:
Title: ***Professional Guide to Diseases, 9th Edition***

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Infection

Introduction

Despite improved treatments and prevention, including potent antibiotics, complex immunizations, and modern sanitation, infection still causes much serious illness, even in highly industrialized countries. In developing countries, infection is a critical health problem.

What is infection?

Infection is the invasion and multiplication of microorganisms in or on body tissue that produce signs and symptoms as well as an immune response. Such reproduction injures the host by causing cellular damage from microorganism-produced toxins or intracellular multiplication or by competing with host metabolism. The host's own immune response may increase tissue damage, which may be localized (as in infected pressure ulcers) or systemic. The infection's severity depends on the pathogenicity and amount of the invading microorganisms and on the strength of host's defenses. The very young and the very old are most susceptible to infections.

Microorganisms that cause infectious diseases are difficult to overcome for many reasons:

- Some bacteria develop a resistance to antibiotics.
- Some microorganisms, such as human immunodeficiency virus, include many different strains and a single vaccine can't provide protection against them all.
- Most viruses resist antiviral drugs.
- Some microorganisms localize in areas that make treatment difficult, such as the central nervous system and bone.

Also, certain factors that normally contribute to improved health, such as availability

of good nutrition, clean living conditions, and advanced medical care, can actually lead to increased risk for infection. For example, travel can expose people to diseases that they have little natural immunity against. Increased use of immunosuppressants, surgery, and other invasive procedures also increases the risk for infection.

Kinds of infections

A laboratory-verified infection that causes no signs and symptoms is called a *subclinical, silent,* or *asymptomatic infection.* A multiplication of microbes that produces no signs, symptoms, or immune response is called a *colonization.* A person with a subclinical infection or colonization may be a carrier and transmit the infection to others. A *latent infection* occurs after a microorganism has been dormant in the host, sometimes for years. An *exogenous infection* results from environmental pathogens or sources other than the host; an *endogenous infection*, results from the host's normal flora (for instance, *Escherichia coli* displaced from the colon, which causes urinary tract infection).

Microorganisms responsible for infectious diseases include bacteria, viruses, rickettsiae, chlamydiae, fungi (yeasts and molds), and protozoa; larger organisms such as helminths (parasitic worms) may also cause infectious disease.

Bacteria are single-cell microorganisms with well-defined cell walls that can grow independently on artificial media without the need for other cells. Bacteria inhabit the intestines of humans and other animals as normal flora used in the digestion of food. Also found in soil, bacteria are vital to soil fertility. These microorganisms break down dead tissue, which allows it to then be used by other organisms.

Despite the many types of known bacteria, only a small percentage are harmful to man. (See *How bacteria damage tissue*.) In developing countries, where poor sanitation increases the risk of infection, bacterial diseases commonly cause death and disability. In industrialized countries, bacterial infections are the most common fatal infectious diseases.

Bacteria are classified by shape. Spherical bacterial cells are called *cocci;* rod-shaped bacteria, *bacilli;* and spiral-shaped bacteria, *spirilla.* Bacteria are also classified according to their response to staining (gram-positive, gram-negative, or acid-fast bacteria); their motility (motile or nonmotile bacteria); their tendency toward encapsulation (encapsulated or nonencapsulated bacteria); and their capacity to form spores (sporulating or nonsporulating bacteria).

Spirochetes are bacteria with flexible, slender, undulating spiral rods that have cell walls. Most are anaerobic. The three forms pathogenic in humans include *Treponema, Leptospira,* and *Borrelia.*

Viruses are subcellular organisms made up only of a ribonucleic acid or a deoxyribonucleic acid nucleus covered with proteins. They're the smallest known organisms (so tiny they're visible only through an electron microscope). Independent of host cells, viruses can't replicate. Rather, they invade a host cell and stimulate it to participate in the formation of additional virus particles. The estimated 400 viruses that infect humans are classified according to their size, shape (spherical, rod shaped, or cubic), or means of transmission (respiratory, fecal, oral, or sexual).

Rickettsiae are relatively uncommon in the United States. They're small, gram-negative organisms classified as bacteria that commonly induce life-threatening infections. Like viruses, they require a host cell for replication. Three genera of rickettsiae include *Rickettsia*, *Coxiella*, and *Rochalimaea*.

Chlamydiae are smaller than rickettsiae and bacteria but larger than viruses. They also depend on host cells for replication but, unlike viruses, they're susceptible to antibiotics.

Fungi are single-cell organisms, with nuclei enveloped by nuclear membranes. They have rigid cell walls like plant cells but lack chlorophyll, the green matter necessary for photosynthesis; they also show relatively little cellular specialization. Fungi occur as yeasts (single-cell, oval-shaped organisms) or molds (organisms with hyphae, or branching filaments). Depending on the environment, some fungi may occur in both forms. Fungal diseases in humans are called *mycoses*.

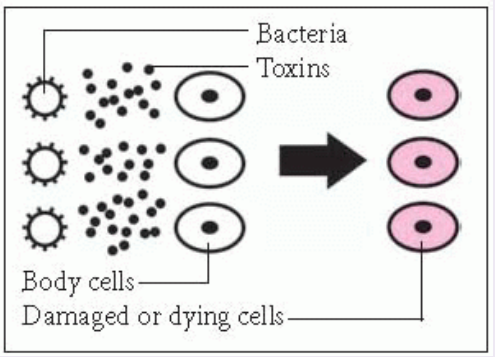
PATHOPHYSIOLOGY

HOW BACTERIA DAMAGE TISSUE

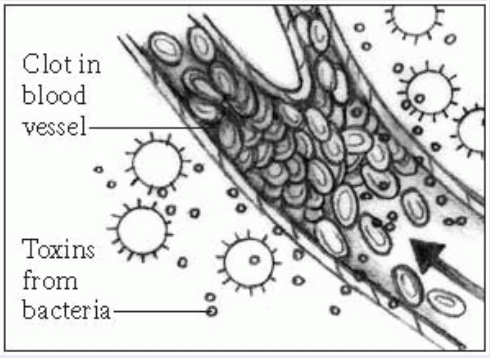
The human body is constantly infected by bacteria and other infectious organisms. Some are beneficial such as the intestinal bacteria that produce vitamins. Others are harmful, causing illnesses ranging from the common cold to life-threatening septic shock.

To infect a host, bacteria must first enter it. They do this either by adhering to the mucosal surface and directly invading the host cell, or by attaching to epithelial cells and producing toxins that eventually invade the host cells. To survive and multiply within a host, bacteria or their toxins adversely affect biochemical reactions in cells. This causes a disruption of normal cell function or cell death (see illustration below). For example, the diphtheria toxin damages heart muscle by inhibiting protein synthesis. Also,

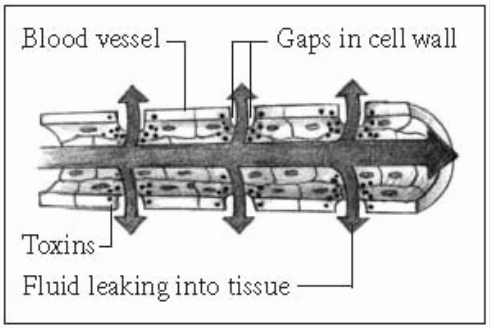
as some organisms multiply, they extend into deeper tissues and eventually gain access to the bloodstream.



Some toxins cause blood to clot in the smaller blood vessels. As a result, the tissues supplied by these vessels may become deprived of blood and subsequently damaged (see illustration below).



Other toxins can damage the cell walls of the smaller blood vessels, causing leakage. This fluid loss can result in decreased blood pressure, which in turn impairs the heart's ability to pump enough blood to the vital organs (see illustration below).



Protozoa are the simplest single-cell organisms among animals. However, they show a high level of cellular specialization. Like other animal cells, they have cell membranes rather than cell walls, and their nuclei are surrounded by nuclear membranes.

In addition to these microorganisms, infectious diseases may also result from larger parasites, such as roundworms or flatworms.

Modes of transmission

Most infectious diseases are transmitted in one of four ways. (See *Standard precautions,* pages 898 and 899 and *CDC isolation precautions*, page 901.)

STANDARD PRECAUTIONS

The Centers for Disease Control and Prevention recommends that the following standard blood and body-fluid precautions be used for *all* patients. This is especially important in emergency care settings, where the risk of blood exposure is high and the patient's infection status is usually unknown.

It's important to remember that implementing standard precautions doesn't eliminate the need for maintaining other transmission-based precautions designed for specific airborne, droplet, and contact infectious diseases.

Sources of potential exposure

Standard precautions apply to blood, semen, vaginal secretions, and cerebrospinal, synovial, pleural, peritoneal, pericardial, and amniotic fluids. Standard precautions also apply to other body substances, such as feces, urine, nasal secretions, saliva, sputum, tears, vomitus, and breast milk.

Barrier precautions

- Wear gloves when touching blood and body fluids, mucous membranes, or the broken skin of patients; when handling items or touching surfaces soiled with blood or body fluids; and when performing venipuncture and other vascular access procedures.
- Change gloves and wash hands after contact with each patient.
- Wear a mask and protective eyewear, or a face shield, to protect the mucous membranes of the mouth, nose, and eyes

during procedures that may generate the splatter of blood or other body fluids.

- In addition to the mask and protective eyewear or face shield, wear a gown or an apron during procedures that are likely to cause splashing of blood or other body fluids.
- After removing gloves and other protective equipment, thoroughly wash hands and other skin surfaces that may be contaminated with blood or other body fluids.

Precautions for invasive procedures

- During all invasive procedures, wear gloves and a surgical mask and goggles or a face shield as appropriate.
- During procedures that commonly cause droplets or splashes of blood or other body fluids, or for those that generate bone chips, wear protective eyewear and a surgical mask or a face shield.
- During invasive procedures that are likely to cause splashing or a splattering of blood or other body fluids, wear a gown or an impervious apron.
- If performing or assisting in a vaginal or cesarean delivery, wear gloves and a gown when handling the placenta or the infant and during umbilical cord care.

Work practice precautions

- To prevent needle-stick injuries, don't recap used needles, bend or break needles, remove needles from their disposable syringes or phlebotomy blood tube holders, or manipulate them.
- Use sharps safety devices. Activate safety mechanisms as directed.
- Place disposable syringes and needles, scalpel blades, and other sharps items in puncture-resistant containers for disposal. Make sure these containers are always located near the area of use.
- Place large-bore reusable needles in a puncture-resistant container for transport to the reprocessing area immediately after a procedure.

- If a glove tears or a needle-stick or other injury occurs, remove the gloves, wash your hands and the site of the needle-stick thoroughly, and put on new gloves as quickly as patient safety permits. Remove the needle or instrument involved in the incident from the sterile field. Promptly report injuries and mucous-membrane exposure to the appropriate infection control practitioner per facility protocol.

Hand-hygiene, either by washing with soap and water or by sanitizing with an alcohol-based sanitizer, is recognized as the most effective method of interrupting the transmission of infection. Plain soap is adequate for removing visible soil. Antimicrobial soap is encouraged for washing after contamination with blood or body fluids. Alcohol-based hand sanitizers, which reduce the number of viable microorganisms on the hands, are designed for waterless use. In the United States, these products usually contain 60% to 95% ethanol or isopropanol.

Indications for washing with either ordinary or antimicrobial soap include:

- when hands are visibly dirty or contaminated with proteinaceous material or visibly soiled with blood or other body fluids (even if gloves were worn)
- before eating and after using the restroom
- exposure to suspected or proven *Bacillus anthracis* (alcohol, chlorhexidine, iodophors, and other antiseptic agents have a poor potency against its spores)
- after caring for a patient with *Clostridium difficile* (alcohol, chlorhexidine, iodophors, and other antiseptic agents are largely ineffective against its spores). Alcohol-based hand sanitizers may be used in all other clinical situations if the hands aren't visibly soiled.

Additional precautions

- Make sure mouthpieces, one-way valve masks, resuscitation bags, and other ventilation devices are available in areas where the need for resuscitation is likely. *Note*: Saliva has not been implicated in human immunodeficiency virus transmission.

- If you have any exudative lesions or weeping dermatitis, refrain from direct patient care and from handling patient care equipment until the condition resolves.

- In *contact transmission*, the susceptible host comes into direct contact (as in contact with blood or body fluids) or indirect contact (contaminated inanimate objects or the close-range spread of respiratory droplets) with the source. The most common method of contact transmission is contaminated hands.
- *Airborne transmission* results from the inhalation of contaminated aerosolized droplet nuclei (as in pulmonary tuberculosis).
- In *enteric (oral-fecal) transmission*, the infecting organisms are found in feces and are ingested, in many cases through fecally contaminated food or water (as in salmonella infections).
- *Vector-borne transmission* occurs when an intermediate carrier (vector), such as a flea, mosquito, or other animal, transfers an organism.

Many actions can be taken to prevent the transmission of infectious diseases including:

- comprehensive immunization (including required immunization of travelers to, or emigrants from, endemic areas)
- drug prophylaxis
- improved nutrition, living conditions, and sanitation
- correction of environmental factors
- widespread disease tracking.

Immunization can now control many diseases, including diphtheria, tetanus, pertussis, measles, rubella, some forms of meningitis, poliovirus, hepatitis B, pneumococcal pneumonia, influenza, rabies, and tetanus. Smallpox (variola)—which killed and disfigured millions—was believed to have been successfully eradicated by a comprehensive World Health Organization program of surveillance and immunization. However, in light of recent concerns regarding bioterrorism, smallpox is considered a potential agent. Health care personnel must recognize potential cases of smallpox and initiate appropriate precautions as well as notify health department officials. Smallpox vaccination may be appropriate for certain emergency and first-response health care providers.

Vaccines, which contain live but attenuated (weakened) or killed microorganisms, and toxoids, which contain modified bacterial exotoxins, induce active immunity against bacterial and viral diseases by stimulating antibody formation. Natural

active immunity is produced as a patient who has the disease forms antibodies against it, thus preventing the recurrence of the disease. Immune globulins contain previously formed antibodies from hyperimmunized donors or pooled plasma and provide temporary passive immunity. Generally, passive immunization is used when active immunization is perilous or impossible or when complete protection requires both active and passive immunization. It may also be appropriate in situations requiring immediate protection such as postexposure in which active immunity from immunizations takes too long to provide the necessary and immediate protection. Maternal passive immunity crosses the placental barrier from mother to fetus and is also provided to the infant by antibodies present in breast milk.

Although preventive antibiotic therapy may prevent certain diseases, the risk of superinfection and the emergence of drug-resistant strains may outweigh the benefits. Therefore, preventive antibiotics are usually reserved for patients at high risk for exposure to dangerous infections. Antibiotic-resistant bacteria are on the rise mainly because antibiotics have been misused and overused. Some bacteria, such as enterococci, have developed mutant strains that don't respond to antibiotic therapy.

Health care-associated infections

A *health care-associated infection* is an infection that develops as a result of health care. Health care-associated infections were previously known as *nosocomial infections,* but the name was updated because these infections may be acquired from, or associated with, any portion of the health care-delivery system, including such areas as outpatient care, ambulatory care, home care, or long-term care.

Health care-associated infections are usually transmitted by direct contact. Less commonly, transmission occurs by inhalation or by contact with contaminated equipment and solutions. Contamination of solutions during the manufacturing process is rare.

Despite facility programs of infection control that include surveillance, prevention, and education, about 5% of patients who enter health care facilities contract a health care-associated infection. Staphylococcal infections, which had been declining since the 1960s, are currently a common cause of infection. In contrast, gram-negative bacilli, resistant enterococci, and fungal infections are also on the rise.

Health care-associated infections continue to be a difficult problem, because today's hospital patients are older and more debilitated with chronic underlying diseases than in the past. Also, the increased use of invasive and surgical procedures, immunosuppressants, and antibiotics predisposes patients to infection

and superinfection. At the same time, the growing number of personnel who can come in contact with each patient makes the risk of exposure greater.

The following measures can help prevent health care-associated infections:

- Follow strict infection-control procedures. (See *Standard precautions*, pages 898 and 899. See also *CDC isolation precautions*.)
- Document hospital infections as they occur.
- Identify outbreaks early, and take steps to prevent their spread.

CDC ISOLATION PRECAUTIONS

To help health care facilities maintain up-to-date isolation practices, the Centers for Disease Control and Prevention (CDC) and the Hospital Infection Control Practices Advisory Committee have developed the CDC's *Guideline for Isolation Precautions in Hospitals.*

Standard precautions

The revised guidelines contain two tiers of precautions. The first tier, called *standard precautions,* is designated for the care of all hospital patients regardless of their diagnosis or presumed infection. Standard precautions are the primary strategy for preventing nosocomial infection and take the place of universal precautions. These precautions apply to:

- blood
- all body fluids, secretions, and excretions —except sweat— regardless of whether or not they contain visible blood
- skin that isn't intact
- mucous membranes.

Transmission-based precautions

The second tier of precautions is known as *transmission-based precautions.* These precautions are instituted for patients who are known to be, or suspected of being, infected with a highly transmissible infection—one that requires precautions beyond those set forth for standard precautions. There are three types of transmission-based precautions: airborne; droplet; and contact precautions.

Airborne precautions

Airborne precautions are designed to reduce the risk of airborne transmission of infectious agents. Microorganisms carried through the air can be widely dispersed by air currents, making them available for inhalation or deposit on a susceptible host in the same room or at a longer distance from the infected patient if ventilation creates shared air space.

Airborne precautions include special air handling and ventilation procedures to prevent the spread of infection. They also require the use of respiratory protection such as a respirator (the N95 or higher disposable respirator or a powered air-purifying respirator) —in addition to standard precautions—when entering an infected patient's room.

Droplet precautions

Droplet precautions are designed to reduce the risk of transmitting infectious agents through large-particle (exceeding 5 micrometers) droplets. Such transmission involves the contact of infectious agents to the conjunctivae or to the nasal or oral mucous membranes of a susceptible person. Large-particle droplets don't remain in the air and generally travel short distances of 3’ (1 m) or less. They require the use of a surgical mask—in addition to standard precautions —to protect the mucous membranes.

Contact precautions

Contact precautions are designed to reduce the risk of transmitting infectious agents by direct or indirect contact. Direct-contact transmission can occur through patient care activities that require physical contact. Indirect-contact transmission involves a susceptible host coming in contact with a contaminated object, usually inanimate, in the patient's environment or with items contaminated with the patient's secretions, excretions, or blood outside of the patient's environment that may have been removed from the environment without appropriate cleaning and disinfection.

Contact precautions require the use of gloves and a gown—in addition to standard precautions—to avoid contact with the infectious agent. A mask is only required if there's a chance of splash or splatter of body fluids to the face. Stringent hand-hygiene is also necessary after removal of the protective items.

IMMUNIZATION SCHEDULE

Before immunization, ask the parents if the child is receiving corticosteroids or other drugs that suppress the immune response or if he had a recent febrile illness. Obtain a history of allergic responses, especially to antibiotics, eggs, feathers, and past immunizations. Keep in mind that a child who's at risk for acquired immunodeficiency syndrome or who tests positive for human immunodeficiency virus infection may need special consideration.

After immunization, tell the parents to watch for and report reactions other than local swelling and pain and mild temperature elevation. Give them the child's immunization record. The following are the 2007 general vaccine recommendations approved by the Advisory Committee on Immunization Practices, the American Academy of Pediatrics, and the American Academy of Family Physicians.

Age	Immunization
Birth	HepB
1 to 4 months	HepB
2 months	DTaP, HIB, IPV, PCV, Rota
4 months	DTaP, HIB, IPV, PCV, Rota
6 months	DTaP, HIB, PCV, Rota
6 to 18 months	HepB, IPV
12 to 15 months	HIB, MMR, PCV, HepA, varicella
6 to 59 months	Influenza (yearly)
12 to 23 months	HepA
15 to 18 months	DTaP, HepA
4 to 6 years	DTaP, IPV, MMR, varicella

11 to 12 years	HPV, Tdap, MCV4
15 years	Tdap, MCV4

- Eliminate unnecessary procedures that contribute to infection.
- Strictly follow necessary isolation techniques.
- Observe *all* patients for signs of infection, especially those patients at high risk.
- Always follow proper hand-hygiene technique and encourage other staff members to follow these guidelines as well.
- Keep staff members and visitors with obvious infection and well-known carriers away from susceptible, high-risk patients.
- Take special precautions with vulnerable patients, such as those with indwelling urinary catheters, mechanical ventilators, or I.V. lines and those recovering from surgery.

Accurate assessment vital

Accurate assessment helps identify infectious diseases and prevents avoidable complications. Complete assessment consists of patient history, physical examination, and laboratory data. The history should include the patient's sex, age, address, occupation, and place of work; known exposure to illness and recent medications, including antibiotics; and date of disease onset. Signs and symptoms, including their duration and whether they occurred suddenly or gradually, should be included in the history as well as precipitating factors, relief measures, and weight loss or gain. Detail information about recent hospitalization, blood transfusions, blood donation denial by the Red Cross or other agencies, recent travel or camping trips, exposure to animals, and vaccinations. (See *Immunization schedule*.) If applicable, ask about possible exposure to sexually transmitted diseases or about drug abuse. Also, try to determine the patient's resistance to infectious disease. Ask about usual dietary patterns, unusual fatigue, and any conditions, such as neoplastic disease or alcoholism, that may predispose him to infection. Notice if the patient is listless or uneasy, lacks concentration, or has any obvious abnormality of mood or affect.

In suspected infection, a physical examination must assess the skin, mucous membranes, liver, spleen, and lymph nodes. Check for and make note of the location and type of drainage from any skin lesions. Record skin color, temperature, and turgor; ask if the patient has pruritus. Take his temperature, using the same route consistently, and watch for a fever, which is the best indicator of many infections. (Keep in mind that some patients, such as those who are immunocompromised, are unable to spike a fever.) Note and record the

pattern of temperature change and the effect of antipyretics. Be aware that certain analgesics may contain antipyretics. With a high fever, especially in children, watch for seizures.

Check the pulse rate. Infection commonly increases the pulse rate, but some infections, notably typhoid fever and psittacosis, may decrease it. Also observe for increased respiratory rate or a change in mental status. In severe infection or when complications are possible, watch for hypotension, hematuria, oliguria, hepatomegaly, jaundice, bleeding from gums or into joints, and an altered level of consciousness. Obtain laboratory studies and appropriate cultures as ordered.

GRAM-POSITIVE COCCI

Staphylococcal infections

Staphylococci are gram-positive bacteria, either coagulase-negative (*Staphylococcus epidermidis*) or coagulase-positive (*Staphylococcus aureus*). Coagulase-negative staphylococci grow abundantly as normal flora on skin, but they can also cause boils, abscesses, and carbuncles. In the upper respiratory tract, they're usually nonpathogenic but can cause serious infections in some individual such as those who are immunocompromised. Pathogenic strains of staphylococci are found in many adult carriers —usually on the nasal mucosa, axilla, or groin. Sometimes, carriers shed staphylococci, infecting themselves or other susceptible people. Coagulase-positive staphylococci tend to form pus and cause many different types of infections. (See *Comparing staphylococcal infections,* pages 904 to 909.)

Methicillin-resistant Staphylococcus aureus infection

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a mutation of very common bacterium spread easily by direct personto-person contact. Once limited to large teaching hospitals and tertiary care centers, MRSA infection is now endemic in nursing homes, long-term care facilities, and community hospitals. It's also seen in patients who haven't been hospitalized, as community-acquired MRSA infections are increasing.

COMPARING STAPHYLOCOCCAL INFECTIONS				

Predisposing factors	Signs and symptoms	Diagnosis	Treatment	Special considerations
Bacteremia				
▪ Infected surgical wounds ▪ Abscesses ▪ Infected I.V. or intra-arterial catheter sites or catheter tips ▪ Infected vascular grafts or prostheses ▪ Infected pressure ulcers ▪ Osteomyelitis ▪ Parenteral drug abuse ▪ Source unknown (primary bacteremia) ▪ Cellulitis ▪ Burns ▪ Immunosuppression ▪ Debilitating diseases, such as chronic renal insufficiency or diabetes ▪ Infective endocarditis (coagulase-positive staphylococci) and subacute bacterial endocarditis (coagulase-negative staphylococci) ▪ Cancer (leukemia) or neutrophil nadir after	▪ Fever (high fever with no obvious source in children younger than age 1), shaking chills, tachycardia ▪ Cyanosis or pallor ▪ Confusion, agitation, stupor ▪ Skin microabscesses ▪ Joint pain ▪ Complications: shock; acute bacterial endocarditis (in prolonged infection; indicated by new or changing systolic murmur); retinal hemorrhages; splinter hemorrhages under nails and small, tender red nodes on pads of fingers and toes (Osler's nodes); abscess formation in skin, bones, lungs, brain, and kidneys; pulmonary emboli if tricuspid valve is infected ▪ Prognosis poor in patients older than age 60 or	▪ Blood cultures (two to four samples from different sites at different times): growing staphylococci and leukocytosis (usually 12,000 white blood cells [WBCs]/µl), with a shift to the left of polymorphonuclear leukocytes (70% to 90% neutrophils) ▪ Urinalysis may show microscopic hematuria ▪ Erythrocyte sedimentation rate (ESR) elevated, especially in chronic or subacute bacterial endocarditis ▪ Prolonged partial thromboplastic time and prothrombin time; low fibrinogen and platelet counts, and low factor assays; possible disseminated intravascular coagulation ▪ Cultures of urine, sputum, and skin lesions with discharge may identify primary	▪ Semisynthetic penicillins (oxacillin, nafcillin) or cephalosporins (cefazolin) given I.V. ▪ Vancomycin I.V. for patients with penicillin allergy or suspected methicillin-resistant organisms ▪ Possibly, probenecid given to partially prevent urinary excretion of penicillin and to prolong blood levels ▪ I.V. fluids to reverse shock ▪ Removal of infected catheter or foreign body ▪ Surgery	▪ Report infection to authorities as required. ▪ *S. aureus* bacteremia can be fatal within 12 hours. Be especially alert for it in debilitated patients with I.V. catheters or in those with a history of drug abuse. ▪ Administer antibiotics on time to maintain adequate blood levels, but give them slowly, using the prescribed amount of diluent, to prevent thrombophlebitis. ▪ Watch for signs of penicillin allergy, especially pruritic rash (anaphylaxis) and breathing difficulties. Keep epinephrine 1:1,000 and resuscitation equipment handy. Monitor the patient's vital signs, urine

chemotherapy or radiation	with chronic illness	infection site; chest X-rays and scans of lungs, liver, abdomen, and brain may assist with identification ▪ Echocardiogram may show heart valve vegetation		output, and mental state for signs of shock. ▪ Obtain cultures carefully, and observe for clues to the primary site of infection. Never refrigerate blood cultures; it delays identification of organisms by slowing their growth. ▪ Impose contact precautions if the primary site of infection is draining. Special blood precautions are not necessary because the number of organisms present, even in fulminant bacteremia, is minimal. Some facilities continue isolation if the patient has methicillin-resistant *Staphylococcus aureus* (MRSA) regardless of site. ▪ Obtain peak and trough levels of vancomycin to determine the adequacy of treatment. ▪ Administer vancomycin I.V. slowly over 1

				hour to avoid any adverse reactions.
Pneumonia				
▪ Immune deficiencies, especially in elderly and in children younger than age 2 ▪ Chronic lung diseases and cystic fibrosis ▪ Malignant tumors ▪ Antibiotics that kill normal respiratory flora but spare *S. aureus* ▪ Viral respiratory infections, especially influenza ▪ Hematogenous (bloodborne) bacteria spread to the lungs from primary sites of infection (such as heart valves, abscesses, and pulmonary emboli) ▪ Recent bronchial or endotracheal suctioning or intubation	▪ High temperature: adults, 103° to 105° F (39.4° to 40.6° C); children, 101° F (38.3° C) or above ▪ Cough, with purulent, yellow, or bloody sputum ▪ Dyspnea, crackles, and decreased breath sounds ▪ Pleuritic pain ▪ In infants: mild respiratory infection that suddenly worsens: irritability, anxiety, dyspnea, anorexia, vomiting, diarrhea, spasms of dry coughing, marked tachypnea, expiratory grunting, sternal retractions, and cyanosis ▪ Complications: necrosis, lung abscess, pyopneumothorax, empyema, pneumatocele, shock, hypotension, oliguria or anuria,	▪ WBC count may be elevated (15,000 to 40,000/µl in adults; 15,000 to 20,000/µl in children), with predominance of polymorphonuclear leukocytes ▪ Sputum Gram stain: mostly gram-positive cocci in clusters, with many polymorphonuclear leukocytes ▪ Sputum culture: mostly coagulase-positive staphylococci ▪ Chest X-rays: usually patchy infiltrates ▪ Arterial blood gas analysis: hypoxia and respiratory acidosis	▪ Semisynthetic penicillins (oxacillin, nafcillin) or cephalosporins (cefazolin) given I.V. ▪ Vancomycin I.V. for patients with penicillin allergy or suspected methicillin-resistant organisms ▪ Isolation until sputum shows minimal numbers of *S. aureus* (about 24 to 72 hours after starting antibiotics)	▪ The Centers for Disease Control and Prevention's isolation guidelines require standard precautions unless MRSA, which requires contact precautions, is present. ▪ Keep the door to the patient's room closed. Don't store extra supplies in his room. Empty suction bottles carefully. Disposable suction containers are preferred. ▪ When obtaining sputum specimens, make sure you're collecting thick sputum, not saliva. The presence of epithelial cells (found in the mouth, not lungs) indicates a poor specimen. ▪ Administer antibiotics strictly on time,

	cyanosis, loss of consciousness			but slowly. Watch for signs of penicillin allergy and for signs of infection at the I.V. sites. Change the I.V. site every third day. ▪ Perform frequent chest physical therapy. Do chest percussion and postural drainage after intermittent positive pressure breathing treatments. Concentrate on consolidated areas (revealed by X-rays or auscultation).
Enterocolitis				
▪ Broad-spectrum antibiotics (tetracycline, chloramphenicol, or neomycin) or aminoglycosides (tobramycin, streptomycin, or kanamycin) as prophylaxis for bowel surgery or treatment of hepatic coma ▪ Usually occurs in elderly patients, but also in neonates (associated with	▪ Sudden onset of profuse, watery diarrhea usually 2 days to several weeks after start of antibiotic therapy, I.V. or by mouth (P.O.) ▪ Nausea, vomiting, abdominal pain and distention ▪ Hypovolemia and dehydration (decreased skin turgor, hypotension, fever)	▪ Stool Gram stain: many gram-positive cocci and polymorphonuclear leukocytes, with few gram-negative rods ▪ Stool culture: *S. aureus* ▪ Sigmoidoscopy: mucosal ulcerations ▪ Blood studies: leukocytosis, moderately increased blood urea nitrogen level, and	▪ Broad-spectrum antibiotics discontinued ▪ Possibly, antistaphylococcal agents such as vancomycin P.O. ▪ Normal flora replenished with yogurt that contains live cultures	▪ Monitor vital signs frequently to detect early signs of shock. ▪ Force fluids to correct dehydration. ▪ Know serum electrolyte levels. Measure and record bowel movements when possible. Check serum chloride level for alkalosis (hypochloremia). Watch for dehydration and

staphylococcal skin lesions)		decreased serum albumin level		electrolyte imbalance. ▪ Collect serial stool specimens for Gram stain and culture to confirm diagnosis. (The effectiveness of therapy is usually measured by clinical response.) ▪ Observe standard precautions. ▪ Follow reporting requirements, especially in a group situation such as a nursing home. They may vary per facility protocol.
Osteomyelitis				
▪ Hematogenous organisms ▪ Skin trauma ▪ Infection spreading from adjacent joint or other infected tissues ▪ *S. aureus* bacteremia ▪ Orthopedic surgery or trauma ▪ Cardiothoracic surgery ▪ Usually occurs in growing bones, especially femur and tibia, of	▪ Abrupt onset of fever— usually 101° F (38.3° C) or above; shaking chills; pain and swelling over infected area; restlessness; headache ▪ About 20% of children develop a chronic infection if not properly treated	▪ Possible history of prior trauma to involved area ▪ Positive bone and pus cultures (and blood cultures in about 50% of patients) ▪ X-ray changes apparent after second or third week ▪ ESR elevated with leukocyte shift to the left	▪ Surgical debridement ▪ Prolonged antibiotic therapy (4 to 8 weeks) ▪ Vancomycin I.V. for patients with penicillin allergy or methicillin-resistant organisms ▪ Possibly, removal of prosthesis or hardware	▪ Identify the infected area, and mark it on the care plan. ▪ Check the penetration wound from which the organism originated for evidence of present infection. ▪ Severe pain may render the patient immobile. If so, perform passive range-of-motion exercises. Apply

children younger than age 12 ▪ More common in males				heat as needed, and elevate the affected part. (Extensive involvement may require casting until the infection subsides.) ▪ Before procedures such as surgical debridement, warn the patient to expect some pain. Explain that drainage is essential for healing, and that he will continue to receive analgesics and antibiotics after surgery. ▪ Obtain a complete history of symptoms, recent meals, and other known cases of food poisoning.
Food poisoning				
▪ Enterotoxin produced by toxigenic strains of *S. aureus* in contaminated food (second most common cause of food poisoning in United States)	▪ Anorexia, nausea, vomiting, diarrhea, and abdominal cramps 1 to 6 hours after ingestion of contaminated food ▪ Symptoms usually subside within 18 hours, with complete	▪ Clinical findings sufficient ▪ Stool cultures: usually negative for *S. aureus* ▪ Epidemiologic history: if others are ill and food history is a commonality; health department	▪ No treatment necessary unless dehydration becomes a problem (usually in infants and elderly); oral rehydrating solution or I.V. therapy may be necessary to replace fluids	▪ Monitor vital signs, fluid balance, and serum electrolyte levels. ▪ Check for dehydration if vomiting is severe or prolonged and for decreased blood pressure.

	recovery occuring in 1 to 3 days	may be contacted for an outbreak		▪ Observe and report the number and color of stools. ▪ Report infection to authorities as required.
Skin infections				
▪ Decreased resistance ▪ Burns or pressure ulcers ▪ Decreased blood flow ▪ Possible skin contamination from nasal discharge ▪ Foreign bodies ▪ Underlying skin diseases, such as eczema and acne ▪ Common in people with poor hygiene living in crowded quarters ▪ Insulin-dependent diabetes mellitus ▪ Hemodialysis ▪ I.V. drug injection	▪ Cellulitis—diffuse, acute inflammation of soft tissue (no discharge) ▪ Pus-producing lesions in and around hair follicles (folliculitis) ▪ Boil-like lesions (furuncles and carbuncles) extend from hair follicles to subcutaneous tissues; these painful, red, indurated lesions are 1 to 2 cm and have a purulent yellow discharge. ▪ Small macules or skin blebs that may develop into vesicles containing pus (bullous impetigo); common in school-age children ▪ Mild or spiking fever ▪ Malaise	▪ Clinical findings and analysis of pus cultures if sites are draining ▪ Cultures of nondraining cellulitis taken from the margin of the reddened area by infiltration with 1 ml sterile saline solution (nonbacteriostatic saline) and immediate fluid aspiration	▪ Topical ointments; gentamicin or bacitracin-neomycinpolymyxin ▪ P.O. cloxacillin, dicloxacillin, or erythromycin; I.V. oxacillin or nafcillin for severe infection; I.V. vancomycin for methicillin-resistant organisms ▪ Application of heat to reduce pain ▪ Surgical drainage ▪ Identification and treatment of sources of reinfection (nostrils, perineum) ▪ Cleaning and covering the area with moist, sterile dressings	▪ Identify the site and extent of infection. ▪ Keep lesions clean with saline solution and peroxide irrigations, as ordered. Cover infections near wounds or the genitourinary tract with gauze pads. Keep pressure off the site to facilitate healing. ▪ Be alert for the extension of skin infections. ▪ Severe infection or abscess may require surgical drainage. Explain the procedure to the patient. Determine if cultures will be taken, and be prepared to collect a specimen. ▪ Impetigo is contagious. Isolate the patient and alert

	his family. Use contact precautions for all draining lesions.

Patients most at risk for MRSA infection include immunosuppressed patients, burn patients, intubated patients, and those with central venous catheters, surgical wounds, or dermatitis. Others at risk include those with prosthetic devices, heart valves, and postoperative wound infections. Other risk factors include prolonged hospital stays; extended therapy with multiple or broad-spectrum antibiotics; and close proximity to those colonized or infected with MRSA. Also at risk are patients with acute endocarditis, bacteremia, cervicitis, meningitis, pericarditis, and pneumonia.

Causes and incidence

MRSA enters health care facilities through an infected or colonized patient or a colonized health care worker. Although MRSA has been recovered from environmental surfaces, it's transmitted mainly by health care workers' hands. Many colonized individuals become silent carriers. The most frequent site of colonization is the anterior nares (40% of adults and most children become transient nasal carriers). Other, less common sites are the groin, axilla, and the gut. Typically, MRSA colonization is diagnosed by isolating bacteria from nasal secretions.

In individuals where the natural defense system breaks down, such as after an invasive procedure, trauma, or chemotherapy, the normally benign bacteria can invade tissue, proliferate, and cause infection. Today, up to 90% of *S. aureus* isolates or strains are penicillin resistant, and about 50% of all *S. aureus* isolates are resistant to methicillin, a penicillin derivative, as well as to nafcillin and oxacillin. These strains may also resist cephalosporins, aminoglycosides, erythromycin, tetracycline, and clindamycin.

MRSA infection has become prevalent with the overuse of antibiotics. Over the years, this has given once-susceptible bacteria the chance to develop defenses against antibiotics. This new capability allows resistant strains to flourish when antibiotics kill their more-sensitive cousins.

Complications

- Sepsis
- Death

Signs and symptoms

MRSA may start as small, red bumps that resemble pimples, boils, or spider bites. They can quickly turn into deep, painful abscesses. The bacteria can remain confined to the skin or they can burrow deep into the body and cause life-threatening infections in joints, bones, surgical wounds, heart valves, lungs, and the bloodstream.

Diagnosis

MRSA can be cultured from the suspected site with the appropriate method. For example, a wound can be swabbed for culture. Cultures of blood, urine, and sputum specimens will reveal sources of MRSA. Many laboratories use oxacillin disks to check for staphylococcus sensitivity when testing culture specimens; resistance to oxacillin indicates MRSA.

Treatment

To eradicate MRSA colonization in the nares, the physician may order topical mupirocin to be applied inside the nostrils. Other protocols involve combining a topical agent and an oral antibiotic. Most facilities keep patients in isolation until surveillance cultures are negative.

To attack MRSA infection, vancomycin is the drug of choice (see *Vancomycin-resistant infections,* page 910). A serious adverse effect (mostly caused by histamine release) is itching, which can progress to anaphylaxis. Some physicians also add rifampin, but whether rifampin acts synergistically or antagonistically when given with vancomycin is controversial.

Special considerations

- People in contact with the patient should perform hand-hygiene before and after patient care.

VANCOMYCIN-RESISTANT INFECTIONS

Some *Staphylococcus aureus* organisms have developed resistance to vancomycin. In some cases, the resistance is considered intermediatestrength resistance and is known as vancomycin-intermediate *S. aureus* (VISA). Another mutation, vancomycin-resistant *S. aureus* (VRSA), is fully resistant to vancomycin.

Researchers believe VISA and VRSA enter health care facilities through an infected or colonized patient or a colonized health

care worker. They spread through direct contact between the patient and caregiver or between patients. They may also be spread through patient contact with contaminated surfaces.

Patients with laboratoryconfirmed VISA or VRSA must be placed in a single room on contact precautions, and the number of health care workers involved in patient care should be limited. Other patients who shared the patient's room should be checked for VISA or VRSA colonization using an anterior nares culture. (Notify the laboratory to look specifically for *S. aureus* and to check sensitivity).

People involved in direct care of the patient before the initiation of contact precautions should be interviewed regarding the extent of their interactions. Those with extensive interaction should have an anterior nares culture. The local health department should be notified immediately. Antimicrobial treatment may include an increased dosage of vancomycin (for VISA only) and linezolid, quinupristin and dalfopristin, or a combination of other antimicrobials according to the sensitivity pattern of the organism.

- Good hand-hygiene is the most effective way to prevent MRSA infection from spreading.
- Use an antiseptic soap such as chlorhexidine. Bacteria have been cultured from worker's hands washed with milder soap. One study showed that, without proper hand-hygiene, MRSA could survive on health care workers' hands for up to 3 hours. Chlorhexidine has a residual antimicrobial effect on the skin.
- Contact isolation precautions should be used when in contact with the patient. A disinfected private room should be made available with dedicated equipment.
- Change gloves when contaminated or when moving from a “dirty” area of the body to a clean one.
- Instruct the patient's family and friends to wear protective clothing when they visit him, and show them how to dispose of it.
- Provide teaching and emotional support to the patient and his family members.
- Consider grouping infected patients together and having the same nursing staff care for them.
- Don't lay equipment used on the patient on the bed or bed stand. Be sure to wipe it with appropriate disinfectant before leaving the room.

- Ensure judicious and careful use of antibiotics. Encourage physicians to limit their use.
- Instruct the patient to take antibiotics for the full period prescribed, even if he begins to feel better.

Streptococcal infections

Streptococci are small gram-positive bacteria, spherical to ovoid in shape, and linked together in pairs or chains. Several species occur as part of normal human flora in the respiratory, GI, and genitourinary tracts. Although researchers have identified 21 species of streptococci, three classes— groups A, B, and D—cause most of the infections. (See *Comparing streptococcal infections*, pages 912 to 917.) Organisms belonging to groups A and B beta-hemolytic streptococci are associated with a characteristic

pattern of human infections. Most disorders due to group D streptococcus are caused by *Enterococcus faecalis*, formerly called *Streptococcus faecalis*, or *S. bovis*. Group C and group G streptococci have been identified as the etiologic agent in such infections as bacteremia, meningitis, pharyngitis, osteomyelitis, and neonatal sepsis.

Clinically, there are three states of streptococcal infection: carrier, acute, and delayed nonsuppurative complications. In the carrier state, the patient is infected with a disease-causing species of streptococci without evidence of infection. In the acute form, streptococci invade the tissues and cause physical symptoms. In the delayed nonsuppurative complications state, specific signs and symptoms associated with streptococcal infection occur. These include those associated with the inflammatory state of acute rheumatic fever, chorea, and glomerulonephritis. If further complications occur, they usually appear about 2 weeks after the acute illness, but they may be evident after a nonsymptomatic illness.

Necrotizing fasciitis

Most commonly referred to as *flesh-eating bacteria,* necrotizing fasciitis is a progressive, rapidly spreading inflammatory infection located in the deep fascia that destroys fascia and fat with secondary necrosis of subcutaneous tissue. Also referred to as *hemolytic streptococcal gangrene, acute dermal gangrene, suppurative fasciitis,* and *synergistic necrotizing cellulites,* necrotizing fasciitis is most commonly caused by the pathogenic bacteria *Streptococcus pyogenes*, also known as group A *Streptococcus* (GAS), although other aerobic and anaerobic pathogens may be present.

This severe and potentially fatal infection may begin at the site of a small insignificant wound or surgical incision. It's characterized by invasive and progressive necrosis of the soft tissue and underlying blood supply. The high

mortality rates associated with it have been attributed to the emergence of more virulent strains of streptococci caused by changes in the bacteria's deoxyribonucleic acid.

This would account for an increase in the frequency and severity of the cases reported since 1985, following a 50- to 60-year span of clinical insignificance. Noted for decades and described in medical literature since the Civil War, necrotizing fasciitis accounts for 8% of reported cases of invasive GAS infections today. The mortality rate is very high, at 70% to 80%. Mortality drops significantly and prognosis improves with early intervention and treatment. Cases treated aggressively with surgery, antibiotics, and hyperbaric oxygen (HBO) therapy have seen mortality rates reduced to as low as 9% to 20%.

Causes and incidence

More than 80 types of the causative bacteria *S. pyrogenes* are in existence, making the epidemiology of GAS infections most complex. Wounds as minor as pinpricks, needle punctures, bruises, blisters, and abrasions or as serious as a traumatic injury or surgical incision can provide an opportunity for bacteria to enter the body.

In necrotizing fasciitis, group A beta-hemolytic *Streptococcus* and *Staphylococcus aureus*, working alone or together, are most commonly the primary infecting bacteria. They can enter the host via local tissue injury or through a breach in the integrity of a mucous membrane barrier. Other aerobic and anaerobic pathogens, including *Bacteroides, Clostridium, Peptostreptococcus,* Enterobacteriaceae, coliforms, *Proteus*, *Pseudomonas,* and *Klebsiella*, may be present. They can proliferate in an environment of tissue hypoxia caused by trauma, recent surgery, or medical compromise. The end product of this invasion is necrosis of the surrounding tissue, which accelerates the disease process by creating an even more favorable environment for the organisms.

