune response to chronic malaria antigenemia.
II. Splenomegaly

Splenomegaly may be a clinical finding in:

1. African trypanosomiasis.
2. Beta thalassemia major: early in the course of the illness until splenectomy.
5. Crimean-Congo hemorrhagic fever.
7. Sickle cell anemia: early in the course of the illness.
8. Trench fever.
9. Tropical splenomegaly syndrome.
10. Tuberculosis.

III. Splenomegaly that may be associated with hepatomegaly

The following diseases may have splenomegaly as a clinical finding, but may also be associated with hepatomegaly:

1. American trypanosomiasis.
2. Babesiosis.
5. Histoplasmosis.
7. Leptospirosis.
8. Malaria.
11. Paratyphoid fever.
13. Psittacosis.
15. Schistosomiasis.
16. Toxoplasmosis: including the congenital form.
17. Tularemia.
18. Typhoid fever.
19. Visceral leishmaniasis: splenomegaly very prominent.
20. West Nile fever.
Bibliography