Men are three times more likely to develop this rare condition than women, and the disease rarely occurs in children except in countries with poor hygienic practices. The mean age of the population contracting the disease is 38 to 44 years.

COMPARING STREPTOCOCCAL INFECTIONS

Causes and incidence	Signs and symptoms	Diagnosis	Complications	Treatment and special considerations
Streptococcus pyogenes (Group A streptococcus)				
Streptococcal pharyngitis (strep throat)				
▪ Accounts for 95% of all cases of bacterial pharyngitis ▪ Most common in children ages 5 to 10, from October to April ▪ Spread by direct person-to-person contact via droplets of saliva or nasal secretions ▪ Organism usually colonizes throats of persons with no symptoms ▪ Up to 20% of school children may be carriers	▪ After 1- to 5-day incubation period: temperature of 101° to 104° F (38.3° to 40° C), sore throat with severe pain on swallowing, beefy red pharynx, tonsillar exudate, edematous tonsils and uvula, swollen glands along the jaw line, generalized malaise and weakness, anorexia, occasional abdominal discomfort ▪ Up to 40% of small children have symptoms too mild for diagnosis ▪ Fever abates in 3 to 5 days; nearly all symptoms subside within a week	▪ Clinically indistinguishable from viral pharyngitis ▪ Throat culture showing group A beta-hemolytic streptococci (carriers have positive throat culture) ▪ Elevated white blood cell (WBC) count ▪ Serology showing a fourfold rise in streptozyme titers during convalescence	▪ Acute otitis media or acute sinusitis occurs most frequently ▪ Rarely, bacteremic spread may cause arthritis, endocarditis, meningitis, osteomyelitis, or liver abscess ▪ Poststreptococcal sequelae: acute rheumatic fever or acute glomerulonephritis ▪ Reye's syndrome	▪ Penicillin or erythromycin, analgesics, and antipyretics may be ordered. ▪ Stress the need for bed rest and isolation from other children for 24 hours after antibiotic therapy begins; the patient should finish his prescription, even if symptoms subside; abscess, glomerulonephritis, and rheumatic fever can occur. ▪ Tell the patient not to skip doses and to properly dispose of soiled tissues.
Scarlet fever (scarlatina)				
▪ Usually follows streptococcal pharyngitis; may follow wound	▪ Streptococcal sore throat, fever, strawberry tongue, fine	▪ Characteristic rash and strawberry tongue	▪ Although rare, complications may include high fever, arthritis,	▪ Penicillin or erythromycin may be ordered.

infections or puerperal sepsis ▪ Caused by streptococcal strain that releases an erythrogenic toxin ▪ Most common in children ages 2 to 10 ▪ Spread by large respiratory droplets or direct contact with items soiled with respiratory secretions	erythematous rash that blanches on pressure and resembles sunburn with goosebumps ▪ Rash usually appearing first on upper chest, then spreading to neck, abdomen, legs, and arms, sparing soles and palms; flushed cheeks, pallor around mouth ▪ Skin sheds during convalescence	▪ Culture and Gram stain showing *S. pyogenes* from nasopharynx ▪ Granulocytosis	jaundice, pneumonia, pericarditis, and peritonsillar abscess	▪ Keep the patient in isolation for the first 24 hours. ▪ Carefully dispose of purulent discharge. ▪ Stress the need for prompt and complete antibiotic treatment.
Erysipelas				
▪ Occurs primarily in infants and adults older than age 30 ▪ Usually follows streptococcal pharyngitis ▪ Exact mode of spread to skin unknown	▪ Sudden onset, with reddened, swollen, raised lesions (skin looks like an orange peel), usually on face and scalp, bordered by areas that often contain easily ruptured blebs filled with yellow-tinged fluid; lesions sting and itch; lesions on the trunk, arms, or legs usually affect incision or wound sites ▪ Other symptoms: vomiting, fever, headache, cervical	▪ Typical reddened lesions ▪ Culture taken from edge of lesions showing group A beta-hemolytic streptococci ▪ Throat culture almost always positive for group A beta-hemolytic streptococci	▪ Untreated lesions on trunk, arms, or legs may involve large body areas and lead to death.	▪ Penicillin or erythromycin I.V. or by mouth (P.O.) may be ordered. ▪ Cold packs, analgesics (aspirin and codeine for local discomfort), and topical anesthetics may be used to increase comfort. ▪ Prevention includes prompt treatment of streptococcal infections and drainage and secretion precautions.

	lymphadenopathy, sore throat			
Impetigo (streptococcal pyoderma)				
▪ Common in children ages 2 to 5 in hot, humid weather; high rate of familial spread ▪ Predisposing factors: close contact in schools, overcrowded living quarters, poor skin hygiene, minor skin trauma ▪ May spread by direct contact, environmental contamination, or arthropod vector	▪ Small macules rapidly develop into vesicles, then become pustular and encrusted, causing pain, surrounding erythema, regional adenitis, cellulitis, and itching; scratching spreads infection ▪ Lesions commonly affect the face, heal slowly, and leave depigmented areas	▪ Characteristic lesions with honey-colored crust ▪ Culture and Gram stain of swabbed lesions showing *S. pyogenes*	▪ Septicemia (rare) ▪ Ecthyma, a form of impetigo with deep ulcers	▪ Penicillin I.V. or P.O., erythromycin, or antibiotic ointments may be ordered. ▪ Perform frequent washing of lesions with antiseptics, such as povidone-iodine or antibacterial soap, followed by thorough drying. ▪ Isolate a patient with draining wounds. ▪ Prevention includes good hygiene and proper wound care.
Streptococcus agalactiae (Group B streptococcus)				
Neonatal streptococcal infections				
▪ Incidence of early-onset infection (age 6 days or younger): 2/1,000 live births ▪ Incidence of late-onset infection (age 7 days to 3 months): 1/1,000 live births ▪ Spread by vaginal delivery or hands of nursery staff	▪ Early onset: bacteremia, pneumonia, and meningitis; mortality from 14% for infants weighing more than 1,500 g at birth to 61% for infants weighing less than 1,500 g at birth ▪ Late onset: bacteremia with meningitis, fever, and bone and	▪ Isolation of group B streptococcus from blood, cerebrospinal fluid (CSF), or skin ▪ Chest X-ray showing massive infiltrate similar to that of respiratory distress syndrome or pneumonia	▪ Overwhelming pneumonia, sepsis, and death	▪ Penicillin or ampicillin and an aminoglycoside I.V. may be ordered. ▪ Patient isolation is unnecessary unless an open draining lesion is present, but proper hand-hygiene is essential; for a draining lesion, take drainage and secretion precautions.

▪ Predisposing factors: maternal genital tract colonization, membrane rupture over 24 hours before delivery, crowded nursery	joint involvement; mortality 15% to 20% ▪ Other signs and symptoms, such as skin lesions, depend on the site affected			▪ Group B streptococcus prophylaxis may be ordered for women who are pregnant if vaginal or rectal cultures are positive at 35 to 37 weeks gestation or if the patient meets other criteria, such as delivery earlier or later than 3 weeks of term, amniotic fluid rupture for 18 hours or more; or an intrapartum temperature greater than or equal to 100.4° F [38.0° C]).
Adult group B streptococcal infection				
▪ Most adult infections occur in postpartum women, usually in the form of endometritis or wound infection following cesarean section ▪ Incidence of group B streptococcal endometritis: 1.3/1,000 live births ▪ Group B streptococcal bacteremia and pneumonia: occur in the elderly and frequently in	▪ Fever, malaise, and uterine tenderness ▪ Change in lochia ▪ Bacteremia and pneumonia patients: can exhibit neurologic symptoms such as a change in mental status	▪ Isolation of group B streptococcus from blood or infection site	▪ Bacteremia followed by meningitis or endocarditis	▪ Ampicillin or penicillin I.V. may be ordered. ▪ Perform careful observation for symptoms of infection following delivery. ▪ Follow drainage and secretion precautions.

patients with diabetes ▪ Invasive group B streptococcal infection: occurs in patients with human immunodeficiency virus ***Streptococcus pneumoniae***				
Pneumococcal pneumonia				
▪ Accounts for 70% of all cases of bacterial pneumonia ▪ More common in men, elderly, Blacks, and Native Americans, in winter and early spring ▪ Spread by droplets and contact with infective secretions ▪ Predisposing factors: trauma, viral infection, underlying pulmonary disease, overcrowded living quarters, chronic diseases, asplenia, and immunodeficiency ▪ Among the 10 leading causes of death in the United States	▪ Sudden onset with severe shaking chills, temperature of 102° to 105° F (38.9° to 40.6° C), bacteremia, cough (with thick, scanty, blood-tinged sputum) accompanied by pleuritic pain ▪ Malaise, weakness, and prostration common ▪ Tachypnea, anorexia, nausea, and vomiting less common ▪ Severity of pneumonia usually caused by host's cellular defenses, not bacterial virulence	▪ Gram stain of sputum showing gram-positive diplococci; culture showing *S. pneumoniae* ▪ Chest X-ray showing lobular consolidation in adults; bronchopneumonia in children and elderly patient ▪ Elevated WBC count ▪ Blood cultures usually positive for *S. pneumoniae*	▪ Pleural effusion (occurs in 25% of patients) ▪ Pericarditis (rare) ▪ Lung abscess (rare) ▪ Bacteremia ▪ Disseminated intravascular coagulation ▪ Death possible if bacteremia is present	▪ Penicillin or erythromycin I.V. or I.M. may be ordered. ▪ Monitor and support respirations, as needed. ▪ Record sputum color and amount. ▪ Prevent dehydration. ▪ Avoid sedatives and opioids to preserve cough reflex. ▪ Carefully dispose of all purulent drainage (standard precautions); advise high-risk patients to receive a vaccine and to avoid infected people.
Otitis media				

▪ High incidence, with about 76% to 95% of all children having otitis media at least once (*S. pneumoniae* causes half of these cases.)	▪ Ear pain, ear drainage, hearing loss, fever, lethargy, and irritability ▪ Other possible symptoms: vertigo, nystagmus, and tinnitus	▪ Fluid in middle ear ▪ Isolation of *S. pneumoniae* from aspirated fluid if necessary	▪ Recurrent attacks (may cause hearing loss)	▪ Amoxicillin or ampicillin and analgesics may be ordered. ▪ Tell the patient to report a lack of response to therapy after 72 hours.
Meningitis				
▪ Can follow bacteremic pneumonia, mastoiditis, sinusitis, skull fracture, or endocarditis ▪ Mortality (30% to 60%) highest in infants and elderly people	▪ Fever, headache, nuchal rigidity, vomiting, photophobia, lethargy, coma, wide pulse pressure, and bradycardia	▪ Isolation of *S. pneumoniae* from CSF or blood culture ▪ Increased CSF cell count and protein level; decreased CSF glucose level ▪ Computed tomography scan of head ▪ EEG	▪ Persistent hearing deficits, seizures, hemiparesis, or other nerve deficits ▪ Encephalitis	▪ Penicillin I.V. or chloramphenicol may be ordered. ▪ Monitor the patient closely for neurologic changes. ▪ Watch for symptoms of septic shock, such as acidosis and tissue hypoxia.
Group D streptococcus				
Endocarditis				
▪ Group D streptococcus (enterococcus): causes 10% to 20% of all bacterial endocarditis ▪ Most common in elderly people and in those who abuse I.V. substances ▪ Typically follows bacteremia from an obvious source, such as a	▪ Weakness, fatigability, weight loss, fever, night sweats, anorexia, arthralgia, splenomegaly, and new systolic murmur	▪ Anemia, increased erythrocyte sedimentation rate and serum immunoglobulin level, and positive blood culture for group D streptococcus ▪ Echocardiogram showing vegetation on valves	▪ Embolization ▪ Pulmonary infarction ▪ Osteomyelitis	▪ Penicillin for *Streptococcus bovis* (non-enterococcal group D streptococcus) may be ordered. ▪ Penicillin or ampicillin *and* an aminoglycoside for enterococcal group D streptococcus may be ordered.

wound infection, urinary tract infection, or I.V. insertion site infection
▪ Most cases are subacute
▪ Also causes urinary tract infection

Complications

- Renal failure
- Septic shock
- Cardiovascular collapse
- Scarring
- Myosis
- Myonecrosis

Signs and symptoms

Pain, out of proportion to the size of the wound or injury it's associated with, is usually the first symptom of necrotizing fasciitis. It generally presents before all other physical findings.

The infective process will usually begin with a mild area of erythema at the site of insult, which will quickly progress within the first 24 hours. During the first 24- to 48-hour period, the erythema changes from red to purple in color and then to blue, with the formation of fluid-filled blisters and bullae that indicate the rapid progression of the necrotizing process. By days 4 and 5, multiple patches of this erythema form, producing large areas of gangrenous skin. By days 7 to 10, dead skin begins to separate at the margins of the erythema, revealing extensive necrosis of the subcutaneous tissue. At this stage, fascial necrosis is typically more advanced than appearance would suggest.

Other clinical symptoms include fever and hypovolemia. In later stages, hypotension and respiratory insufficiency, which

are signs of overwhelming sepsis requiring supportive care, occur. In the most severe cases, necrosis advances rapidly until several large areas of the body are

involved. This may cause the patient to become mentally cloudy, delirious, or even unresponsive secondary to the intoxication rendered.

Other complications include renal failure, septic shock with cardiovascular collapse, and scarring with cosmetic deformities. Without treatment, involvement of deeper muscle layers may occur, resulting in myositis or myonecrosis.

Diagnosis

Tissue biopsy is the best method of diagnosing necrotizing fasciitis. Cultures of microorganisms can be obtained locally from the periphery of the spreading infection or from deeper tissues during surgical debridement. Gram's staining and culturing of biopsied tissue are useful in establishing the type of invasive organisms and the effective treatment against them.

Radiographic studies can pinpoint the presence of subcutaneous gases, and computed tomography scans can locate the anatomic site of involvement by locating the necrosis. In combination with clinical assessment, magnetic resonance imaging determines areas of necrosis and the need for surgical debridement.

Other supportive studies include laboratory values such as complete blood count with differential, electrolytes, glucose, blood urea nitrogen and creatinine, urinalysis, and arterial blood gas levels.

Other conditions to consider in the differential diagnosis include cellulitis, testicular torsion, epididymitis and orchitis (as related to Fournier's gangrene), gas gangrene, hernias, and toxic shock syndrome (TSS).

Treatment

Prompt and aggressive exploration and debridement of suspected necrotizing fasciitis is mandatory for early, definitive diagnosis and to improve prognosis. Ninety percent of patients that present with clinical signs and symptoms will need immediate surgical debridement, fasciotomy, or amputation.

Penicillin, clindamycin, metronidazole, ceftriaxone, gentamicin, chloramphenicol, and ampicillin are among the medications given orally, I.V., or I.M. to treat the organisms involved with necrotizing fasciitis. The specific drug is determined by the sensitivity of the cultured organisms. When the infection is polymicrobial, medications must be used in combination. Recommendations for specific drugs continue to change as new antibiotics are developed and new resistance emerges.

Reports suggest that the use of HBO therapy decreases the mortality rate, significantly improves the tissues defense against infection, and prevents the necrosis from spreading by increasing the normal oxygen saturations of infected wounds by a thousandfold, causing a bactericidal effect. Typical treatment

involves aggressively starting HBO therapy after the first surgical debridement and continuing for a total of 10 to 15 sessions.

Special considerations

- Antibiotic therapy should be initiated immediately.
- Accurate and frequent assessment of the patient's pain level, mental status, wound status, and vital signs is essential in order to recognize the progression of the wound changes or the development of new signs and symptoms. Changes must be reported and documented immediately.
- The need for supportive care, such as endotracheal intubation, cardiac monitoring, fluid replacement, and supplemental oxygen should be assessed and provided as warranted.
- Care of postoperative patients and patients with trauma wounds requires strict sterile technique, good hand hygiene, and barriers between health care providers and patients to prevent contamination.
- Health care workers with sore throats should see their physician to determine if they have a streptococcal infection. If they are diagnosed positive, they shouldn't return to work until 24 hours after the initiation of antibiotic therapy.
- Risk factors for contracting necrotizing fasciitis include patients with advanced age, human immunodeficiency infection, history of alcohol abuse, and varicellar infection. Patients with chronic illnesses, such as cancer, diabetes, cardiopulmonary disease, and kidney disease requiring hemodialysis, and those using steroids are more susceptible to GAS infection due to their debilitated immune response.

ALERT

Watch for signs and symptoms of TSS, which is associated with any streptococcal soft tissue infection, and the development of shock, acute respiratory distress syndrome, renal impairment, or bacteremia, any of which can lead to sudden death.

Vancomycin intermittent-resistant Staphylococcus aureus

Vancomycin intermittent-resistant *Staphylococcus aureus* (VISA) is a mutation of a bacterium that's easily spread by direct person-to-person contact. It was first discovered in mid-1996 in a Japanese infant's surgical wound; similar isolates were later reported in Michigan and New Jersey. The U.S. patients had received multiple courses of vancomycin for methicillin-resistant *S. aureus* infections.

Another mutation, vancomycin-resistant *S. aureus* (VRSA), is fully resistant to vancomycin.

Causes and incidence

VISA or VRSA enters a health care facility through an infected or colonized (symptom-free but infected) patient or colonized health care worker. It's spread during direct contact between the patient and caregiver or patient-to-patient. It may also be spread through patient contact with contaminated surfaces, such as an overbed table. It's able to live for weeks on surfaces. It has been detected on patient gowns, bed linens, and handrails.

A colonized patient is more than 10 times as likely to become infected with the organism, such as through a breach in the immune system. Patients most at risk for infection include immunosuppressed patients or those with severe underlying disease; patients with a history of taking vancomycin, third-generation cephalosporins, or antibiotics targeted at anaerobic bacteria (such as *Clostridium difficile*); patients with indwelling urinary or central venous catheters; elderly patients, especially those with prolonged or repeated facility admissions; patients with malignancies or chronic renal failure; patients undergoing cardiothoracic or intra-abdominal surgery or organ transplants; patients with wounds with an opening to the pelvic or intra-abdominal area, such as surgical wounds, burns, and pressure ulcers; patients with enterococcal bacteremia, often associated with endocarditis; and patients exposed to contaminated equipment or to a patient with the infecting microbe.

Complications

- Sepsis
- Multisystem organ involvement
- Death

Signs and symptoms

The carrier patient is commonly asymptomatic but may have signs and symptoms related to the primary diagnosis. Depending on the source of the infection and the reason for treatment, the patient may exhibit cardiac, respiratory, or other major symptoms. Assessment should focus on the affected body system.

Diagnosis

The causative agent may be found incidentally when culture results show the organism. A person with no signs or symptoms of infection is considered colonized

if VISA or VRSA can be isolated from stool or a rectal swab.

Treatment

Because no single antibiotic to combat VISA or VRSA is currently available, the physician may opt not to treat an infection at all. Instead, he may stop all antibiotics and wait for normal bacteria to repopulate and replace the strain. Combinations of various drugs may also be used, depending on the source of the infection.

To prevent the spread of VISA and VRSA, some hospitals perform weekly surveillance cultures on at-risk patients in intensive care units or oncology units and those transferred from a long-term care facility. Any colonized patient is then placed in contact isolation until his culture is negative or he's discharged. Colonization can last indefinitely; no protocol has been established for the length of time a patient should remain in isolation.

In 1996, the CDC and the Hospital Infection Control Practices Advisory Committee proposed a two-level system of precautions to simplify isolation for resistant organisms. The first level calls for standard precautions, which incorporate features of universal blood and body fluid precautions and body substance isolation precautions. The second level calls for transmission-based precautions, implemented when a particular infection is suspected.

Special considerations

- Wash your hands before and after providing patient care. Good hand washing is the most effective way to prevent VISA and VRSA from spreading. Use an antiseptic soap such as chlorhexidine; bacteria have been cultured from workers' hands after washing with milder soap.
- Consider grouping infected patients together and having the same nursing staff care for them.
- Contact isolation precautions should be used when in contact with the patient. A private room should be used. Use dedicated equipment and disinfect the environment.
- Change gloves when contaminated or when moving from a soiled area of the body to a clean one.
- Don't touch potentially contaminated surfaces, such as a bed or bed stand, after removing gown and gloves.
- Be particularly cautious in caring for a patient with an ileostomy, colostomy, or draining wound that isn't contained by a dressing.

- Equipment used on the patient shouldn't be laid on the bed or bed stand and should be wiped with appropriate disinfectant before leaving the room.
- Ensure judicious and careful use of antibiotics. Encourage physicians to limit the use of antibiotics.
- Instruct family and friends to wear protective garb when they visit the patient. Demonstrate how to dispose of it.
- Provide teaching and emotional support to the patient and his family members.
- Instruct the patient to take antibiotics for the full prescription period, even if he begins to feel better.

Vancomycin-resistant enterococcus infection

Vancomycin-resistant enterococcus (VRE) is a mutation of a common bacterium normally found in the GI tract that's spread easily by direct person-to-person contact. Facilities in more than 40 states have reported VRE infection, with 30% of enterococcus infections in intensive care units (ICUs) and 25% of enterococcus infections in non-ICU areas reporting as vancomycin-resistant.

Patients most at risk for VRE infection include:

- immunosuppressed patients or those with severe underlying disease
- patients with a history of taking vancomycin, third-generation cephalosporins, antibiotics targeted at anaerobic bacteria (such as *Clostridium difficile*), or multiple courses of antibiotics
- patients with indwelling urinary or central venous catheters
- elderly patients, especially those with prolonged or repeated hospital admissions
- patients with cancer or chronic renal failure
- patients undergoing cardiothoracic or intra-abdominal surgery or organ transplant
- patients with wounds opening into the pelvic or intra-abdominal area, including surgical wounds, burns, and pressure ulcers
- patients with enterococcal bacteremia, typically associated with endocarditis
- patients exposed to contaminated equipment or to another VRE-positive patient.

Causes and incidence

VRE enters health care facilities through an infected or colonized patient or a colonized health care worker. It can also develop following treatment with vancomycin. VRE spreads through direct contact between the patient and caregiver or between patients. It can also spread through patient contact with

contaminated surfaces such as an overbed table where it's capable of living for weeks. VRE has also been detected on patient gowns, bed linens, and handrails.

Complications

- Sepsis
- Multisystem dysfunction
- Death (in immunocompromised patients)

Signs and symptoms

There are no specific signs and symptoms related to VRE infection. The causative agent may be found incidentally with culture results.

Diagnosis

Persons with no signs or symptoms of infection are considered colonized if VRE can be isolated from stool or a rectal swab.

Once colonized, a patient is more than 10 times as likely to become infected with VRE, for example, through a breach in the immune system.

Treatment

New antimicrobials, such as linezolid and quinupristin and dalfopristin, are available for treatment of VRE infection. Patients who are already colonized with VRE usually

aren't treated with antimicrobials. Instead, the physician may stop all antibiotics and simply wait for normal bacteria to repopulate and replace the VRE strain. Combinations of various drugs may also be used, depending on the source of the infection.

To prevent the spread of VRE, some facilities perform weekly surveillance cultures on at-risk patients in the intensive care or oncology units and on patients who have been transferred from a long-term care facility. Any colonized patient is then placed in contact isolation until culturenegative or until discharged. Colonization can last indefinitely; no protocol has been established for the length of time a patient should remain in isolation.

Special considerations

- Hand hygiene before and after care of the patient is crucial. Good hand hygiene is the most effective way to prevent VRE from spreading. Use an antiseptic soap such as chlorhexidine. Bacteria have been cultured from workers' hands after

they've washed with milder soap. Alcohol-based hand sanitizers are effective as well.

- Use contact precautions when in contact with the patient or his support equipment. Provide the patient with a private room and dedicated equipment. Disinfect the environment and the equipment frequently.
- Change gloves when contaminated or when moving from a “dirty” area of the body to a clean one.
- Don't touch potentially contaminated surfaces such as an overbed table after removing your gown and gloves.
- Be particularly prudent in caring for a patient with an ileostomy, colostomy, or draining wound that isn't contained by a dressing.
- Instruct the patient's family and friends to wear protective garb when they visit him, and teach them how to dispose of it. Instruct them on proper hand hygiene.
- Provide teaching and emotional support to the patient and his family members.
- Consider grouping (“cohorting”) infected or colonized patients together and assigning the same nursing staff to them.
- Don't lay equipment used on the patient on the bed or on the overbed table. Wipe the equipment with the appropriate disinfectant before leaving the room.
- Ensure judicious and careful use of antibiotics. Encourage physicians to limit their use.
- Instruct patients to take antibiotics for the full period prescribed, even if they begin to feel better.

Scarlet fever

Although scarlet fever (scarlatina) usually follows streptococcal pharyngitis, it may also occur after other streptococcal infections, such as wound infections, urosepsis, and puerperal sepsis. It's most common in children ages 3 to 15. The incubation period commonly lasts from 2 to 4 days, but may be only 1 day or extend to 7 days.

Causes and incidence

Group A beta-hemolytic streptococci cause scarlet fever. The infecting strain produces one of three erythrogenic toxins, which triggers a sensitivity reaction in the patient.

Complications

- Severe disseminated toxic illness
- Septicemia
- Rheumatic heart disease
- Liver damage

Signs and symptoms

The patient may report a sore throat, headache, chills, anorexia, abdominal pain, and malaise. He's likely to have a temperature of 100° to 103° F (37.8° to 39.4° C). In addition, he commonly has had contact with a person with a sore throat.

Inspection of the patient's mouth initially shows an inflamed and heavily coated tongue. As the disease progresses, note a strawberrylike tongue. As it progresses further, the tongue begins to peel and becomes beefy red. It returns to normal by the end of the second week. The uvula, tonsils, and posterior oropharynx appear red and edematous, with mucopurulent exudate.

Inspection of the skin may reveal a fine, erythematous rash that appears first on the upper chest and back. It later spreads to the neck, abdomen, legs, and arms but doesn't

appear on the soles and palms. The rash resembles sunburn with goose bumps and blanches when pressure is applied. The patient's face appears flushed, except around the mouth, which remains pale. During convalescence, desquamation of the skin occurs at the tips of the fingers and toes and, occasionally, over wide areas of the trunk and limbs. It's more pronounced where the erythematous rash was most severe.

The cervical lymph nodes feel enlarged and tender on palpation. The liver may also feel slightly enlarged and tender, and tachycardia may be noted.

Diagnosis

A pharyngeal culture is positive for group A beta-hemolytic streptococci. A complete blood count reveals granulocytosis and, possibly, a reduced red blood cell count.

Treatment

Antibiotic therapy with penicillin or erythromycin is given for 10 days, along with antipyretics.

Special considerations

- Implement droplet precautions for 24 hours after starting antibiotic therapy.

- Keep the patient on complete bed rest while he's febrile to prevent complications, promote recovery, and help conserve his energy.
- Offer frequent oral fluids and oral hygiene and give antipyretics as ordered.
- Apply topical anesthetics on the patient's tongue and throat to relieve pain.
- Provide skin care to relieve discomfort from the rash.
- Instruct the patient (or his parents) to make sure he takes his oral antibiotics for the prescribed length of time.

GRAM-NEGATIVE COCCI

Meningococcal infections

Two major meningococcal infections (meningitis and meningococcemia) are caused by the gram-negative bacteria *Neisseria meningitidis*, which also causes primary pneumonia, purulent conjunctivitis, endocarditis, sinusitis, and genital infection. Meningococcemia occurs as simple bacteremia, fulminating meningococcemia and, rarely, chronic meningococcemia. It commonly accompanies meningitis. (See "Meningitis," page 196.) Meningococcal infections may occur sporadically or in epidemics; particularly virulent infections may be fatal within a matter of hours.

Causes and incidence

Meningococcal infections usually occur among children (ages 6 months to 1 year) and men, usually military recruits or those enrolled at institutions, such as colleges, because of overcrowding.

N. meningitidis has seven serogroups (A, B, C, D, X, Y, and Z); group A causes most epidemics. Transmission takes place through inhalation of an infected droplet from a carrier (an estimated 2% to 38% of the population). The bacteria localize in the nasopharynx. After incubating about 3 to 4 days, they spread through the bloodstream to joints, skin, adrenal glands, lungs, and the central nervous system. The tissue damage that results (possibly due to the effects of bacterial endotoxins) produces symptoms and, in fulminating meningococcemia and meningococcal bacteremia, hemorrhage, thrombosis, and necrosis.

Complications

- Respiratory failure
- Disseminated intravascular coagulation (DIC)
- Septic arthritis
- Pericarditis

- Endophthalmitis
- Neurologic deterioration
- Death

Signs and symptoms

Features of *meningococcal bacteremia* include sudden spiking fever, headache, sore throat, cough, chills, myalgia (in back and legs), arthralgia, tachycardia, tachypnea, mild hypotension, and a petechial, nodular, or maculopapular rash. Headache and stiff

neck can also occur as the infection extends to the meninges.

In about 10% to 20% of patients, the disease progresses to *fulminating meningococcemia*, with extreme prostration, enlargement of skin lesions, DIC, and shock. Without prompt treatment, death from respiratory or heart failure occurs in 6 to 24 hours.

Characteristics of the rare *chronic meningococcemia* include intermittent fever, rash, joint pain, and an enlarged spleen.

Diagnosis

CONFIRMING DIAGNOSIS

Gram-negative diplococci, on blood or cerebrospinal fluid (CSF) Gram stain, are highly suspicious for N. meningitidis. *Isolation of* N. meningitidis *through a positive blood culture, CSF culture, or lesion scraping confirms the diagnosis, except in nasopharyngeal infections because* N. meningitidis *is part of the normal nasopharyngeal flora.*

Tests that support the diagnosis include counterimmunoelectrophoresis of CSF or blood, low white blood cell count and, in patients with skin or adrenal hemorrhages, decreased platelet and clotting levels. Diagnostic evaluation must rule out Rocky Mountain spotted fever and vascular purpuras.

Treatment

As soon as meningococcal infection is suspected, treatment begins with high doses of aqueous penicillin G, ampicillin, or cephalosporins such as ceftriaxone; or, for the patient who is allergic to penicillin, I.V. chloramphenicol. Therapy may also include mannitol for cerebral edema, I.V. heparin for DIC, dopamine for shock, and digoxin and a diuretic if heart failure develops. Supportive measures include fluid and electrolyte maintenance, ventilation (maintenance of a patent airway and

oxygen, if necessary), insertion of an arterial or central venous pressure (CVP) line to monitor cardiovascular status, and bed rest.

Prophylaxis with ciprofloxacin or rifampin aids health care personnel who work in close contact with the patient, such as those administering cardiopulmonary resuscitation or assisting with intubation or suctioning without wearing a surgical mask.

Special considerations

- Give I.V. antibiotics, as ordered, to maintain blood and CSF drug levels.
- Enforce bed rest in early stages. Provide a dark, quiet, restful environment.
- Maintain adequate ventilation with oxygen or a ventilator, if necessary. Suction and turn the patient frequently.
- Keep accurate intake and output records to maintain proper fluid and electrolyte levels. Monitor blood pressure, pulse, arterial blood gas levels, and CVP.
- Watch for complications, such as DIC, arthritis, endocarditis, and pneumonia.
- If the patient is receiving chloramphenicol, monitor complete blood count.
- Check the patient's drug history for allergies before giving antibiotics.

To prevent the infection's spread:

- Impose droplet precautions until the patient has had antibiotic therapy for 24 hours.
- Label all meningococcal specimens. Deliver them to the laboratory quickly because meningococci are very sensitive to changes in humidity and temperature.
- Report all meningococcal infections to public health department officials.

PREVENTION

As a preventive measure, the meningococcal vaccine can be administered to first-year college students living in dormitories.

GRAM-POSITIVE BACILLI

Diphtheria

Diphtheria is an acute, highly contagious toxin-mediated infection caused by *Corynebacterium diphtheriae*, a gram-positive rod that usually infects the respiratory tract, primarily the tonsils, nasopharynx, and larynx. The GI and urinary tracts, conjunctivae, and ears are rarely involved.

Causes and incidence

Transmission usually occurs through intimate contact or by airborne respiratory droplets from asymptomatic carriers or convalescing patients. Many more people carry this disease than contract active infection. Diphtheria is more prevalent during the colder months because of closer person-to-person indoor contact, however it may be contracted at any time during the year.

Thanks to effective immunization, diphtheria is rare in many parts of the world, including the United States. Since 1972, the incidence of cutaneous diphtheria has been increasing, especially in the Pacific Northwest and the Southwest, in areas where crowding and poor hygienic conditions prevail. Most victims are children younger than age 15; about 10% of patients die.

Signs and symptoms

Most infections go unrecognized, especially in partially immunized individuals. After an incubation period of less than a week, clinical cases of diphtheria characteristically show a thick, patchy, grayish green membrane over the mucous membranes of the pharynx, larynx, tonsils, soft palate, and nose; fever; sore throat; and a rasping cough, hoarseness, and other symptoms similar to croup. Attempts to remove the membrane usually cause bleeding, which is highly characteristic of diphtheria. If this membrane causes airway obstruction (particularly likely in laryngeal diphtheria), symptoms include tachypnea, stridor, possibly cyanosis, suprasternal retractions, and suffocation, if untreated. Adenopathy and cervical swelling can occur. In cutaneous diphtheria, skin lesions resemble impetigo.

Complications

- Thrombocytopenia
- Myocarditis
- Neurologic involvement (primarily affecting motor fibers but possibly also sensory neurons)
- Renal involvement
- Pulmonary involvement (bronchopneumonia) caused by *C. diphtheriae* or other superinfecting organisms

Diagnosis

CONFIRMING DIAGNOSIS

Examination showing the characteristic membrane and a throat culture, or culture of other suspect lesions growing C. diphtheriae, *confirm this diagnosis.*

Treatment

Treatment must not wait for confirmation by culture. Standard treatment includes diphtheria antitoxin administered I.M. or I.V.; antibiotics, such as penicillin or erythromycin, to eliminate the organisms from the upper respiratory tract and other sites and terminate the carrier state; measures to prevent complications; and possible tracheotomy if airway obstruction occurs.

Special considerations

Diphtheria requires comprehensive supportive care with psychological support.

- To prevent the spread of this disease, stress the need for droplet precautions. Teach proper disposal of nasopharyngeal secretions. Maintain infection precautions until after two consecutive negative nasopharyngeal cultures — at least 1 week after discontinuing drug therapy. Treatment of exposed individuals with antitoxin remains controversial. Suggest that the patient's family receive diphtheria toxoid if they haven't been immunized.
- Give drugs as ordered. Although time-consuming and risky, desensitization should be attempted if tests are positive, because diphtheria antitoxin is the only *specific* treatment available. If sensitivity tests are negative, the antitoxin is given before laboratory confirmation, because mortality increases directly with any delay in antitoxin administration. Before giving diphtheria antitoxin, which is made from horse serum, obtain eye and skin tests to determine sensitivity. After giving antitoxin or penicillin, be alert for anaphylaxis; keep epinephrine 1:1,000 and resuscitation equipment handy. In patients who receive erythromycin, watch for thrombophlebitis.
- Monitor respirations carefully, especially in laryngeal diphtheria (usually, such patients are in a high-humidity environment). Watch for signs of airway obstruction, and be ready to give immediate life support, including intubation and tracheotomy.
- Watch for signs of shock, which can develop suddenly.
- Obtain cultures as ordered.
- If neuritis develops, tell the patient it's usually transient. Be aware that peripheral neuritis may not develop until 2 to 3 months after the onset of illness.

ALERT

Be alert for signs of myocarditis, such as the development of heart murmurs or electrocardiogram changes. Ventricular fibrillation is a common cause of sudden death in patients with diphtheria.

PREVENTION

Stress the need for childhood immunizations to all parents. Protective immunity doesn't last longer than 10 years after the last vaccination, so it's important to get tetanus-diphtheria boosters every 10 years.

- Report all cases to local public health authorities.

Listeriosis

Listeriosis is an infection caused by the weakly hemolytic, gram-positive bacillus *Listeria monocytogenes*. It occurs most commonly in fetuses, in neonates (during the first 3 weeks of life), and in older or immunosuppressed adults. The infected fetus is usually stillborn or is born prematurely, almost always with lethal listeriosis. This infection produces milder illness in pregnant women and varying degrees of illness in older and immunosuppressed patients; their prognoses depend on the severity of underlying illness.

Causes and incidence

The primary method of person-to-person transmission is neonatal infection in utero (through the placenta) or during passage through an infected birth canal. Other modes of transmission may include inhaling contaminated dust; drinking contaminated, unpasteurized milk; eating unprocessed soft cheese or deli meats; coming in contact with infected animals, contaminated sewage or mud, or soil contaminated with feces containing *L. monocytogenes*; and, possibly, person-to-person transmission.

Complications

- Sepsis
- Diffuse clotting dyscrasias
- Respiratory insufficiency
- Circulatory insufficiency
- Meningitis

- Cerebritis
- Nonpurulent conjunctivitis
- Granulomatous skin infection

Signs and symptoms

Contact with *L. monocytogenes* commonly causes a transient asymptomatic carrier state. But sometimes it produces bacteremia and a febrile, generalized illness. In a pregnant woman, especially during the third trimester, listeriosis causes a mild illness with malaise, chills, fever, and back pain. However, her fetus may suffer severe uterine infection, abortion, premature delivery, or stillbirth. Transplacental infection may also cause early neonatal death or granulomatosis infantiseptica, which produces organ abscesses in infants.

Infection with *L. monocytogenes* commonly causes meningitis (especially in immunocompromised patients), resulting in tense fontanels, irritability, lethargy, seizures, and coma in neonates and low-grade fever and personality changes in adults. Fulminant manifestations with coma are rare.

Diagnosis

CONFIRMING DIAGNOSIS

L. monocytogenes *is identified by its diagnostic tumbling motility on a wet mount of the culture.*

Other supportive diagnostic results include positive blood culture, spinal fluid, drainage from cervical or vaginal lesions, or lochia from a mother with an infected infant, but isolation of the organism from these specimens is generally difficult. Listeriosis also causes monocytosis.

Treatment

The treatment of choice is ampicillin or penicillin I.V. infusion for 3 to 6 weeks, possibly with gentamicin to increase its effectiveness.

Alternate treatments include erythromycin (E-mycin), chloramphenicol, tetracycline, or co-trimoxazole.

Ampicillin or penicillin G is best for treating meningitis due to *L. monocytogenes*, because they can easily cross the blood-brain barrier. Pregnant women require prompt, vigorous treatment to combat fetal infection.

Special considerations

- Deliver specimens to the laboratory promptly. Because few organisms may be present, take at least 10 ml of spinal fluid for culture.
- Use standard precautions until a series of cultures are negative. Be especially careful when handling lochia from an infected mother and secretions from her infant's eyes, nose, mouth, and rectum, including meconium.
- Evaluate neurologic status at least every 2 hours. In an infant, check fontanels for bulging. Maintain adequate I.V. fluid intake; measure intake and output accurately.
- If the patient has central nervous system depression and becomes apneic, provide respiratory assistance, monitor respirations, and obtain frequent arterial blood gas measurements.
- Provide adequate nutrition by total parenteral nutrition, nasogastric tube feedings, or a soft diet, as ordered.
- Allow the patient's parents to see and, if possible, hold their infant in the neonatal intensive care unit. Be flexible about visiting privileges. Keep the parents informed of the infant's status and prognosis at all times.
- Reassure the parents of an infected neonate who may feel guilty about the infant's illness.

PREVENTION

- *Advise pregnant women to avoid infective materials on farms where listeriosis is endemic among livestock.*
- *To avoid infection, instruct the patient and his family to avoid soft cheeses and to cook such foods as hot dogs thoroughly. Immunocompromised patients should avoid soft cheeses and deli meats.*

Tetanus

Tetanus, also known as *lockjaw,* is an acute exotoxin-mediated infection caused by the anaerobic, spore-forming, gram-positive bacillus *Clostridium tetani*. This infection is usually systemic; less commonly, localized. Tetanus is fatal in up to 60% of unimmunized people, usually within 10 days of onset. When symptoms develop within 3 days after exposure, the prognosis is poor.

Causes and incidence

Normally, transmission occurs through a puncture wound that's contaminated by soil, dust, or animal excreta containing *C. tetani* or by way of burns and minor wounds. After *C. tetani* enters the body, it causes local infection and tissue

necrosis. It also produces toxins that then enter the bloodstream and lymphatics and eventually spread to central nervous system tissue.

Tetanus occurs worldwide, but is more prevalent in agricultural regions and developing countries that lack mass immunization programs. It's one of the most common causes of neonatal deaths in developing countries, where infants of unimmunized mothers are delivered under unsterile conditions. In such infants, the unhealed umbilical cord is the portal of entry.

In the United States, about 75% of all cases occur between April and September.

Complications

- Atelectasis
- Pneumonia
- Pulmonary emboli
- Acute gastric ulcers
- Seizures
- Flexion contractures
- Cardiac arrhythmias

Signs and symptoms

The incubation period varies from 3 to 4 weeks in mild tetanus to under 2 days in severe tetanus. When symptoms occur within 3 days after injury, death is more likely. If tetanus remains localized, signs of onset are spasm and increased muscle tone near the wound.

If tetanus is generalized (systemic), indications include marked muscle hypertonicity, hyperactive deep tendon reflexes, tachycardia, profuse sweating, low-grade fever, and painful, involuntary muscle contractions:

- neck and facial muscles, especially cheek muscles — locked jaw (trismus), painful spasms of masticatory muscles, difficulty opening the mouth, and *risus sardonicus,* a grotesque, grinning expression produced by spasm of facial muscles
- somatic muscles — arched-back rigidity (opisthotonos); boardlike abdominal rigidity
- intermittent tonic seizures lasting several minutes, which may result in cyanosis and sudden death by asphyxiation.

Despite such pronounced neuromuscular symptoms, cerebral and sensory functions remain normal. Complications can include atelectasis, pneumonia, pulmonary emboli, acute gastric ulcers, flexion contractures, and cardiac arrhythmias.

Neonatal tetanus is always generalized. The first clinical sign is difficulty in sucking, which usually appears 3 to 10 days after birth. It progresses to total inability to suck with excessive crying, irritability, and nuchal rigidity.

Diagnosis

In many cases, diagnosis must rest on clinical features, a history of trauma, and no previous tetanus immunization. Blood cultures and tetanus antibody tests are often negative; only a third of patients have a positive wound culture. Cerebrospinal fluid pressure may rise above normal. Diagnosis must also rule out meningitis, rabies, phenothiazine or strychnine toxicity, and other conditions that mimic tetanus.

Treatment

Within 72 hours after a puncture wound, a patient with no previous history of tetanus immunization first requires tetanus immune globulin (TIG) or tetanus antitoxin to neutralize the toxins and to confer temporary protection. Next, he needs active immunization with tetanus toxoid. If he hasn't received tetanus immunization within 10 years, a booster injection of tetanus toxoid is necessary. If tetanus develops despite immediate postinjury treatment, the patient will require airway maintenance and a muscle relaxant, such as diazepam, to decrease muscle rigidity and spasm. If muscle contractions aren't relieved by muscle relaxants, a neuromuscular blocker, such as metocurine iodide, may be prescribed. The patient with tetanus needs high-dose antibiotics (penicillin administered I.V. if he isn't allergic to it or such alternatives as clindamycin, erythromycin, and metronidazole.) The source of the toxin needs to be removed and destroyed through surgical exploration and wound debridement.

Special considerations

- Thoroughly debride and clean the injury site, and check the patient's immunization history. Record the cause of injury. If it's an animal bite, report the case to local public health authorities.
- Before giving penicillin and TIG, antitoxin, or toxoid, obtain an accurate history of allergies to immunizations or penicillin. If the patient has a history of allergies, keep epinephrine 1:1,000 and resuscitation equipment available.
- Stress the importance of maintaining active immunization with a booster dose of tetanus toxoid every 10 years.

After tetanus develops:

- Maintain an adequate airway and ventilation to prevent pneumonia and atelectasis. Suction often and watch for signs of respiratory distress. Keep emergency airway equipment on hand because the patient may require artificial ventilation or oxygen administration.
- Maintain an I.V. line for medications and emergency care, if necessary.
- Monitor the electrocardiogram frequently for arrhythmias. Record intake and output accurately, and check vital signs often.
- Turn the patient frequently to prevent pressure ulcers and pulmonary stasis.
- Because even minimal external stimulation provokes muscle spasms, keep the patient's room quiet and only dimly lighted. Warn visitors not to upset or overly stimulate the patient.

- If urine retention develops, insert an indwelling urinary catheter.
- Give muscle relaxants and sedatives, as ordered, and schedule patient care — such as passive range-of-motion exercises — to coincide with periods of heaviest sedation.
- Insert an artificial airway, if necessary, to prevent tongue injury and maintain airway during spasms.
- Provide adequate nutrition to meet the patient's increased metabolic needs. The patient may need nasogastric feedings or total parenteral nutrition. (See *Preventing tetanus.*)

PREVENTION

PREVENTING TETANUS

Tetanus can be easily prevented through immunization against the toxin. Almost all cases of tetanus occur in people who've never been immunized or who haven't had a tetanus booster shot within the last 10 years. Having had a tetanus infection doesn't provide immunity. Following recommendations for vaccinations is necessary to prevent recurrence of tetanus.

The tetanus vaccine is usually given to children as part of the diphtheria and tetanus toxoids and pertussis (DTP) shot.

It's recommended that adolescents get a booster shot between the ages of 11 and 18, and that adults receive a routine tetanus booster shot every 10 years. In the event of a deep or dirty wound and it has been more than five years since the last booster shot, it is recommended that another booster be given.

Tell your patient to foloow these guidelines to help prevent tetnus infection.

Take care of a wound

Tell the patient to do the following:

- Rinse the wound thoroughly with clean water.
- Clean the wound and the area around it with soap and a washcloth.
- Contact a practitioner if debris is embedded in the wound.

Consider the wound source

- Tell the patient to contact a practitioner if the wound is deep, especially dirty, or if it's a result of an animal bite.
- Tell the patient to contact a practitioner if the date of his most recent tetanus shot is uncertain.

Use antibiotic ointment or cream

- After the wound has been cleaned, the patient should apply a thick layer of an antibiotic cream or ointment, such as the multi-ingredient antibiotics Neosporin or Polysporin.

Cover the wound

- Bandages can help keep the wound clean and keep harmful bacteria out. Have the patient cover blisters that are draining; they're vulnerable to infection until a scab forms.

Change the dressing

- To help prevent infection, tell the patient to apply a new dressing at least once per day or whenever the dressing becomes wet or dirty.

Botulism

Botulism, a life-threatening paralytic illness, results from an exotoxin produced by the gram-positive, anaerobic bacillus *Clostridium botulinum*. It occurs with botulism food poisoning, wound botulism, and infant botulism. The mortality from botulism is about 25%; death is usually caused by respiratory failure during the first week of illness.

Causes and incidence

Botulism is usually the result of ingesting inadequately cooked contaminated foods,

especially those with low acid content, such as home-canned fruits and vegetables, sausages, and smoked or preserved fish or meat. Rarely, it's a result of wound infection with *C. botulinum*.

Botulism occurs worldwide and affects more adults than children. Recently, findings have shown that an infant's GI tract can become colonized with *C. botulinum* from some unknown source, and then the exotoxin is produced within the infant's intestine. Infant botulism is usually attributed to the ingestion of honey or corn syrup. Incidence had been declining, but the current trend toward home canning has resulted in an upswing (approximately 250 cases per year in the United States) in recent years.

Complications

- Respiratory failure
- Paralytic ileus

Signs and symptoms

Symptoms usually appear within 12 to 36 hours (range is 6 hours to 8 days) after the ingestion of contaminated food. Severity varies with the amount of toxin ingested and the patient's degree of immunocompetence. Generally, early onset (within 24 hours) signals critical and potentially fatal illness. Initial signs and symptoms include dry mouth, sore throat, weakness, dizziness, vomiting, and diarrhea. The cardinal sign of botulism, though, is acute symmetrical cranial nerve impairment (ptosis, diplopia, and dysarthria), followed by descending weakness or paralysis of muscles in the extremities or trunk, and dyspnea from respiratory muscle paralysis. Such impairment doesn't affect mental or sensory processes and isn't associated with fever.

Infant botulism usually afflicts infants between 3 and 20 weeks of age and can produce hypotonic (floppy) infant syndrome. Signs and symptoms are constipation, feeble cry, depressed gag reflex, and inability to suck. Cranial nerve deficits also occur in infants and are manifested by a flaccid facial expression, ptosis, and ophthalmoplegia. Infants also develop generalized muscle weakness, hypotonia, and areflexia. Loss of head control may be striking. Respiratory arrest is likely.

Diagnosis

CONFIRMING DIAGNOSIS

Identification of the offending toxin in the patient's serum, stool, gastric content, or the suspected food, confirms the diagnosis. An electromyogram showing diminished muscle action potential after a single supramaximal nerve stimulus is also diagnostic.

Diagnosis also must rule out other diseases commonly confused with botulism, such as Guillain-Barré syndrome, myasthenia gravis, stroke, staphylococcal food poisoning, tick paralysis, chemical intoxications, carbon monoxide poisoning, fish poisoning, trichinosis, and diphtheria.

Treatment

Treatment consists of I.V. or I.M. administration of botulinus antitoxin (available through the Centers for Disease Control and Prevention).

If breathing difficulty develops, intubation and mechanical ventilation may be required. I.V. fluids can be given if there are swallowing problems, and a nasogastric tube can also be ordered.

Special considerations

If you suspect ingestion of contaminated food:

- Obtain a careful history of the patient's food intake for the past several days. See if other family members exhibit similar symptoms and share a common food history.
- Observe carefully for abnormal neurologic signs. Tell the patient's family to watch for signs of weakness, blurred vision, and slurred speech after he has returned home. If such signs appear, the patient must return to the hospital immediately.
- If ingestion has occurred within several hours, induce vomiting, begin gastric lavage, and give a high enema to purge any unabsorbed toxin from the bowel.

If clinical signs of botulism appear:

- Admit the patient to the intensive care unit, and monitor cardiac and respiratory functions carefully.
- Administer botulinus antitoxin, as ordered, to neutralize any circulating toxin. Before giving the antitoxin, be sure to obtain an accurate patient history of allergies,

  especially to horses, and perform a skin test. Afterward, watch for anaphylaxis or other hypersensitivity and serum sickness. Keep epinephrine 1:1,000 (for subcutaneous administration) and emergency airway equipment available.

- Assess respiratory function every 4 hours. Report decreased vital capacity on inspiratory effort and any signs of respiratory distress.
- Closely assess and accurately record neurologic function, including bilateral motor status (reflexes, ability to move arms and legs).
- Give I.V. fluids as ordered. Turn the patient often, and encourage deep-breathing exercises. Isolation isn't required.
- As botulism is sometimes fatal, keep the patient and his family informed regarding the course of the disease.
- Immediately report all cases of botulism to local public health authorities.

PREVENTION

Encourage patients to observe proper techniques in processing and preserving foods. Warn them to avoid even tasting food from a bulging can or one with a peculiar odor and to sterilize by boiling any utensil that comes in contact with suspected food. Ingestion of even a small amount of food contaminated with botulism toxin can prove fatal.

Gas gangrene

Gas gangrene results from local infection with the anaerobic, spore-forming, gram-positive rod *Clostridium perfringens* (or another clostridial species). It occurs in devitalized tissues and results from compromised arterial circulation after trauma, surgery, compound fractures, or lacerations. This rare infection carries a high mortality unless therapy begins immediately; however, with prompt treatment, 80% of patients with gas gangrene of the extremities survive. The prognosis is poorer for gas gangrene in other sites, such as the abdominal wall or the bowel. The usual incubation period is 1 to 4 days but can vary from 3 hours to 6 weeks or longer.

Causes and incidence

C. perfringens is a normal inhabitant of the GI and female genital tracts; it's also prevalent in soil. Transmission occurs by entry of organisms during trauma or surgery. Because *C. perfringens* is anaerobic and spore forming, gas gangrene is usually found in deep wounds, especially those in which tissue necrosis further reduces oxygen supply. Clostridium bacteria produce four different toxins (alpha, beta, epsilon, iota) that can cause potentially fatal symptoms. When *C. perfringens* invades soft tissues, it produces thrombosis of regional blood vessels, tissue necrosis, localized edema, and damage to the myocardium, liver, and kidneys. (See *Growth cycle of* Clostridium perfringens.) Such necrosis releases both carbon dioxide and hydrogen subcutaneously, producing interstitial gas

bubbles. Gas gangrene usually occurs in the extremities and in abdominal wounds; it's less common in the uterus.

Gas gangrene is rare, with only 1,000 to 3,000 cases occurring in the United States annually.

Complications

- Renal failure
- Hypotension
- Hemolytic anemia
- Shock
- Tissue death
- Amputation

Signs and symptoms

True gas gangrene produces myositis and another form of this disease, involving only soft tissue, called *anaerobic cellulitis*. Most signs of infection develop within 72 hours of trauma or surgery. The hallmark of gas gangrene is crepitation (a crackling sensation when the skin is touched), a result of carbon dioxide and hydrogen accumulation as a metabolic by-product in necrotic tissues. Other typical indications are severe localized pain, swelling, and discoloration (usually dusky brown or reddish), with formation of bullae and necrosis within 36 hours from onset of symptoms. The skin over the wound may rupture, revealing dark red or black necrotic muscle, a foul-smelling watery or frothy discharge, intravascular hemolysis, thrombosis of

blood vessels, and evidence of infection spread.

In addition to these local symptoms, gas gangrene produces early signs of toxemia and hypovolemia (tachycardia, tachypnea, and hypotension), with moderate fever usually not above 101° F (38.3° C). Although pale, prostrate, and motionless, patients with gas gangrene may exhibit toxic delirium and are extremely apprehensive. Possible sudden death is preceded by delirium and coma and is sometimes accompanied by vomiting, profuse diarrhea, and circulatory collapse.

ELDER TIP

Absence of a fever doesn't necessarily mean absence of infection in an elderly person. Many older adults develop a subnormal temperature in response to infection.

Diagnosis

CONFIRMING DIAGNOSIS

A history of recent surgery or a deep puncture wound and the rapid onset of pain and crepitation around the wound suggest gas gangrene.

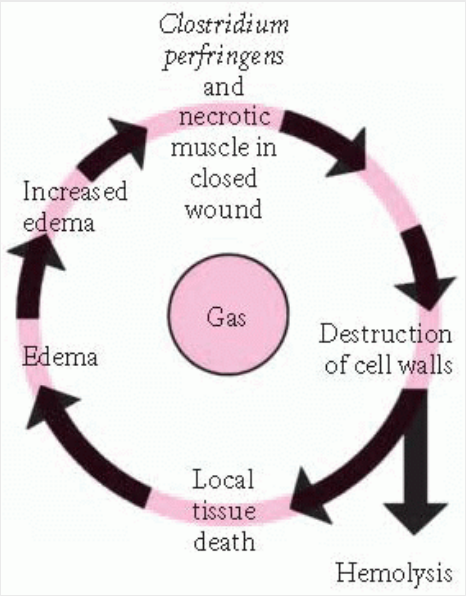
Gas gangrene is confirmed by anaerobic cultures of wound drainage showing *C. perfringens;* a Gram stain of wound drainage showing large, gram-positive, rodshaped bacteria; X-rays showing gas in tissues; and blood studies showing leukocytosis and, later, hemolysis.

Diagnosis must rule out synergistic gangrene and necrotizing fasciitis; unlike gas gangrene, both these disorders anesthetize the skin around the wound.

Treatment

Treatment includes careful observation for signs of myositis and cellulitis and *immediate treatment* if these signs appear; *immediate* wide surgical excision of all affected tissues and necrotic muscle in myositis (*delayed or inadequate surgical excision is a fatal mistake*); I.V. administration of high-dose penicillin; and, after adequate debridement, hyperbaric oxygenation, if available. For 1 to 3 hours every 6 to 8 hours, the patient is placed in a hyperbaric chamber, exposing him to pressures designed to increase oxygen tension and prevent multiplication of the anaerobic clos-tridia. Surgery may be performed within the hyperbaric chamber if the chamber is large enough.

GROWTH CYCLE OF *CLOSTRIDIUM PERFRINGENS*



Special considerations

Careful observation may result in early diagnosis. Look for signs and symptoms of ischemia (cool skin; pallor or cyanosis; sudden, severe pain; sudden edema; and loss of pulses in involved limb).

After the diagnosis:

- Throughout this illness, provide adequate fluid replacement, and assess pulmonary and cardiac functions often. Maintain airway and ventilation.
- To prevent skin breakdown and further infection, give good skin care. After surgery, provide meticulous wound care.
- Before penicillin administration, obtain a patient history of allergies; afterward, watch closely for signs of hypersensitivity.
- Psychological support is critical, because these patients can remain alert until death, knowing that death is imminent and unavoidable.
- Deodorize the room to control foul odor from the wound. Prepare the patient emotionally for a large wound after surgical excision,

  and refer him for physical rehabilitation, as necessary.
- Institute standard precautions. Dispose of drainage material properly, and wear sterile gloves when changing dressings. Spore-forming bacteria aren't destroyed by ordinary disinfecting methods. Contaminated items should be cleaned and disinfected or sterilized, as appropriate.

PREVENTION

- *Routinely take precautions to render all wound sites unsuitable for growth of clostridia by attempting to keep granulation tissue viable; adequate debridement is imperative to reduce anaerobic growth conditions. The surgeon may delay closure of wounds.*
- *Be alert for devitalized tissues, and notify the surgeon promptly.*
- *Position the patient to facilitate drainage, and eliminate all dead spaces in closed wounds.*

Actinomycosis

Actinomycosis is a rare infection primarily caused by the gram-positive anaerobic bacillus *Actinomyces israelii*, which produces granulomatous, suppurative lesions with abscesses. Common infection sites are the head, neck, thorax, and abdomen, but it can spread to contiguous tissues, causing multiple draining sinuses.

Causes and incidence

A. israelii occurs as part of the normal flora of the throat, tonsillar crypts, and mouth (particularly around carious teeth); infection results from its traumatic introduction into body tissues.

Actinomycosis affects twice as many males — especially those ages 15 to 35 — as females. People with dental disease or human immunodeficiency virus infection are at increased risk.

Complications

- Sinus and maxilla facial subcutaneous tissue involvement
- Abscesses and fistulas of the brain
- Pneumonia
- Empyema

Signs and symptoms

Symptoms appear from days to months after injury and may vary, depending on the site of infection.

In *cervicofacial actinomycosis* (lumpy jaw), painful, indurated swellings appear in the mouth or neck up to several weeks after dental extraction or trauma. They gradually enlarge and form fistulas that open onto the skin. Sulfur granules (yellowish gray masses that are actually colonies of *A. israelii*) appear in the exudate.

In *pulmonary actinomycosis*, aspiration of bacteria from the mouth into areas of the lungs already anaerobic from infection or atelectasis produces a fever and a cough that becomes productive and occasionally causes hemoptysis. Eventually, empyema follows, a sinus forms through the chest wall, and septicemia may occur.

In *GI actinomycosis*, ileocecal lesions are caused by swallowed bacteria, which produce abdominal discomfort, fever, sometimes a palpable mass, and an external sinus. This follows intestinal mucosa disruption, usually by surgery or an inflammatory bowel condition such as appendicitis.

Rare sites of actinomycotic infection are the bones, brain, liver, kidneys, and female reproductive organs. Symptoms reflect the organ involved.

Diagnosis

CONFIRMING DIAGNOSIS

Isolation of A. israelii *in exudate or tissue confirms actinomycosis. Other tests that help identify this condition are:*

- *microscopic examination of sulfur granules*
- *Gram staining of excised tissue or exudate to reveal branching gram-positive rods*
- *chest X-ray to show lesions in unusual locations such as the shaft of a rib.*

Treatment

Treatment is long term, with 1 to 2 months of penicillin I.V. followed by 6 to 12 months of penicillin taken by mouth. Doxycycline usually isn't prescribed for children until after permanent teeth have erupted. In some cases, surgical drainage of the lesion may be required.

Special considerations

- Dispose of all dressings in a sealed plastic bag.
- After surgery, provide proper sterile wound management.
- Administer antibiotics as ordered. Before giving the first dose, obtain an accurate patient history of allergies. Watch for hypersensitivity reactions, such as rash, fever, itching, and signs of anaphylaxis. If the patient has a history of any allergies, keep epinephrine 1:1,000 and resuscitation equipment available.

PREVENTION

Stress the importance of good oral hygiene and proper dental care.

Nocardiosis

Nocardiosis is an acute, subacute, or chronic bacterial infection caused by a weakly gram-positive species of the genus *Nocardia* — usually *Nocardia asteroides*. It's most common in men, especially those with a compromised immune system. In patients with brain infection, mortality exceeds 80%; in other forms, mortality is 50%, even with appropriate therapy.

Causes and incidence

Nocardia are aerobic gram-positive bacteria with branching filaments resembling fungi. Normally found in soil, these organisms cause occasional sporadic disease in

humans and animals throughout the world. Their incubation period is unknown but is probably several weeks. The usual mode of transmission is inhalation of organisms suspended in dust. Transmission by direct inoculation through puncture wounds or abrasions is less common.

Complications

- Meningitis
- Seizures
- Cardiac arrhythmias

Signs and symptoms

Nocardiosis originates as a pulmonary infection with a cough that produces thick, tenacious, purulent, mucopurulent, and possibly blood-tinged sputum. It may also cause a fever as high as 105° F (40.6° C), chills, night sweats, anorexia, malaise, and weight loss. This infection may lead to pleurisy, intrapleural effusions, and empyema. Other potential complications include tracheitis, bronchitis, pericarditis, endocarditis, peritonitis, mediastinitis, septic arthritis, and keratoconjunctivitis.

If the infection spreads through the blood to the brain, abscesses form, causing confusion, disorientation, dizziness, headache, nausea, and seizures. Rupture of a brain abscess can cause purulent meningitis. Extrapulmonary, hematogenous spread may cause endocarditis or lesions in the kidneys, liver, subcutaneous tissue, and bone.

Diagnosis

Identifying *Nocardia* by culture of sputum or discharge is difficult. In many cases, special staining techniques must be used to make the diagnosis, in conjunction with a typical clinical picture (usually progressive pneumonia, despite antibiotic therapy). Occasionally, diagnosis requires biopsy of lung or other tissue. Chest X-rays and computed tomography may show fluffy or interstitial infiltrates, nodules, or abscesses.

In brain infection with meningitis, lumbar puncture shows nonspecific changes such as increased opening pressure. Cerebrospinal fluid shows increased white blood cell and protein levels and decreased glucose levels compared with serum glucose.

Treatment

Nocardiosis requires at least 6 months of treatment, preferably with co-trimoxazole or high doses of sulfonamides. In patients who don't respond to

sulfonamide treatment, other drugs, such as ampicillin, erythromycin, or minocycline, may be added. Treatment also includes surgical drainage of abscesses and excision of necrotic tissue. The acute phase requires complete bed rest; as the patient improves, activity can increase.

Special considerations

Because it isn't transmitted from person to person, nocardiosis requires no isolation.

- Provide adequate nourishment through total parenteral nutrition, nasogastric tube feedings, or a balanced diet.
- Give the patient tepid sponge baths and antipyretics, as ordered, to reduce his fever.
- Monitor for allergic reactions to antibiotics.
- High-dose sulfonamide therapy (especially sulfadiazine) predisposes the patient to crystalluria and oliguria; so assess him frequently, force fluids, and alkalinize the urine with sodium bicarbonate, as ordered, to prevent these complications.
- In patients with pulmonary infection, administer chest physiotherapy. Auscultate the lungs daily, checking for increased crackles or consolidation. Note and record the amount, color, and thickness of sputum.
- In brain infection, regularly assess neurologic function. Watch for signs of increased intracranial pressure, such as a decreased level of consciousness and respiratory abnormalities.
- In long-term hospitalization, turn the patient often, and assist with range-of-motion exercises.
- Before the patient is discharged, stress the need to follow a regular medication schedule to maintain therapeutic blood levels and to continue drugs even after symptoms subside. Explain the importance of frequent follow-up examinations.
- Provide support and encouragement to help the patient and his family cope with this long-term illness.

Clostridium difficile infection

Clostridium difficile is a gram-positive anaerobic bacterium that typically causes antibiotic-associated diarrhea. Symptoms may range from asymptomatic carrier states to severe pseudomembranous colitis and are caused by the exotoxins produced by the organism. Toxin A is an enterotoxin and toxin B is a cytotoxin.

Causes and incidence

C. difficile colitis can be caused by almost any antibiotic that disrupts the bowel flora, but it's classically associated with clindamycin use. High-risk groups include individuals on greater numbers of antibiotics, those having abdominal surgery, patients receiving antineoplastics that have an antibiotic activity, immunocompromised individuals, pediatric patients (commonly in day-care centers), and nursing home patients.

Other factors that alter normal intestinal flora include enemas and intestinal stimulants. *C. difficile* is most often transmitted directly from patient to patient by contaminated hands of facility personnel; it may also be indirectly spread by contaminated equipment such as bedpans, urinals, call bells, rectal thermometers, nasogastric tubes, and contaminated surfaces such as bed rails, floors, and toilet seats.

Complications

- Electrolyte abnormalities
- Hypovolemic shock
- Anasarca (caused by hypoalbuminemia)
- Toxic megacolon
- Colonic perforation
- Peritonitis
- Sepsis
- Hemorrhage
- Death (rare)

Signs and symptoms

Risk of *C. difficile* begins 1 to 2 days after antibiotic therapy is started and extends for as long as 2 to 3 months after the last dose. The patient may be asymptomatic or may exhibit any of the following symptoms: soft, unformed, or watery diarrhea (more than 3 stools in a 24-hour period) that may be foul smelling or grossly bloody; abdominal pain, cramping, or tenderness; and fever. The patient's white blood cell count may be elevated to 20,000. In severe cases, toxic megacolon, colonic perforation, and peritonitis may develop.

Diagnosis

Diagnosis is by identification of the toxin through one of these acceptable methods:

- *cell cytotoxin test* — tests for both toxin A and B; this takes 2 days to perform. It's highly sensitive and specific for *C. difficile*.
- *enzyme immunoassays* — slightly less sensitive than the cell cytotoxin test but has

  a turnaround time of only a few hours. Specificity is excellent.
- *stool culture* — the most sensitive test; has a turnaround time of 2 days to obtain results. Non-toxin-producing strains of *C. difficile* can be easily identified and must be further tested for presence of the toxin.
- *endoscopy (flexible sigmoidoscopy)* — may be used in a patient who presents with an acute abdomen but no diarrhea, making it difficult to obtain a stool specimen. If pseudomembranes are visualized, treatment for *C. difficile* is usually initiated.

Treatment

After withdrawing the causative antibiotic (if possible), symptoms resolve in patients who are mildly symptomatic. This is usually the only treatment needed. In more severe cases, metronidazole or vancomycin are effective therapies; metronidazole is the preferred treatment. Retesting for *C. difficile* is unnecessary if symptoms resolve.

About 10% to 20% of patients experience recurrence with the same organism within 14 to 30 days of treatment. Beyond 30 days, a recurrence may be a relapse or reinfection of *C. difficile*. If the previous treatment was metronidazole, low-dose vancomycin may be an effective choice.

There's no evidence to support the effectiveness of eating yogurt or taking lactobacillus. Other experimental treatments involve the administration of yeast *Saccharomyces boulardii* with metronidazole or vancomycin and biologic vaccines to restore the normal GI flora. I.V. immunoglobulin has been used successfully in children and adults with relapsing infections, although it's still being studied.

Special considerations

- Patients with known or suspected *C. difficile* diarrhea who are unable to practice good hygiene should be placed on contact precautions in a single room or in a room with other patients with similar status.
- Use contact precautions for contact with blood and body fluids and for all direct contact with the patient and his immediate environment.
- Wash your hands with an antiseptic soap after direct contact with the patient or the immediate environment.

- A patient who is asymptomatic, without diarrhea or fecal incontinence for 72 hours, and who is able to practice good hygiene may have contact precautions discontinued.

ALERT

The spores of C. difficile *are resistant to most common facility disinfectants; therefore, contamination will remain in the room even though the patient may be discharged. The immediate environment should be thoroughly cleaned and disinfected with 0.5% sodium hypochlorite.*

- Make sure reusable equipment is disinfected before it's used on another patient.
- Teach good hand-washing technique to prevent the spread of the infection.
- Review proper disinfection of contaminated clothing or household items.
- Tell the patient to inform health care workers of his condition before admission.

GRAM-NEGATIVE BACILLI

Salmonellosis

A common infection in the United States, salmonellosis is caused by gram-negative bacilli of the genus *Salmonella*, a member of the Enterobacteriaceae family. It occurs as enterocolitis, bacteremia, localized infection, typhoid, or paratyphoid fever. Nontyphoidal forms usually produce mild to moderate illness with low mortality. (See *Types of salmonellosis*, page 936.)

Typhoid, the most severe form of salmonellosis, usually lasts from 1 to 4 weeks. Mortality is about 3% in patients who are treated. In those who are untreated, 10% of cases result in fatality, usually as a result of intestinal perforation or hemorrhage, cerebral thrombosis, toxemia, pneumonia, or acute circulatory failure. An attack of typhoid confers lifelong immunity, although the patient may become a carrier. Salmonellosis is 20 times more common in patients with acquired immunodeficiency syndrome. Features are increased incidence of bacteremia, inability to identify the infection source, and tendency of infection to recur after therapy is stopped.

TYPES OF SALMONELLOSIS

Type	Cause	Clinical features
Bacteremia	Any *Salmonella* species, but most commonly *S. choleraesuis*. Incubation period: variable	Fever, chills, anorexia, weight loss (without GI symptoms), and joint pain

Enterocolitis	Any species of nontyphoidal *Salmonella,* but usually *S. enteritidis.* Incubation period: 6 to 48 hours	Mild to severe abdominal pain, diarrhea, sudden fever of up to 102° F (38.9° C), nausea, and vomiting; usually self-limiting, but may progress to enteric fever (resembling typhoid), local abscesses (usually abdominal), dehydration, and septicemia
Localized infections	Usually follows bacteremia caused by *Salmonella* species	Site of localization determines symptoms; localized abscesses may cause osteomyelitis, endocarditis, bronchopneumonia, pyelonephritis, and arthritis
Paratyphoid	*S. paratyphi* and *S. schottmuelleri* (formerly *S. paratyphi B*). Incubation period: 3 weeks or more	Fever and transient diarrhea; generally resembles typhoid but less severe
Typhoid fever	*S. typhi* enters the GI tract and invades the bloodstream via the lymphatics, setting up intracellular sites. During this phase, infection of the biliary tract leads to intestinal seeding with millions of bacilli. Involved lymphoid tissues (especially Peyer's patches in the ilium) enlarge, ulcerate, and necrose, resulting in hemorrhage. Incubation period: usually 1 to 2 weeks	Symptoms of enterocolitis may develop within hours of ingestion of *S. typhi;* they usually subside before onset of typhoid fever symptoms *First week:* gradually increasing fever, anorexia, myalgia, malaise, headache, and slow pulse *Second week:* remittent fever up to 104° F (40° C) usually in the evening, chills, diaphoresis, weakness, delirium, increasing abdominal pain and distention, diarrhea or constipation, cough, moist crackles, tender abdomen with enlarged spleen, and maculopapular rash (especially on abdomen) *Third week:* persistent fever, increasing fatigue and weakness; usually subsides by end of third week, although relapses may occur *Complications:* intestinal perforation or hemorrhage, abscesses, thrombophlebitis, cerebral thrombosis, pneumonia, osteomyelitis, myocarditis, acute circulatory failure, and chronic carrier state

Causes and incidence

Of an estimated 1,700 serotypes of *Salmonella*, 10 cause the diseases most common in the United States; all 10 can survive for weeks in water, ice, sewage, or food. Nontyphoidal salmonellosis generally follows the ingestion of contaminated or inadequately processed foods, especially eggs, chicken, turkey, and duck. Proper cooking reduces

the risk of contracting salmonellosis. Other causes include contact with infected people or animals or ingestion of contaminated dry milk, chocolate bars, or drugs of animal origin. Salmonellosis may occur in children younger than age 5 from fecal-oral spread. Enterocolitis and bacteremia are common (and more virulent) among infants, elderly persons, and people already weakened by other infections; paratyphoid fever is rare in the United States.

Typhoid usually results from drinking water contaminated by excretions of a carrier or from ingesting contaminated shellfish. (Contamination of shellfish occurs by leakage of sewage from offshore disposal depots.) Most typhoid patients are younger than age 30; most carriers are women older than age 50. Incidence of typhoid in the United States is increasing as a result of travelers returning from endemic areas.

Complications

- Intestinal perforation
- Intestinal hemorrhage
- Cerebral thrombosis
- Pneumonia
- Endocarditis
- Myocarditis
- Meningitis
- Pyelonephritis
- Osteomyelitis
- Cholecystitis
- Hepatitis
- Septicemia
- Acute circulatory failure
- Reiter's syndrome

Signs and symptoms

Clinical manifestations of salmonellosis vary but usually include fever, abdominal pain, and severe diarrhea with enterocolitis. Headache, increasing fever, and constipation are more common in typhoidal infection.

Diagnosis

Generally, diagnosis depends on isolation of the organism in a culture, particularly blood (in typhoid, paratyphoid, and bacteremia) or feces (in enterocolitis, paratyphoid, and typhoid). Other appropriate culture specimens include urine, bone marrow, pus, and vomitus. In endemic areas, clinical symptoms of enterocolitis allow a working diagnosis before the cultures are positive. The presence of *Salmonella typhi* in stool 1 or more years after treatment indicates that the patient is a carrier, which is true of 3% of patients.

Widal's test, an agglutination reaction against somatic and flagellar antigens, may suggest typhoid with a fourfold rise in titer. However, drug use or hepatic disease can also increase these titers and invalidate test results. Other supportive laboratory values may include transient leukocytosis during the 1st week of typhoidal salmonellosis, leukopenia during the 3rd week, and leukocytosis in local infection.

Treatment

Antimicrobial therapy for typhoid, paratyphoid, and bacteremia depends on organism sensitivity. It may include amoxicillin, chloramphenicol and, in severely toxemic patients, co-trimoxazole, ciprofloxacin, or ceftriaxone. Localized abscesses may also need surgical drainage. Enterocolitis requires a short course of antibiotics only if it causes septicemia or prolonged fever. Other treatments include bed rest and fluid and electrolyte replacement. The administration of camphorated opium tincture, kaolin with pectin, diphenoxylate, codeine, or small doses of morphine may be necessary to relieve diarrhea and control cramps in patients who must remain active.

Special considerations

- All infections caused by *Salmonella* must be reported to the state health department.
- Follow contact precautions if the patient is incontinent or diapered; otherwise, standard precautions are appropriate. Always wash your hands thoroughly before and after any contact with the patient, and advise other facility personnel to do the same. Teach the patient to use proper hand washing, especially after defecating and before eating or handling food. Wear gloves and a gown when disposing of feces or fecally contaminated objects. Continue precautions until three consecutive stool cultures are negative — the first one taken 48 hours after antibiotic treatment ends, followed by two more at 24-hour intervals.

- Observe the patient closely for signs and symptoms of bowel perforation from erosion of intestinal ulcers: sudden pain in the lower right side of the abdomen and abdominal rigidity, possibly after one or more rectal bleeding episodes; sudden fall in temperature or blood pressure; and rising pulse rate (indicating shock).
- During acute infection, plan care and activities to allow the patient as much rest as possible. Raise the side rails and use other safety measures, because the patient may become delirious. Assign him a room close to the nurses' station so he can be checked often. Use a room deodorizer (preferably electric) to minimize odor from diarrhea and to provide a comfortable atmosphere for rest.
- Accurately record intake and output. Maintain adequate I.V. hydration. When the patient can tolerate oral feedings, encourage high-calorie fluids such as milkshakes. Watch for constipation.
- Provide good skin and mouth care. Turn the patient frequently, and perform mild passive exercises, as indicated. Apply mild heat to the abdomen to relieve cramps.
- *Don't* administer antipyretics. These mask fever and lead to possible hypothermia. Instead, to promote heat loss through the skin without causing shivering (which keeps fever high by vasoconstriction), apply tepid, wet towels (don't use alcohol or ice) to the patient's groin and axillae. To promote heat loss by vasodilation of peripheral blood vessels, use additional wet towels on the arms and legs, wiping with long, vigorous strokes.
- After draining the abscesses of a joint, provide heat, elevation, and passive range-of-motion exercises to decrease swelling and maintain mobility.
- If the patient has positive stool cultures on discharge, tell him to be sure to wash his hands after using the bathroom and to avoid preparing uncooked foods, such as salads, for family members. He also shouldn't work as a food handler until cultures are negative. (See *Preventing recurrence of salmonellosis*.)

PREVENTION

PREVENTING RECURRENCE OF SALMONELLOSIS

Take the following actions to help your patient prevent a recurrence of salmonellosis:

- Explain the causes of salmonella infection.
- Show the patient how to wash his hands by wetting them under running water, lathering with soap and scrubbing, rinsing under running water with his fingers pointing down, and drying with a clean towel or paper towel.

- Tell the patient to wash his hands after using the bathroom and before eating.
- Tell the patient to cook foods thoroughly — especially eggs and chicken — and to refrigerate them at once.
- Teach the patient how to avoid cross-contaminating foods by cleaning preparation surfaces with hot, soapy water and drying them thoroughly after use; cleaning surfaces between foods when preparing more than one food; and washing his hands before and after handling each food.
- Tell the patient with a positive stool culture to avoid handling food and to use a separate bathroom or clean the bathroom after each use.
- Tell the patient to report dehydration, bleeding, or recurrence of signs of salmonella infection.

Shigellosis

Shigellosis, also known as *bacillary dysentery,* is an acute intestinal infection caused by the bacteria *Shigella*, a short, nonmotile, gram-negative rod. *Shigella* can be classified into four groups, all of which may cause shigellosis: group A (*S. dysenteriae*), which is most common in Central America and causes particularly severe infection and septicemia; group B (*S. flexneri*); group C (*S. boydii*); and group D (*S. sonnei*). Typically,

shigellosis causes a high fever (especially in children), acute self-limiting diarrhea with tenesmus (ineffectual straining at stool) and, possibly, electrolyte imbalance and dehydration. It's most common in children ages 1 to 4; however, many adults acquire the illness from children.

The prognosis is good. Mild infections usually subside within 10 days; severe infections may persist for 2 to 6 weeks. With prompt treatment, shigellosis is fatal in only 1% of cases, although in severe *S. dysenteriae* epidemic mortality may reach 8%.

Causes and incidence

Transmission occurs through the fecal-oral route; by direct contact with contaminated objects; or through ingestion of contaminated food or water. Occasionally, the housefly is a vector.

Shigellosis is endemic in North America, Europe, and the tropics. In the United States, about 18,000 cases appear annually, usually in children or in elderly,

debilitated, or malnourished people. Shigellosis commonly occurs among confined populations, such as those in mental institutions or day-care centers.

Complications

- Electrolyte imbalances
- Metabolic acidosis
- Shock
- Conjunctivitis
- Uritis
- Arthritis
- Rectal prolapse
- Bacterial infection

Signs and symptoms

After an incubation period of 1 to 7 days (3 days is the average), *Shigella* organisms invade the intestinal mucosa and cause inflammation. In children, shigellosis usually produces high fever, diarrhea with tenesmus, nausea, vomiting, irritability, drowsiness, and abdominal pain and distention. Within a few days, the child's stool may contain pus, mucus, and — from the superficial intestinal ulceration typical of this infection — blood. Without treatment, dehydration and weight loss are rapid and overwhelming.

In adults, shigellosis produces sporadic, intense abdominal pain, which may be relieved at first by passing formed stools. Eventually, however, it causes rectal irritability, tenesmus and, in severe infection, headache and prostration. Stools may contain pus, mucus, and blood. Fever may be present.

Diagnosis

CONFIRMING DIAGNOSIS

Fever (in children) and diarrhea with stools containing blood, pus, and mucus point to this diagnosis; microscopic bacteriologic studies and culture help confirm it.

Microscopic examination of a fresh stool may reveal mucus, red blood cells, and polymorphonuclear leukocytes; direct immunofluorescence with specific antisera will demonstrate *Shigella*. Severe infection increases hemagglutinating antibodies. Sigmoidoscopy or proctoscopy may reveal typical superficial ulcerations.

Diagnosis must rule out other causes of diarrhea, such as enteropathogenic *Escherichia coli* infection, malabsorption diseases, and amebic or viral diseases.

Treatment

Treatment of shigellosis includes enteric precautions, low-residue diet and, most important, replacement of fluids and electrolytes with I.V. infusions of normal saline solution (with electrolytes) in sufficient quantities to maintain a urine output of 40 to 50 ml/hour. Antibiotics are of questionable value but may be used in an attempt to eliminate the pathogen and thereby prevent further spread. Ampicillin, tetracycline, or co-trimoxazole may be useful in severe cases, especially in children with overwhelming fluid and electrolyte loss. Sulfamethoxazole-trimethoprim and ciprofloxacin are also used.

Antidiarrheals that slow intestinal motility are contraindicated in shigellosis because they delay fecal excretion of *Shigella* and prolong fever and diarrhea. An investigational vaccine containing attenuated strains of *Shigella* appears promising in preventing shigellosis.

Special considerations

Supportive care can minimize complications and increase patient comfort.

- To prevent dehydration, administer I.V. fluids as ordered. Measure intake and output (including stools) carefully.
- Correct identification of *Shigella* requires examination and culture of fresh stool specimens. Therefore, hand carry specimens directly to the laboratory. Because shigellosis is suspected, include this information on the laboratory slip.
- Use a disposable hot-water bottle to relieve abdominal discomfort, and schedule care to conserve patient strength.

PREVENTION

- *To help prevent spread of this disease, maintain enteric precautions until microscopic bacteriologic studies confirm that the stool specimen is negative. If a risk of exposure to the patient's stool exists, put on a gown and gloves before entering the room. Keep the patient's (and your own) nails short to avoid harboring organisms. Change soiled linen promptly and store in an isolation container.*
- *During shigellosis outbreaks, obtain stool specimens from all potentially infected staff, and instruct those infected to remain away from work until two stool specimens are negative.*

- *Report cases to the local health department.*

Escherichia coli and other Enterobacteriaceae infections

The Enterobacteriaceae—a group of mostly aerobic, gram-negative bacilli — cause local and systemic infections, including an invasive diarrhea resembling shigella and, more commonly, a noninvasive toxinmediated diarrhea resembling cholera. With other Enterobacteriaceae, *Escherichia coli* causes most nosocomial infections. Noninvasive, enterotoxin-producing *E. coli* infections may be a major cause of diarrheal illness in children in the United States. (See *Enterobacterial infections.*)

The prognosis in mild to moderate infection is good. Severe infection requires immediate fluid and electrolyte replacement to avoid fatal dehydration, especially among children, in whom mortality may be quite high.

Causes and incidence

Although some strains of *E. coli* exist as part of the normal GI flora, infection usually results from certain nonindigenous strains. For example, noninvasive diarrhea results from two toxins produced by strains called enterotoxic or enteropathogenic *E. coli*. Enteropathogenic *E. coli* serotype 0157:H7 is the most well-known strain in the United States. These toxins interact with intestinal juices and promote excessive loss of chloride and water. In the invasive form, *E. coli* directly invades the intestinal mucosa without producing enterotoxins, thereby causing local irritation, inflammation, and diarrhea. Normal strains can cause infection in immunocompromised patients.

Transmission can occur directly from an infected person or indirectly by ingestion of contaminated food or water or contact with contaminated utensils. Incubation takes 12 to 72 hours.

Incidence of *E. coli* infection is highest among travelers returning from other countries, particularly Mexico, Southeast Asia, and South America. *E. coli* infection also induces other diseases, especially in people whose resistance is low. The strain *E. coli* 0157:H7 has been associated with undercooked hamburger and with animals and petting zoos.

Complications

- Bacteremia
- Severe dehydration
- Life-threatening electrolyte disturbances

- Acidosis
- Shock

Signs and symptoms

Effects of noninvasive diarrhea depend on the causative toxin but may include the abrupt onset of watery diarrhea with cramping abdominal pain and, in severe illness, acidosis. Invasive infection produces

chills, abdominal cramps, and diarrheal stools containing blood and pus.

Infantile diarrhea from an *E. coli* infection is usually noninvasive; it begins with loose, watery stools that change from yellow to green and contain little mucus or blood. Vomiting, listlessness, irritability, and anorexia commonly precede diarrhea. This condition can progress to fever, severe dehydration, acidosis, and shock. Bloody diarrhea may occur from infection with *E. coli* 0157:H7, which has also been associated with hemolytic uremic syndrome in children.

Diagnosis

Because certain strains of *E. coli* normally reside in the GI tract, culturing is of little value; a working diagnosis depends on clinical observation. However, if *E. coli* 0157:H7 is suspected, notify the laboratory so that appropriate testing of stool specimens can be performed.

A firm diagnosis requires sophisticated identification procedures, such as bioassays, that are expensive, time-consuming and, consequently, not widely available. Diagnosis must rule out salmonellosis and shigellosis, other common infections that produce similar signs and symptoms.

Treatment

Treatment consists of correction of fluid and electrolyte imbalances; for an infant or immunocompromised patient, I.V. antibiotics based on the organism's drug sensitivity; and salicylates or opium tincture for cramping and diarrhea.

Special considerations

- Keep accurate intake and output records. Measure stool volume and note the presence of blood or pus. Replace fluids and electrolytes as needed, monitoring for decreased serum sodium and chloride levels and signs of gram-negative shock. Watch for signs of dehydration, such as poor skin turgor and dry mouth.
- For infants, use contact precautions, give nothing by mouth, administer antibiotics as ordered, and maintain body warmth.

To prevent spread of this infection:

ENTEROBACTERIAL INFECTIONS

The Enterobacteriaceae include *Escherichia coli, Arizona, Citrobacter, Enterobacter, Erwinia, Hafnia, Klebsiella, Morganella, Proteus, Providencia, Salmonella, Serratia, Shigella,* and *Yersinia.*

Enterobacterial infections are exogenous (from other people or the environment), endogenous (from one part of the body to another), or a combination of both. Enterobacteriaceae infections may cause any of a long list of bacterial diseases: bacterial (gram-negative) pneumonia, empyema, endocarditis, osteomyelitis, septic arthritis, urethritis, cystitis, bacterial prostatitis, urinary tract infection, pyelonephritis, perinephric abscess, abdominal abscesses, cellulitis, skin ulcers, appendicitis, gastroenterocolitis, diverticulitis, eyelid and periorbital cellulitis, corneal conjunctivitis, meningitis, bacteremia, and intracranial abscesses.

Appropriate antibiotic therapy depends on the results of culture and sensitivity tests. Generally, the aminoglycosides, cephalosporins, and penicillins — such as ampicillin, mezlocillin, and piperacillin — are most effective. Cefepime and quinones are also effective for some infections.

- Prevent direct patient contact during epidemics. Report cases to local public health authorities. *E. coli* 0157:H7 is a reportable disease.
- Use proper hand-washing technique. Teach health care personnel, patients, and their families to do the same.
- Follow standard precautions. Provide the patient with a private room, wear protective clothing as necessary, such as when handling feces or soiled linens, and perform scrupulous hand washing before entering and after leaving the patient's room.
- Advise travelers to foreign countries to avoid unbottled water and uncooked fruits and vegetables.

MELIOIDOSIS

Melioidosis results from wound penetration, inhalation, or ingestion of the gram-negative bacteria produced by the Burkholderia species (gram-negative rods). Although it was once confined to Southeast Asia, Central America, South America,

Madagascar, and Guam, incidence in the United States has risen as a result of the influx of Southeast Asians.

Melioidosis occurs in two forms: chronic melioidosis, which causes osteomyelitis and lung abscesses, and the rare acute melioidosis, which causes pneumonia, bacteremia, and prostration. Acute melioidosis is commonly fatal; however, most melioidosis infections are chronic and asymptomatic, producing clinical symptoms only with accompanying malnutrition, major surgery, or severe burns.

Diagnostic measures consist of isolation of *P. pseudomallei* in a culture of exudate, blood, or sputum; serology tests (complement fixation, passive hemagglutination); and chest X-ray (findings resemble tuberculosis). Treatment includes oral tetracycline and co-trimoxazole, abscess drainage and, in severe cases, chloramphenicol until X-rays show resolution of primary abscesses.

The prognosis is good because most patients have a mild infection and acquire permanent immunity; aggressive use of antibiotics and sulfonamides has improved the prognosis in acute melioidosis.

Pseudomonas infections

Pseudomonas is a small gram-negative bacillus that produces nosocomial infections, superinfections of various parts of the body, and a rare disease called melioidosis. This bacillus is also associated with bacteremia, endocarditis, and osteomyelitis in drug addicts. In local *Pseudomonas* infections, treatment is usually successful and complications rare; however, in patients with any type of lowered immunologic resistance — premature neonates; elderly patients; patients with debilitating disease, burns, or wounds; or patients receiving chemotherapy or radiation therapy — septicemic *Pseudomonas* infections are serious and commonly fatal.

Causes and incidence

The most common species of *Pseudomonas* is *P. aeruginosa*. Other species that typically cause disease in humans include *Xanthomonas maltophilia* (formerly known as *P. maltophilia*), *Burkholderia cepacia* (formerly known as *P. cepacia*), *P. fluorescens*, *P. testosteroni*, *P. acidovorans*, *P. alcaligenes*, *P. stutzeri*, *P. putrefaciens*, and *P. putida*. These organisms are commonly found in liquids that have been allowed to stand for a long time, such as benzalkonium chloride, saline

solution, penicillin, water in flower vases, and fluids in incubators, humidifiers, and inhalation therapy equipment. *P. aeruginosa* is associated with chronic obstructive pulmonary disease. *B. cepacia* is the organism most closely associated with cystic fibrosis, although *P. aeruginosa* is also associated with it. In elderly patients, *Pseudomonas* infection usually enters through the genitourinary tract; in neonates and infants, through the umbilical cord, skin, and GI tract.

Complications

- Septic shock
- Severe mucopurulent pneumonia
- Systemic inflammatory response syndrome
- Multiple organ dysfunction
- Death

PREVENTION

PREVENTING *PSEUDOMONAS* INFECTION

Maintain proper endotracheal and tracheostomy suctioning technique by doing the following:

- Use strict sterile technique when caring for I.V. lines, catheters, and other tubes.
- Use suction catheters only once.
- Properly dispose of suction bottle contents.
- Label and date solution bottles and change them frequently, according to policy.
- Change water for fresh flowers in the patient's room daily.
- Avoid using humidifiers in the patient's room.

Signs and symptoms

The most common infections associated with *Pseudomonas* include skin infections (such as burns and pressure ulcers), urinary tract infections, infant epidemic diarrhea and other diarrheal illnesses, bronchitis, pneumonia, bronchiectasis, meningitis, corneal ulcers, mastoiditis, otitis externa, otitis media, endocarditis, and bacteremia.

Drainage in *Pseudomonas* infections has a distinct, sickly sweet odor and a greenish blue pus that forms a crust on wounds. Other symptoms depend on the

site of infection. (See *Melioidosis*.) For example, when it invades the lungs, *Pseudomonas* causes pneumonia with fever, chills, and a productive cough.

Diagnosis

CONFIRMING DIAGNOSIS

Diagnosis requires isolation of the Pseudomonas *organism in blood, spinal fluid, urine, exudate, or sputum culture.*

Treatment

In the debilitated or otherwise vulnerable patient with clinical evidence of *Pseudomonas* infection, treatment should begin immediately, without waiting for results of laboratory tests. Antibiotic treatment includes aminoglycosides, such as gentamicin or tobramycin, combined with an antipseudomonal penicillin, such as ticarcillin or piperacillin. An alternative combination is amikacin and a similar penicillin or imipenem and cilastatin. Such combination therapy is necessary because *Pseudomonas* quickly becomes resistant to ticarcillin alone.

Local *Pseudomonas* infections or septicemia secondary to wound infection requires 1% acetic acid irrigations; topical applications of colistimethate, polymyxin B, and silver sulfadiazine cream; and debridement or drainage of the infected wound.

Special considerations

- Observe and record the character of wound exudate and sputum.
- Before administering antibiotics, ask the patient about a history of drug allergies, especially to penicillin. If combinations of piperacillin or ticarcillin and an aminoglycoside are ordered, schedule the doses 1 hour apart (ticarcillin may decrease the antibiotic effect of the aminoglycoside). *Don't* give both antibiotics through the same administration set.
- Monitor the patient's renal function (output, blood urea nitrogen level, specific gravity, urinalysis, and creatinine level) during treatment with aminoglycosides. Obtain drug levels to ensure effectiveness.
- Protect immunocompromised patients from exposure to this infection. Proper hand washing and sterile techniques prevent further spread. (See *Preventing* Pseudomonas *infection*.)

VIBRIO PARAHAEMOLYTICUS FOOD POISONING

Vibrio parahaemolyticus is a common cause of gastroenteritis in Japan. Outbreaks also occur on American cruise ships and in the

eastern and southeastern coastal areas of the United States, especially during the summer.

V. parahaemolyticus, which thrives in a salty environment, is transmitted by ingesting uncooked or undercooked contaminated shellfish, particularly crab and shrimp. After an incubation period of 2 to 48 hours, *V. parahaemolyticus* causes watery diarrhea, moderately severe cramps, nausea, vomiting, headache, weakness, chills, and fever. Food poisoning is usually self-limiting and subsides spontaneously within 2 days. Occasionally, however, it's more severe, and may even be fatal in debilitated or elderly persons.

Diagnosis requires bacteriologic examination of vomitus, blood, stool smears, or fecal specimens collected by rectal swab. Diagnosis must rule out not only other causes of food poisoning but also other acute GI disorders.

Treatment is supportive, consisting primarily of bed rest and oral fluid replacement. I.V. replacement therapy is seldom necessary, but oral tetracycline may be prescribed. Thorough cooking of seafood prevents this infection.

Cholera

Cholera (also known as *Asiatic cholera* or *epidemic cholera*) is an acute enterotoxinmediated GI infection caused by the gramnegative bacillus *Vibrio cholerae*. It produces profuse diarrhea, vomiting, massive fluid and electrolyte loss and, possibly, hypovolemic shock, metabolic acidosis, and death. A similar bacterium, *Vibrio parahaemolyticus,* causes food poisoning. (See Vibrio parahaemolyticus *food poisoning*.)

Even with prompt diagnosis and treatment, cholera is fatal in up to 2% of children; in adults, it's fatal in less than 1%. However, untreated cholera may be fatal in as many as 50% of patients. Cholera infection confers only transient immunity.

Causes and incidence

Humans are the only hosts and victims of *V. cholerae*, a motile, aerobic organism. It's transmitted through food and water contaminated with fecal material from carriers or people with active infections. Cholera is most common in Africa, southern and Southeast Asia, and the Middle East, although outbreaks have occurred in Japan, Australia, and Europe. Infection also occurs after eating shellfish from recognized environmental reservoirs of cholera, including one that's along the United States' Gulf of Mexico coast.

Cholera occurs during the warmer months and is most prevalent among lower socioeconomic groups. In India, it's common among children ages 1 to 5, but in other endemic areas, it's equally distributed among all age-groups. Susceptibility to cholera may be increased by a deficiency or an absence of hydrochloric acid.

Complications

- Hypoglycemia
- Severe electrolyte depletion
- Hypovolemic shock
- Metabolic acidosis
- Renal failure
- Liver failure
- Bowel ischemia
- Bowel infarction

Signs and symptoms

After an incubation period ranging from several hours to 5 days, cholera produces acute, painless, profuse, watery diarrhea and effortless vomiting (without preceding nausea). As diarrhea worsens, the stools contain white flecks of mucus (rice-water stools). Because of massive fluid and electrolyte losses from diarrhea and vomiting (fluid loss in adults may reach 1 L/hour), cholera causes intense thirst, weakness, loss of skin turgor, wrinkled skin, sunken eyes, pinched facial expression, muscle cramps (especially in the extremities), cyanosis, oliguria, tachycardia, tachypnea, thready or absent peripheral pulses, falling blood pressure, fever, and inaudible, hypoactive bowel sounds.

Patients usually remain oriented but apathetic, although small children may become stuporous or develop seizures. If complications don't occur, the symptoms subside and the patient recovers within a week. However, if treatment is delayed or inadequate, cholera may lead to metabolic acidosis, uremia and, possibly, coma and death. About 3% of patients who recover continue to carry *V. cholerae* in the gallbladder; however, most patients are free from the infection after about 2 weeks.

Diagnosis

In endemic areas or during epidemics, characteristic clinical features strongly suggest cholera.

CONFIRMING DIAGNOSIS

A culture of V. cholerae *from feces or vomitus indicates cholera; however, definitive diagnosis requires agglutination and other clear reactions to group- and typespecific antisera.*

A dark-field microscopic examination of fresh feces showing rapidly moving bacilli (like shooting stars) allows for a quick, tentative diagnosis. Immunofluorescence also allows rapid diagnosis. Diagnosis must rule out *Escherichia coli* infection, salmonellosis, and shigellosis.

Treatment

Improved sanitation and the administration of cholera vaccine to travelers in endemic areas can control this disease. Unfortunately, the vaccine now available confers only 60% to 80% immunity and is effective for only 3 to 6 months. Consequently, vaccination is impractical for residents of endemic areas.

Treatment requires rapid I.V. infusion of large amounts (50 to 100 ml/minute) of isotonic saline solution, alternating with isotonic sodium bicarbonate or sodium lactate. Potassium replacement may be added to the I.V. solution. Antibiotic therapy using such drugs as tetracycline, bactrim, erythromycin, and ciprofloxacin can shorten the course of infection and reduce the rehydration requirement.

When I.V. infusions have corrected hypovolemia, fluid infusion decreases to quantities sufficient to maintain normal pulse and skin turgor or to replace fluid loss through diarrhea. An oral glucoseelectrolyte solution can substitute for I.V. infusions. In mild cholera, oral fluid replacement is adequate. If symptoms persist despite fluid and electrolyte replacement, treatment includes tetracycline.

Special considerations

A cholera patient requires contact precautions, supportive care, and close observation during the acute phase.

- Wear a gown and gloves when handling feces-contaminated articles or when a danger of contaminating clothing exists, and wash your hands after leaving the patient's room.
- Monitor output (including stool volume) and I.V. infusion accurately. To detect overhydration, carefully observe neck veins, take serial patient weights, and auscultate the lungs (fluid loss in cholera is massive, and improper replacement may cause potentially fatal renal insufficiency).
- Protect the patient's family by administering oral tetracycline or doxycycline, if ordered.

- Advise anyone traveling to an endemic area to boil all drinking water and avoid uncooked vegetables and unpeeled fruits.

Septic shock

Second only to cardiogenic shock as the leading cause of shock-related death, septic shock causes inadequate tissue perfusion, abnormalities of oxygen supply and demand, metabolic changes, and circulatory collapse. It typically occurs among hospitalized patients, usually as a result of bacterial infection. About 25% of patients who develop gram-negative bacteremia go into shock. Unless vigorous treatment begins promptly, preferably before symptoms fully develop, septic shock rapidly progresses to death (in many cases within a few hours) in up to 80% of these patients. Septic shock is the most common cause of death in acute care units in the United States.

Causes and incidence

In two-thirds of patients, septic shock results from infection with the gram-negative bacteria *Escherichia coli*, *Klebsiella*, *Enterobacter*, *Proteus*, *Pseudomonas*, or *Bacteroides*; in a few, from the gram-positive bacteria *Streptococcus pneumoniae*, *Streptococcus pyogenes*, *Staphylococcus aureus,* or *Actinomyces*. Infections with viruses, rickettsiae, chlamydiae, and protozoa may be complicated by shock.

These organisms produce septicemia in people whose resistance is already compromised by an existing condition; infection also results from translocation of bacteria from other areas of the body through surgery, I.V. therapy, and catheters. Septic shock commonly occurs in patients hospitalized for primary infection of the genitourinary, biliary, GI, or gynecologic tract. Other predisposing factors include immunodeficiency, advanced age, trauma, burns, diabetes mellitus, cirrhosis, and disseminated cancer.

Complications

- Disseminated intravascular coagulation (DIC)
- Renal failure
- Heart failure
- GI ulcers
- Hepatic dysfunction

Signs and symptoms

Signs and symptoms of septic shock vary according to the stage of shock, the organism causing it, and the patient's immune response and age:

- *early stage* — oliguria, sudden fever (over 101° F [38.3° C]), and chills; tachypnea, tachycardia, full bounding pulse, hyperglycemia, nausea, vomiting, diarrhea, and prostration
- *late stage* — restlessness, apprehension, irritability, thirst from decreased cerebral tissue perfusion, hypoglycemia, hypothermia, and anuria. Hypotension, altered level of consciousness, and hyperventilation may be the *only* signs among infants and the elderly.

Diagnosis

One or more typical symptoms (fever, confusion, nausea, vomiting, and hyperventilation) in a patient suspected of having an infection suggests septic shock and necessitates immediate treatment.

In early stages, arterial blood gas (ABG) levels indicate respiratory alkalosis (low partial pressure of carbon dioxide [$PaCO_2$], low or normal bicarbonate [HCO_3-], and high pH). As shock progresses, metabolic acidosis develops with hypoxemia, indicated by decreasing $PaCO_2$ (may increase as respiratory failure ensues); partial pressure of oxygen; HCO_3-, and pH.

The following tests support the diagnosis and determine the treatment:

- blood cultures to isolate the organism
- decreased platelet count and leukocytosis (15,000 to 30,000/µl)
- increased blood urea nitrogen and creatinine levels, decreased creatinine clearance
- abnormal prothrombin consumption and partial thromboplastin time
- simultaneous measurement of urine and plasma osmolalities for renal failure (urine osmolality below 400 mOsm, with a ratio of urine to plasma below 1.5)
- decreased central venous pressure (CVP), pulmonary artery pressure, and pulmonary artery wedge pressure (PAWP); decreased cardiac output (in early septic shock, cardiac output increases); low systemic vascular resistance
- on electrocardiogram, ST-segment depression, inverted T waves, and arrhythmias resembling myocardial infarction.

Treatment

The first goal of treatment is to monitor for and then reverse shock through volume expansion with I.V. fluids and insertion of a pulmonary artery catheter to check PAWP. Administration of whole blood or plasma can then raise the PAWP to a

high normal to slightly elevated level of 14 to 18 mm Hg. A ventilator may be necessary to overcome hypoxia. Urinary catheterization allows accurate measurement of hourly urine output.

Treatment also requires immediate administration of I.V. antibiotics to control the infection. Depending on the organism, the antibiotic combination usually includes an aminoglycoside (such as amikacin, gentamicin, or tobramycin) for gram-negative bacteria combined with a penicillin (such as ticarcillin or piperacillin). Sometimes treatment includes a cephalosporin or vancomycin for suspected staphylococcal infection. Therapy may include chloramphenicol for nonsporulating anaerobes (*Bacteroides*), which may cause bone marrow depression, and clindamycin, which may produce pseudomembranous enterocolitis. Metronidazole can also be used for anaerobic infection. Appropriate antibiotics for other causes of septic shock depend on the suspected organism. Other measures to combat infection include surgery to drain and excise abscesses and debridement.

If shock persists after fluid infusion, treatment with vasopressors, such as dopamine, maintains adequate blood perfusion in the brain, liver, GI tract, kidneys, and skin. In adults with severe sepsis who have a high risk of death, drotrecogin alfa may be used to interrupt the sepsis cascade. Other treatment includes I.V. bicarbonate to correct acidosis and corticosteroids (especially in patients with gram-negative septic shock). Other experimental treatments include opioid antagonists, prostaglandin inhibitors, and calcium channel blockers, which are used to block the rapid inflammatory process.

Special considerations

Determine which of your patients are at high risk for developing septic shock. Know the signs of impending septic shock, but don't rely solely on technical aids to judge the patient's status. Consider any change in mental status and urine output as significant as a change in CVP. Report such changes promptly.

- Carefully maintain the pulmonary artery catheter. Check ABG levels for adequate oxygenation or gas exchange, and report any changes immediately.
- Record intake and output and daily weight. Maintain adequate urine output (0.5 to 1 ml/kg/hour) and systolic pressure. Avoid fluid overload.
- Monitor serum antibiotic levels, and administer drugs as ordered.

ALERT

Watch closely for signs of DIC (abnormal bleeding), renal failure (oliguria, increased specific gravity), heart failure (dyspnea, edema, tachycardia, distended neck veins), GI ulcers

(hematemesis and melena), and hepatic abnormality (jaundice, hypoprothrombinemia, and hypoalbuminemia).

Haemophilus influenzae infection

Haemophilus influenzae causes diseases in many organ systems but usually attacks the respiratory system. It's a common cause of epiglottitis, laryngotracheobronchitis, pneumonia, bronchiolitis, otitis media, and meningitis. Less commonly, it causes bacterial endocarditis, conjunctivitis, facial cellulitis, septic arthritis, and osteomyelitis.

Causes and incidence

H. influenzae, the cause of this infection, is a small, gram-negative, pleomorphic aerobic bacillus. Transmission occurs by direct contact with secretions or by respiratory droplets. It infects about half of all children before age 1 and virtually all children by age 3, although a haemophilus influenza b vaccine given at ages 2, 4, and 6 months has reduced this number.

Complications

- Subdural effusions
- Permanent neurologic sequelae
- Upper airway obstruction
- Cellulitis
- Pericarditis
- Pleural effusion
- Respiratory failure

Signs and symptoms

H. influenzae provokes a characteristic tissue response — acute suppurative inflammation. When *H. influenzae* infects the larynx, trachea, or bronchial tree, it leads to irritable cough, dyspnea, mucosal edema, and thick, purulent exudate. When it invades the lungs, it leads to bronchopneumonia.

In the pharynx, *H. influenzae* usually produces no remarkable changes, except when it causes epiglottitis, which generally affects both the laryngeal and pharyngeal surfaces. The pharyngeal mucosa may be reddened, rarely with soft yellow exudate. Usually, though, it appears normal or shows only slight diffuse redness, even while severe pain makes swallowing difficult or impossible. *H. influenzae* infections typically cause high fever and generalized malaise.

Meningitis, the most serious infection caused by *H. influenzae,* is indicated by fever and altered mental status. In young children, nuchal rigidity may be absent.

Diagnosis

CONFIRMING DIAGNOSIS

Isolation of the organism, usually with a blood culture, confirms the diagnosis of H. influenzae *infection.*

Other laboratory findings include:

- polymorphonuclear leukocytosis (15,000 to 30,000/µl)
- leukopenia (2,000 to 3,000/µl) in young children with severe infection
- *H. influenzae* bacteremia, found in many patients with meningitis.

Treatment

H. influenzae infections usually respond to a course of ampicillin, cefotaxime, gatifloxacin, moxifloxacin, or ceftriaxone as an initial treatment, although resistant strains are becoming more common. As an alternative, a combination of chloramphenicol and ampicillin is prescribed. If the strain proves susceptible to ampicillin, chloramphenicol is discontinued.

Special considerations

- Maintain adequate respiratory function through proper positioning, humidification (croup tent) in children, and suctioning, as needed. Monitor rate and type of respirations. Watch for signs of cyanosis and dyspnea, which require intubation or a tracheotomy. Monitor the patient's level of consciousness (LOC); decreased LOC may indicate hypoxemia. For home treatment, suggest using a room humidifier or breathing moist air from a shower or bath, as necessary.
- Check the patient's history for drug allergies before administering antibiotics. Monitor his complete blood count for signs of bone marrow depression when therapy includes ampicillin or chloramphenicol.
- Monitor the patient's intake (including I.V. infusions) and output. Watch for signs of dehydration, such as decreased skin turgor, parched lips, concentrated urine, decreased urine output, and increased pulse rate.
- Organize your physical care measures beforehand, and do them quickly so as not to disrupt the patient's rest.

PREVENTION

Advise your patient on preventive measures, such as vaccinating infants, maintaining droplet precautions, using proper hand-hygiene technique, properly disposing of respiratory secretions, placing soiled tissues in a plastic bag, and decontaminating all equipment.

Whooping cough

Whooping cough, also known as *pertussis*, is a highly contagious respiratory infection usually caused by the nonmotile, gram-negative coccobacillus *Bordetella pertussis* and, occasionally, by the related similar bacteria *B. parapertussis* and *B. bronchiseptica*. Characteristically, whooping cough produces an irritating cough that becomes paroxysmal and commonly ends in a highpitched inspiratory whoop.

Since the 1940s, immunization and aggressive diagnosis and treatment have significantly reduced mortality from whooping cough in the United States. Mortality in children younger than age 1 is usually a result of pneumonia and other complications. The disease is also dangerous in the elderly but tends to be less severe in older children and adults.

Causes and incidence

Whooping cough is usually transmitted by the direct inhalation of contaminated droplets from a patient in the acute stage; it

may also be spread indirectly through soiled linen and other articles contaminated by respiratory secretions.

Whooping cough is endemic throughout the world, usually occurring in late winter and early spring. In about 50% of cases, it strikes unimmunized children younger than age 1, because the immunization series hasn't been completed and the child has had contact with an adult harboring the organisms.

Complications

- Epistasis
- Periorbital edema
- Conjunctival hemorrhage
- Detached retina
- Rectal prolapse
- Inguinal or umbilical hernia
- Atelectasis

- Seizures

Signs and symptoms

After an incubation period of about 7 to 10 days, *B. pertussis* enters the tracheobronchial mucosa, where it produces progressively tenacious mucus. Whooping cough follows a classic 6-week course that includes three stages, each of which lasts about 2 weeks.

First, the *catarrhal stage* characteristically produces an irritating hacking, nocturnal cough, anorexia, sneezing, listlessness, infected conjunctiva and, occasionally, a low-grade fever. This stage is highly communicable.

After a period of 7 to 14 days, the *paroxysmal stage* produces spasmodic and recurrent coughing that may expel tenacious mucus. Each cough characteristically ends in a loud, crowing inspiratory whoop; excessive coughing; and choking on mucus, causing vomiting. (Patients with persistent cough should be evaluated for whooping cough, because not every patient will develop paroxysms or the distinctive whooping sound.) Paroxysmal coughing may induce such complications as nosebleed, increased venous pressure, periorbital edema, conjunctival hemorrhage, hemorrhage of the anterior chamber of the eye, detached retina (and blindness), rectal prolapse, inguinal or umbilical hernia, seizures, atelectasis, and pneumonitis. In infants, choking spells may cause apnea, anoxia, and disturbed acid-base balance. During this stage, patients are highly vulnerable to fatal secondary bacterial or viral infections. Suspect such secondary infection (usually otitis media or pneumonia) in any whooping cough patient with a fever during this stage, because whooping cough itself seldom causes fever.

During the *convalescent stage,* paroxysmal coughing and vomiting gradually subside. However, for months afterward, even a mild upper respiratory tract infection may trigger paroxysmal coughing. (Paroxysmal coughing may not be present in partially immunized individuals.)

Diagnosis

Classic clinical findings, especially during the paroxysmal stage, suggest this diagnosis; laboratory studies will confirm it. Nasopharyngeal swabs and sputum cultures show *B. pertussis* only in the early stages of this disease; fluorescent antibody screening of nasopharyngeal smears provides quicker results than cultures but is less reliable. In addition, the white blood cell (WBC) count is usually increased, especially in children older than age 6 months and early in the paroxysmal stage. Sometimes, the WBC count may reach 175,000 to 200,000/µl, with 60% to 90% lymphocytes.

Treatment

Vigorous supportive therapy requires hospitalization of infants (commonly in the intensive care unit) and fluid and electrolyte replacement. Other measures include adequate nutrition; codeine and mild sedation to decrease coughing; oxygen therapy in apnea; and antibiotics, such as erythromycin and, possibly, ampicillin, to shorten the period of communicability and prevent secondary infections.

Because very young infants (younger than age 1) are particularly susceptible to whooping cough, immunization — most commonly with the diphtheria-tetanus acellular-pertussis vaccine — begins at ages 2, 4, and 6 months. Boosters follow at age 18 months and at ages 4 to 6. The risk of pertussis is greater than the risk of vaccine complications such as neurologic damage. However, seizures or unusual and persistent

crying may be a sign of a severe neurologic reaction, and the physician may not order the other doses. The vaccine is contraindicated in children older than age 6 because it can cause a severe fever.

Special considerations

Whooping cough calls for aggressive, supportive care and droplet precautions (surgical masks only) for 5 to 7 days after initiation of antibiotic therapy.

- Monitor acid-base, fluid, and electrolyte balances.
- Carefully suction secretions, and monitor oxygen therapy. *Remember:* Suctioning removes oxygen as well as secretions.
- Create a quiet environment to decrease coughing stimulation. Provide small, frequent meals, and treat constipation or nausea caused by codeine.
- Offer emotional support to the parents of children with whooping cough.
- To decrease exposure to organisms, empty the suction bottle and change the trash bag at least once each shift. Change soiled linens as often as needed.

Plague

Plague, also known as the *black death,* is an acute infection caused by the gram-negative, nonmotile, nonsporulating bacillus *Yersinia pestis* (formerly called *Pasteurella pestis*).

Plague occurs in several forms. *Bubonic plague*, the most common, causes the characteristic swollen, and sometimes suppurating, lymph glands (buboes) that give this infection its name. Other forms include *septicemic plague*, a severe, rapid systemic form, and *pneumonic plague*, which can be primary or secondary to the other two forms. *Primary pneumonic plague* is an acutely fulminant, highly contagious form that causes acute prostration, respiratory distress, and death — in many cases within 2 to 3 days after onset.

Without treatment, mortality is about 60% in bubonic plague and approaches 100% in both septicemic and pneumonic plagues. With treatment, mortality is approximately 18% for all forms of plague, largely due to the delay between onset and treatment and the patient's age and physical condition.

Causes and incidence

Plague is usually transmitted to humans through the bite of a flea from an infected rodent host, such as a rat, squirrel, prairie dog, or hare. (See *Carrier of bubonic plague*.) Occasionally, transmission occurs from handling infected animals or their tissues. Bubonic plague is notorious for the historic pandemics in Europe and Asia during the Middle Ages, which in some areas killed up to two-thirds of the population. This form is rarely transmitted from person to person. However, the untreated bubonic form may progress to a secondary pneumonic form, which is transmitted by contaminated respiratory droplets (coughing) and is highly contagious. In the United States, the primary pneumonic form usually occurs after inhalation of *Y. pestis* in a laboratory.

Sylvatic (wild rodent) plague remains endemic in South America, the Near East, central and Southeast Asia, north central and southern Africa, Mexico, and western United States and Canada. In the United States, its incidence has been rising, a possible reflection of different bacterial strains or environmental changes that favor rodent growth in certain areas. Plague tends to occur between May and September; between October and February it usually occurs in hunters who skin wild animals. One attack confers permanent immunity.

Signs and symptoms

The incubation period, early symptoms, severity at onset, and clinical course vary in the three forms of plague. In *bubonic plague*, the incubation period is 2 to 8 days. The milder form begins with malaise, fever, and pain or tenderness in regional lymph nodes, possibly associated with swelling. Lymph node damage (usually axillary or inguinal) eventually produces painful, inflamed, and possibly suppurative buboes. The classic sign of plague is an excruciatingly painful bubo. Hemorrhagic areas may become necrotic; in the skin, such areas appear dark — hence the name “black death.”

This infection can progress extremely rapidly. A seemingly mildly ill person with only fever and adenitis may become moribund within hours. Plague may also begin dramatically, with a sudden high fever of 103° to 106° F (39.4° to 41.1° C), chills, myalgia, headache, prostration, restlessness, disorientation, delirium, toxemia, and staggering gait. Occasionally, it causes abdominal pain, nausea, vomiting, and constipation followed by diarrhea (frequently bloody), skin mottling, petechiae, and circulatory collapse.

In *primary pneumonic plague*, the incubation period is 2 to 3 days followed by a typically acute onset, with high fever, chills, severe headache, tachycardia, tachypnea, dyspnea, and a productive cough (first mucoid sputum, later frothy pink or red).

Secondary pneumonic plague, the pulmonary extension of the bubonic form, complicates about 5% of cases of untreated plague. A cough producing bloody sputum signals this complication. Both the primary and secondary forms of pneumonic plague rapidly cause severe prostration, respiratory distress and, usually, death.

Septicemic plague usually develops without overt lymph node enlargement. In this form, the patient shows toxicity, hyperpyrexia, seizures, prostration, shock, and disseminated intravascular coagulation (DIC). Septicemic plague causes widespread nonspecific tissue damage — such as peritoneal or pleural effusions, pericarditis, and meningitis. It's rapidly fatal unless promptly and correctly treated.

Diagnosis

Because plague is rare in the United States, it's commonly overlooked until after the patient dies or multiple cases develop. Characteristic buboes and a history of exposure to rodents in known endemic areas strongly suggest bubonic plague.

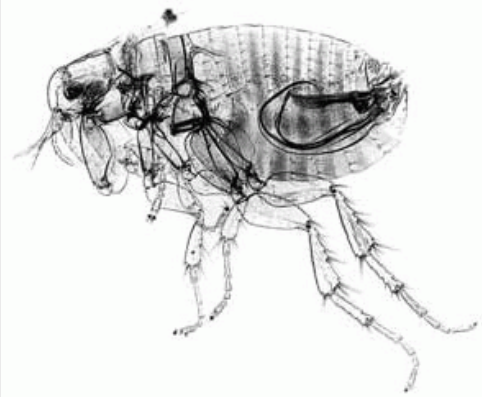
CONFIRMING DIAGNOSIS

Stained smears and cultures of Y. pestis *obtained from a needle aspirate of a small amount of fluid from skin lesions confirm this diagnosis.*

Postmortem examination of a guinea pig inoculated with a sample of blood or purulent drainage allows isolation of the organism. Other laboratory findings include a white blood cell count of over 20,000/µl with increased polymorphonuclear leukocytes and hemoagglutination reaction (antibody titer) studies. Diagnosis should rule out tularemia, typhus, and typhoid.

CARRIER OF BUBONIC PLAGUE

Bubonic plague is usually transmitted to humans through the bite of a flea (*Xenopsylla cheopis*), shown here.



In pneumonic plague, diagnosis requires a chest X-ray to show fulminating pneumonia and stained smear and culture of sputum to identify *Y. pestis*. Other bacterial pneumonias and psittacosis must be ruled out. Stained smear and blood culture containing *Y. pestis* are diagnostic in septicemic plague. However, cultures of *Y. pestis* grow slowly; so, in suspected plague (especially pneumonic and septicemic plagues), treatment should begin without waiting for laboratory confirmation. For a presumptive diagnosis of plague, a fluorescent antibody test may be ordered.

Treatment

Antimicrobial treatment of suspected plague must begin immediately after blood specimens have been taken for culture and shouldn't be delayed for laboratory confirmation. Generally, treatment consists of large doses of streptomycin, the drug proven most effective against *Y. pestis*. Other effective drugs include gentamicin, doxycycline,

tetracycline, and chloramphenicol. Penicillins are ineffective against plague.

In both septicemic and pneumonic plagues, life-saving antimicrobial treatment must begin within 18 hours of onset. Supportive management aims to control fever, shock, and seizures and to maintain fluid balance.

After the start of antimicrobial therapy, glucocorticoids can be used to combat life-threatening toxemia and shock; diazepam relieves restlessness; and, if the patient develops DIC, treatment may include heparin.

Special considerations

- Patients with bubonic plague require standard precautions.
- Use an approved insecticide to rid the patient and his clothing of fleas. Carefully dispose of soiled dressings and linens, feces, and sputum. If the patient has pneumonic plague, wear a mask and follow droplet precautions. Handle all

exudates, purulent discharge, and laboratory specimens with gloves. For more information, consult your infection control officer.

- Give drugs and treat complications as ordered.
- Treat buboes with hot, moist compresses. Never excise or drain them because this could spread the infection.
- When septicemic plague causes peripheral tissue necrosis, prevent further injury to necrotic tissue. Avoid using restraints or armboards, and pad the bed's side rails.
- For patients with pneumonic plague, obtain a history of patient contacts so that they can be quarantined for 7 days of observation. Administer prophylactic tetracycline or chloramphenicol, as ordered.
- Report suspected cases of plague to local public health department officials so they can identify the source of infection.

PREVENTION

To help prevent plague, discourage contact with wild animals (especially those that are sick or dead), and support programs aimed at reducing insect and rodent populations. Recommend immunization with plague vaccine to travelers to, or residents of, endemic areas, even though the effect of immunization is transient.

Brucellosis

Brucellosis (also known as *undulant fever, Malta fever,* or *Bang's disease*) is an acute febrile illness transmitted to humans from animals. It's caused by the nonmotile, nonspore-forming, gram-negative coccobacilli of the genus *Brucella*, notably *B. suis* (found in swine), *B. melitensis* (in goats and sheep), *B. abortus* (in cattle), and *B. canis* (in dogs). Brucellosis causes fever; profuse sweating; anxiety; general aching; and bone, spleen, liver, kidney, or brain abscesses.

The prognosis is good. With treatment, brucellosis is seldom fatal, although complications can cause permanent disability.

Causes and incidence

Brucellosis is transmitted through the consumption of unpasteurized dairy products and through contact with infected animals or their secretions or excretions. It's most common among farmers, stock handlers, butchers, and veterinarians. Because of such occupational risks, brucellosis infects six times more men than women, especially those between ages 20 and 50; it's less common in children.

Because hydrochloric acid in gastric juices kills *Brucella* bacteria, people with achlorhydria are particularly susceptible to this disease.

Although brucellosis occurs throughout the world, it's most prevalent in the Middle East, Africa, the former Soviet Union, India, South America, and Europe; it's seldom found in the United States. The incubation period usually lasts from 5 to 60 days, but in some cases it can last for months.

Complications

- Endocarditis
- Orchitis
- Hepatosplenomegaly
- Arthritis
- Osteomyelitis
- Eczematous rashes
- Petechiae
- Purpura
- Pleural effusions
- Pneumothorax
- Abscesses in testes, ovaries, kidneys, spleen, liver, bone, and brain

Signs and symptoms

Onset of brucellosis is usually insidious, but the disease course falls into two distinct phases. Characteristically, the acute phase causes fever, chills, profuse sweating, fatigue, headache, backache, enlarged lymph nodes, hepatosplenomegaly, weight loss, and abscess and granuloma formulation in subcutaneous tissues, lymph nodes, liver, and spleen. Despite this disease's common name — undulant fever — few patients have a truly intermittent (undulant) fever; in fact, fever is commonly insignificant. It may be observed if the patient goes without treatment for a long time.

The chronic phase produces recurrent depression, sleep disturbances, fatigue, headache, sweating, and sexual impotence; hepatosplenomegaly and enlarged lymph nodes persist. In addition, abscesses may form in the testes, ovaries, kidneys, and brain (meningitis and encephalitis). About 10% to 15% of patients with such brain abscesses develop hearing and visual disorders, hemiplegia, and ataxia. Other complications include osteomyelitis, orchitis and, rarely, subacute bacterial endocarditis, which is difficult to treat.

Diagnosis

In patients with characteristic clinical features, a history of exposure to animals, occupational exposure, or ingestion of high-risk foods suggests brucellosis. Multiple agglutination tests help to confirm the diagnosis. Approximately 90% of patients with brucellosis have agglutinin titers of 1:160 or more within 3 weeks of developing this disease. However, elevated agglutinin titers also follow vaccination against tularemia, *Yersinia* infection, or cholera; skin tests; or relapse. Agglutinin titers testing can also monitor effectiveness of treatment.

CONFIRMING DIAGNOSIS

Three to six cultures of blood and bone marrow and biopsies of infected tissue (for example, the spleen) may provide a definite diagnosis. Culturing is best done during the acute phase.

Hematologic studies indicate an increased erythrocyte sedimentation rate and normal or reduced white blood cell count. Diagnosis must rule out infectious diseases that produce similar symptoms, such as typhoid and malaria.

Treatment

Treatment consists of bed rest during the febrile phase. Antibiotic therapy includes a combination of doxycycline and gentamicin or doxycycline and rifampin. In severe cases, I.V. corticosteroids are given for 3 days, followed by oral corticosteroids. Standard precautions are required until lesions stop draining.

Special considerations

In suspected cases of brucellosis, take a full history. Ask the patient about his occupation and if he has recently traveled or eaten unprocessed food such as dairy products (especially unpasteurized dairy products).

- During the acute phase, monitor and record the patient's temperature every 4 hours. Be sure to use the same route (oral or rectal) every time. Ask the dietary department to provide between-meal milk shakes and other supplemental foods to counter weight loss. Watch for heart murmurs, muscle weakness, vision loss, and joint inflammation — which may signal complications.
- During the chronic phase, watch for depression and disturbed sleep patterns. Administer sedatives as ordered, and plan your care to allow adequate rest.
- Keep suppurative granulomas and abscesses dry. Properly dispose of all secretions and soiled dressings. Reassure the patient that this infection *is* curable.

- Before discharge, stress the importance of continuing medication for the prescribed duration. To prevent recurrence, advise the patient to avoid using unpasteurized milk or other dairy products. Warn meat packers and other people at risk of occupational exposure to wear gloves and goggles.

Anthrax

Anthrax is an acute bacterial infection that most commonly occurs in grazing animals, such as cattle, sheep, goats, and horses. It can also affect people who come in contact with contaminated animals or their hides,

bones, fur, hair, or wool. It's also used as an agent for bioterrorism and biological warfare.

Anthrax occurs worldwide but is most common in developing countries. In humans, anthrax occurs in three forms, depending on the mode of transmission: cutaneous, inhalational, and GI.

Causes and incidence

Anthrax is caused by the bacteria *Bacillus anthracis,* which exists in the soil as spores that can live for years. Transmission to humans usually occurs through exposure to, or handling of, infected animals or animal products. Anthrax spores can enter the body through abraded or broken skin (*cutaneous anthrax*), by inhalation (*inhalational anthrax*), or through ingestion of undercooked meat from an infected animal (*GI anthrax*). Anthrax isn't known to spread from person to person.

Complications

- Septicemia
- Death

Signs and symptoms

From the time of exposure, signs and symptoms of infection usually occur within 1 to 7 days but may take as long as 60 days to appear. The signs and symptoms of anthrax depend on the form acquired:

- *Cutaneous anthrax:* This is the most common form of anthrax. Skin infection may begin as a small, elevated, itchy lesion that resembles an insect bite, develops into a vesicle in 1 to 2 days, and finally becomes a small, painless ulcer with a necrotic center. Enlarged lymph glands in the surrounding area are common. Without treatment, mortality from cutaneous anthrax is 20%; it's less than 1% with treatment.

- *Inhalational anthrax:* The patient may initially report flulike signs and symptoms, such as malaise, fever, headache, myalgia, and chills. These mild signs and symptoms may progress to severe respiratory difficulties, such as dyspnea, stridor, chest pain, and cyanosis, followed by the onset of shock. Even with treatment, inhalational anthrax is usually fatal.
- *GI anthrax:* Ingestion of anthrax spores can cause acute inflammation of the intestinal tract. The patient may present with nausea, vomiting, decreased appetite, and fever, which then progress to abdominal pain, vomiting blood, and severe diarrhea. With treatment, death occurs in 25% to 60% of cases.

Diagnosis

Anthrax can be diagnosed through cultures of the blood, skin lesions, or sputum of an exposed patient. If *B. anthracis* is isolated, the diagnosis is confirmed. Additionally, specific antibodies may be detected in the blood.

Treatment

Treatment that's initiated as soon as exposure to anthrax is suspected is essential to preventing anthrax infection; early treatment may also help prevent fatality. Many antibiotics are effective against anthrax. The most widely used are penicillin, ciprofloxacin, and doxycycline.

Special considerations

- Any case of anthrax in either livestock or a person must be reported to the appropriate public health department.
- Supportive measures are geared toward the type of anthrax exposure.
- An anthrax vaccine is available but, due to limited supplies, it's now administered only to United States military personnel and isn't for routine civilian use.
- Anthrax isn't transmitted from person to person.

Campylobacteriosis

Campylobacteriosis is an intestinal infection caused by the *Campylobacter* organism, a spiral-shaped bacteria that invades and destroys the epithelial cells of the jejunum, ileum, and colon. It may spread to the bloodstream in persons with compromised immune systems, causing a life-threatening infection.

Causes and incidence

Campylobacteriosis is transmitted by the consumption of contaminated food, such as raw poultry, fresh produce, water, or unpasteurized milk; and through contact with an infected person's stool. Transmission is also possible through contact with infected pets and wild animals. Risk factors include recent family infection with *C. jejuni* and travel to an area with poor hygiene or sanitation practices.

Campylobacteriosis, which is more common in the summer months, is the most common bacterial cause of diarrheal illness in the United States.

Complications

- Bacteremia
- Severe dehydration
- Electrolyte disturbances
- Sepsis, endocarditis, meningitis and thrombophlebitis in immunocompromised patients

Signs and symptoms

Signs and symptoms usually develop 2 to 4 days after exposure to *Campylobacter*. The patient's history typically reveals consumption of contaminated food or water, followed by an acute onset of mild or severe diarrhea. There may also be a history of recent close contact with a person experiencing diarrhea.

On examination, the patient may complain of cramping, abdominal pain, nausea, and vomiting. Fever may be present, and there may be traces of blood in the stool. Complications associated with campylobacteriosis include bacteremia; severe dehydration and electrolyte disturbances; Guillain-Barré syndrome; and Reiter's syndrome. Patients with campylobacteriosis who are immunocompromised are more susceptible to sepsis, endocarditis, meningitis, and thrombophlebitis because of the spread of the bacteria into the bloodstream.

Diagnosis

CONFIRMING DIAGNOSIS

Stool culture identifying Campylobacter *confirms the diagnosis of campylobacteriosis.*

History and physical examination help in diagnosing campylobacteriosis.

Treatment

Campylobacteriosis typically resolves on its own and isn't usually treated with antibiotics unless severe signs and symptoms are present. If severe symptoms are present, antibiotics such as ciprofloxacin and azithromycin may be ordered. Fluid and electrolyte imbalances are corrected with increased fluid intake or I.V. fluid replacement, as indicated.

Special considerations

- Monitor the patient's intake and output and vital signs. Assess for signs of dehydration, such as tachycardia, tachypnea, and decreased urine output.
- Monitor the patient's electrolyte levels and assess the effects of replacement electrolyte therapy and I.V. fluids.
- Observe standard precautions.
- Instruct the patient and his family on hand-washing techniques and preventive measures, including the proper handling and preparing of foods.

Tularemia

Tularemia is a highly infectious disease that can be caused by as little as 10 *Francisella tularensis* organisms, a gram-negative pleomorphic bacterium. There are six forms: ulceroglandular, glandular, oculoglandular, oropharyngeal, pneumonic, and septicemic. The disease is fatal in about 5% of patients who don't receive treatment and in less than 1% of patients who do receive treatment.

F. tularensis is considered a potential bioterrorism agent. If dispersed in aerosol form (the most likely method of dispersion), infected persons would generally develop signs and symptoms of severe respiratory illness, including pneumonia and systemic infection.

Causes and incidence

Tularemia is transmitted by the bites of infected ticks and deerflies, consuming contaminated food or water, and through contact with the blood of an infected animal (such as by skinning or handling infected carcasses), especially rabbits. The organism gains access to the host by skin or mucous membrane inoculation, inhalation, or ingestion. After inoculation, a papule and high fever develop. (The papule eventually

develops into an ulcer.) The incubation period is 3 to 4 days.

In the United States, there are about 200 human cases of tularemia reported annually, with most occurring in the south-central and western areas of the country.

Complications

- Pneumonia
- Lung abscess
- Respiratory failure
- Rhabdomyolysis
- Meningitis
- Pericarditis
- Osteomyelitis

Signs and symptoms

Common signs and symptoms are ulcer and fever, but signs and symptoms also vary according to the form of tularemia the patient is infected with. In the ulceroglandular form, ulcers occur at the site of inoculation and are accompanied by swollen regional lymph nodes. In the glandular form, swollen regional lymph nodes are present. In the oculoglandular form, the patient exhibits a painful, red eye with purulent exudates and swollen submandibular, preauricular, or cervical lymph nodes. In the oropharyngeal form, the patient has a sore throat, abdominal pain, nausea, vomiting, diarrhea and, occasionally, GI bleeding. In the pneumonic form, the patient presents with a dry cough, dyspnea, and pleuritic chest pain. In the septicemic form, the patient has fever, chills, myalgia, malaise, and weight loss.

Complications of tularemia include pneumonia, lung abscess, respiratory failure, rhabdomyolysis, meningitis, pericarditis, and osteomyelitis.

Diagnosis

Diagnosis is based on presenting signs and symptoms, as well as the patient's history, which may include a tick bite and exposure to contaminated food or water or to contaminated blood. Other results may include a white blood cell count that is normal or elevated and blood or sputum cultures that are positive for *F. tularensis*. Chest X-ray may show pneumonia.

Treatment

General treatment involves proper skin care and increased fluid intake or supportive therapy with I.V. fluids. Medications include I.V., I.M., or oral antibiotic therapy with streptomycin or tetracycline. Antipyretics may be administered for fever.

Special considerations

- Monitor the patient's intake and output and vital signs.
- Assess the patient for signs of dehydration, such as tachycardia, tachypnea, and decreased urine output.
- Monitor for complications, such as meningitis, pneumonia, pericarditis, and osteomyelitis.

Ehrlichiosis

Human ehrlichiosis, an infectious rickettsial disease that's transmitted by the bite of an infected tick, was first diagnosed in 1986. The genus Ehrlichia contains an emerging number of species that can transmit potentially life-threatening infections.

Causes and incidence

Ehrlichiosis is caused by *Ehrlichia* organisms, specifically *E. chaffeensis* and granulocytic *Ehrlichia*. Known vectors include the lone star tick (*Amblyomma americanum*), the American dog tick (*Dermacentor variabilis*), and deer ticks (*Ixodes dammini* and *Ixodes scapularis*).

In the United States, most cases of ehrlichiosis are reported in the south-central and southern Atlantic areas of the country, but it has also been reported in the upper midwest. Persons at highest risk include those who live in endemic and highly wooded areas, engage in activities in high grassy areas, and own a pet that may introduce a tick into the home.

Complications

- Acute respiratory distress syndrome
- Disseminated intravascular coagulation
- Seizures
- Coma

Signs and symptoms

The incubation period for ehrlichiosis is 9 days from the time of the tick bite. Early symptoms include fever, chills, headache, muscle pain (myalgia), and nausea. A maculopapular or petechial rash appears in about half of the cases.

Most people infected with ehrlichiosis don't seek medical help, but it can be fatal.

Diagnosis

Diagnosis of ehrlichiosis is based on evaluation of signs and symptoms and supporting laboratory data. A fluorescent antibody test may return positive for *E. chaffeensis* or granulocytic *Ehrlichia*. A complete blood cell count shows decreased white blood cells (WBCs), indicative of leukopenia, and a low platelet count, indicative of thrombocytopenia. Granulocyte stain shows clumps of bacteria inside the WBCs. Liver enzymes show elevated levels of transaminase.

Treatment

Ehrlichiosis is treated with tetracycline or doxycycline, producing rapid improvement when used early in the disease's course. Death can occur if treatment is delayed.

PEDIATRIC TIP

Oral tetracycline usually isn't prescribed for children until all permanent teeth have erupted because it can permanently discolor teeth that are still forming.

Supportive therapy is provided to help relieve signs and symptoms.

Special considerations

- Review with the patient measures to prevent tick bites when outdoors, such as wearing long-sleeve shirts and pants, tucking pants inside boots, and using insect repellent.
- Advise the patient to stick to trails and avoid dense brush when hiking. Also tell him to avoid standing under overhanging foliage.
- Tell the patient that he should examine himself for ticks after being outdoors and that he should remove any ticks found on his body; studies suggest that a tick must be attached for at least 24 hours in order to cause ehrlichiosis.

SPIROCHETES AND MYCOBACTERIA

Lyme disease

A multisystemic disorder, Lyme disease is caused by the spirochete *Borrelia burgdorferi*, which is carried by *Ixodes dammini, I. Pacificus,* and other ticks in the Ixodidae family. It commonly begins in the summer with a papule that becomes red and warm but isn't painful. This classic skin lesion is called *erythema chronicum migrans* (ECM), which may be confused with a similar rash caused by Southern tick-associated rash illness. (See *Southern tick-associated rash illness,*

page 958.) Weeks or months later, cardiac or neurologic abnormalities sometimes develop, possibly followed by arthritis of the large joints.

Causes and incidence

Lyme disease occurs when a tick injects spirochete-laden saliva into the bloodstream. After incubating for 3 to 32 days, the spirochetes migrate out to the skin, causing ECM. Then they disseminate to other skin sites or organs via the bloodstream or lymph system. They may survive for years in the joints, or they may trigger an inflammatory response in the host and then die.

Initially, Lyme disease was identified in a group of children in Lyme, Connecticut. Now it's known to occur primarily in three parts of the United States: in the northeast, from Massachusetts to Maryland; in the Midwest, in Wisconsin and Minnesota; and in the west, in California and Oregon. Although it's endemic to these areas, cases have been reported in all 50 states and in 20 other countries, including Germany, Switzerland, France, and Australia.

Complications

- Myocarditis
- Pericarditis
- Arrhythmias
- Heart block
- Meningitis
- Encephalitis
- Cranial or peripheral neuropathies
- Arthritis

SOUTHERN TICK-ASSOCIATED RASH ILLNESS

Southern tick-associated rash illness (STARI) is a newly recognized tick-borne disease that produces a rash similar to the rash caused by Lyme disease. STARI is associated with the bite of the lone star tick, *Ambylomma americanum*, and occurs primarily in the southeastern and south-central states of the United States. Deoxyribonucleic acid analysis of the spirochetes found in the *A. americanum* tick has indicated that they aren't *Borrelia burgdorferi*, the agent of Lyme disease, but are a different, newly recognized species, *Borrelia lonestari*.

Individuals living or traveling to southeastern or south-central states who develop a red, expanding rash with central clearing following a tick bite should see a physician. Mild illness, characterized by such signs and symptoms as fatigue, headache, stiff neck and, occasionally, fever, may accompany the rash.

Currently, there's no specific diagnostic test for STARI. It's suspected if diagnostic tests rule out Lyme disease, the patient's travel history is as indicated above, and a known lone star tick bite has been reported or the patient has participated in activities that may have resulted in exposure to a tick.

There are no current recommendations for treating STARI, but the rash and other accompanying signs and symptoms usually resolve with doxycycline therapy.

Signs and symptoms

Typically, Lyme disease has three stages. ECM heralds stage one with a red macule or papule, commonly at the site of a tick bite. This lesion typically feels hot and itchy and may grow to over 20″ (50.8 cm) in diameter; it resembles a bull's eye or target. Within a few days, more lesions may erupt, and a migratory, ringlike rash, conjunctivitis, or diffuse urticaria occurs. In 3 to 4 weeks, lesions are replaced by small red blotches, which persist for several more weeks. Malaise and fatigue are constant, but other findings are intermittent: headache, neck stiffness, fever, chills, achiness, and regional lymphadenopathy. Less common effects are meningeal irritation, mild encephalopathy, migrating musculoskeletal pain, hepatitis, and splenomegaly. A persistent sore throat and dry cough may appear several days before ECM.

Weeks to months later, the second stage (disseminated infection) begins, and patients may develop additional symptoms depending on the system affected. Neurologic abnormalities — fluctuating meningoencephalitis with peripheral and cranial neuropathy — usually resolve after days or months. Facial palsy is especially noticeable. Cardiac abnormalities, such as a brief, fluctuating atrioventricular heart block, left ventricular dysfunction, or cardiomegaly may also develop. Cardiac involvement lasts only a few weeks but can be fatal.

Stage three (persistent infection) usually begins weeks or years later and is characterized by arthritis in about 80% of patients. Migrating musculoskeletal pain leads to frank arthritis with marked swelling, especially in the large joints. Recurrent attacks may precede chronic arthritis with severe cartilage and bone erosion.

Diagnosis

Because isolation of *B. burgdorferi* is difficult in humans and serologic testing isn't standardized, diagnosis is usually based on the characteristic ECM lesion and related clinical findings, especially in endemic areas. Antibodies to *B. burgdorferi* are indentified by immunofluorescence or enzyme-linked immunosorbent assay (ELISA). ELISAs are confirmed with Western blot tests. Mild anemia and an elevated erythrocyte sedimentation rate, leukocyte count, serum immunoglobulin M, and aspartate aminotransferase levels support the diagnosis.

Clinicians must differentiate between Lyme disease and arthritis, encephalopathy, or polyneuropathy.

PREVENTION

PREVENTING LYME DISEASE

Advise your patient to do the following to help prevent him from contracting Lyme disease.

Protect yourself from ticks

- Wear long pants and sleeves.
- Wear shoes, long pants with cuffs tucked into the socks, a long-sleeved shirt, a hat, and gloves when walking in wooded or grassy areas.
- Stay on trails and avoid walking through low bushes and long grass.
- Keep dogs on a leash.
- Use insect repellents with a 20% to 30% concentration of DEET on the skin and clothing.
- Choose an insect repellent with the concentration based on the hours of protection that's needed, for example, a 20% concentration is effective for about 2 hours, while higher concentrations protect longer.
- Don't use DEET on the hands of young children or on infants younger than age 2 months.
- According to the Centers for Disease Control and Prevention, oil of lemon eucalyptus, a more natural product, offers the same protection as DEET when used in similar concentrations.

Tick-proof your yard

- Clear brush and leaves where ticks live.
- Keep woodpiles in sunny areas.
- Check yourself, your children, and your pets for ticks especially after spending time in wooded or grassy areas.
- Search carefully for deer ticks. Deer ticks are usually no bigger than the head of a pin, so they're difficult to find.
- Shower as soon as you come indoors because ticks commonly remain on the skin for hours before attaching themselves.
- Be aware that even if you've had Lyme disease before, you can get it again.

Carefully remove ticks on yourself or others

- Remove the tick with tweezers. Gently grasp the tick near its head or mouth. Don't squeeze or crush the tick; pull carefully and steadily.
- Once the entire tick has been removed, dispose of it and apply antiseptic to the bite area.

Treatment

A 28-day course of an antibiotic, such as doxycycline, is the treatment of choice for nonpregnant adults. Amoxicillin is usually prescribed for children. Alternatives include cefuroxime and ceftriaxone. When given in the early stages, these drugs can minimize later complications. When given during the late stages, high-dose I.V. ceftriaxone may be successful.

Special considerations

- Take a detailed patient history, asking about travel to endemic areas and exposure to ticks.
- Check for drug allergies, and administer antibiotics carefully.
- For a patient with arthritis, help with range-of-motion and strengthening exercises, but avoid overexertion. Ibuprofen helps relieve joint stiffness.
- Assess the patient's neurologic function and level of consciousness frequently. Watch for signs of increased intracranial pressure and cranial nerve involvement, such as ptosis, strabismus, and diplopia. Also check for cardiac abnormalities, such as arrhythmias and heart block. (See *Preventing Lyme disease*.)

Relapsing fever

An acute infectious disease caused by spirochetes of the genus *Borrelia*, relapsing fever (also called *tick, fowl-nest, cabin, or vagabond fever* or *bilious typhoid*) is transmitted to humans by lice or ticks and is characterized by relapses and remissions. Rodents and other wild animals serve as the primary reservoirs for the *Borrelia* spirochetes. Humans can become secondary reservoirs but cannot transmit this infection by ordinary contagion; however, congenital infection and transmission by contaminated blood are possible.

Untreated louse-borne relapsing fever normally carries a mortality of more than 10%, but during an epidemic, the mortality rate may rise to 50%. With treatment, however, the prognosis for both louse- and tick-borne relapsing fevers is excellent.

Causes and incidence

The body louse (*Pediculus humanus corporis*) carries louse-borne relapsing fever (*B. recurrentis*), which typically occurs in epidemics during wars, famines, and mass migrations. Cold weather and crowded living conditions also favor the spread of body lice.

Inoculation takes place when the victim crushes the louse, causing its infected blood or body fluid to soak into the victim's bitten or abraded skin or mucous membranes.

Louse-borne relapsing fever is most common in North and Central Africa, Europe, Asia, and South America. No cases of louse-borne relapsing fever have been reported in the United States since 1900.

Tick-borne relapsing fever, however, is found in the United States and is caused by at least 15 *Borrelia* species; the three species most commonly identified with tick carriers are *B. hermsii* (associated with *Ornithodoros hermsi*), *B. turicatae* (associated with *O. turicata*), and *B. parkeri* (associated with *O. parkeri*). This form of the disease is most prevalent in Texas and other western states, usually during the summer when ticks and their hosts (chipmunks, goats, squirrels, rabbits, mice, rats, owls, lizards, and prairie dogs) are most active. In the colder weather, outbreaks sometimes afflict people such as campers who sleep in tick-infested cabins.

Because tick bites are virtually painless and most *Ornithodoros* ticks feed at night but don't imbed themselves in the victim's skin, many people are bitten unknowingly.

Complications

- Nephritis

- Bronchitis
- Pneumonia
- Endocarditis
- Seizures
- Cranial nerve lesions
- Paralysis
- Coma
- Death

Signs and symptoms

The incubation period for relapsing fever is 5 to 15 days (the average is 7 days). Clinically, tick- and louse-borne diseases are similar. Both begin suddenly, with a temperature approaching 105° F (40.6° C), prostration, headache, severe myalgia, arthralgia, diarrhea, vomiting, coughing, and eye or chest pains. Splenomegaly is common; hepatomegaly and lymphadenopathy may occur. During febrile periods, the victim's pulse and respiratory rates rise, and a transient macular rash may develop over his torso.

The first attack usually lasts from 3 to 6 days; then the patient's temperature drops quickly and is accompanied by profuse sweating. A skin rash on the trunk lasting 1 to 2 days is common after the primary febrile episode. The rash may be petechiae, macular, or papular. About 5 to 10 days later, a second febrile, symptomatic period begins. In louse-borne infection, additional relapses are unusual; but, in tick-borne cases, a second or third relapse is common. As the afebrile intervals become longer, relapses become shorter and milder because of antibody accumulation. Relapses are possibly due to antigenic changes in the *Borrelia* organism.

Complications from relapsing fever include nephritis, bronchitis, pneumonia, endocarditis, seizures, cranial nerve lesions, paralysis, and coma. Death may occur from hyperpyrexia, massive bleeding, circulatory

failure, splenic rupture, or a secondary infection.

Diagnosis

CONFIRMING DIAGNOSIS

Diagnosis requires demonstration of the spirochetes in peripheral blood smears during febrile periods, using Wright's or Giemsa stain.

Borrelia spirochetes may be more difficult to detect in later relapses because their number declines in the blood. In such cases, injecting the patient's blood or tissue into a young rat and incubating the organism in the rat's blood for 1 to 10 days commonly allows spirochete identification.

In severe infection, spirochetes are found in the urine and cerebrospinal fluid. Other abnormal laboratory results usually include a white blood cell (WBC) count as high as 25,000/µl, with increases in lymphocytes and erythrocyte sedimentation rate; however, the WBC count may be normal. Because the *Borrelia* organism is a spirochete, relapsing fever may cause a false-positive test for syphilis in 5% to 10% of cases.

Treatment

Doxycycline or erythromycin is the treatment of choice and should continue for 4 to 5 days. In cases of drug allergy or resistance, penicillin G may be administered as an alternative. However, neither drug should be given at the height of a severe febrile attack because it may cause Jarisch-Herxheimer reaction, resulting in malaise, rigors, leukopenia, flushing, fever, tachycardia, rising respiration rate, and hypotension. This reaction, which is caused by toxic by-products from massive spirochete destruction, can mimic septic shock and may prove fatal. Antimicrobial therapy should be postponed until the fever subsides. Until then, supportive therapy (consisting of parenteral fluids and electrolytes) should be given.

Special considerations

- During the initial evaluation period, obtain a complete history of the patient's travels and activities.
- Throughout febrile periods, monitor vital signs, level of consciousness (LOC), and temperature every 4 hours. Watch for and immediately report any signs of neurologic complications, such as decreasing LOC or seizures. To reduce fever, give tepid sponge baths and antipyretics, as ordered.
- Maintain adequate fluid intake to prevent dehydration. Provide I.V. fluids as ordered. Measure intake and output accurately, especially if the patient is vomiting or has diarrhea.
- Administer antibiotics carefully. Document and report any hypersensitive reactions (rash, fever, anaphylaxis), especially a Jarisch-Herxheimer reaction.
- Treat flushing, hypotension, or tachycardia with vasopressors or fluids, as ordered.
- Look for symptoms of relapsing fever in family members and in others who may have been exposed to ticks or lice along with the victim.

- Use proper hand-washing technique, and teach it to the patient. Isolation is unnecessary because the disease isn't transmitted from person to person.
- Report all cases of louse- or tick-borne relapsing fever to the local public health department, as required by law.

PREVENTION

To prevent relapsing fever, advise anyone traveling to tick-infested areas (Asia, North and Central Africa, and South America) to wear clothing that covers as much skin as possible and to tuck pant legs into boots or socks. Advise the use of insect repellant to reduce risk.

Leprosy

Leprosy, also known as *Hansen's disease,* is a chronic, systemic infection characterized by progressive cutaneous lesions. It's caused by *Mycobacterium leprae*, an acidfast bacillus that attacks cutaneous tissue and peripheral nerves, producing skin lesions, anesthesia, infection, and deformities.

With timely and correct treatment, leprosy has a good prognosis and is seldom fatal. Untreated, however, it can cause severe disability. The lepromatous type may lead to blindness and deformities.

Leprosy occurs in three distinct forms:

- *Lepromatous leprosy,* the most serious type, causes damage to the upper respiratory tract, eyes, and testes as well as to the nerves and skin.
- *Tuberculoid leprosy* affects peripheral nerves and sometimes the surrounding skin, especially the face, arms, legs, and buttocks.
- *Borderline (dimorphous) leprosy* has characteristics of both lepromatous and tuberculoid leprosies. Skin lesions in this type of leprosy are diffuse and poorly defined.

Causes and incidence

Contrary to popular belief, leprosy isn't highly contagious; it actually has a low rate of infectivity. Continuous, close contact is needed to transmit it. In fact, 9 out of 10 persons have a natural immunity to it. Susceptibility appears highest during childhood and seems to decrease with age. Presumably, transmission occurs through nasal droplets containing *M. leprae* or by inoculation through skin breaks (with a contaminated hypodermic or tattoo needle, for example). The incubation period is unusually long—2 to 40 years with an average of 5 to 7 years.

Leprosy is most prevalent in the underdeveloped areas of Asia (especially India and China), Africa, South America, and the islands of the Caribbean and Pacific. About 6 million people worldwide suffer from this disease; approximately 7,000 are in the United States, mostly in California, Texas, Louisiana, Florida, New York, and Hawaii.

Complications

- Fever
- Malaise
- Lymphadenopathy
- Ulceration into muscle and fascia
- Secondary bacterial infection

Signs and symptoms

M. leprae attacks the peripheral nervous system, especially the ulnar, radial, facial, anterior-tibial, and posterior-popliteal nerves. The central nervous system appears highly resistant. When the bacilli damage the skin's fine nerves, they cause anesthesia, anhidrosis, and dryness. If they attack a large nerve trunk, motor nerve damage, weakness, and pain occur, followed by peripheral anesthesia, muscle paralysis, or atrophy. In later stages, clawhand, footdrop, and ocular complications — such as corneal insensitivity and ulceration, conjunctivitis, photophobia, and blindness — can occur. Injury, ulceration, infection, and disuse of the deformed parts cause scarring and contracture. Neurologic complications occur in both lepromatous and tuberculoid leprosy but are less extensive and develop more slowly in the lepromatous form. Lepromatous leprosy can invade tissue in virtually every organ of the body, but the organs generally remain functional.

The lepromatous and tuberculoid forms affect the skin in markedly different ways. In lepromatous disease, early lesions are multiple, symmetrical, and erythematous, sometimes appearing as macules or papules with smooth surfaces. Later, they enlarge and form plaques or nodules called *lepromas* on the earlobes, nose, eyebrows, and forehead, giving the patient a characteristic leonine appearance. In advanced stages, *M. leprae* may infiltrate the entire skin surface. Lepromatous leprosy also causes loss of eyebrows, eyelashes, and sebaceous and sweat gland function and, in advanced stages, conjunctival and scleral nodules. Upper respiratory lesions cause epistaxis, ulceration of the uvula and tonsils, septal perforation, and nasal collapse. Lepromatous leprosy can lead to hepatosplenomegaly and orchitis. Fingertips and toes deteriorate as bone resorption follows trauma and infection in these insensitive areas.

When tuberculoid leprosy affects the skin (sometimes its effect is strictly neural), it produces raised, large, erythematous plaques or macules with clearly defined borders. As they grow, they become rough, hairless, and hypopigmented and leave anesthetic scars.

In borderline leprosy, skin lesions are numerous but smaller, less anesthetic, and less sharply defined than tuberculoid lesions. Untreated, borderline leprosy may deteriorate into lepromatous disease.

Occasionally, acute episodes intensify leprosy's slowly progressing course. Whether such exacerbations are part of the disease process or a reaction to therapy remains controversial. Erythema nodosum leprosum (ENL), seen in lepromatous leprosy,

produces fever, malaise, lymphadenopathy, and painful red skin nodules, usually during antimicrobial treatment, although it may occur in untreated people. In Mexico and other Central American countries, some patients with lepromatous disease develop Lucio's phenomenon. This malady produces generalized punched-out ulcers that may extend into muscle and fascia. Leprosy may also lead to secondary bacterial infection of skin ulcers and to amyloidosis.

Diagnosis

Early clinical indications of skin lesions and muscular and neurologic deficits are usually sufficiently diagnostic in patients from endemic areas. Biopsies of skin lesions are also diagnostic. Peripheral nerve biopsy or smears of the skin or of ulcerated mucous membranes help confirm the diagnosis. Blood tests show increased erythrocyte sedimentation rate; decreased albumin, calcium, and cholesterol levels; and, possibly, anemia.

Treatment

Treatment consists of antimicrobial therapy using sulfones, primarily oral dapsone, which may cause hypersensitivity reactions. Hepatitis and exfoliative dermatitis, although uncommon, are especially dangerous reactions. If they occur, sulfone therapy should be stopped immediately.

Failure to respond to sulfone or the occurrence of respiratory involvement or other complications requires the use of alternative therapy, such as rifampin in combination with clofazimine or ethionamide. Clawhand, wristdrop, or footdrop may require surgical correction.

When a patient's disease becomes inactive, as determined by the morphologic and bacterial index, treatment is discontinued according to the following schedule: tuberculoid, 3 years; borderline, depends on the severity of the disease but may be as long as 10 years; lepromatous, requires lifetime therapy.

Because ENL is commonly considered a sign that the patient is responding to treatment, antimicrobial therapy should be continued. Thalidomide and clofazimine have been used successfully to treat ENL at the National Hansen's Disease Center (NHDC); however, this treatment requires a signed consent form and strict adherence to established NHDC protocols. Corticosteroids may also be given as part of ENL therapy.

Any patient suspected of having leprosy may be referred to the Gillis W. Long Hansen's Disease Center in Carville, Louisiana, or to a regional center. At this international research and educational center, patients undergo diagnostic studies and treatment and are educated about their disease. Patients are encouraged to return home as soon as their medical condition permits. The federal government pays the full cost of their medical and nursing care.

Special considerations

Patient care is supportive and consists of measures to control acute infection, prevent complications, speed rehabilitation and recovery, and provide psychological support.

- Give antipyretics, analgesics, and sedatives, as needed. Watch for and report ENL or Lucio's phenomenon.
- Although leprosy isn't highly contagious, take precautions against the possible spread of infection. Tell patients to cover coughs or sneezes with a paper tissue and to dispose of it properly. Take infection precautions when handling clothing or articles that have been in contact with open skin lesions.
- Patients with borderline or lepromatous leprosy may suffer associated eye complications, such as iridocyclitis and glaucoma. Decreased corneal sensation and lacrimation may also occur, requiring patients to use a tear substitute daily and protect their eyes to prevent corneal irritation and ulceration.
- Stress the importance of adequate nutrition and rest. Watch for fatigue, jaundice, and other signs of anemia and hepatitis.
- Tell the patient to be careful not to injure an anesthetized leg by putting too much weight on it. Advise testing bath water carefully to prevent scalding. To prevent ulcerations, suggest the use of sturdy footwear and soaking feet in warm water after any kind of exercise, even a short walk. Advise rubbing the feet with petroleum jelly, oil, or lanolin.
- For patients with deformities, an interdisciplinary rehabilitation program employing a physiotherapist and plastic surgeon may be necessary. Teach the patient and help him with prescribed therapies.

- Provide emotional support throughout treatment. Communicating accurate information about leprosy to the general public, especially to health care professionals, is a function of primary importance for the entire staff at the NHDC.

MYCOSES

Candidiasis

Candidiasis (also called *candidosis* or *moniliasis*) is usually a mild, superficial fungal infection caused by the genus *Candida*. It usually infects the nails (onychomycosis), skin (diaper rash), or mucous membranes, especially the oropharynx (thrush), vagina (moniliasis), esophagus, and GI tract. Rarely, these fungi enter the bloodstream and invade the kidneys, lungs, endocardium, brain, or other structures, causing serious infections. Such systemic infection is most prevalent among drug abusers and patients already hospitalized, particularly diabetics, immunosuppressed patients, or patients receiving broad-spectrum antibiotics. The prognosis varies, depending on the patient's resistance.

Causes and incidence

Most cases of *Candida* infection result from *C. albicans*. Other infective strains include *C. parapsilosis*, *C. tropicalis*, *C. glabrata*, and *C. guillermondii*. These fungi are part of the normal flora of the GI tract, mouth, vagina, and skin. They cause infection when some change in the body (rising glucose levels from diabetes mellitus; lowered resistance from an immunosuppressive drug, radiation, aging, or a disease, such as cancer or human immunodeficiency virus [HIV] infection) permits their sudden proliferation or when they're introduced systemically by I.V. or urinary catheters, drug abuse, hyperalimentation, or surgery. However, the most common predisposing factor remains the use of broad-spectrum antibiotics, which decrease the number of normal flora and permit an increasing number of candidal organisms to proliferate. The baby of a mother with vaginal candidiasis can contract oral thrush while passing through the birth canal. Thrush is also found in many infants who are breast-fed. The incidence of candidiasis is rising because of wider use of I.V. therapy and a greater number of immunocompromised patients, especially those with HIV infection.

Complication

- Candida dissemination with kidney, brain, GI tract, eyes, lungs, and heart failure.

Signs and symptoms

Symptoms of superficial candidiasis correspond to the site of infection:

- skin — scaly, erythematous, papular rash, sometimes covered with exudate, appearing below the breast, between fingers, and at the axillae, groin, and umbilicus; in diaper rash, papules at the edges of the rash
- nails — red, swollen, darkened nail bed; occasionally, purulent discharge and the separation of a pruritic nail from the nail bed
- oropharyngeal mucosa (thrush) — cream-colored or bluish white curdlike patches of exudate on the tongue, mouth, or pharynx that reveal bloody engorgement when scraped. They may swell, causing respiratory distress in infants, or they may be painful or cause a burning sensation in the throats and mouths of adults. (See *Recognizing candidiasis*.)
- esophageal mucosa — dysphagia, retrosternal pain, regurgitation and, occasionally, scales in the mouth and throat
- vaginal mucosa — white or yellow discharge, with pruritus and local excoriation; white or gray raised patches on vaginal walls, with local inflammation; dyspareunia.

Systemic infection produces chills; high, spiking fever; hypotension; prostration; myalgias; arthralgias; and a rash. Specific

signs and symptoms depend on the site of infection:

- pulmonary — hemoptysis, cough, fever
- renal — fever, flank pain, dysuria, hematuria, pyuria, cloudy urine
- brain — headache, nuchal rigidity, seizures, focal neurologic deficits
- endocardium — systolic or diastolic murmur, fever, chest pain, embolic phenomena
- eye — endophthalmitis, blurred vision, orbital or periorbital pain, scotoma, and exudate.

Diagnosis

Diagnosis of superficial candidiasis depends on clinical signs and symptoms plus evidence of *Candida* on a Gram stain of skin, vaginal scrapings, pus, or sputum or on skin scrapings prepared in potassium hydroxide solution. Systemic infections require obtaining a specimen for blood or tissue culture.

Treatment

Treatment first aims to improve the underlying condition that predisposes the patient to candidiasis, such as controlling diabetes or discontinuing antibiotic

therapy and catheterization, if possible.

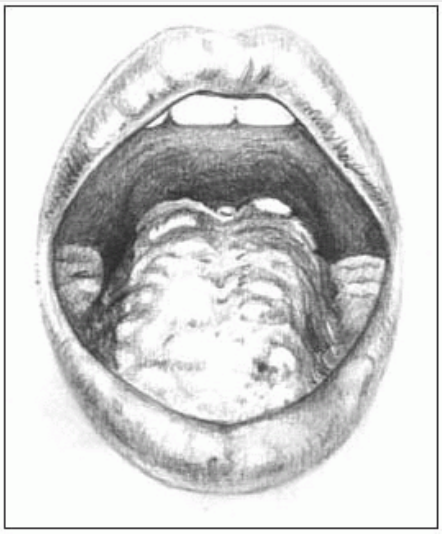
Nystatin is an effective antifungal for superficial candidiasis. Clotrimazole, fluconazole, ketoconazole, and miconazole are effective in mucous-membrane and vaginal candidal infections. Ketoconazole or fluconazole is the treatment of choice for chronic candidiasis of the mucous membranes. Treatment for systemic infection consists of I.V. amphotericin B or fluconazole.

Special considerations

- Instruct the patient using nystatin solution to swish it around in his mouth for several minutes before he swallows it.
- Swab nystatin on the oral mucosa of an infant with thrush. Treat the infant after a feeding because feedings will wash the medication away. The infant's mother should also be treated to prevent the infection from being passed back and forth.
- Provide the patient with a nonirritating mouthwash to loosen tenacious secretions and a soft toothbrush to avoid irritation.
- Relieve the patient's mouth discomfort with a topical anesthetic, such as lidocaine, at least 1 hour before meals. (It may suppress the gag reflex and cause aspiration.)
- Provide a soft diet for the patient with severe dysphagia. Tell the patient with mild dysphagia to chew food thoroughly, and make sure he doesn't choke.
- Use dry padding in intertriginous areas of obese patients to prevent irritation.
- Note dates of insertion of I.V. catheters, and replace them according to your hospital's policy to prevent phlebitis.
- Assess the patient with candidiasis for underlying causes such as diabetes mellitus. If the patient is receiving amphotericin B for systemic candidiasis, he may have severe chills, fever, anorexia, nausea, and vomiting. Premedicate with acetaminophen, antihistamines, or antiemetics to help reduce adverse effects.
- Frequently check vital signs of patients with systemic infections. Provide appropriate supportive care. In patients with renal involvement, carefully monitor intake and output and urine blood and protein levels.
- Check high-risk patients daily, especially those receiving antibiotics, for patchy areas, irritation, sore throat, bleeding of the mouth or gums, or other signs of superinfection. Check for vaginal discharge; record color and amount.
- Encourage women in their third trimester of pregnancy to be examined for vaginal candidiasis to protect their neonate from infection at birth.

RECOGNIZING CANDIDIASIS

Candidiasis of the oropharyngeal mucosa (thrush) causes creamcolored or bluish white pseudomembranous patches on the tongue, mouth, or pharynx. Fungal invasion may extend to circumoral tissues.



Cryptococcosis

Cryptococcosis, also called *torulosis* or *European blastomycosis,* is caused by the fungus *Cryptococcus neoformans*. Usually beginning as an asymptomatic pulmonary infection, it disseminates to extrapulmonary sites, usually to the central nervous system (CNS) but also to the skin, bones, prostate gland, liver, or kidneys.

With appropriate treatment, the prognosis in pulmonary cryptococcosis is good. CNS infection, however, can be fatal, but treatment dramatically reduces mortality.

Complications

- Optic atrophy
- Ataxia
- Hydrocephalus
- Deafness
- Paralysis
- Organic mental syndrome
- Personality changes

Causes and incidence

Because *C. neoformans* is transmitted in particles of dust contaminated by pigeon feces that harbor this organism, cryptococcosis is primarily an urban infection. It's most prevalent in men, usually those between ages 30 and 60, and is rare in children.

Cryptococcosis is especially likely to develop in immunocompromised patients, such as those with Hodgkin's lymphoma, sarcoidosis, leukemia, or lymphoma, and in those who are receiving immunosuppressive agents. Currently, patients with acquired immunodeficiency syndrome (AIDS) are by far the most commonly affected group.

Signs and symptoms

Typically, signs and symptoms of pulmonary cryptococcosis include fever, cough with pleuritic pain, weight loss, and CNS disturbances. CNS involvement occurs gradually (cryptococcal meningitis) and causes progressively severe frontal and temporal headache, diplopia, blurred vision, dizziness, ataxia, aphasia, vomiting, tinnitus, memory changes, inappropriate behavior, irritability, psychotic symptoms, seizures, and fever. If untreated, symptoms progress to coma and death, usually as a result of cerebral edema or hydrocephalus.

Skin involvement produces red facial papules and other skin abscesses, with or without ulcerations; bone involvement produces painful osseous lesions of the long bones, skull, spine, and joints.

Diagnosis

Although a routine chest X-ray showing a pulmonary lesion may point to pulmonary cryptococcosis, this infection usually escapes diagnosis until it disseminates.

CONFIRMING DIAGNOSIS

Firm diagnosis requires identification of C. neoformans *by culture of sputum, urine, prostatic secretions, bone marrow aspirate or biopsy, or pleural biopsy; in CNS infection, by an India ink preparation of cerebrospinal fluid (CSF) and culture. Blood cultures are positive only in severe infection.*

Supportive values include increased antigen titer in serum and CSF in disseminated infection; increased CSF pressure, protein, and white blood cell count in CNS infection; and moderately decreased CSF glucose levels in about half these patients. Diagnosis must rule out cancer and tuberculosis.

Treatment

The patient with pulmonary cryptococcosis will require close medical observation for a year after diagnosis. Treatment is unnecessary unless extrapulmonary lesions develop or pulmonary lesions progress.

Treatment of disseminated infection calls for I.V. amphotericin B, flucytosine, or fluconazole. Patients with AIDS will also need long-term therapy, usually with oral fluconazole.

Special considerations

Cryptococcosis doesn't require isolation.

- Check the patient's vital functions, and note any changes in his mental status, orientation, pupillary response, and motor function.
- Watch for headache, vomiting, and nuchal rigidity.
- Before giving I.V. amphotericin B, check for phlebitis. Infuse slowly and dilute as ordered — rapid infusion may cause circulatory collapse.
- Before therapy, draw blood for a serum electrolyte analysis to determine baseline renal status.
- During drug therapy, watch for decreased urine output, elevated blood urea nitrogen and creatinine levels, and hypokalemia.
- Monitor results of complete blood count, urinalysis, magnesium and potassium levels, and hepatic function tests. Ask the patient to report hearing loss, tinnitus, or dizziness.
- Give analgesics, antihistamines, and antiemetics, as ordered, for fever, chills, nausea, and vomiting.
- Provide psychological support to help the patient cope with long-term hospitalization.

Aspergillosis

Aspergillosis is an opportunistic infection caused by fungi of the genus *Aspergillus*, usually *A. fumigatus*, *A. flavus*, and *A. niger*. It occurs in four major forms: *aspergilloma*, which produces a fungus ball in the lungs (called a *mycetoma*); *allergic aspergillosis*, a hypersensitive asthmatic reaction to aspergillus antigens; *aspergillosis endophthalmitis*, an infection of the anterior and posterior chambers of the eye that can lead to blindness; and *disseminated aspergillosis*, an acute infection that produces septicemia, thrombosis, and infarction of virtually any organ, but especially the heart, lungs, brain, and kidneys.

Aspergillus may cause infection of the ear (otomycosis), cornea (mycotic keratitis), and prosthetic heart valves (endocarditis); pneumonia (especially in

patients receiving immunosuppressants, such as antineoplastic agents or high-dose steroids); sinusitis; and brain abscesses.

The prognosis varies with each form. Occasionally, aspergilloma causes fatal hemoptysis.

Causes and incidence

Aspergillus is found worldwide, commonly in decaying vegetation, such as fermenting compost piles and damp hay. It's transmitted by inhalation of fungal spores or, in aspergillosis endophthalmitis, by the invasion of spores through a wound or other tissue injury. It's a common laboratory contaminant.

Aspergillus produces clinical infection only in people who become especially vulnerable to it. Such vulnerability can result from excessive or prolonged use of antibiotics, glucocorticoids, or other immunosuppressive agents; from radiation; from such conditions as acquired immunodeficiency syndrome, Hodgkin's disease, leukemia, azotemia, alcoholism, sarcoidosis, bronchitis, or bronchiectasis; from organ transplants; and, in aspergilloma, from tuberculosis or another cavitary lung disease.

Complications

- Respiratory failure
- Death

Signs and symptoms

The incubation period in aspergillosis ranges from a few days to weeks. In aspergilloma, colonization of the bronchial tree with *Aspergillus* produces plugs and atelectasis and forms a tangled ball of hyphae (fungal filaments), fibrin, and exudate in a cavity left by a previous illness such as tuberculosis. Characteristically, aspergilloma either causes no symptoms or mimics tuberculosis, causing a productive cough and purulent or blood-tinged sputum, dyspnea, empyema, and lung abscesses.

Allergic aspergillosis causes wheezing, dyspnea, cough with some sputum production, pleural pain, and fever.

Aspergillosis endophthalmitis usually appears 2 to 3 weeks after an eye injury or surgery and accounts for half of all cases of endophthalmitis. It causes clouded vision, eye pain, and reddened conjunctiva. Eventually, *Aspergillus* infects the anterior and posterior chambers, where it produces purulent exudate.

ALERT

In disseminated aspergillosis, Aspergillus *invades blood vessels and causes thrombosis, infarctions, and the typical signs and symptoms of septicemia (chills, fever, hypotension, delirium), with azotemia, hematuria, urinary tract obstruction, headaches, seizures, bone pain and tenderness, and soft-tissue swelling. It's rapidly fatal.*

Diagnosis

In patients with aspergilloma, a chest X-ray reveals a crescent-shaped radiolucency surrounding a circular mass, but this isn't definitive for aspergillosis.

CONFIRMING DIAGNOSIS

In aspergillosis endophthalmitis, a history of ocular trauma or surgery and a culture or exudate showing Aspergillus *is diagnostic. In disseminated aspergillosis, culture and microscopic examination of affected tissue can confirm the diagnosis, but this form is usually diagnosed at autopsy.*

In allergic aspergillosis, sputum examination shows eosinophils. Culture of mouth scrapings or sputum showing *Aspergillus* is inconclusive because even healthy people harbor this fungus.

Treatment

Aspergillosis doesn't require isolation. Treatment requires local excision of the lesion and supportive therapy, such as chest physiotherapy and coughing, to improve pulmonary function. Endocarditis caused by *Aspergillus* is treated by surgical removal of infected heart valves and long-term amphotericin B therapy. Allergic aspergillosis requires desensitization and, possibly, steroids. Disseminated aspergillosis and aspergillosis endophthalmitis require a 2- to 3-week course of I.V. amphotericin B (as well as prompt cessation of immunosuppressive therapy). Voriconazole or itraconazole can also be used for treatment. However, the disseminated form results in an infection that's so virulent that amphotericin B therapy can't stop the systemic involvement; eventually, death ensues.

Special considerations

- Assist with chest physiotherapy and instruct the patient to cough effectively.
- Monitor the patient's vital signs, intake and output, and diagnostic test results.
- Provide emotional support for the patient and his family.

Histoplasmosis

Histoplasmosis is a fungal infection caused by *Histoplasma capsulatum*. This disease may also be called *Ohio Valley, Central Mississippi Valley, Appalachian Mountain,* or *Darling's disease*. In the United States, it occurs in three forms: primary acute histoplasmosis, progressive disseminated histoplasmosis (acute disseminated or chronic disseminated disease), and chronic pulmonary (cavitary) histoplasmosis, which produces cavitations in the lung similar to those in pulmonary tuberculosis.

A fourth form, African histoplasmosis, occurs only in Africa and is caused by the fungus *Histoplasma capsulatum duboisii*.

The prognosis varies with each form. The primary acute disease is benign; the progressive disseminated disease is fatal in approximately 90% of patients; and, without proper chemotherapy, chronic pulmonary histoplasmosis is fatal in 50% of patients within 5 years.

Causes and incidence

H. capsulatum is found in the feces of birds and bats or in soil contaminated by their feces, such as that near roosts, chicken coops, barns, caves, or underneath bridges. Transmission occurs through inhalation of *H. capsulatum* or *H. duboisii* spores or through the invasion of spores after minor skin trauma. Possibly, oral ingestion of spores may cause the disease.

The incubation period is from 5 to 18 days, although chronic pulmonary histoplasmosis may progress slowly for many years. Probably because of occupational exposure, histoplasmosis is more common in adult males. Fatal disseminated

disease, however, is more common in infants and elderly men.

Histoplasmosis occurs worldwide, especially in the temperate areas of Asia, Africa, Europe, and North and South America. In the United States, it's most prevalent in the central and eastern states, especially in the Mississippi and Ohio River valleys.

Complications

- Vascular or bronchial obstruction
- Acute pericarditis
- Pleural effusion
- Mediastinal fibrosis or granuloma
- Intestinal ulceration

- Addison's disease
- Endocarditis
- Meningitis

Signs and symptoms

Symptoms vary with each form of this disease. Primary acute histoplasmosis may be asymptomatic or may cause symptoms of a mild respiratory illness similar to a severe cold or influenza. Typical clinical effects may include fever, malaise, headache, myalgia, anorexia, cough, chest pain, anemia, leukopenia, thrombocytopenia, and oropharyngeal ulcers.

Progressive disseminated histoplasmosis causes hepatosplenomegaly, general lymphadenopathy, anorexia, weight loss, fever and, possibly, ulceration of the tongue, palate, epiglottis, and larynx, with resulting pain, hoarseness, and dysphagia. It may also cause endocarditis, meningitis, pericarditis, and adrenal insufficiency.

Chronic pulmonary histoplasmosis mimics pulmonary tuberculosis and causes a productive cough, dyspnea, and occasional hemoptysis. Eventually, it produces weight loss, extreme weakness, breathlessness, and cyanosis.

African histoplasmosis produces cutaneous nodules, papules, and ulcers; lesions of the skull and long bones; lymphadenopathy; and visceral involvement without pulmonary lesions.

Diagnosis

A history of exposure to contaminated soil in an endemic area, miliary calcification in the lung or spleen, a positive histoplasmin skin test or urine antigen test indicate exposure to histoplasmosis. Rising complement fixation and agglutination titers (more than 1:32) strongly suggest histoplasmosis. A histoplasmosis antigen assay test can help in diagnosis.

CONFIRMING DIAGNOSIS

The diagnosis of histoplasmosis requires a morphologic examination of tissue biopsy and culture of H. capsulatum *from sputum in acute primary and chronic pulmonary histoplasmosis and from bone marrow, lymph node, blood, and infection sites in disseminated histoplasmosis. However, cultures take several weeks to grow these organisms. Faster diagnosis is possible with stained biopsies. Also available is a deoxyribonucleic acid probe for histoplasma that can be used for difficult isolates.*

Findings must rule out tuberculosis and other diseases that produce similar symptoms. The diagnosis of histoplasmosis caused by *H. duboisii* necessitates examination of tissue biopsy and culture of the affected site.

Treatment

Treatment consists of antifungal therapy, surgery, and supportive care. Antifungal therapy is most important. Except for asymptomatic primary acute histoplasmosis (which resolves spontaneously) and the African form, histoplasmosis requires high-dose or long-term (10-week) therapy with amphotericin B, fluconazole, ketoconazole, or itraconazole. For a patient who also has acquired immunodeficiency syndrome, lifelong therapy with fluconazole is indicated.

Supportive care usually includes oxygen for respiratory distress, glucocorticoids for adrenal insufficiency, and parenteral fluids for dysphagia due to oral or laryngeal ulcerations. Histoplasmosis doesn't require isolation.

Special considerations

Patient care is primarily supportive.

- Give medications as ordered, and teach a patient about possible adverse effects. Because amphotericin B may cause chills, fever, nausea, and vomiting, give appropriate antipyretics and antiemetics, as ordered.

- A patient with chronic pulmonary or disseminated histoplasmosis also needs psychological support because of long-term hospitalization. As needed, refer the patient to a social worker or occupational therapist. Help the parents of children with this disease arrange for a visiting teacher.

PREVENTION

Teach a patient in an endemic area to watch for early signs and to seek treatment promptly. Instruct a patient who's at risk for occupational exposure to contaminated soil to wear a face mask.

Blastomycosis

Blastomycosis (sometimes called *North American blastomycosis* or *Gilchrist' s disease*) is caused by the yeastlike fungus *Blastomyces dermatitidis*, which usually infects the lungs and produces bronchopneumonia. Less commonly, this fungus may disseminate through the blood and cause osteomyelitis and central nervous system (CNS), skin, and genital disorders. Untreated blastomycosis is slowly progressive and usually fatal; however, spontaneous remissions occasionally occur. With antifungal drug therapy and supportive treatment, the prognosis for patients with blastomycosis is good.

Causes and incidence

B. dermatitidis is probably inhaled by people who are in close contact with the soil. The incubation period may range from weeks to months. Blastomycosis is generally found in North America (where *B. dermatitidis* normally inhabits the soil), and is endemic to the southeastern United States. Sporadic cases have also been reported in Africa. Blastomycosis usually infects men ages 30 to 50, but no occupational link has been found.

Complications

- Skin abscesses
- Skin fistulas
- Meningitis
- Cerebral abscesses
- Addison's disease
- Pericarditis
- Arthritis

Signs and symptoms

Fifty percent of cases are asymptomatic. Initial signs and symptoms of pulmonary blastomycosis mimic those of a bacterial upper respiratory tract infection. These findings typically include a dry, hacking, or productive cough (occasionally hemoptysis), pleuritic chest pain, fever, shaking, chills, night sweats, malaise, anorexia, weight loss, and arthralgia. It can progress to pneumonia.

Cutaneous blastomycosis causes small, painless, nonpruritic, and nondistinctive macules or papules on exposed body parts. These lesions become raised and reddened and occasionally progress to draining skin abscesses or fistulas.

Dissemination to the bone causes soft-tissue swelling, tenderness, and warmth over bony lesions, which generally occur in the thoracic, lumbar, and sacral regions; long bones of the legs; and, in children, the skull.

Genital dissemination produces painful swelling of the testes, epididymis, or prostate; deep perineal pain; pyuria; and hematuria. CNS dissemination, which usually only occurs in immunocompromised hosts, causes meningitis or cerebral abscesses with resulting decreased level of consciousness (LOC), lethargy, and change in mood or affect. Other forms of dissemination may result in Addison's disease (adrenal insufficiency), pericarditis, and arthritis.

Diagnosis

Diagnosis of blastomycosis requires:

- culture of *B. dermatitidis* from skin lesions, pus, sputum, or pulmonary secretions
- microscopic examination of tissue specimens from the skin or the lungs or of bronchial washings, sputum, or pus, as the physician finds appropriate
- immunodiffusion testing, which detects antibodies for the A and B antigen of blastomycosis.

In addition, suspected pulmonary blastomycosis requires a chest X-ray, which may show pulmonary infiltrates. Other abnormal laboratory findings include increased white blood cell count and erythrocyte sedimentation rate, slightly increased serum globulin levels, mild normochromic anemia and, with bone lesions, increased alkaline phosphatase.

Treatment

All forms of blastomycosis respond to amphotericin B. Ketoconazole or fluconazole may be used as alternative agents and may be more effective with the patients who are immunocompromised. Patient care is mainly supportive.

Special considerations

- In severe pulmonary blastomycosis, check for hemoptysis. If the patient is febrile, provide a cool room and give tepid sponge baths.
- If blastomycosis causes joint pain or swelling, elevate the joint and apply heat. In CNS infection, watch the patient carefully for decreasing LOC and unequal pupillary response. In men with disseminated disease, watch for hematuria.
- Infuse I.V. antifungal agents slowly (toorapid infusion may cause circulatory collapse). During infusion, monitor vital signs (temperature may rise but should subside within 1 to 2 hours). Watch for decreased urine output and monitor laboratory results for increased blood urea nitrogen and creatinine levels. Monitor serum potassium levels for signs of amphotericin B-induced hypokalemia, which may indicate renal toxicity. Report any hearing loss, tinnitus, or dizziness immediately. To relieve the adverse effects of amphotericin B, give antiemetics and antipyretics, as ordered.

Coccidioidomycosis

Coccidioidomycosis, also called *valley fever* or *San Joaquin Valley fever,* is caused by the fungus *Coccidioides immitis* and occurs primarily as a respiratory infection. Secondary sites include the skin, bones, joints, and meninges. Generalized dissemination is also possible. The *primary* pulmonary form is usually self-limiting and seldom fatal. The rare *secondary* (progressive, disseminated) form produces abscesses throughout the body and carries a mortality of up to 60%, even with treatment. Such dissemination is more common in darkskinned men, pregnant women, and patients who are receiving immunosuppressants.

Causes and incidence

Coccidioidomycosis is endemic to the southwestern United States, especially between the San Joaquin Valley in California and southwestern Texas; it's also found in Mexico, Guatemala, Honduras, Venezuela, Colombia, Argentina, and Paraguay. It may result from inhalation of *C. immitis* spores found in the soil in these areas or from inhalation of spores from dressings or plaster casts of infected people. It's most prevalent during warm, dry months.

Because of population distribution and an occupational link (it's common in migrant farm laborers), coccidioidomycosis generally strikes Philippinos, Mexicans, Native Americans, and Blacks. In primary infection, the incubation period is from 1 to 4 weeks.

Complications

- Bronchiectasis
- Osteomyelitis
- Meningitis
- Hepatosplenomegaly
- Liver failure

Signs and symptoms

Primary coccidioidomycosis usually produces acute or subacute respiratory signs and symptoms (dry cough, pleuritic chest pain, and pleural effusion), fever, sore throat, dyspnea, chills, malaise, headache, and an itchy macular rash. Chest pain, night sweats, and arthralgias can occur as well. Occasionally, the only sign is a fever that persists for weeks. From 3 days to several weeks after onset, some patients, particularly white women, may develop tender red nodules (erythema nodosum) on their legs, especially the shins, with joint pain in the knees and ankles. Generally, primary disease heals spontaneously within a few weeks.

In rare cases, coccidioidomycosis disseminates to other organs several weeks or months after the primary infection. Disseminated coccidioidomycosis causes fever

and abscesses throughout the body, especially in skeletal, central nervous system (CNS), splenic, hepatic, renal, and subcutaneous tissues. Depending on the location of these abscesses, disseminated coccidioidomycosis may cause bone pain and meningitis. Chronic pulmonary cavitation, which can occur in both the primary and the disseminated forms, causes hemoptysis with or without chest pain.

Diagnosis

CONFIRMING DIAGNOSIS

Typical clinical features and skin and serologic studies confirm this diagnosis. The primary form — and sometimes the disseminated form — produces a positive coccidioidin skin test.

In the first week of illness, complement fixation for immunoglobulin G antibodies or, in the first month, positive serum precipitins (immunoglobulins) also establish this diagnosis. Examination or, more recently, immunodiffusion testing of sputum, pus from lesions, and a tissue biopsy may show *C. immitis* spores. The presence of antibodies in pleural and joint fluid and a rising serum or body fluid antibody titer indicate dissemination.

Other abnormal laboratory results include increased white blood cell (WBC) count, eosinophilia, increased erythrocyte sedimentation rate, and a chest X-ray showing bilateral diffuse infiltrates.

In coccidioidal meningitis, examination of cerebrospinal fluid shows WBC count increased to more than 500/µl (primarily due to mononuclear leukocytes), increased protein levels, and decreased glucose levels. Ventricular fluid obtained from the brain may contain complement fixation antibodies.

After diagnosis, the results of serial skin tests, blood cultures, and serologic testing may document the therapy's effectiveness.

Treatment

Usually, mild primary coccidioidomycosis requires only bed rest and relief of symptoms. Severe primary disease and dissemination, however, also require long-term I.V. infusion (or, in CNS dissemination, intrathecal administration) of amphotericin B, fluconazole, or itraconazole and, possibly, excision or drainage of lesions. Severe pulmonary lesions may require lobectomy. Miconazole and ketoconazole suppress *C. immitis* but don't eradicate it. Ketoconazole and itraconazole are used for oral treatment of nonmeningeal infection and for long-term therapy.

Special considerations

- Don't wash off the circle marked on the skin for serial skin tests, because this aids in reading test results.
- In mild primary disease, encourage bed rest and adequate fluid intake. Record the amount and color of sputum. Watch for shortness of breath that may point to pleural effusion. In patients with arthralgia, provide analgesics as ordered.
- Coccidioidomycosis requires standard precautions, such as gloves for contact with drainage or broken skin, and good hand hygiene.
- In CNS dissemination, monitor the patient carefully for decreased level of consciousness or change in mood or affect.
- Before intrathecal administration of amphotericin B, explain the procedure to the patient, and reassure him that he'll receive analgesics before a lumbar puncture. If the patient is to receive I.V. amphotericin B, infuse it slowly, as ordered, because rapid infusion may cause circulatory collapse. During infusion, monitor vital signs (temperature may rise but should return to normal within 1 to 2 hours). Watch for decreased urine output, and monitor laboratory results for elevated blood urea nitrogen and creatinine levels and for hypokalemia. Tell the patient to immediately report hearing loss, tinnitus, dizziness, and all signs of toxicity. To ease adverse effects of amphotericin B, give antiemetics and antipyretics, as ordered.

Sporotrichosis

Sporotrichosis is a chronic disease caused by the fungus *Sporothrix schenckii*. It occurs in three forms: *cutaneous lymphatic*, which produces nodular erythematous primary lesions and secondary lesions along lymphatic channels; *pulmonary*, a rare form that produces a productive cough and pulmonary lesions; and *disseminated*, another rare form that may cause arthritis or

osteomyelitis. The course of sporotrichosis is slow, the prognosis is good, and fatalities are rare. However, untreated skin lesions may cause secondary bacterial infection.

Causes and incidence

S. schenckii is found in soil, wood, sphagnum moss, and decaying vegetation throughout the world. Because this fungus usually enters through broken skin (the pulmonary form through inhalation), sporotrichosis is more common in horticulturists, agricultural workers, and home gardeners. Perhaps because of occupational exposure, it's more prevalent in adult men than in women and children.

Complications

- Arthritis
- Osteomyelitis
- Meningitis

Signs and symptoms

After an incubation period that lasts from 1 week to 3 months, cutaneous lymphatic sporotrichosis produces characteristic skin lesions, usually on the hands or fingers. Each lesion begins as a small, painless, movable subcutaneous nodule but grows progressively larger, discolors, and eventually ulcerates. (See *Recognizing sporotrichosis.*) Later, additional lesions form along the adjacent lymph node chain.

Pulmonary sporotrichosis causes a productive cough, lung cavities and nodules, hilar adenopathy, pleural effusion, fibrosis, and the formation of a fungus ball. It's commonly associated with sarcoidosis and tuberculosis.

Disseminated sporotrichosis produces multifocal lesions that spread from the primary lesion in the skin or lungs. The disease begins insidiously, typically causing weight loss, anorexia, synovial or bony lesions and, possibly, arthritis or osteomyelitis.

Diagnosis

CONFIRMING DIAGNOSIS

Typical clinical findings and a culture of S. schenckii *in sputum, pus, or bone drainage confirm the diagnosis of sporotrichosis.*

RECOGNIZING SPOROTRICHOSIS

Ulceration, swelling, and crusting of nodules on fingers is characteristic of cutaneous lymphatic sporotrichosis.

Histologic identification is difficult. Diagnosis must rule out tuberculosis, sarcoidosis and, in patients with the disseminated form of sporotrichosis, bacterial osteomyelitis and neoplasm.

Treatment

Sporotrichosis doesn't require isolation. The cutaneous lymphatic form usually responds to itraconazole (Sporanox). Occasionally, cutaneous lesions must be excised or drained. The disseminated form responds to amphotericin B and itraconazole (Sporanox). Local heat application relieves pain. Cavitary pulmonary lesions may require surgery.

Special considerations

- Keep lesions clean, make the patient as comfortable as possible, and carefully dispose of contaminated dressings.
- Warn the patient about the possible adverse effects of drugs. Because amphotericin B may cause fever, chills, nausea, and vomiting, give antipyretics and antiemetics, as ordered.

PREVENTION

Advise a patient who gardens to wear gloves while working.

RESPIRATORY VIRUSES

Common cold

The common cold (also known as *acute coryza*) is an acute, usually afebrile viral infection that causes inflammation of the upper respiratory tract. It's the most common infectious disease, accounting for more time lost from school or work than any other cause. Although a cold is benign and self-limiting, it can lead to secondary bacterial infections.

Causes and incidence

About 90% of colds stem from a viral infection of the upper respiratory passages and consequent mucous membrane inflammation; occasionally, colds result from a mycoplasmal infection. (See *What happens in the common cold.*)

Over a hundred viruses can cause the common cold. Major offenders include rhinoviruses, coronaviruses, myxoviruses, adenoviruses, coxsackieviruses, and echoviruses.

Transmission occurs through airborne respiratory droplets, contact with contaminated objects, and hand-to-hand transmission. Children acquire new strains from their schoolmates and pass them on to family members. Fatigue or drafts don't increase susceptibility.

The common cold is more prevalent in children than in adults; in adolescent boys than in girls; and in women than in men. In temperate zones, it's more common in the colder months; in the tropics, during the rainy season.

Complications

- Sinusitis
- Otitis media
- Pharyngitis
- Lower respiratory tract infection

Signs and symptoms

After a 1- to 4-day incubation period, the common cold produces pharyngitis, nasal congestion, coryza, headache, and burning, watery eyes. Additional effects may include fever (in children), chills, myalgia, arthralgia, malaise, lethargy, and a hacking, nonproductive, or nocturnal cough.

As the cold progresses, clinical features develop more fully. After a day, symptoms include a feeling of fullness with a copious nasal discharge that commonly irritates the nose, adding to discomfort. About 3 days after onset, major signs diminish, but the “stuffed up” feeling generally persists for about a week. Reinfection (with productive cough) is common, but complications (sinusitis, otitis media, pharyngitis, and lower respiratory tract infection) are rare. A cold is communicable for 2 to 3 days after the onset of symptoms.

Diagnosis

No explicit diagnostic test exists to isolate the specific organism responsible for the common cold. Consequently, diagnosis rests on the typically mild, localized, and afebrile upper respiratory symptoms. Despite infection, white blood cell counts and differential are within normal limits. Diagnosis must rule out allergic rhinitis, measles, rubella, and other disorders that produce similar early symptoms. A temperature higher than 100° F (37.8° C), severe malaise, anorexia, tachycardia, exudate on the tonsils or throat, petechiae, and tender lymph glands may point to more serious disorders and require additional diagnostic tests.

Treatment

The primary treatments—aspirin, acetaminophen or ibuprofen, fluids, and rest—are purely symptomatic because the common cold has no cure. Aspirin eases myalgia and headache; fluids help loosen accumulated respiratory secretions and maintain hydration; and rest combats fatigue and weakness. In a child with a fever, acetaminophen is the drug of choice.

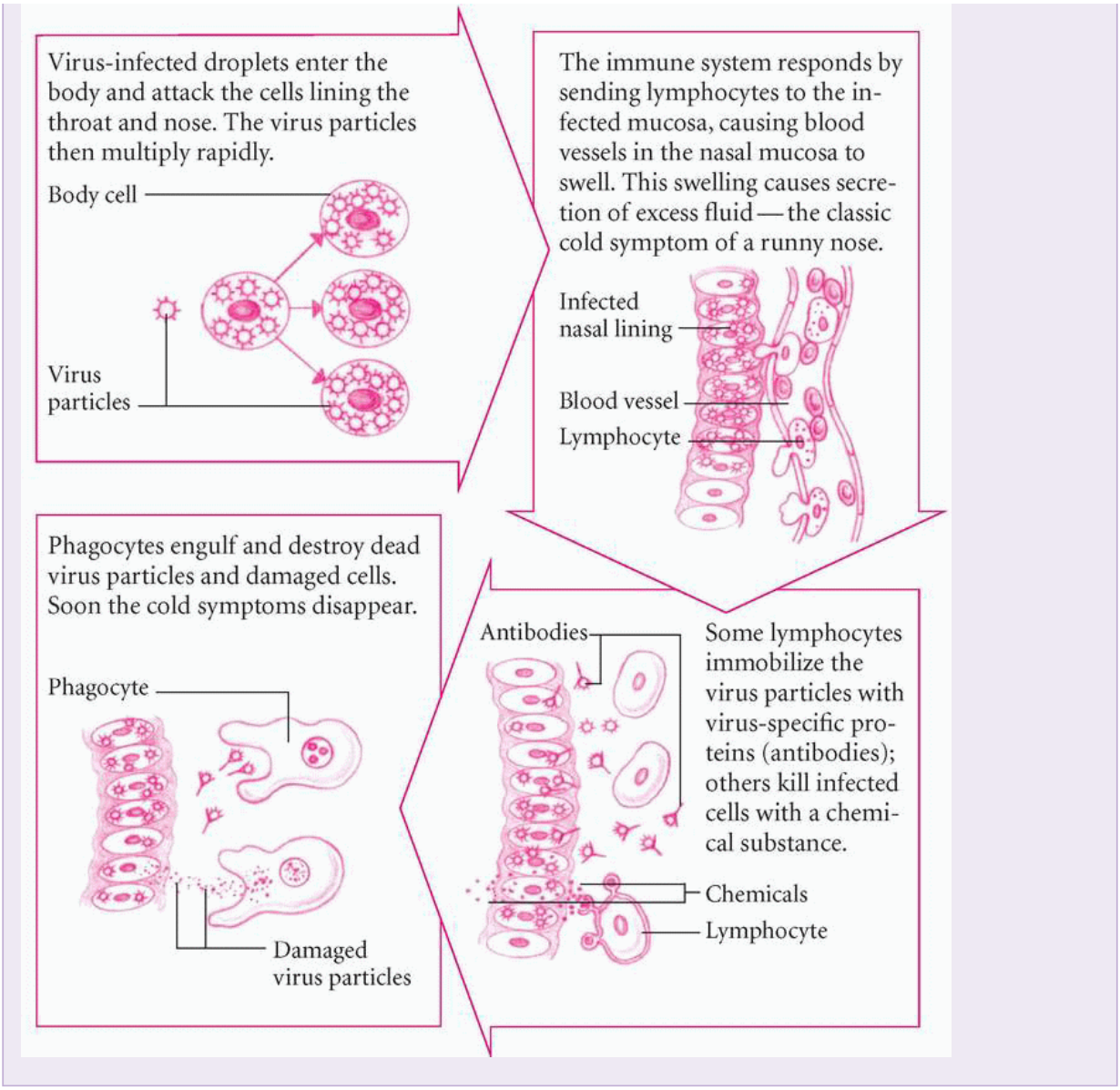
Decongestants can relieve congestion, and throat lozenges relieve soreness. Steam encourages expectoration. Nasal douching, sinus drainage, and antibiotics aren't necessary except in complications or chronic illness. Pure antitussives relieve severe coughs but are contraindicated in productive coughs, when cough suppression is harmful. The role of vitamin C remains

controversial. In infants, saline nose drops and mucus aspiration with a bulb syringe may be beneficial.

PATHOPHYSIOLOGY

WHAT HAPPENS IN THE COMMON COLD

Virus-infected droplets enter the body and attack the cells lining the throat and nose. The virus particles then multiply rapidly.



Special considerations

- Emphasize that antibiotics don't cure the common cold.
- Tell the patient to maintain bed rest during the first few days, to use a lubricant on his nostrils to decrease irritation, to relieve throat irritation with hard candy or cough drops, to increase fluid intake, and to eat light meals.
- Warm baths or heating pads can reduce aches and pains but won't hasten a cure. Suggest hot- or cold-steam vaporizers. Commercial expectorants are available, but their effectiveness is questionable.

- Advise against overuse of nose drops or sprays because they may cause rebound congestion.

PREVENTION

Warn the patient to minimize contact with people who have colds. To avoid spreading colds, teach the patient to wash his hands often and before touching his eyes, to cover coughs and sneezes, and to avoid sharing towels and drinking glasses.

Respiratory syncytial virus infection

Respiratory syncytial virus (RSV) infection results from a subgroup of the myxoviruses that resemble paramyxovirus. RSV is the leading cause of lower respiratory tract infections in infants and young children. It's the major cause of pneumonia, tracheobronchitis, and bronchiolitis in this agegroup and a suspected cause of the fatal respiratory diseases of infancy.

Causes and incidence

The organism that causes RSV is transmitted from person to person by respiratory secretions and has an incubation period of 4 to 5 days. Antibody titers seem to indicate that few children younger than age 4 escape contracting some form of RSV, even if it's mild. In fact, RSV is the only viral disease that has its maximum impact during the first few months of life (incidence of RSV bronchiolitis peaks at age 2 months). School-age children, adolescents, and young adults with mild reinfections are probably the source of infection for infants and young children.

This virus occurs in annual epidemics during the late winter and early spring in temperate climates and during the rainy season in the tropics. It can also be seen in immunocompromised adults, especially patients with bone marrow transplants.

Complications

- Pneumonia
- Bronchiolitis
- Tracheobronchitis
- Otitis media
- Apnea
- Respiratory failure

Signs and symptoms

Clinical features of RSV infection vary in severity from mild, coldlike symptoms to bronchiolitis or bronchopneumonia and, in a few patients, severe, life-threatening lower respiratory tract infections. Symptoms usually include coughing, wheezing, malaise, pharyngitis, dyspnea, and inflamed mucous membranes in the nose and throat. Reinfection is common, producing milder symptoms than the primary infection.

Otitis media is a common complication of RSV in infants. RSV has also been identified in patients with a variety of central nervous system disorders, such as meningitis and myelitis.

Diagnosis

Diagnosis is usually based on clinical findings and epidemiologic information.

- Many facilities can perform rapid tests for the virus using fluid obtained from the nose.
- Cultures of nasal and pharyngeal secretions may show RSV; however, the virus is labile, so cultures aren't always reliable.
- Chest X-rays help detect pneumonia.

Treatment

Treatment aims to support respiratory function, maintain fluid balance, and relieve symptoms. Ribavirin in aerosol form may be administered to severely ill patients or those at high risk for complications.

Special considerations

Your care plan should provide support and relief of symptoms.

- Monitor respiratory status, including rate and pattern. Watch for nasal flaring or retraction, cyanosis, pallor, and dyspnea; listen or auscultate for wheezing, rhonchi, or other signs of respiratory distress. Monitor arterial blood gas levels and oxygen saturation.
- Maintain a patent airway, and be especially watchful when the patient has periods of acute dyspnea. Perform percussion and provide drainage and suction, when necessary. Use a croup tent to provide a high-humidity atmosphere. Semi-Fowler's position may help prevent aspiration of secretions.
- Monitor intake and output carefully. Observe for signs of dehydration such as decreased skin turgor. Encourage the patient to drink plenty of high-calorie fluids. Administer I.V. fluids as needed.
- Promote bed rest. Plan your nursing care to allow uninterrupted rest.

- Hold and cuddle infants; talk to and play with toddlers. Offer diversionary activities that are appropriate for the child's condition and age. Encourage parental visits and cuddling. Restrain the child only as necessary.
- Impose droplet precautions. Enforce strict hand hygiene, because RSV may be transmitted from fomites. Avoid hand contact with nose or eyes; wear a surgical mask and eye protection.
- Make sure that staff members with respiratory illnesses don't care for infants.

Parainfluenza

Parainfluenza refers to any of a group of respiratory illnesses caused by paramyxoviruses, a subgroup of the myxoviruses. Affecting both the upper and lower respiratory tracts, these self-limiting diseases resemble influenza but are milder and seldom fatal. They primarily affect young children.

Causes and incidence

Parainfluenza is transmitted by direct contact or by inhalation of contaminated airborne droplets. Paramyxoviruses occur in four forms—Para 1 to 4—that are linked to several diseases: croup (Para 1, 2, 3); acute febrile respiratory illnesses (1, 2, 3); the common cold (1, 3, 4); pharyngitis (1, 3, 4); bronchitis (1, 3); and bronchopneumonia (1, 3). Para 3 ranks second to respiratory syncytial viruses as the most common cause of lower respiratory tract infections in children. Para 4 rarely causes symptomatic infections in humans.

Parainfluenza is rare among adults but widespread among children, especially males. By age 8, most children demonstrate antibodies to Para 1 and Para 3. Most adults have antibodies to all four types as a result of childhood infections and subsequent multiple exposures. Incidence rises in the winter and spring.

Complications

- Croup
- Bronchiolitis
- Pneumonia

Signs and symptoms

After a short incubation period (usually 3 to 6 days), signs and symptoms emerge that are similar to those of other respiratory diseases: sudden fever, nasal discharge, reddened throat (with little or no exudate), chills, and muscle pain. Bacterial complications are uncommon, but in infants and very young children,

parainfluenza may lead to croup or laryngotracheobronchitis. Reinfection is usually less severe and affects only the upper respiratory tract.

Diagnosis

Parainfluenza infections are usually clinically indistinguishable from similar viral infections. A swab of nasal secretions is useful for rapid viral testing. Isolation of the virus and serum antibody titers differentiate parainfluenza from other respiratory illness but is rarely done.

Treatment

Parainfluenza may require no treatment or bed rest, antipyretics, analgesics, and antitussives, depending on the severity of the symptoms. Complications, such as croup and pneumonia, require appropriate treatment. No vaccine is effective against parainfluenza. Give corticosteroids and nebulization to treat respiratory symptoms and to reduce airway edema.

Special considerations

- Throughout the illness, monitor respiratory status and temperature, and ensure adequate fluid intake and rest.

Adenovirus infection

Adenoviruses cause acute, self-limiting febrile infections, with inflammation of the respiratory or ocular mucous membranes, or both. (See *Major adenovirus infections*, page 978.)

MAJOR ADENOVIRUS INFECTIONS

Disease	Age-group	Clinical features
Acute febrile respiratory illness	Children	Nonspecific coldlike symptoms, similar to other viral respiratory illnesses: fever, pharyngitis, tracheitis, bronchitis, and pneumonitis
Acute respiratory disease	Adults (usually military recruits)	Malaise, fever, chills, headache, pharyngitis, hoarseness, and dry cough
Viral pneumonia	Children and adults	Sudden onset of high fever, rapid infection of upper and lower respiratory tracts, rash, diarrhea, and intestinal intussusception
Acute	Children	Spiking fever lasting several days, headache, pharyngitis,

pharyngoconjunctival fever	(particularly after swimming in pools or lakes)	conjunctivitis, rhinitis, and cervical adenitis
Acute follicular conjunctivitis	Adults	Unilateral tearing and mucoid discharge; later, milder symptoms in other eye
Epidemic keratoconjunctivitis	Adults	Unilateral or bilateral ocular redness and edema, periorbital swelling, local discomfort, and superficial opacity of the cornea without ulceration
Hemorrhagic cystitis	Children (boys)	Adenovirus in urine, hematuria, dysuria, and urinary frequency
Diarrhea	Infants	Fever and watery diarrhea

Causes and incidence

Adenovirus has 35 known serotypes; it causes 5 major infections, all of which occur in epidemics. These organisms are common and can remain latent for years; they infect almost everyone early in life, although maternal antibodies offer some protection during the first 6 months of life.

Transmission of adenovirus can occur by direct inoculation into the eye; by the oral-fecal route (adenovirus may persist in the GI tract for years after infection); or by inhalation of an infected droplet.

Complications

- Acute conjunctivitis
- Sinusitis
- Pharyngitis
- Bronchiolitis
- Pneumonia

Signs and symptoms

The incubation period—usually lasting less than 1 week—is followed by acute illness lasting less than 5 days. Clinical features vary, depending on the type of infection. Prolonged asymptomatic reinfection may occur.

Diagnosis

CONFIRMING DIAGNOSIS

Definitive diagnosis requires isolation of the virus from respiratory or ocular secretions or fecal smears; during epidemics, however, typical symptoms alone can confirm the diagnosis.

Because adenoviral illnesses resolve rapidly, serum antibody titers aren't useful for diagnosis. Adenoviral diseases cause lymphocytosis in children. When they cause respiratory disease, chest X-ray may show pneumonitis.

Treatment

Supportive treatment includes bed rest, antipyretics, and analgesics. Ocular infections may require corticosteroids and direct supervision by an ophthalmologist. Hospitalization is required in cases of pneumonia (in infants) to prevent death and in epidemic keratoconjunctivitis (EKC) to prevent blindness.

Special considerations

- During acute illness, monitor respiratory status and intake and output. Give analgesics and antipyretics, as needed. Stress the need for bed rest.
- To help minimize the incidence of adenoviral disease, instruct all patients in proper hand washing to reduce fecal-oral transmission and eye inoculation.
- EKC can be prevented by sterilization of ophthalmic instruments, adequate chlorination in swimming pools, and avoidance of swimming pools during epidemics. Killed virus vaccine (not widely available) or a live oral virus vaccine can prevent adenoviral infection and are recommended for high-risk groups.

Influenza

Influenza (also called the *grippe* or the *flu*), an acute, highly contagious infection of the respiratory tract, results from three different types of *Myxovirus influenzae*. It occurs sporadically or in epidemics (usually during the colder months). Epidemics tend to peak within 2 to 3 weeks after initial cases and subside within a month.

Although influenza affects all agegroups, its incidence is highest in schoolchildren. However, its effects are most severe in persons who are young, elderly, or suffering from chronic disease. In these groups, influenza may even lead to death. The catastrophic pandemic of 1918 was responsible for an estimated 20 million deaths. The most recent pandemics (in 1957, 1968, and 1977) began in mainland China.

Causes and incidence

Transmission of influenza occurs through inhalation of a respiratory droplet from an infected person or by indirect contact with a contaminated object, such as a drinking glass or other items contaminated with respiratory secretions. The influenza virus then invades the epithelium of the respiratory tract, causing inflammation and desquamation. (See *How influenza viruses multiply,* page 980.)

One of the remarkable features of the influenza virus is its capacity for *antigenic variation* into numerous distinct strains, allowing it to infect new populations that have little or no immunologic resistance. Antigenic variation is characterized as *antigenic drift* (minor changes that occur yearly or every few years) and *antigenic shift* (major changes that lead to pandemics). Influenza viruses are classified into three groups:

- Type A, the most prevalent, strikes every year, with new serotypes causing epidemics every 3 years.
- Type B also strikes annually but causes epidemics only every 4 to 6 years.
- Type C is endemic and causes only sporadic cases.

Each year, tens of millions of people in the United States get the flu; about 114,000 people get sick enough to be hospitalized, and about 36,000 people die.

Complications

- Pneumonia
- Myositis
- Exacerbation of chronic obstructive pulmonary disease
- Reye's syndrome
- Myocarditis (rare)
- Pericarditis (rare)
- Transverse myelitis (rare)
- encephalitis (rare)

Signs and symptoms

After an incubation period of 24 to 48 hours, flu symptoms begin to appear: sudden onset of chills, temperature of 101° to 104° F (38.3° to 40° C), headache, malaise, myalgia (particularly in the back

and limbs), a nonproductive cough and, occasionally, laryngitis, hoarseness,

conjunctivitis, rhinitis, and rhinorrhea. These symptoms usually subside in 3 to 5 days, but cough and weakness may persist. Fever is usually higher in children than in adults. Also, cervical adenopathy and croup are likely to be associated with influenza in children. In some patients (especially elderly patients), lack of energy and easy fatigability may persist for several weeks.

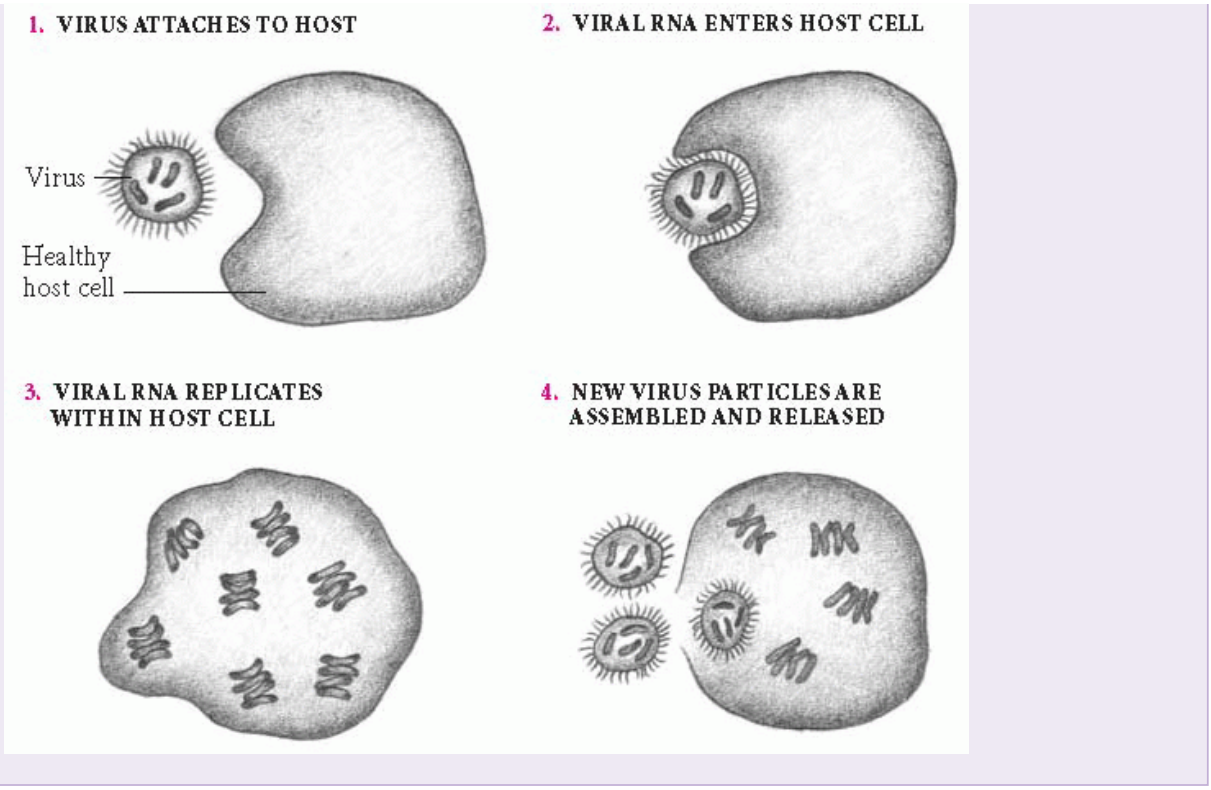
PATHOPHYSIOLOGY

HOW INFLUENZA VIRUSES MULTIPLY

An influenza virus, classified as type A, B, or C, contains the genetic material ribonucleic acid (RNA), which is covered and protected by protein. RNA is arranged in genes that carry the instruction for viral replication. This genetic material has an extraordinary ability to mutate, causing the generation of new serologically distinct strains of influenza virus. Being a virus, the pathogen can't reproduce or carry out chemical reactions on its own. It needs a host cell.

After attaching to the host cell, the viral RNA enters the host cell and uses host components to replicate its genetic material and protein, which are then assembled into the new virus particles. These newly produced viruses can burst forth to invade other healthy cells.

The viral invasion destroys the host cells, impairing respiratory defenses, especially the mucociliary transport system, and predisposing the patient to secondary bacterial infection.



Fever that persists longer than 3 to 5 days signals the onset of complications. The most common complication is pneumonia, which occurs as primary influenza virus pneumonia or secondary to bacterial infection. Influenza may also cause myositis, exacerbation of chronic obstructive pulmonary disease, Reye's syndrome and, rarely, myocarditis, pericarditis, transverse myelitis, and encephalitis.

Diagnosis

At the beginning of an influenza epidemic, early cases are usually mistaken for other respiratory disorders.

CONFIRMING DIAGNOSIS

Because signs and symptoms of influenza aren't pathognomonic, isolation of M. influenzae *through nose and throat cultures and increased serum antibody titers help confirm this diagnosis. Also, rapid diagnostic methods for detecting influenza are now available and help confirm this diagnosis.*

After these measures confirm an influenza epidemic, diagnosis requires only observation of clinical signs and symptoms. Uncomplicated cases show a decreased white blood cell count with an increase in lymphocytes.

Treatment

Treatment of uncomplicated influenza includes bed rest, adequate fluid intake, aspirin or acetaminophen (in children) to relieve fever and muscle pain, and dextromethorphan or another antitussive to relieve nonproductive coughing. Prophylactic antibiotics aren't recommended because they have no effect on the influenza virus.

Amantadine and rimantadine (antiviral agents) have proven to be effective in reducing the duration of signs and symptoms of influenza A infection. Oseltamivir and zanamivir are effective against influenza A and B infection. In influenza complicated by pneumonia, supportive care (fluid and electrolyte supplements, oxygen, and assisted ventilation) and treatment of bacterial superinfection with appropriate antibiotics are necessary. No specific therapy exists for cardiac, central nervous system, or other complications.

Special considerations

Unless complications occur, influenza doesn't require hospitalization; patient care focuses on relief of symptoms:

- Advise the patient to increase his fluid intake. Warm baths or heating pads may relieve myalgia. Give him nonopioid analgesics-antipyretics as ordered.
- Screen visitors to protect the patient from bacterial infection and the visitors from influenza. Use droplet precautions.
- Teach the patient proper disposal of tissues and proper hand-washing technique to prevent the virus from spreading.
- Watch for signs and symptoms of developing pneumonia, such as crackles, another temperature rise, or coughing accompanied by purulent or bloody sputum. Assist the patient to gradually resume his normal activities.
- Educate patients about influenza immunizations. For high-risk patients and health care personnel, suggest annual inoculations at the start of the flu season (late autumn). Remember, however, that such vaccines are made from chicken embryos and must not be given to people who are hypersensitive to eggs. (For people who are hypersensitive to eggs, amantadine is an effective alternative to the vaccine; however it must be started before the flu season and continued throughout the season.) The vaccine administered is based on the previous year's virus and is usually about 75% effective.
- Inform people receiving the vaccine of possible adverse effects (discomfort at the vaccination site, fever, malaise and, rarely, Guillain-Barré syndrome). Influenza vaccine (inactivated) is recommended for women who are pregnant and who will be in the second or third trimester during influenza season.

- Live-attenuated influenza vaccine is now available as a nasal spray. Criteria and contraindications for use vary from the inactivated, injectable vaccine. Recipients of live-attenuated influenza vaccine may shed influenza virus for up to 21 days post-immunization.

Hantavirus pulmonary syndrome

Mainly occurring in the southwestern United States, but not confined to that area, *Hantavirus* pulmonary syndrome is a viral disease first reported in May 1993. The syndrome, which rapidly progresses from flulike symptoms to respiratory failure and, possibly, death, is known for its high mortality. The hantavirus strain that causes disease in Asia and Europe—mainly hemorrhagic fever and renal disease—is distinctly different from the one currently described in North America.

Causes and incidence

A member of the Bunyaviridae family, the genus *Hantavirus* (first isolated in 1977) is responsible for *Hantavirus* pulmonary syndrome. Disease transmission is associated with exposure to aerosols (such as dust) contaminated by urine or feces from infected rodents, the primary reservoir for this virus. Data suggest that the deer mouse is the main source, but pinon mice, brush mice, and western chipmunks in close proximity to humans in rural areas are also sources. Hantavirus infections have been documented in people whose activities are associated with rodent contact, such as farming, hiking or camping in rodentinfested areas, and occupying rodentinfested dwellings.

Infected rodents manifest no apparent illness but shed the virus in feces, urine, and saliva. Human infection may occur from inhalation, ingestion (of contaminated food or water, for example), contact with rodent excrement, or rodent bites. Transmission from person to person or by mosquitoes, fleas, or other arthropods hasn't been reported.

Complications

- Respiratory failure
- Death

Signs and symptoms

Noncardiogenic pulmonary edema distinguishes the syndrome. Common chief complaints include myalgia, fever, headache, nausea, vomiting, and cough. Respiratory distress typically follows the onset of a cough. Fever, hypoxia and, in some patients, serious hypotension typify its course.

Other signs and symptoms include a rising respiratory rate (28 breaths/minute or more) and an increased heart rate (120 beats/minute or more).

Diagnosis

Despite ongoing efforts to identify clinical and laboratory features that distinguish *Hantavirus* pulmonary syndrome from other infections with similar features, diagnosis currently is based on clinical suspicion along with a process of elimination developed by the Centers for Disease Control and Prevention (CDC) with the Council of State and Territorial Epidemiologists. (See *Screening for* Hantavirus *pulmonary syndrome*.) Serologic testing for hantavirus can be performed.

Laboratory tests usually reveal an elevated white blood cell count with a predominance of neutrophils, myeloid precursors, and atypical lymphocytes; elevated hematocrit; decreased platelet count; elevated partial thromboplastin time; and a normal fibrinogen level. Usually, laboratory findings demonstrate only minimal abnormalities in renal function, with serum creatinine levels no higher than 2.5 mg/dl.

Chest X-rays eventually show bilateral diffuse infiltrates in almost all patients (findings consistent with acute respiratory distress syndrome).

Treatment

Primarily supportive, treatment consists of maintaining adequate oxygenation, monitoring vital signs, and intervening to stabilize the patient's heart rate and blood pressure.

Drug therapy includes vasopressors, such as dopamine or epinephrine, for hypotension. Fluid volume replacement may also be ordered (with precautions not to overhydrate the patient). Ribavirin in aerosol form has been used for children, but its efficacy for adults has not been proven.

Special considerations

- Assess the patient's respiratory status and arterial blood gas values often.
- Monitor serum electrolyte levels and correct imbalances as appropriate.
- Maintain a patent airway by suctioning. Ensure adequate humidification, and check ventilator settings frequently.
- In patients with hypoxemia, assess neurologic status frequently along with heart rate and blood pressure.

SCREENING FOR *HANTAVIRUS* PULMONARY SYNDROME

The Centers for Disease Control and Prevention (CDC) has developed a screening procedure to track cases of *Hantavirus* pulmonary syndrome. The screening criteria identify potential and actual cases.

Potential cases

For a diagnosis of possible *Hantavirus* pulmonary syndrome, a patient must have one of the following:

- febrile illness (temperature equal to or above 101° F [38.3° C]) occurring in a previously healthy person and characterized by unexplained acute respiratory distress syndrome
- bilateral interstitial pulmonary infiltrates that develop within 1 week of hospitalization with respiratory compromise that requires supplemental oxygen
- an unexplained respiratory illness resulting in death and autopsy findings demonstrating noncardiogenic pulmonary edema without an identifiable specific cause of death.

Exclusions

Of the patients who meet the criteria for having potential *Hantavirus* pulmonary syndrome, the CDC excludes those who have any of the following:

- a predisposing underlying medical condition (for example, severe underlying pulmonary disease), solid tumors or hematologic cancers, congenital or acquired immunodeficiency disorders, or medical conditions or treatments— such as rheumatoid arthritis or organ transplantation—requiring immunosuppressive drug therapy (for example, steroids or cytotoxic chemotherapy)
- an acute illness that provides a likely explanation for the respiratory illness (for example, a recent major trauma, burn, or surgery; recent seizures or history of aspiration; bacterial sepsis; another respiratory disorder such as respiratory syncytial virus in young children; influenza; or legionella pneumonia).

Confirmed cases

Cases of confirmed *Hantavirus* pulmonary syndrome must include the following:

- at least one serum or tissue specimen available for laboratory testing for evidence of hantavirus infection
- in a patient with a compatible clinical illness, serologic evidence (presence of hantavirus-specific immunoglobulin [Ig] M or rising titers of IgG), polymerase chain reaction for hantavirus ribonucleic acid, or positive immunohistochemistry for hantavirus antigen.

- Administer drug therapy, and monitor the patient's response.
- Provide I.V. fluid therapy based on results of hemodynamic monitoring.
- Provide emotional support for the patient and his family.
- Report cases of *Hantavirus* pulmonary syndrome to the appropriate state health department.
- Provide the patient with prevention guidelines. (Until more is known about *Hantavirus* pulmonary syndrome, preventive measures currently focus on rodent control.)

Rotavirus

Rotavirus is the most common cause of severe diarrhea among children. The disease is characterized by vomiting and watery diarrhea for 3 to 8 days, commonly with fever and abdominal pain.

In the United States and other countries with a temperate climate, the disease has a winter seasonal pattern, with annual epidemics occurring from November to April. The illness occurs most often in infants and young children; most children in the United States are infected by age 2. Rotavirus is responsible for the hospitalization

of about 55,000 children each year in the United States and for the death of more than 600,000 children annually worldwide.

Causes and incidence

The primary mode of transmission is fecal-oral, although some have reported low titers of virus in respiratory tract secretions and other body fluids. Because of the endurance of the virus in the environment, transmission may occur through ingestion of contaminated water or food and contact with contaminated surfaces.

Billions of rotavirus particles are passed in the stool of the infected individual. Small numbers of the rotavirus may lead to infection if a baby puts fingers or other

objects contaminated with the virus into the mouth. Young children can pass it on to siblings and parents.

Immunity after infection is incomplete, but recurrent infections tend to be less severe than the original infection.

Complications

- Severe dehydration
- Shock
- Skin breakdown

Signs and symptoms

The incubation period for rotavirus disease is about 2 days. Rotavirus gastroenteritis commonly starts with a fever, nausea, and vomiting, followed by diarrhea. The illness can range from mild to severe and last from 3 to 9 days. Diarrhea and vomiting may cause dehydration.

Diagnosis

The diagnosis is determined by rapid antigen detection of rotavirus in stool specimens.

Rotavirus is the most common diagnosis for young children with acute diarrhea, but other causes may include bacteria (*Salmonella, Shigella,* and *Campylobacter* are the most common), parasites (*Giardia* and *Cryptosporidium* are the most common), localized infection elsewhere, antibiotic-associated adverse effects (such as those related to treatment for *Clostridium difficile*), and food poisoning. Noninfectious causes include overfeeding (particularly of fruit juices), irritable bowel syndrome, celiac disease, milk protein intolerance, lactose intolerance, cystic fibrosis, and inflammatory bowel syndrome.

Treatment

For a person with a healthy immune system, rotavirus gastroenteritis is a self-limited illness, lasting only days. Treatment is nonspecific and consists of oral rehydration therapy to prevent dehydration.

Special considerations

- Enforce strict hand-washing and careful cleaning of all equipment, including the child's toys to prevent the spread of rotavirus.
- Implement contact precautions.

- Help the patient maintain adequate hydration. Remember that dehydration occurs rapidly in infants and young children. Ice pops, gelatin, and ice chips may be included in the diet to maintain hydration.
- Breast-fed infants should continue to nurse without restrictions. Lactose-free soybean formulas may be used for infants who are bottle-fed.
- Carefully monitor intake and output (including stools).
- Clean the perineum thoroughly to prevent skin breakdown.
- Instruct parents about proper hand-washing techniques for themselves and the infant. Provide instructions about diaper changing and cleaning all affected surfaces.
- Teach parents and caregivers how to measure intake and output. Tell them to notify their physician about any increased diarrhea or dehydration.

Avian influenza

Avian influenza (flu) mainly infects birds, but is of concern to humans, who have no immunity against it. The virus that causes this infection in birds can mutate and easily infect humans and potentially start a deadly worldwide epidemic. The first avian flu virus to infect humans directly occurred in Hong Kong in 1997 and has since spread across Asia. In October 2005 it was discovered in Turkey and Romania. About 161

people have been infected by H5N1 and the current death rate with confirmed infection is more than 50%. The H7N7 avian flu outbreak in the Netherlands resulted in 89 confirmed cases but only one death; an avian flu virus designated H9N2 infected 3 children in Asia and all three recovered. Prognosis depends on the severity of infection as well as the type of avian flu virus that caused it.

Causes and incidence

Avian influenza A (H5N1) virus is commonly referred to as *bird flu virus*. Highly infective avian flu viruses, such as H5N1, have been shown to survive in the environment for long periods of time, and infection may be spread simply by touching contaminated surfaces. Birds who recover from flu can continue to shed the virus in their feces and saliva for as long as 10 days.

Those at risk include:

- Farmers and other people working with poultry
- Travelers visiting affected countries
- People who handle infected birds
- People who eat raw or undercooked poultry meat

- Health care workers and others in contact with patients who have avian flu

Complications

- Conjunctivitis
- Pneumonia
- Acute respiratory distress
- Viral pneumonia
- Sepsis
- Organ failure

Signs and symptoms

Symptoms of avian flu infection in humans depend on the particular strain of virus. In case of the H5N1 virus, infection causes more classic flulike symptoms, which might include headache, malaise, dry or productive cough, sore throat, fever greater than 100.4°F (38°C), runny nose, difficulty breathing, diarrhea, and muscle aches.

Diagnosis

CONFIRMING DIAGNOSIS

Influenza A/H5 (Asian lineage) Virus Real-time RT-PCR Primer and Probe Set. The test gives preliminary results within 4 hours; older tests required 2 to 3 days.

Chest X-ray, nasopharyngeal culture, and blood differential can also aid in diagnosis.

Treatment

Antiviral medication oseltamivir (Tamiflu), and perhaps zanamivir (Relenza), may decrease the severity of the disease, if started within 48 hours after symptoms begin. Oseltamivir may also be prescribed for household contacts of people diagnosed with avian flu. Provide supportive treatment with mechanical ventilation, I.V. fluids, and symptomatic treatment.

It's currently recommended that people diagnosed with H5N1 infection be put in isolation.

Currently, there's no available vaccine against avian flu. However, a vaccine against H5N1 is being tested in clinical trials.

A flu shot may be administered to reduce the chance of an avian flu virus mixing with a human flu virus, which would create a new virus that may easily spread.

Special considerations

- Tell patients to call their health care provider if they develop flulike symptoms within 10 days of handling infected birds or traveling to an area with a known avian flu outbreak.
- Travelers should avoid visits to live-bird markets in areas with an avian flu outbreak.
- Those who work with birds who might be infected should use protective clothing and special breathing masks.
- Watch for signs and symptoms of complications.

PREVENTION

- *Tell the patient that avoiding undercooked or uncooked meat reduces the risk of exposure to avian flu and other food borne diseases.*
- *Teach the patient about proper disposal of tissues and proper hand-washing technique.*

RASH-PRODUCING VIRUSES

Varicella

Varicella, commonly known as *chickenpox*, is a common, acute, and very contagious infection caused by the herpesvirus varicella-zoster, the same virus that, in its latent stage, causes herpes zoster (shingles).

Causes and incidence

Chickenpox can occur at any age, but it's most common in children ages 2 to 8. Congenital varicella may affect infants whose mothers had acute infections in their first or early second trimester. Neonatal infection is rare, probably because of transient maternal immunity. However, neonates born to mothers who develop varicella 5 days before delivery or up to 2 days after delivery are at risk for developing severe generalized varicella. Second attacks are also rare. This infection is transmitted by direct contact (primarily with respiratory secretions; less commonly, with skin lesions) and indirect contact (airborne). The incubation period usually lasts 14 to 17 days but can be as short as 10 days and as long as 20 days. (See *Incubation and duration of common rash-producing infections.*)

Chickenpox is probably communicable from 1 day before lesions erupt to 6 days after vesicles form (it's most contagious in the early stages of eruption of skin lesions).

Chickenpox occurs worldwide and is endemic in large cities. Outbreaks occur sporadically, usually in areas with large groups of susceptible children. It affects all races and both sexes equally. Seasonal distribution varies; in temperate areas, incidence is higher during late autumn, winter, and spring.

Most children recover completely. Potentially fatal complications may affect children on corticosteroids, antimetabolites, or other immunosuppressants and those with leukemia, other neoplasms, or immunodeficiency disorders. Congenital and adult varicella may also have severe effects.

Complications

- Pneumonia
- Encephalitis

Signs and symptoms

Chickenpox produces distinctive signs and symptoms, notably a pruritic rash. During the prodromal phase, the patient has slight fever, malaise, and anorexia. Within 24 hours, the rash typically begins as crops of small, erythematous macules on the trunk or scalp. It progress to papules and then clear vesicles on an erythematous base (the so-called dewdrop on a rose petal). These become cloudy and break easily; then scabs form.

The rash spreads to the face and over the trunk of the body, then to the limbs, buccal mucosa, axillae, upper respiratory tract, conjunctivae and, occasionally, the genitalia. New vesicles continue to appear for 3 or 4 days, so the rash contains a combination of red papules, vesicles, and scabs in various stages.

Congenital varicella causes hypoplastic deformity and limb scarring; retarded growth; and central nervous system and eye manifestations. In progressive varicella, an immunocompromised patient may have lesions and a high fever for over 7 days.

Severe pruritus with this rash may provoke persistent scratching, which can lead to infection, scarring, impetigo, furuncles, and cellulitis. Rare complications include pneumonia, myocarditis, fulminating encephalitis (Reye's syndrome), bleeding disorders, arthritis, nephritis, hepatitis, and acute myositis.

Diagnosis

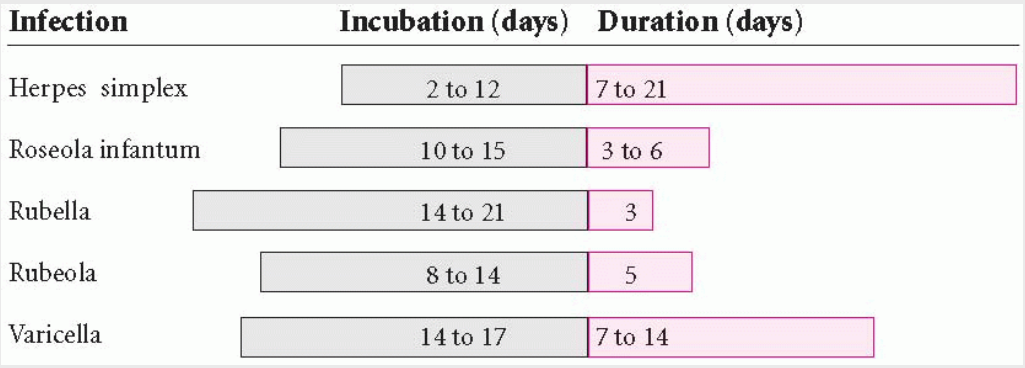
Diagnosis rests on the characteristic clinical signs and usually doesn't require laboratory tests. However, the virus can be isolated from vesicular fluid within the

first 3 or 4 days of the rash; Giemsa stain distinguishes varicella-zoster from vaccinia and variola viruses. Serum contains antibodies 7 days after onset.

Treatment

Chickenpox calls for droplet and contact isolation until all vesicles and most of the scabs are dry (no new lesions; usually 1 week after the onset of the rash). Children with only a few remaining scabs are no longer contagious and can return to school. Congenital chickenpox requires no isolation.

INCUBATION AND DURATION OF COMMON RASH-PRODUCING INFECTIONS



In most cases, treatment consists of local or systemic antipruritics: lukewarm oatmeal baths, calamine lotion, or diphenhydramine (or another antihistamine). Antibiotics are unnecessary unless bacterial infection develops. Salicylates are contraindicated because of their link with Reye's syndrome.

Susceptible patients may need special treatment. When given up to 72 hours after exposure to varicella, varicella-zoster immunoglobulin may provide passive immunity. Acyclovir and famciclovir, antiviral agents, may slow vesicle formation, speed skin healing, and control the systemic spread of infection.

Special considerations

Care is supportive and emphasizes patient and family teaching and preventive measures.

- Teach the child and his family how to apply topical antipruritic medications correctly. Stress the importance of good hygiene.
- Tell the patient not to scratch the lesions. However, because the need to scratch may be overwhelming, parents should trim the child's fingernails or tie mittens on his hands.

- Warn parents to watch for and immediately report signs of complications. Severe skin pain and burning may indicate a serious secondary infection and require prompt medical attention.

PREVENTION

- *Varicella vaccine, part of the recommended childhood immunization schedule, effectively prevents infection. It's also effective if given 5 days post-exposure.*
- *To help prevent chickenpox, don't admit a child exposed to chickenpox to a unit that contains children who receive immunosuppressants or who have leukemia or immunodeficiency disorders. A vulnerable child who has been exposed to chickenpox should be evaluated for administration of varicella-zoster immunoglobulin to lessen the severity of the disease.*

Herpes simplex

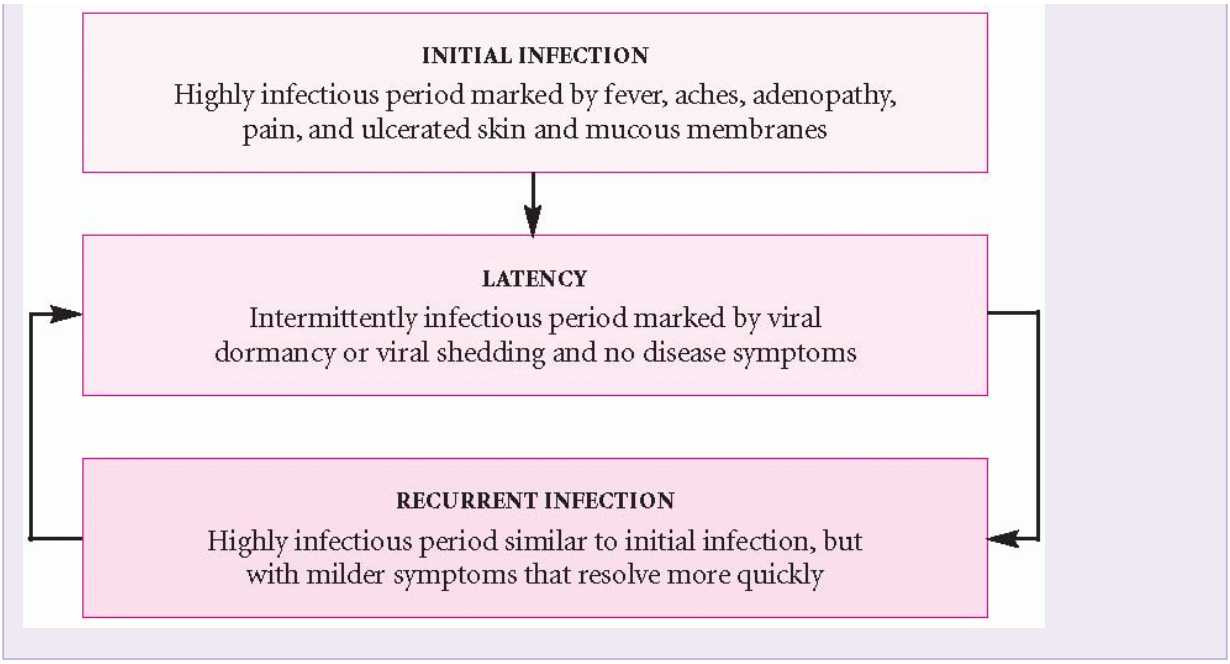
Herpes simplex, a recurrent viral infection, is caused by *Herpesvirus hominis* (HVH), a widespread infectious agent. Herpes type I, which is transmitted by oral and respiratory secretions, affects the skin and mucous membranes, commonly producing cold sores and fever blisters. Herpes type II primarily affects the genital area and is transmitted by sexual contact. However, cross-infection may result from orogenital sex or autoinoculation from one site to another. (See *Understanding the genital herpes cycle*, page 988.)

PATHOPHYSIOLOGY

UNDERSTANDING THE GENITAL HERPES CYCLE

After a patient is infected with genital herpes, a latency period follows. The virus resides permanently in the nerve cells surrounding the lesions, and intermittent viral shedding may take place.

Repeated outbreaks may develop at any time, again followed by a latent stage during which the lesions heal completely. Outbreaks may recur as often as three to eight times per year. Although the cycle continues indefinitely, some people remain asymptomatic for years.



Causes and incidence

About 85% of all HVH infections are subclinical; the others produce localized lesions and systemic reactions. After the first infection, a patient is a carrier susceptible to recurrent infections, which may be provoked by fever, menses, stress, heat, and cold. However, the patient usually has no constitutional signs and symptoms in recurrent infections.

Primary HVH is the leading cause of childhood gingivostomatitis in children ages 1 to 3. It causes the most common form of nonepidemic encephalitis and is the second most common viral infection in pregnant women. It can pass to the fetus transplacentally and, in early pregnancy, may cause spontaneous abortion or premature birth. (See *Recognizing herpetic whitlow.*)

Herpes infection is equally common in males and females. Worldwide in distribution, it's most prevalent among children in lower socioeconomic groups who live in crowded environments. Saliva, stool, skin lesions, purulent eye exudate, and urine are potential sources of infection.

Complications

- Abortion
- Premature labor
- Microcephaly
- Uterine growth retardation
- Increased risk for cervical cancer

- Perianal ulcers
- Colitis
- Esophagitis
- Pneumonitis

Signs and symptoms

In neonates, HVH symptoms usually appear 1 to 2 weeks after birth. They range from localized skin lesions to a disseminated infection of organs, such as the liver, lungs, or brain. Common complications include seizures, mental retardation, blindness, chorioretinitis, deafness, microcephaly, diabetes insipidus, and spasticity. Up to 90% of infants with disseminated disease die.

Primary infection in childhood may be localized or generalized and occurs after an incubation period of 2 to 12 days. After brief prodromal tingling and itching, localized infection causes typical primary lesions. These erupt as vesicles on an erythematous base, eventually rupture and leave a painful ulcer, followed by a yellowish crust. Vesicles may form on any part of the oral mucosa, especially the tongue, gingiva, and cheeks. Healing begins 7 to 10 days after onset and is complete in 3 weeks.

Generalized infection begins with fever, pharyngitis, erythema, and edema. Vesicles occur with submaxillary lymphadenopathy, increased salivation, halitosis, anorexia, and a fever of up to 105° F (40.6° C). Herpetic stomatitis may lead to severe dehydration in children. A generalized infection usually runs its course in 4 to 10 days. In this form, virus reactivation causes cold sores—a single or group of vesicles in and around the mouth.

Genital herpes usually affects adolescents and young adults. Typically painful, the initial attack produces fluid-filled vesicles that ulcerate and heal in 1 to 3 weeks. Fever, regional lymphadenopathy, and dysuria may also occur.

Usually, herpetic keratoconjunctivitis is unilateral and causes only local signs and symptoms: conjunctivitis, regional adenopathy, blepharitis, and vesicles on the lid. Other ocular effects may include excessive lacrimation, edema, chemosis, photophobia, and purulent exudate.

Both types of HVH can cause acute sporadic encephalitis with altered level of consciousness, personality changes, and seizures. Other effects may include smell and taste hallucinations and neurologic abnormalities such as aphasia.

RECOGNIZING HERPETIC WHITLOW

Herpetic whitlow is a finger infection caused by the microorganism that causes herpes simplex virus (HSV). It

commonly affects nurses, usually only in one finger. Typical signs and symptoms in the affected finger begin with tingling followed by:

- pain, redness, and swelling
- vesicular eruptions bordered by red halos
- vesicular ulceration or coales-cence—related effects include satellite vesicles, fever, chills, malaise, and a red streak up the arm.

Healing occurs in 2 to 3 weeks. Health care workers with herpetic whitlow must be restricted from patient care. In health care workers, the infecting organism usually is HSV-1. In others, the infection usually is secondary to HSV-2 infection.

Herpetic whitlow, an HVH finger infection, affects many nurses. First the finger tingles and then it becomes red, swollen, and painful. Vesicles with a red halo erupt and may ulcerate or coalesce. Other effects may include satellite vesicles, fever, chills, malaise, and a red streak up the arm.

Diagnosis

CONFIRMING DIAGNOSIS

Typical lesions may suggest HVH infection. However, confirmation requires isolation of the virus from local lesions.

A rise in antibodies and moderate leukocytosis may support the diagnosis.

Treatment

No cure for herpes exists; however, recurrences tend to be milder and of shorter duration than the primary infection. Symptomatic and supportive therapy is essential. Generalized primary infection usually requires an analgesic-antipyretic to reduce fever and relieve pain. Anesthetic mouth-washes,

such as viscous lidocaine, may reduce the pain of gingivostomatitis, enabling the patient to eat and preventing dehydration. (Avoid alcohol-based mouthwashes.) Drying agents, such as calamine lotion, ease the pain of labial or skin lesions. Avoid petroleum-based ointments, which promote viral spread and slow healing.

Refer patients with eye infections to an ophthalmologist. Topical corticosteroids are contraindicated in active infection, but idoxuridine, trifluridine, and vidarabine are effective.

Oral acyclovir may bring relief to patients with genital herpes. Frequent prophylactic use of acyclovir in immunosuppressed transplant patients prevents disseminated disease. Foscarnet can be used to treat HVH that's resistant to acyclovir. Anti-viral agents similar to acyclovir are valacyclovir and famciclovir. These agents are more active than acyclovir.

Special considerations

- Teach the patient with genital herpes to use warm compresses or take sitz baths several times per day; to use a drying agent, such as povidone-iodine solution; to increase fluid intake; and to avoid all sexual contact during the active stage.
- For pregnant women with active HVH infection at the time of delivery, a cesarean delivery is recommended to decrease the risk of infecting the neonate.
- Health care personnel should use standard precautions, such as gloves, for contact with mucous membranes to prevent acquisition of herpetic whitlow.
- Instruct patients with herpetic whitlow not to share towels or eating utensils. Educate staff members and other susceptible people about the risk of contagion. Abstain from direct patient care if you have herpetic whitlow.
- Tell patients with cold sores not to kiss people. (Those with genital herpes pose no risk to infants if their hygiene is meticulous.)
- Patients with central nervous system infection alone need no isolation.

Herpes zoster

Herpes zoster (also called *shingles*) is an acute unilateral and segmental inflammation of the dorsal root ganglia caused by infection with the herpesvirus varicella-zoster, which also causes chickenpox. This infection usually occurs in adults. It produces localized vesicular skin lesions, confined to a dermatome, and severe neuralgic pain in peripheral areas innervated by the nerves arising in the inflamed root ganglia.

The prognosis is good unless the infection spreads to the brain. Eventually, most patients recover completely, except for possible scarring and, in corneal damage, visual impairment. Occasionally, neuralgia may persist for months or years.

Causes and incidence

Herpes zoster results from reactivation of varicella virus that has lain dormant in the cerebral ganglia (extramedullary ganglia of the cranial nerves) or the ganglia of posterior nerve roots since a previous episode of chickenpox. Exactly how or why this reactivation occurs isn't clear. Some believe that the virus multiplies as it's reactivated and that antibodies remaining from the initial infection neutralize it. However, if effective antibodies aren't present, the virus continues to multiply

in the ganglia, destroy the host neuron, and spread down the sensory nerves to the skin.

Herpes zoster occurs primarily in adults, especially those older than age 50. It seldom recurs. It's also seen in patients with human immunodeficiency virus and other immunodeficiency disorders.

Signs and symptoms

Herpes zoster begins with fever and malaise. Within 2 to 4 days, severe deep pain, pruritus, and paresthesia or hyperesthesia develop, usually on the trunk and occasionally on the arms and legs in a dermatomal distribution. Pain may be continuous or intermittent and usually lasts from 1 to 4 weeks. Up to 2 weeks after the first symptoms, small red nodular skin lesions erupt on the painful areas. (These lesions typically spread unilaterally around the thorax or vertically over the arms or legs.) Sometimes nodules don't appear at all, but

when they do, they quickly become vesicles filled with clear fluid or pus. About 10 days after they appear, the vesicles dry and form scabs. (See *Recognizing shingles.*) When ruptured, such lesions usually become infected and, in severe cases, may lead to the enlargement of regional lymph nodes; they may even become gangrenous. Intense pain may occur before the rash appears and after the scabs form.

Occasionally, herpes zoster involves the cranial nerves, especially the trigeminal and geniculate ganglia or the oculomotor nerve. Geniculate zoster may cause vesicle formation in the external auditory canal, ipsilateral facial palsy, hearing loss, dizziness, and loss of taste. Trigeminal ganglion involvement causes eye pain and, possibly, corneal and scleral damage and impaired vision. Rarely, oculomotor involvement causes conjunctivitis, extraocular weakness, ptosis, and paralytic mydriasis.

In rare cases, herpes zoster leads to generalized central nervous system infection, muscle atrophy, motor paralysis (usually transient), acute transverse myelitis, and ascending myelitis. More commonly, generalized infection causes acute urine retention and unilateral diaphragm paralysis. In postherpetic neuralgia, most common in elderly persons, intractable neurologic pain may persist for years. Scars may be permanent.

Patients with immunodeficiency disorders may develop disseminated zoster. Lesions are bilateral and not limited to dermatomal distribution.

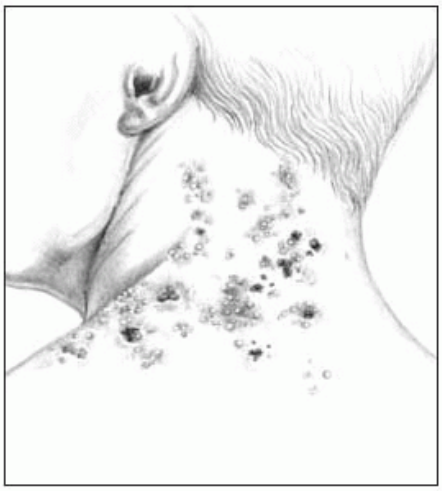
Diagnosis

Diagnosis of herpes zoster usually isn't possible until the characteristic skin lesions develop. Before then, the pain may mimic that of appendicitis, pleurisy, or other

conditions. Individuals who are susceptible to varicella may develop a varicella infection following exposure to patients with zoster. Examination of vesicular fluid and infected tissue shows eosinophilic intranuclear inclusions and varicella virus. Also, a lumbar puncture shows increased pressure; examination of cerebrospinal fluid shows increased protein levels and, possibly, pleocytosis. Differentiation of herpes zoster from localized herpes simplex requires staining antibodies from vesicular fluid and identification under fluorescent light.

RECOGNIZING SHINGLES

The characteristic skin lesions in herpes zoster (shingles) are fluidfilled vesicles that dry and form scabs after about 10 days.



Treatment

Antiviral therapy is the mainstay of treatment. Acyclovir seems to stop the rash's progression and prevent visceral complications. Capsaicin, transcutaneous electrical nerve stimulation, and low-dose amitriptyline are the current treatments of choice for postherpetic neuralgia. Topical antiviral ointment is helpful if started early in the disease process.

Herpes zoster can resolve spontaneously and may only require symptomatic treatment, the goal of which is to relieve itching and neuralgic pain with calamine lotion or another antipruritic; aspirin, possibly with codeine or another analgesic; and, occasionally, collodion or compound benzoin tincture applied to unbroken lesions.

If bacteria have infected ruptured vesicles, the treatment plan usually includes an appropriate systemic antibiotic.

Trigeminal zoster with corneal involvement calls for instillation of idoxuridine ointment or another antiviral agent. To help a patient cope with the intractable

pain of postherpetic neuralgia, the physician may order systemic corticosteroids—such as cortisone or possibly corticotropin—to reduce inflammation (although their use is controversial). He also may prescribe tranquilizers, sedatives, or tricyclic antidepressants with phenothiazines. In some immunocompromised patients— both children and adults—acyclovir I.V. appears to prevent disseminated, life-threatening disease. High doses of interferon (an antiviral glycoprotein) have been used in patients with cancer when the herpetic lesions are limited to the dermatome.

Special considerations

Your care plan should emphasize keeping the patient comfortable, maintaining meticulous hygiene, and preventing further infection. During the acute phase, adequate rest and supportive care can promote proper healing of lesions.

- If calamine lotion has been ordered, apply it liberally to the lesions. If lesions are severe and widespread, apply a wet dressing. Drying therapies, such as oxygen or air-loss bed, and Silvadene ointment, may also be used.
- Instruct the patient to avoid scratching the lesions.
- If vesicles rupture, apply a cold compress as ordered.
- To decrease the pain of oral lesions, tell the patient to use a soft toothbrush, eat soft foods, and use a saline or bicarbonate mouthwash.
- To minimize neuralgic pain, never withhold or delay administration of analgesics. Give them exactly on schedule because the pain of herpes zoster can be severe. In postherpetic neuralgia, consult a pain specialist to maximize pain relief without risking tolerance to the analgesic.
- Repeatedly reassure the patient that herpetic pain will eventually subside. Encourage diversionary or relaxation activity.
- Institute droplet and contact precautions. Disseminated zoster requires the same isolation precautions as primary varicella.

PREVENTION

Vaccination with Zostavax is recommended to prevent the occurrence of shingles in adults over the age of 60.

Rubella

Rubella, commonly called *German measles,* is an acute, mildly contagious viral disease that produces a distinctive 3-day rash and lymphadenopathy. It usually occurs among children ages 5 to 9, adolescents, and young adults. Rubella

flourishes worldwide during the spring (particularly in big cities), and epidemics occur sporadically. This disease is self-limiting, with an excellent prognosis.

Causes and incidence

The rubella virus is transmitted through contact with the blood, urine, stools, or nasopharyngeal secretions of infected people and, possibly, by contact with contaminated articles of clothing. Transplacental transmission, especially in the first trimester of pregnancy, can cause serious birth defects, such as microcephaly, mental retardation, patent ductus arteriosus, glaucoma, and bone defects. (See *Congenital rubella syndrome*.) Humans are the only known hosts for the rubella virus. The disease is contagious from about 10 days before the rash appears until 5 days after it has appeared.

Complications

- Arthritis (females)
- Encephalitis
- Myocarditis
- Thrombocytopenia
- Hepatitis

Signs and symptoms

In children, after an incubation period of from 14 to 21 days, an exanthematous, maculopapular rash erupts abruptly. (See *Incubation and duration of common rash-producing infections*, page 987.) In adolescents and adults, prodromal signs and symptoms—headache, malaise, anorexia, low-grade fever, coryza, lymphadenopathy and, sometimes, conjunctivitis—are the first to appear. Suboccipital, postauricular, and postcervical lymph node enlargement is a hallmark of this disease and precedes the rash.

Typically, the rubella rash begins on the face and spreads rapidly, in many cases covering the trunk and extremities within

hours. Small, red, petechial macules on the soft palate (Forschheimer spots) may precede or accompany the rash but aren't diagnostic of rubella. By the end of the second day, the facial rash begins to fade, but the rash on the trunk may become confluent and be mistaken for scarlet fever. The rash continues to fade in the downward order in which it appeared. It generally disappears on the third day, but it may persist for 4 or 5 days—sometimes accompanied by mild coryza and conjunctivitis. The rapid appearance and disappearance of the rubella rash distinguishes it from rubeola. In rare cases, rubella can occur without a rash. Low-

grade fever may accompany the rash (99° to 101° F [37.2° to 38.3° C]), but it usually doesn't persist after the first day of the rash; rarely, temperature may reach 104° F (40° C).

Complications seldom occur in children with rubella, but when they do, they commonly appear as hemorrhagic problems such as thrombocytopenia. Many young women, however, experience transient joint pain or arthritis, usually just as the rash is fading. Fever may then recur. These complications usually subside spontaneously within 5 to 30 days.

A significant number of cases, 20% to 50%, are asymptomatic.

Diagnosis

The rubella rash, lymphadenopathy, other characteristic signs, and a history of exposure to infected people usually permit clinical diagnosis without laboratory tests.

CONFIRMING DIAGNOSIS

The rubella rash has been confused with scarlet fever, measles (rubeola), infectious mononucleosis, roseola, erythema infectiosum, and other viral exanthems. Therefore, without exposure history, laboratory confirmation is beneficial. Cell cultures of the throat, blood, urine, and cerebrospinal fluid can confirm the virus' presence. Convalescent serum that shows a fourfold rise in antibody titers corroborates the diagnosis.

Treatment

Because the rubella rash is self-limiting and only mildly pruritic, it doesn't require topical or systemic medication. Treatment consists of aspirin for fever and joint pain. Bed

rest isn't necessary, but the patient should be isolated until the rash disappears.

CONGENITAL RUBELLA SYNDROME

Congenital rubella is by far the most serious form of the disease. Intrauterine rubella infection, especially during the first trimester, can lead to spontaneous abortion or stillbirth as well as single or multiple birth defects. (As a rule, the earlier the infection occurs during pregnancy, the greater the damage to the fetus.)

The combination of cataracts, deafness, and cardiac disease characterizes congenital rubella syndrome. Low birth weight, microcephaly, and mental retardation are other common

manifestations. However, researchers now believe that congenital rubella can cause more disorders, many of which don't appear until later in life. These include dental abnormalities, thrombocytopenic purpura, hemolytic and hypoplastic anemia, encephalitis, giant-cell hepatitis, seborrheic dermatitis, and diabetes mellitus. Indeed, it now appears that congenital rubella may be a lifelong disease. This theory is supported by the fact that the rubella virus has been isolated from urine 15 years after its acquisition in the uterus.

Neonates born with congenital rubella should be isolated immediately because they excrete the virus for several months to a year after birth. Cataracts and cardiac defects may require surgery. The prognosis depends on the particular malformations that occur. The overall mortality for neonates with rubella is 6%, but it's higher for neonates born with thrombocytopenic purpura, congenital cardiac disease, or encephalitis. Parents of affected children need emotional support and guidance in finding help from community resources and organizations.

Special considerations

- Make the patient with active rubella as comfortable as possible. If the patient is a child, give him children's books to read or games to play to keep him occupied.
- Explain to the patient why droplet precautions are necessary. Congenital rubella requires contact precautions until age 1. Make sure the patient understands how important it is to avoid exposing women who are pregnant to this disease.
- Report confirmed cases of rubella to local public health officials.

Before giving the rubella vaccine:

- Obtain a history of allergies, especially to neomycin. If the patient has this allergy or has had a reaction to immunization in the past, check with the physician before giving the vaccine.
- Ask women of childbearing age if they're pregnant. If they are or think they may be, *don't* give the vaccine or perform a pregnancy test first. Warn women who receive rubella vaccine to use an effective means of birth control for at least 3 months after immunization.
- Give the vaccine at least 3 months after any administration of immune globulin or blood, which could have antibodies that neutralize the vaccine.

- Don't vaccinate patients who are immunocompromised, patients with immunodeficiency diseases, or those receiving immunosuppressive, radiation, or corticosteroid therapy. Instead, administer immune serum globulin as ordered, to prevent or reduce infection in susceptible patients.

After giving the rubella vaccine:

- Observe the patient for signs of anaphylaxis for at least 30 minutes. Keep epinephrine 1:1,000 handy.
- Warn the patient about possible mild fever, slight rash, transient arthralgia (in adolescents), and arthritis (in elderly patients). Suggest aspirin or acetaminophen for fever.
- If swelling persists after the initial 24 hours, suggest a cold compress to promote vasoconstriction and prevent antigenic cyst formation.

PREVENTION

Immunization with live virus vaccine RA27/3, the only rubella vaccine available in the United States, is necessary for prevention and appears to be more immunogenic than previous vaccines. The rubella vaccine should be given with measles and mumps vaccines at age 15 months to decrease the cost and number of injections.

Rubeola

Rubeola, also known as the *measles* or *morbilli*, is an acute, highly contagious paramyxovirus infection that may be one of the most common and most serious of all communicable childhood diseases. Use of the vaccine has reduced the occurrence of measles during childhood; as a result, measles is becoming more prevalent in adolescents and adults. (See *Administering measles vaccine*.) In the United States, the prognosis is usually excellent; however, measles is a major cause of death in children in underdeveloped countries.

Causes and incidence

Measles is spread by direct contact or by contaminated airborne respiratory droplets. The portal of entry is the upper respiratory tract. In temperate zones, incidence is highest in late winter and early spring. Before the availability of measles vaccine, epidemics occurred every 2 to 5 years in large urban areas.

Complications

- Otitis media

- Cervical adinitis
- Laryngitis
- Pneumonia
- Encephalitis

Signs and symptoms

Incubation is from 8 to 14 days. Initial symptoms begin and greatest communicability occurs during the prodromal phase, about 11 days after exposure to the virus. This phase lasts from 4 to 5 days; signs and symptoms include fever, photophobia, malaise, anorexia, conjunctivitis, coryza, hoarseness, and hacking cough.

ADMINISTERING MEASLES VACCINE

- Ask the patient about known allergies, especially to neomycin (each dose contains a small amount). However, a patient who's allergic to eggs may receive the vaccine because it contains only minimal amounts of albumin and yolk components.
- Avoid giving the vaccine to a pregnant woman (ask for date of last menstrual period). Warn female patients to avoid pregnancy for at least 3 months following vaccination.
- Don't vaccinate children with untreated tuberculosis, immunodeficiencies, leukemia, or lymphoma or those receiving immunosuppressants. If such children are exposed to the virus, recommend that they receive gamma globulin (gamma globulin won't prevent measles but will lessen its severity). Older unimmunized children who have been exposed to measles for more than 5 days may also require gamma globulin. Be sure to immunize them 3 months later.
- Delay vaccination for 8 to 12 weeks after administration of whole blood, plasma, or gamma globulin because measles antibodies in these components may neutralize the vaccine.
- Watch for signs of anaphylaxis for 30 minutes after vaccination. Keep epinephrine 1:1,000 handy.
- Warn the patient or his parents that possible adverse effects are anorexia, malaise, rash, mild thrombocytopenia or leukopenia, and fever. Advise them that mild reactions may occur, usually within 7 to 10 days. If swelling occurs within 24

hours after vaccination, tell the patient to apply cold compresses to the injection site to promote vasoconstriction and to prevent antigenic cyst formation.

- Generally, one bout of measles provides immunity (a second infection is extremely rare and may indicate a misdiagnosis); infants younger than age 4 months may be immune because of circulating maternal antibodies. Under normal conditions, measles vaccine isn't administered to children younger than age 15 months. However, during an epidemic, infants as young as 6 months may receive the vaccine and then be reimmunized at age 15 months. An alternative approach calls for administering gamma globulin to infants between ages 6 and 15 months who are likely to be exposed to measles.

At the end of the prodrome, Koplik's spots, the hallmark of the disease, appear. These spots look like tiny, bluish white specks surrounded by a red halo. They appear on the oral mucosa opposite the molars and occasionally bleed. About 5 days after Koplik's spots appear, temperature rises sharply, spots slough off, and a slightly pruritic rash appears. This characteristic rash starts as faint macules behind the ears and on the neck and cheeks. The macules become papular and erythematous, rapidly spreading over the entire face, neck, eyelids, arms, chest, back, abdomen, and thighs. When the rash reaches the feet (2 to 3 days later), it begins to fade in the same sequence that it appeared, leaving a brownish discoloration that disappears in 7 to 10 days. (See *Incubation and duration of common rash-producing infections*, page 987.)

The disease climax occurs 3 to 4 days after the rash appears and is marked by a fever of 103° to 105° F (39.4° to 40.6° C), severe cough, puffy red eyes, and rhinorrhea. About 5 days after the rash appears, other symptoms disappear and communicability ends. Symptoms are usually mild in patients with partial immunity (conferred by administration of gamma globulin) or infants with transplacental antibodies. More severe symptoms and complications are more likely to develop in young infants, adolescents, adults, and patients who are immunocompromised than in young children.

Atypical measles may appear in patients who received the killed measles vaccine. These patients are acutely ill with a fever and maculopapular rash that's most obvious in the arms and legs or with pulmonary involvement and no skin lesions.

Severe infection may lead to secondary bacterial infection and to autoimmune reaction or organ invasion by the virus, resulting in otitis media, pneumonia, and encephalitis. Subacute sclerosing panencephalitis, a rare and invariably fatal

complication, may develop several years after measles but it's less common in patients who have received the measles vaccine.

Diagnosis

Diagnosis rests on distinctive clinical features, especially the pathognomonic Koplik's spots. Mild measles may resemble rubella, roseola infantum, enterovirus infection, toxoplasmosis, and drug eruptions; laboratory tests are required for a differential diagnosis. If necessary, measles virus may be isolated from the blood, nasopharyngeal secretions, and urine during the febrile period. Serum antibodies appear within 3 days after onset of the rash and reach peak titers 2 to 4 weeks later.

Treatment

Treatment for measles requires bed rest, relief of symptoms, and droplet isolation throughout the communicable period. Vaporizers and a warm environment help reduce respiratory irritation, and antipyretics can reduce fever. Cough preparations and antibiotics are generally ineffective. I.V. ribavirin has reduced the severity of illness in adults. Therapy must also combat complications.

Special considerations

- Teach the patient's parents supportive measures, and stress the need for isolation, plenty of rest, and increased fluid intake. Advise them to cope with photophobia by darkening the room or providing sunglasses and to reduce fever with antipyretics and tepid sponge baths.
- Warn the patient's parents to watch for and report the early signs and symptoms of complications, such as encephalitis, otitis media, and pneumonia.

Variola

Variola, or *smallpox,* was an acute, highly contagious infectious disease caused by the poxvirus variola. After a global eradication program, the World Health Organization pronounced smallpox eradicated on October 26, 1979, 2 years after the last naturally occurring case was reported in Somalia. Vaccination is no longer recommended, except for certain laboratory workers. The last known case in the United States was reported in 1949. Although naturally occurring smallpox has been eradicated, variola virus preserved in laboratories remains an unlikely source of infection. In response to bioterrorism concerns, smallpox vaccination was offered to members of the military, health department officials, first responders, and key health care providers. If a bioterrorism event involving smallpox is suspected or occurs, vaccination programs can be initiated.

Smallpox developed in three major forms: *variola major* (classic smallpox), which carried a high mortality; *variola minor*, a mild form that occurred in nonvaccinated people and resulted from a less virulent strain; and *varioloid*, a mild variant of smallpox that occurred in previously vaccinated people who had only partial immunity.

Causes and incidence

Smallpox affected people of all ages. In temperate zones, incidence was highest during the winter; in the tropics, during the hot, dry months. Smallpox was transmitted directly by respiratory droplets or dried scales of virus-containing lesions or indirectly through contact with contaminated linens or other objects. Variola major was contagious from onset until after the last scab was shed.

Complications

- Arthritis
- Pneumonia

- Bronchitis
- Pneumonitis
- Encephalitis

Signs and symptoms

Characteristically, after an incubation period of 7 to 14 days, smallpox caused an abrupt onset of chills (and possible seizures in children), high fever (above 104° F [40° C]), headache, backache, severe malaise, vomiting (especially in children), marked prostration and, occasionally, violent delirium, stupor, or coma. Two days after onset, symptoms became more severe, but by the third day the patient began to feel better.

However, he soon developed a sore throat and cough as well as lesions on the mucous membranes of the mouth, throat, and respiratory tract. Within days, skin lesions also appeared, progressing from macular to papular, vesicular, and pustular (pustules were as large as 8.5 mm in diameter). All skin lesions were in the same stage of development. During the pustular stage, the patient's temperature again rose, and early symptoms returned. By day 10, the pustules began to rupture and eventually dried and formed scabs. Symptoms finally subsided about 14 days after onset. Desquamation of the scabs took another 1 to 2 weeks, caused intense pruritus, and commonly left permanently disfiguring scars.

In fatal cases, a diffuse dusky appearance came over the patient's face and upper chest. Death resulted from encephalitic manifestations, extensive bleeding from

any or all orifices, or secondary bacterial infections.

Diagnosis

Before the global eradication program, smallpox was readily recognizable, especially during an epidemic or after known contact. However, most of today's health care workers aren't familiar with the disease's telltale signs and symptoms. The most conclusive laboratory test is a culture of variola virus isolated from an aspirate of vesicles and pustules. Other laboratory tests include microscopic examination of smears from lesion scrapings and complement fixation to detect virus or antibodies to the virus in the patient's blood.

Treatment

Treatment for smallpox requires hospitalization with droplet and contact precautions, antimicrobial therapy to treat bacterial complications, vigorous supportive measures, and symptomatic treatment of lesions with antipruritics, starting during the pustular stage. If the smallpox vaccination is given within 1 to 4 days of exposure to the disease, it may prevent illness or lessen symptoms. Treatment once the disease has started is limited.

Special considerations

- Give aspirin, codeine, or (as needed) morphine to relieve pain.
- I.V. infusions and gastric tube feedings provide fluids, electrolytes, and calories because pharyngeal lesions make swallowing difficult.
- If smallpox is suspected, the state health department should be notified immediately.

Monkeypox

Monkeypox is a rare viral disease identified mostly in the rainforest countries of central and west Africa. The virus was originally discovered in laboratory monkeys in 1958. It was later recovered from an African squirrel, which was thought to be the natural host. It may also infect other rodents, such as rats, mice, and rabbits. The first human cases of monkeypox were reported in remote African locations in 1970. In June 2003, there was an outbreak in the United States involving people who had gotten ill following contact with infected prairie dogs.

Causes and incidence

The *monkeypox virus,* belonging to the orthopoxvirus group of viruses, causes monkeypox. It's related to variola and cowpox. People can contract monkeypox from an infected animal through a bite or direct contact with the animal's blood,

body fluids, or lesions. It's spread person to person via respiratory droplets during direct and

prolonged face-to face contact. It's less infectious than smallpox, but it can also be spread through direct contact with an infected person's body fluids or with viruscontaminated objects, such as bedding or clothing.

Complications

- Encephalitis (rare)
- Death (rare)

Signs and symptoms

The signs and symptoms of monkeypox are similar to smallpox, but milder. After an incubation period of about 12 days, the patient may report fever, headache, muscle aches, backache, swollen lymph nodes, and a general feeling of discomfort and exhaustion. A papular rash begins on the face or other area of the body within 1 to 3 days after onset of the fever. The lesions go through several stages before crusting and falling off. The illness' duration is 2 to 4 weeks.

In Africa, monkeypox is fatal in 10% of those who contract the disease.

Diagnosis

Diagnosis is based on history and presenting signs and symptoms. The virus may be isolated from vesicular fluid to aid in diagnosis and differentiation from other rash-producing viruses.

Treatment

There is no specific treatment for monkeypox, but the smallpox vaccine appears to reduce the risk of contracting the disease. The Centers for Disease Control and Prevention recommends that persons who are investigating monkeypox outbreaks and caring for infected individuals or animals should receive smallpox vaccination. Persons exposed to individuals or animals confirmed to have monkeypox should also receive vaccinations (up to 14 days after exposure).

Vaccinia immune globulin may be considered in some cases, such as in patients who are severely immunocompromised. There is no data available on the effectiveness of cidofovir in the treatment of human monkeypox cases.

Special considerations

- Notify the local health department immediately if you suspect monkeypox.

- A combination of standard, contact, and droplet precautions should be applied in all health care settings. Because of the risk of airborne transmission, droplet precautions should be applied whenever possible using a NIOSH-certified N95 (or comparable) filtering disposable respirator that has been fit-tested. Surgical masks may be worn if the respirator is not available. Isolation continues until all lesions are crusted over or until the local or state health department advises that isolation is no longer necessary.
- Perform scrupulous hand washing after contact with an infected patient or contaminated objects. Teach the patient and his family members proper hand washing as well.
- Eye protection should be used if splash or spray of body fluids is possible.
- Place the patient in a private room. Use a negative-pressure room if available.
- When transporting the patient, place a mask over his nose and mouth, and cover the exposed skin lesions with a sheet or gown. If the patient is to remain at home, he should maintain the same precautions.

Roseola infantum

Roseola infantum (exanthema subitum), an acute, benign, presumably viral infection, usually affects infants and young children ages 6 months to 3 years. Characteristically, it first causes a high fever and then a rash that accompanies an abrupt drop to normal temperature. It's also known as *sixth disease*.

Causes and incidence

Human herpesvirus 6 causes roseola. It affects boys and girls alike and occurs year-round. Overt roseola, the most common exanthem in children younger than age 2, affects 30% of all children; inapparent roseola (febrile illness without a rash) may affect the rest. The mode of transmission isn't known, but oral secretions are suspected. Rarely does an infected child transmit roseola to a sibling.

Signs and symptoms

After a 5- to 15-day incubation period (average, 10 days) the infant with roseola develops an abruptly rising, unexplainable fever and, sometimes, seizures. Temperature peaks at 103° to 105° F (39.4° to 40.6° C) for 3 to 5 days, then drops suddenly. In the early febrile period, the infant may be anorexic, irritable, and listless but doesn't appear particularly ill. Simultaneously, with an abrupt drop in temperature, the infant develops a maculopapular, nonpruritic rash that blanches on pressure. The rash is profuse on the trunk, arms, and neck and mild on the face and legs. It fades within 24 hours. Complications are extremely rare.

Diagnosis

Diagnosis requires observation of the characteristic rash that appears about 48 hours after fever subsides. Serologic evidence of primary infection can be determined by checking antibody levels in acute and convalescent sera.

Treatment

Because roseola is self-limiting, treatment is supportive and symptomatic: antipyretics to lower fever and, if necessary, anticonvulsants to relieve seizures.

Special considerations

- Teach parents how to reduce their infant's fever by giving tepid baths, keeping him in lightweight clothes, and maintaining normal room temperature. Stress the need for adequate fluid intake. Strict bed rest and isolation are unnecessary. Tell parents that a short febrile seizure will not cause brain damage. Explain that seizures will cease after fever subsides and that phenobarbital is likely to cause drowsiness; if it causes stupor, tell the parents to call their physician immediately.

ENTEROVIRUSES

Herpangina

Herpangina is an acute infection caused by group A coxsackieviruses (usually types 1 through 10, 16, and 22) and, less commonly, by group B coxsackieviruses and echoviruses. The disease typically produces vesicular lesions on the mucous membranes of the soft palate, tonsillar pillars, and throat.

Causes and incidence

Because fecal-oral transfer is the main mode of transmission, herpangina usually affects children younger than age 10 (except neonates because of maternal antibodies). It's also transmitted via contact with nose and throat discharges. It's slightly more common in late summer and fall and can be sporadic, endemic, or epidemic. It generally subsides in 4 to 7 days.

Complication

- Dehydration

Signs and symptoms

After a 2- to 9-day incubation period, herpangina begins abruptly with a sore throat, pain on swallowing, and a temperature of 101° to 104° F (38.3° to 40° C) that persists for 1 to 4 days and may cause seizures, headache, anorexia, malaise, and pain in the stomach, back of the neck, legs, or arms. Grayish white papulovesicles (up to 12) appear on the soft palate and, less commonly, on the tonsils, uvula, tongue, and larynx. These lesions grow from 1 to 2 mm in diameter to large, punched-out ulcers that are several millimeters in diameter and surrounded by small, inflamed margins.

Diagnosis

CONFIRMING DIAGNOSIS

Characteristic oral lesions suggest this diagnosis; isolation of the virus from mouth washings or feces and elevated specific antibody titer confirm it.

Other routine test results are normal except for slight leukocytosis.

Diagnosis requires distinguishing the mouth lesions in herpangina from those in streptococcal tonsillitis (no ulcers; lesions confined to tonsils).

Treatment

Treatment for herpangina is entirely symptomatic, emphasizing measures to reduce fever and prevent seizures and possible dehydration.

Herpangina doesn't require isolation or hospitalization but does require careful hand hygiene and sanitary disposal of excretions. Topical anesthetic agents, such as benzocaine or Xylocaine, may be applied to the mouth for discomfort.

Special considerations

- Teach parents to provide adequate fluids and a nonirritating diet, to enforce bed rest, and to administer tepid sponge baths and antipyretics.

Poliomyelitis

Poliomyelitis, also called *polio* or *infantile paralysis,* is an acute communicable disease caused by the poliovirus. It ranges in severity from inapparent infection to fatal paralytic illness. First recognized in 1840, poliomyelitis became epidemic in Norway and Sweden in 1905. Outbreaks reached pandemic proportions in Europe, North America, Australia, and New Zealand during the first half of this century. Incidence peaked during the 1940s and early 1950s, and led to the development of the Salk vaccine. (See *Polio protection.*)

Minor polio outbreaks still occur, usually among nonimmunized groups such as the Amish of Pennsylvania. The disease usually strikes during the summer and fall. Once confined mainly to infants and children, poliomyelitis mostly occurs today in people older than age 15. Adults and girls are at greater risk for infection; boys, for paralysis.

If the central nervous system (CNS) is spared, the prognosis is excellent. However, CNS infection can cause paralysis and death. The mortality for all types of poliomyelitis is 5% to 10%.

Causes and incidence

The poliovirus has three antigenically distinct serotypes—types I, II, and III—all of which cause poliomyelitis. These viruses are found worldwide and are transmitted from person to person by direct contact with infected oropharyngeal secretions or feces. The incubation period ranges from 5 to 35 days—7 to 14 days on average.

The virus usually enters the body through the alimentary tract, multiplies in the oropharynx and lower intestinal tract, and then spreads to regional lymph nodes and the blood. Factors that increase the risk of paralysis include pregnancy; old age; localized trauma, such as a recent tonsillectomy, tooth extraction, or inoculation; and unusual physical exertion at or just before the clinical onset of poliomyelitis.

Complications

- Respiratory failure
- Pulmonary edema
- Pulmonary embolism
- Urinary tract infection
- Urolithiasis
- Atelectasis
- Pneumonia
- Cor pulmonale
- Soft tissue and skeletal deformities
- Paralytic shock
- Hypertension

Signs and symptoms

Manifestations of poliomyelitis follow three basic patterns. Inapparent (subclinical) infections constitute 95% of all poliovirus infections. Abortive poliomyelitis (minor illness), which accounts for 4% to 8% of all cases, causes slight fever, malaise, headache, sore throat, inflamed pharynx, and vomiting. The patient usually recovers within 72 hours. Most cases of inapparent or abortive poliomyelitis go unnoticed.

Major poliomyelitis, however, involves the CNS and takes two forms: nonparalytic and paralytic. Children commonly show a biphasic course, in which the onset of major illness occurs after recovery from the minor illness stage. *Nonparalytic poliomyelitis* produces moderate fever, headache, vomiting, lethargy, irritability, and pains in the neck, back, arms, legs, and abdomen. It also causes muscle tenderness, weakness, and spasms in the extensors of the neck and back and sometimes in the hamstring and other muscles. (These spasms may be observed during maximum range-of-motion exercises.) Nonparalytic polio usually lasts about a week, with meningeal irritation persisting for about 2 weeks.

Paralytic poliomyelitis usually develops within 5 to 7 days of the onset of fever. The patient displays symptoms similar to those of nonparalytic poliomyelitis, with asymmetrical weakness of various muscles, loss of superficial and deep reflexes, paresthesia, hypersensitivity to touch, urine retention, constipation, and abdominal distention. The extent of paralysis depends on the level of the spinal cord lesions, which may be cervical, thoracic, or lumbar.

Resistance to neck flexion is characteristic in nonparalytic and paralytic poliomyelitis. The patient will “tripod”—extend his arms behind him for support—when he sits up. He'll display Hoyne's sign—his head will fall back when he's supine and his shoulders are elevated. From a supine position, he won't be able to raise his legs a full 90 degrees. Paralytic poliomyelitis also causes positive Kernig's and Brudzinski's signs.

When the disease affects the medulla of the brain, it's called *bulbar paralytic poliomyelitis*, which is the most perilous type. This form affects the respiratory muscle nerves, leading to respiratory paralysis, and weakens the muscles supplied by the cranial nerves (particularly IX and X), producing symptoms of encephalitis. Other signs and symptoms include facial weakness, diplopia, dysphasia, difficulty in chewing, inability to swallow or expel saliva, regurgitation of food through the nasal passages, and dyspnea as well as abnormal respiratory rate, depth, and rhythm, which may lead to respiratory arrest. Fatal pulmonary edema and shock are possible.

Diagnosis

CONFIRMING DIAGNOSIS

Diagnosis requires isolation of the poliovirus from throat washings early in the disease, from stools throughout the disease, and from cerebrospinal fluid (CSF) cultures in CNS infection.

Coxsackievirus and echovirus infections must be ruled out. (See *Enterovirus facts*, page 1002.) Convalescent serum antibody titers four times greater than acute titers support a diagnosis of poliomyelitis. Routine laboratory tests are usually within normal limits. However, CSF pressure and protein levels may be slightly increased and white blood cell count elevated initially, mostly due to polymorphonuclear leukocytes, which constitute 50% to 90% of the total count. Thereafter, mononuclear cells constitute most of the diminished number of cells.

POLIO PROTECTION

Dr. Jonas Salk's poliomyelitis vaccine, which became available in 1955, has been rightly called one of the miracle drugs of modern medicine. The vaccine contains dead (formalin-inactivated) polioviruses that stimulate production of circulating antibodies in the human body. This vaccine so effectively eliminated poliomyelitis that today it's difficult to appreciate how fearful people once were of this disease.

Oral polio vaccine is no longer used in the United States because polio had been eliminated from the Western Hemisphere and there were new cases of vaccine-associated polio. Instead, inactivated poliovirus vaccine (IPV) has been the recommended form of vaccine since January 2000. Children should receive 4 doses of IPV at ages 2, 4, and 6 to 18 months and at age 4 to 6 years. Routine poliovirus vaccination isn't generally recommended for people ages 18 or older residing in the United States.

Treatment

Treatment is supportive and includes analgesics to ease headache, back pain, and leg spasms; morphine is contraindicated because of the danger of additional respiratory suppression. Moist heat applications may also reduce muscle spasm and pain.

Bed rest is necessary only until extreme discomfort subsides; in paralytic polio, this may take a long time. Paralytic polio also requires long-term rehabilitation using physical therapy, braces, corrective shoes and, in some cases, orthopedic surgery.

ENTEROVIRUS FACTS

Enteroviruses (polioviruses, coxsackieviruses, and echoviruses) infect the GI tract. These viruses, among the smallest that affect humans, include 3 known polioviruses, 23 group A coxsackieviruses, 6 group B coxsackieviruses, and 34 echoviruses. They usually infect humans as a result of ingestion of fecally contaminated material, causing a wide range of diseases (hand, foot, and mouth disease; aseptic meningitis; myocarditis; pericarditis; gastroenteritis; and poliomyelitis). They can appear in the pharynx, feces, blood, cerebrospinal fluid, and central nervous system tissue. Enterovirus infections are more prevalent in the summer and fall.

Special considerations

Your care plan must be comprehensive to help prevent complications and to assist polio patients—physically and emotionally—during their prolonged convalescence.

- Observe the patient carefully for signs of paralysis and other neurologic damage, which can occur rapidly. Maintain a patent airway, and watch for respiratory weakness and difficulty in swallowing. A tracheotomy is typically done at the first sign of respiratory distress, after which the patient is placed on a mechanical ventilator. Remember to reassure the patient that his breathing is being supported. Practice strict sterile technique during suctioning, and use only sterile solutions to nebulize medications.
- Perform a brief neurologic assessment at least once a day, but don't demand any vigorous muscle activity. Encourage a return to mild activity as soon as the patient is able.
- Check blood pressure frequently, especially in bulbar poliomyelitis, which can cause hypertension or shock because of its effect on the brain stem.
- Watch for signs of fecal impaction due to dehydration and intestinal inactivity. To prevent this, give sufficient fluids to ensure an adequate daily output of low-specificgravity urine (1.5 to 2 L/day for adults).
- Monitor the bedridden patient's food intake for an adequate, well-balanced diet. If tube feedings are required, give liquid baby foods, juices, lactose, and vitamins.
- Be sure to prevent pressure ulcers by providing good skin care, repositioning the patient often, and keeping the bed dry. Remember, muscle paralysis may cause

bladder weakness or transient bladder paralysis.

- Apply high-top sneakers or use a footboard to prevent footdrop. To alleviate discomfort, use foam rubber pads and sandbags, as needed, and light splints as ordered.
- To control the spread of poliomyelitis, wash your hands thoroughly after contact with the patient, especially after contact with excretions. Instruct the ambulatory patient to do the same. (Only hospital personnel who have been vaccinated against poliomyelitis should have direct contact with the patient.)
- Provide emotional support to the patient and his family. Reassure the nonparalytic patient that his chances for recovery are good. Long-term support and encouragement are essential for maximum rehabilitation.
- An interdisciplinary rehabilitation program should be set up for a paralytic patient. It should include physical and occupational therapists, physicians and, if necessary, a psychiatrist to help manage the emotional problems suddenly facing severe physical disabilities.
- Report all cases of poliomyelitis to the appropriate health department.

ARBOVIRUS

Colorado tick fever

Colorado tick fever is a benign infection caused by the Colorado tick fever arbovirus and transmitted to humans by a tick. It occurs in the Rocky Mountain region of the United States, mostly in April and May

at lower altitudes and in June and July at higher altitudes. Because of occupational or recreational exposure, it's more common in men than in women. Colorado tick fever apparently confers long-lasting immunity against reinfection.

Causes and incidence

Colorado tick fever is transmitted to humans by a hard-shelled wood tick called *Dermacentor andersoni*. The adult tick acquires the virus when it bites infected rodents and remains permanently infective.

Incidence is high in Colorado, where up to 15% of people who regularly camp show past exposure. It's much less common in the rest of the United States.

Complications

- Pericarditis

- Myocarditis
- Epididymitis
- Orchitis
- Atypical pneumonia
- Meningoencephalitis

Signs and symptoms

After a 3- to 6-day incubation period, Colorado tick fever begins abruptly with chills; temperature of 104° F (40° C); severe aching of back, arms, and legs; lethargy; and headache with eye movement such as extraocular movement. Photophobia, abdominal pain, nausea, and vomiting may occur. Rare effects include petechial or maculopapular rashes and central nervous system involvement. Symptoms subside after several days but return within 2 to 3 days and continue for 3 more days before slowly disappearing. Complete recovery usually follows.

Diagnosis

CONFIRMING DIAGNOSIS

A history of recent exposure to ticks along with moderate to severe leukopenia, complement fixation tests, or virus isolation confirm the diagnosis.

Treatment

After correct removal of the tick, supportive treatment focuses on relieving symptoms, combating secondary infection, and maintaining fluid balance. Colorado tick fever needs to be differentiated from Rocky Mountain spotted fever and tularemia.

Special considerations

- Carefully remove the tick by grasping it with forceps or gloved fingers and pulling gently. Be careful not to crush the tick's body. Keep it for identification. Thoroughly wash the wound with soap and water. If the tick's head remains embedded, surgical removal is necessary. Give a tetanus-diphtheria booster as ordered.
- Be alert for secondary infection.
- Monitor the patient's fluid and electrolyte balance, and provide replacement therapy, as indicated.

- Reduce fever with antipyretics and tepid sponge baths.

PREVENTION

To prevent tickborne infection, tell the patient to avoid tick bites by wearing long-sleeved clothing, tucking pant bottoms into the top of his boots, and carefully checking his body and scalp for ticks several times a day whenever in tick-infested areas. He should also use insect repellant.

MISCELLANEOUS VIRUSES

Mumps

Mumps, also known as *infectious* or *epidemic parotitis*, is an acute viral disease caused by a paramyxovirus. It causes painful enlargement of the salivary or parotid glands. It may also infect other organs, such as the testes, the central nervous system (CNS), and the pancreas. The prognosis for complete recovery is good, although mumps sometimes causes complications.

Causes and incidence

The mumps paramyxovirus is found in the saliva of an infected person and is transmitted by droplets or by direct contact. The virus is present in the saliva 6 days before to 9 days after onset of parotid gland swelling; the 48-hour period immediately preceding onset of swelling is probably the

time of highest communicability. The incubation period ranges from 14 to 25 days (the average is 18). One attack of mumps (even if unilateral) almost always confers lifelong immunity.

Mumps is most prevalent in children between ages 6 and 8. Infants younger than age 1 seldom get this disease because of the passive immunity received from maternal antibodies. Peak incidence occurs during late winter and early spring.

Complications

- Mumps meningitis
- Epididymo-orchitis, producing abrupt onset of testicular swelling and tenderness, scrotal erythema, lower abdominal pain, nausea, vomiting, fever, and chills in about 25% of postpubertal males

Signs and symptoms

The clinical features of mumps vary widely. An estimated 30% of susceptible people have subclinical illness.

Mumps usually begins with prodromal symptoms that last for 24 hours and include myalgia, anorexia, malaise, headache, and low-grade fever followed by an earache that's aggravated by chewing; parotid gland tenderness and swelling; a temperature of 101° to 104° F (38.3° to 40° C); and pain when chewing or when drinking sour or acidic liquids. Simultaneously with the swelling of the parotid gland or several days later, one or more of the other salivary glands may become swollen.

Mumps meningitis complicates the disease in 10% of patients and affects three to five times more males than females. Signs and symptoms include fever, meningeal irritation (nuchal rigidity, headache, and irritability), vomiting, drowsiness, and a cerebrospinal fluid lymphocyte count ranging from 500 to 2,000/µl. Recovery is usually complete. Less common effects are pancreatitis, deafness, arthritis, myocarditis, encephalitis, pericarditis, oophoritis, and nephritis.

Diagnosis

Diagnosis is usually made after the characteristic signs and symptoms develop, especially parotid gland enlargement with a history of exposure to mumps. Serologic antibody testing can verify the diagnosis when parotid or other salivary gland enlargement is absent. If comparison between a blood specimen obtained during the acute phase of illness and another specimen obtained 3 weeks later shows a fourfold rise in antibody titer, the patient most likely had mumps.

Treatment

Treatment includes analgesics for pain, antipyretics for fever, and adequate fluid intake to prevent dehydration from fever and anorexia. If the patient can't swallow, consider I.V. fluid replacement. Warm salt-water gargles, soft foods, and extra fluids may also help relieve symptoms.

Special considerations

- Stress the need for bed rest during the febrile period. Give analgesics and apply warm or cool compresses to the neck to relieve pain. Give antipyretics and tepid sponge baths for fever. To prevent dehydration, encourage the patient to drink fluids; to minimize pain and anorexia, advise him to avoid spicy, irritating foods and those that require a lot of chewing. Offer a soft, bland diet.
- During the acute phase, observe the patient closely for signs of CNS involvement, such as altered level of consciousness and nuchal rigidity.
- Because the mumps virus is present in the saliva throughout the course of the disease, follow droplet precautions until symptoms subside.

- Report all cases of mumps to local public health authorities.

PREVENTION

Emphasize the importance of routine immunization with live attenuated mumps virus (paramyxovirus) at age 15 months and for susceptible patients (especially males) who are approaching or are past puberty. Remember, immunization within 24 hours of exposure may prevent or attenuate the actual disease. Immunity against mumps lasts at least 12 years.

Infectious mononucleosis

Infectious mononucleosis is an acute infectious disease caused by the Epstein-Barr virus (EBV), a member of the herpes group. It primarily affects young adults and children, although in children it's usually so mild that it's generally overlooked. This infection characteristically produces fever, sore throat, and cervical lymphadenopathy (the hallmarks of the disease) as well as hepatic dysfunction, increased lymphocyte and monocyte counts, and development and persistence of heterophil antibodies. The prognosis is excellent, and major complications are uncommon.

Causes and incidence

Apparently, the reservoir of EBV is limited to humans. Infectious mononucleosis probably spreads by the oral-pharyngeal route because about 80% of patients carry EBV in the throat during the acute infection and for an indefinite period afterward. It can also be transmitted by blood transfusions and has been reported after cardiac surgery as the post-pump perfusion syndrome. Infectious mononucleosis is probably contagious from before symptoms develop until the fever subsides and oral-pharyngeal lesions disappear.

Infectious mononucleosis is fairly common in the United States, Canada, and Europe and affects both sexes equally. Incidence varies seasonally among college students but not among the general population.

Complications

- Splenic rupture
- Aseptic meningitis
- Encephalitis
- Hemolytic anemia

- Pericarditis
- Guillain-Barré syndrome

Signs and symptoms

The symptoms of mononucleosis mimic those of many other infectious diseases, including hepatitis, rubella, and toxoplasmosis. Typically, after an incubation period of about 10 days in children and from 30 to 50 days in adults, infectious mononucleosis produces prodromal symptoms, such as headache, malaise, and fatigue. After 3 to 5 days, patients typically develop a triad of symptoms: sore throat, cervical lymphadenopathy, and temperature fluctuations, with an evening peak of 101° to 102° F (38.3° to 38.9° C). Splenomegaly, hepatomegaly, stomatitis, exudative tonsillitis, or pharyngitis may also develop.

Sometimes, early in the illness, a maculopapular rash that resembles rubella develops; also, jaundice occurs in about 5% of patients. Major complications are rare but may include splenic rupture, aseptic meningitis, encephalitis, hemolytic anemia, idiopathic thrombocytopenic purpura, and Guillain-Barré syndrome. Symptoms usually subside about 6 to 10 days after onset of the disease but may persist for weeks.

Diagnosis

Physical examination demonstrating the clinical triad suggests infectious mononucleosis.

CONFIRMING DIAGNOSIS

The following abnormal laboratory results confirm the diagnosis:

- *Monospot test is positive for infectious mononucleosis.*
- *Leukocyte count increases to 10,000 to 20,000/µl during the second and third weeks of illness. Lymphocytes and monocytes account for 50% to 70% of the total white blood cell (WBC) count; 10% of the lymphocytes are atypical.*
- *Heterophil antibodies (agglutinins for sheep red blood cells) in serum drawn during the acute illness and at 3- to 4-week intervals rise to four times the normal number.*
- *Indirect immunofluorescence shows antibodies to EBV and cellular antigens. Such testing is usually more definitive than heterophil antibodies.*
- *Liver function studies are abnormal.*

Treatment

Infectious mononucleosis resists prevention and antimicrobial treatment. Therapy is essentially supportive: relief of symptoms; bed rest during the acute febrile period; and acetaminophen or ibuprofen for headache and sore throat. Sore throat can also be helped with warm salt-water gargles. If severe throat inflammation causes airway obstruction, steroids can be used to

relieve swelling and avoid tracheotomy. Splenic rupture, marked by sudden abdominal pain, requires splenectomy. About 20% of patients with infectious mononucleosis will also have streptococcal pharyngotonsillitis; these patients should receive antibiotic therapy.

Special considerations

Because uncomplicated infectious mononucleosis doesn't require hospitalization, patient teaching is essential. Convalescence may take several weeks, usually until the patient's WBC count returns to normal.

- During the acute illness, stress the need for bed rest. If the patient is a student, tell him he may continue less demanding school assignments and see his friends but should avoid long, difficult projects until after recovery.
- To minimize throat discomfort, encourage the patient to drink milk shakes, fruit juices, and broths and to eat cool, bland foods. Suggest gargling with saline mouthwash and taking aspirin, as needed.

Rabies

Rabies, also known as *hydrophobia,* is an acute central nervous system (CNS) infection caused by a virus that's transmitted by the saliva of an infected animal (especially wild animals). If symptoms occur, rabies is almost always fatal. Treatment soon after exposure, however, may prevent fatal CNS invasion.

Causes and incidence

The rabies virus is usually transmitted to a human through the bite of an infected animal. The virus begins to replicate in the striated muscle cells at the bite site. Then it spreads up the nerve to the CNS and replicates in the brain. Finally, it moves through the nerves into other tissues, including the salivary glands. Occasionally, airborne droplets and infected tissue transplants can transmit the virus.

Rabies symptoms appear earlier if the head or face is severely bitten. If the bite is on the face, the risk of developing rabies is about 60%; on the upper extremities,

15% to 40%; and on the lower extremities, about 10%.

In the United States, dog vaccinations have reduced the incidence of rabies transmission to humans. Wild animals, such as skunks, foxes, raccoons, and bats, account for 70% of rabies cases.

Signs and symptoms

After an incubation period of a few days to several years, but usually 30 to 90 days, rabies typically produces local or radiating pain or burning and a sensation of cold, pruritus, and tingling at the bite site. It also produces prodromal signs and symptoms, such as a slight fever (100° to 102° F [37.8° to 38.9° C]), malaise, headache, anorexia, nausea, sore throat, and persistent loose cough. After this, the patient begins to display nervousness, anxiety, irritability, hyperesthesia, photophobia, sensitivity to loud noises, pupillary dilation, tachycardia, shallow respirations, pain and paresthesia in the bitten area, and excessive salivation, lacrimation, and perspiration.

About 2 to 10 days after the onset of prodromal symptoms, a phase of excitation (neurologic phase) begins. It's characterized by agitation, aphasia, incoordination, marked restlessness, anxiety, and apprehension and cranial nerve dysfunction that causes ocular palsies, strabismus, asymmetrical pupillary dilation or constriction, absence of corneal reflexes, weakness of facial muscles, and hoarseness. Severe systemic symptoms include tachycardia or bradycardia, cyclic respirations, hypotension, disseminated intravascular coagulation, coma, cardiac arrest, urine retention, and a temperature of about 103° F (39.4° C).

About 50% of affected patients exhibit hydrophobia (literally, “fear of water”). Forceful, painful pharyngeal muscle spasms expel liquids from the mouth and cause dehydration and, possibly, apnea, cyanosis, and death. Difficulty swallowing causes frothy saliva to drool from the patient's mouth. Eventually, even the sight, mention, or thought of water causes uncontrollable pharyngeal muscle spasms and excessive salivation. Between episodes of excitation and hydrophobia, the patient commonly is cooperative and lucid. After

about 3 days, excitation and hydrophobia subside, and the progressively paralytic, terminal phase of this illness begins.

ALERT

The patient experiences progressive, generalized, flaccid paralysis that ultimately leads to peripheral vascular collapse, coma, and death.

Diagnosis

Because rabies is fatal unless treated promptly, always suspect rabies in any person who suffers an unprovoked animal bite until you can prove otherwise.

CONFIRMING DIAGNOSIS

Virus isolation from the patient's saliva or throat and examination of his blood for fluorescent rabies antibody (FRA) are diagnostic.

Other results typically include elevated white blood cell count, with increased polymorphonuclear and large mononuclear cells, and elevated urinary glucose, acetone, and protein levels.

Confinement of the suspected animal for 10 days of observation by a veterinarian also helps support this diagnosis. If the animal appears rabid, it should be killed and its brain tissue tested for FRA and Negri bodies (oval or round masses that conclusively confirm rabies).

Treatment

Treatment consists of wound care and immunization as soon as possible after exposure. Thoroughly wash all bite wounds and scratches with soap and water to remove any infected saliva. (See *First aid for animal bites.*) Check the patient's immunization status, and administer tetanus-diphtheria prophylaxis if needed. Take measures to control bacterial infection as ordered. If the wound requires suturing, special treatment and suturing techniques must be used to allow proper drainage.

After rabies exposure, a patient who has not been immunized before must receive passive immunization with rabies immune globulin (RIG) and active immunization with human diploid cell vaccine (HDCV). If the patient has received HDCV before and has an adequate rabies antibody titer, he doesn't need RIG immunization, just an HDCV booster.

FIRST AID FOR ANIMAL BITES

Immediately wash the bite vigorously with soap and water for at least 10 minutes to remove the animal's saliva. As soon as possible, flush the wound with a viricidal agent, followed by a clear-water rinse. After cleaning the wound, apply a sterile dressing. If possible, don't suture the wound, and don't immediately stop the bleeding (unless it's massive) because blood flow helps to clean the wound.

Question the patient about the bite. Ask if he provoked the animal (if so, chances are it isn't rabid) and if he can identify it or its owner (because the animal may be confined for observation).

Special considerations

- When injecting rabies vaccine, rotate injection sites on the deltoid muscle. Watch for and symptomatically treat redness, itching, pain, and tenderness at the injection site. Half of the RIG should be infiltrated into and around the bite wound, with the remainder given I.M.
- Cooperate with public health authorities to determine the vaccination status of the animal. If the animal is proven rabid, help identify others at risk.

If rabies develops:

- Monitor cardiac and pulmonary function continuously.
- Isolate the patient. Wear a gown, gloves, and protection for the eyes and mouth when handling saliva and articles contaminated with saliva. Take precautions to avoid being bitten by the patient during the excitation phase.
- Keep the room dark and quiet.
- Establish communication with the patient and his family. Provide psychological support to help them cope with the patient's symptoms and probable death.

PREVENTION

Take these actions to help prevent rabies:

- *Stress the need for vaccination of household pets that may be exposed to rabid wild animals.*
- *Warn the patient not to try to touch wild animals, especially if they appear ill or overly docile (possible signs of rabies)*
- *Assist in the prophylactic administration of rabies vaccine to high-risk people, such as farm workers, forest rangers, spelunkers (cave explorers), and veterinarians.*

Cytomegalovirus infection

Cytomegalovirus (CMV) infection is caused by the cytomegalovirus, a deoxyribonucleic acid, ether-sensitive virus belonging to the herpes family. Also known as *generalized salivary gland disease* or *cytomegalic inclusion disease*, CMV infection occurs worldwide and is transmitted by human contact.

Causes and incidence

CMV has been found in the saliva, urine, semen, breast milk, feces, blood, and vaginal and cervical secretions of infected people. The virus is usually transmitted through contact with these infected secretions, which can harbor the virus for months or even years. It may be transmitted by sexual contact and can travel across the placenta, causing a congenital infection. Immunosuppressed patients, especially those who have received transplanted organs, run a 90% chance of contracting CMV infection. Recipients of blood transfusions from donors with positive CMV antibodies are at some risk.

About four out of five people older than age 35 have been infected with CMV, usually during childhood or early adulthood. In most of these people, the disease is so mild that it's overlooked. However, CMV infection during pregnancy can be hazardous to the fetus, possibly leading to stillbirth, brain damage, and other birth defects or to severe neonatal illness. About 1% of all neonates have CMV.

Signs and symptoms

CMV probably spreads through the body in lymphocytes or mononuclear cells to the lungs, liver, GI tract, eyes, and central nervous system, where it commonly produces inflammatory reactions.

Most patients with CMV infection have mild, nonspecific complaints or none at all, even though antibody titers indicate infection. In these patients, the disease usually runs a self-limiting course. However, immunodeficient patients and those receiving immunosuppressants may develop pneumonia or other secondary infections. In patients with acquired immunodeficiency syndrome, disseminated CMV infection may cause chorioretinitis (resulting in blindness), colitis, encephalitis, abdominal pain, diarrhea, or weight loss. Infected infants ages 3 to 6 months usually appear asymptomatic but may develop hepatic dysfunction, hepatosplenomegaly, spider angiomas, pneumonitis, and lymphadenopathy. (See *CMV infection in immunosuppressed patients.*)

Congenital CMV infection is seldom apparent at birth, although the neonate's urine contains the virus. CMV can cause brain damage that may not show up for months after birth. It also can produce a rapidly fatal neonatal illness characterized by jaundice, petechial rash, hepatosplenomegaly, thrombocytopenia, hemolytic anemia, microcephaly, psychomotor retardation, mental deficiency, and hearing loss. Occasionally, this form is rapidly fatal.

In some adults, CMV may cause cytomegalovirus mononucleosis, with 3 weeks or more of irregular, high fever. Other findings may include a normal or elevated white blood cell (WBC) count, lymphocytosis, and increased atypical lymphocytes.

Diagnosis

CONFIRMING DIAGNOSIS

Although virus isolation in urine is the most sensitive laboratory method, diagnosis can also be made with virus isolated from saliva, throat, cervix, WBCs, or biopsy specimens. Chest X-ray typically shows bilateral, diffuse, white infiltrates.

Other laboratory tests supporting the diagnosis include complement fixation studies, hemagglutination inhibition antibody tests and, for congenital infections, indirect immunofluorescent tests for CMV immunoglobulin M antibody.

CMV INFECTION IN IMMUNOSUPPRESSED PATIENTS

The table below lists immunosuppressed patients, their risk factors for cytomegalovirus (CMV) infection, associated disorders, and prevention tips.

Patient	Risk factors	Associated disorders	Prevention
Fetus	Primary maternal infection in early pregnancy	Cytomegalic inclusion disease	Avoidance of exposure
Organ transplant recipient	Seropositive donor, seronegative recipient; intensive immunosuppression, particularly with antilymphocyte globulins, cyclosporine	Febrile leukopenia, pneumonia, GI disease	Donor matching, CMV immunoglobulin, ganciclovir or high-dose acyclovir
Bone marrow transplant recipient	Graft-versus-host disease, older age, seropositive recipient, viremia	Pneumonia, GI disease	Ganciclovir or high-dose acyclovir
Patient with acquired immunodeficiency syndrome	Less than 100 CD4+ cells/µl; CMV seropositivity	Retinitis, GI disease, neurologic disease	Ganciclovir or foscarnet

Treatment

Treatment aims to relieve symptoms and prevent complications. In the immunosuppressed patient, CMV may be treated with acyclovir, ganciclovir, valganciclovir, cidofovir and, possibly, foscarnet. Most important, parents of children with severe congenital CMV infection need support and counseling to help them cope with the possibility of brain damage or death.

Special considerations

To help prevent CMV infection:

- Because many patients who excrete CMV are asymptomatic, standard precautions should be maintained at all times.
- Warn immunosuppressed patients and pregnant women to avoid exposure to confirmed or suspected CMV infection. (Maternal CMV infection can cause fetal abnormalities: hydrocephaly, microphthalmia, seizures, encephalitis, hepatosplenomegaly, hematologic changes, microcephaly, and blindness.)
- Urge patients with CMV infection to use good hand hygiene to prevent spreading it. Stress this particularly with young children.
- Be sure to observe standard precautions when handling body secretions.

Lassa fever

Lassa fever is an epidemic hemorrhagic fever caused by the Lassa virus, an extremely virulent arenavirus. This highly fatal disorder kills 10% to 50% of its victims, but those who survive its early stages usually recover and acquire immunity to secondary attacks.

Causes and incidence

A chronic infection in rodents, Lassa virus is transmitted to humans by contact with infected rodent urine, feces, and saliva. The virus enters the bloodstream, lymph vessels, and respiratory and digestive tracts. It

then multiplies in the cells of the reticuloendothelial system. In the early stages of this illness, when the virus is in the throat, human transmission may occur through inhalation of infected droplets.

As many as 100 cases of Lassa fever occur annually in western Africa; the disease is rare in the United States.

Signs and symptoms

After a 7- to 18-day incubation period, this disease produces a fever that persists for 2 to 3 weeks, exudative pharyngitis, oral ulcers, lymphadenopathy with swelling of the face and neck, purpura, conjunctivitis, and bradycardia. Severe infection may also cause hepatitis, myocarditis, pleural infection, encephalitis, and permanent unilateral or bilateral deafness.

Virus multiplication in reticuloendothelial cells causes capillary lesions that lead to erythrocyte and platelet loss; mild to moderate thrombocytopenia (with a tendency toward bleeding); and secondary bacterial infection. These capillary lesions may also cause focal hemorrhage in the stomach, small intestine, kidneys, lungs, and brain and, possibly, hemorrhagic shock and peripheral vascular collapse.

Diagnosis

CONFIRMING DIAGNOSIS

Isolation of the Lassa virus from throat washings, pleural fluid, or blood confirms the diagnosis.

Recent travel to an endemic area and specific antibody titer support the diagnosis.

Treatment

Treatment of Lassa fever includes I.V. ribavirin, I.V. colloids for shock, analgesics for pain, and antipyretics for fever. Infusion of immune plasma from patients who have recovered from Lassa fever may be useful, but test results on the benefit of this type of therapy are inconclusive.

Special considerations

- Carefully monitor fluid and electrolyte status, vital signs, and intake and output. Watch for and immediately report signs of infection or shock.
- Contact precautions are necessary for at least 3 weeks, until the patient's throat washings and urine are free of the virus. To prevent the spread of this contagious disease, carefully dispose of, or disinfect, all materials contaminated with the infected patient's urine, feces, respiratory secretions, or exudates. Watch known contacts closely for at least 3 weeks for signs of the disease.
- Provide good oral care. Remember to clean the patient's mouth with a softbristled toothbrush to avoid irritating any oral ulcers. Ask your facility's dietary department to supply a soft, bland, nonirritating diet.
- Immediately contact the Viral Diseases Division of the Centers for Disease Control and Prevention in Atlanta to get specific guidelines for managing suspected or confirmed cases of Lassa fever.
- Report all cases of Lassa fever to the local health department.

Ebola virus infection

One of the most frightening viruses to come out of the African subcontinent, the Ebola virus first appeared in 1976. More than 400 people in Zaire (now known as Democratic Republic of Congo) and the neighboring Sudan were killed by the

hemorrhagic fever that it caused. Ebola virus has been responsible for several outbreaks in the years since then, including one in Zaire in the summer of 1995.

An unclassified ribonucleic acid (RNA) virus, Ebola is morphologically similar to the Marburg virus. Both can cause headache, malaise, myalgia, and high fever, progressing to severe diarrhea, vomiting, and internal and external hemorrhage.

Four strains of the Ebola virus are known to exist: Ebola Zaire, Ebola Sudan, Ebola Tai, and Ebola Reston. All four types are structurally similar, although they have different antigenic properties. However, Ebola Reston causes illness only in monkeys, not in humans, as do the other three.

The prognosis for Ebola virus infection is extremely poor, with mortality as high as 90%. The incubation period ranges from 2 to 21 days.

PREVENTION

PREVENTING THE SPREAD OF EBOLA VIRUS

The Center for Disease Control and Prevention recommends the following guidelines to help prevent the spread of this deadly disease:

- Keep the patient on contact precautions throughout the course of the disease.
- If possible, place the patient in a negative-pressure room at the beginning of hospitalization to avoid the need for transfer as the disease progresses.
- Keep frequently used items (such as a thermometer and stethoscope) in the patient's room. Thoroughly disinfect this equipment or discard it upon the patient's discharge or death.
- Restrict nonessential staff members from entering the patient's room.
- Make sure that anyone who enters the patient's room wears gloves and a gown to prevent contact with any surface in the room that may have been soiled.

Causes and incidence

Ebola virus infection is caused by an unclassified RNA virus that's passed from person to person by direct contact with infected blood, body secretions, or organs. Nosocomial and community-acquired transmission can occur. Contaminated needles can also cause the infection. Transmission through semen may occur up to

7 weeks after clinical recovery. The virus remains contagious even after the patient has died. (See *Preventing the spread of Ebola virus.*)

Complications

- Liver and kidney dysfunction
- Dehydration
- Hemorrhage

Signs and symptoms

The patient's health history usually reveals contact with an infected person. However, no clear line of infection may be apparent at the beginning of an Ebola virus outbreak. The patient usually complains of flulike signs and symptoms (such as headache, malaise, myalgia, fever, cough, and sore throat), which first appear within 3 days of infection.

As the virus spreads through the body, inspection reveals bruising as capillaries rupture and dead blood cells infiltrate the skin. A maculopapular eruption appears after the fifth day of infection. The patient may also display melena, hematemesis, epistaxis, and bleeding gums. As the infection progresses, severe complications, including liver and kidney dysfunction, dehydration, and hemorrhage, may develop. In pregnant women, the Ebola virus leads to abortion and massive hemorrhage.

In the final stages of the disease, the skin blisters and sloughs off, blood seeps from all body orifices, and the patient begins vomiting his liquefied internal organs. Death usually results during the second week of illness from organ failure or hemorrhage.

Diagnosis

Specialized laboratory tests reveal specific antigens or antibodies and may show the isolated virus such as an enzyme-linked immunosorbent assay (ELISA) test using EBO-2 viral antigens and Immunoglobulin G ELISA. As with other types of hemorrhagic fever, tests also demonstrate neutrophil leukocytosis, hypofibrinogenemia, thrombocytopenia, and microangiopathic hemolytic anemia.

Treatment

No cure exists for Ebola virus infection; treatment consists mainly of intensive supportive care. Administration of I.V. fluids helps offset the effects of severe dehydration. The patient may receive replacement

of plasma heparin before the onset of clinical shock.

Experimental treatments include the administration of plasma that contains Ebola virus-specific antibodies. Although this treatment has resulted in diminished levels of Ebola virus in the body, further evaluation is needed.

Throughout treatment, the patient should remain on contact precautions. If diagnostic tests indicate that the patient is free from the virus—which typically occurs 21 days after onset in those few who survive—the patient can be released.

Special considerations

- Follow the guidelines for standard precautions formulated by the Centers for Disease Control and Prevention (CDC) when assessing a patient who may have Ebola virus.
- Check the results of complete blood count and coagulation studies for signs of blood loss and coagulopathy.
- Assess the patient daily for petechiae, ecchymoses, and oozing blood. Note and document the size of ecchymoses at least every 24 hours.
- Protect all areas of petechiae and ecchymoses from further injury.
- Test stools, urine, and vomitus for occult blood.
- Watch for frank bleeding, including GI bleeding and, in women, menorrhagia. Note and document the amount of bleeding every 24 hours or more often.
- Monitor the patient's family and other close contacts for fever and other signs of infection.
- Provide emotional support for the patient and his family during the course of this devastating disease. Encourage them to ask questions and discuss any concerns they have about the disease and its treatment.

West Nile encephalitis

West Nile encephalitis is categorized as an infectious disease that primarily causes an inflammation or “encephalitis” of the brain. West Nile virus (WNV), a flavivirus commonly found in humans, birds, and other vertebrates in Africa, west Asia, and the Middle East, causes the disease, which is a part of a family of vector-borne diseases that also includes malaria, yellow fever, and Lyme disease.

The virus had not been previously documented in the Western Hemisphere until late August 1999. A virus found in numerous dead birds in New York, New Jersey, and Connecticut was definitively identified by genetic sequencing as the West Nile virus. Scientists in the United States discovered the rare strain initially in and

around the Bronx Zoo and believe that infected birds may have carried the disease and that it was spread as mosquitoes fed on them.

In the temperate areas of the world, West Nile encephalitis cases occur mainly in the late summer or early fall. In the southern climates where temperatures are milder, West Nile encephalitis can occur all year round.

The risk of contracting West Nile encephalitis is greater for all residents of areas where active cases have been identified, but persons older than age 50 or those with compromised immune systems have the greatest risk. At present, there is no documented evidence that a pregnant woman's fetus is at risk due to an infection with WNV. The mortality rate of West Nile encephalitis is measured by case-fatality rates, which range from 3 % to 15 % (higher in the elderly population.)

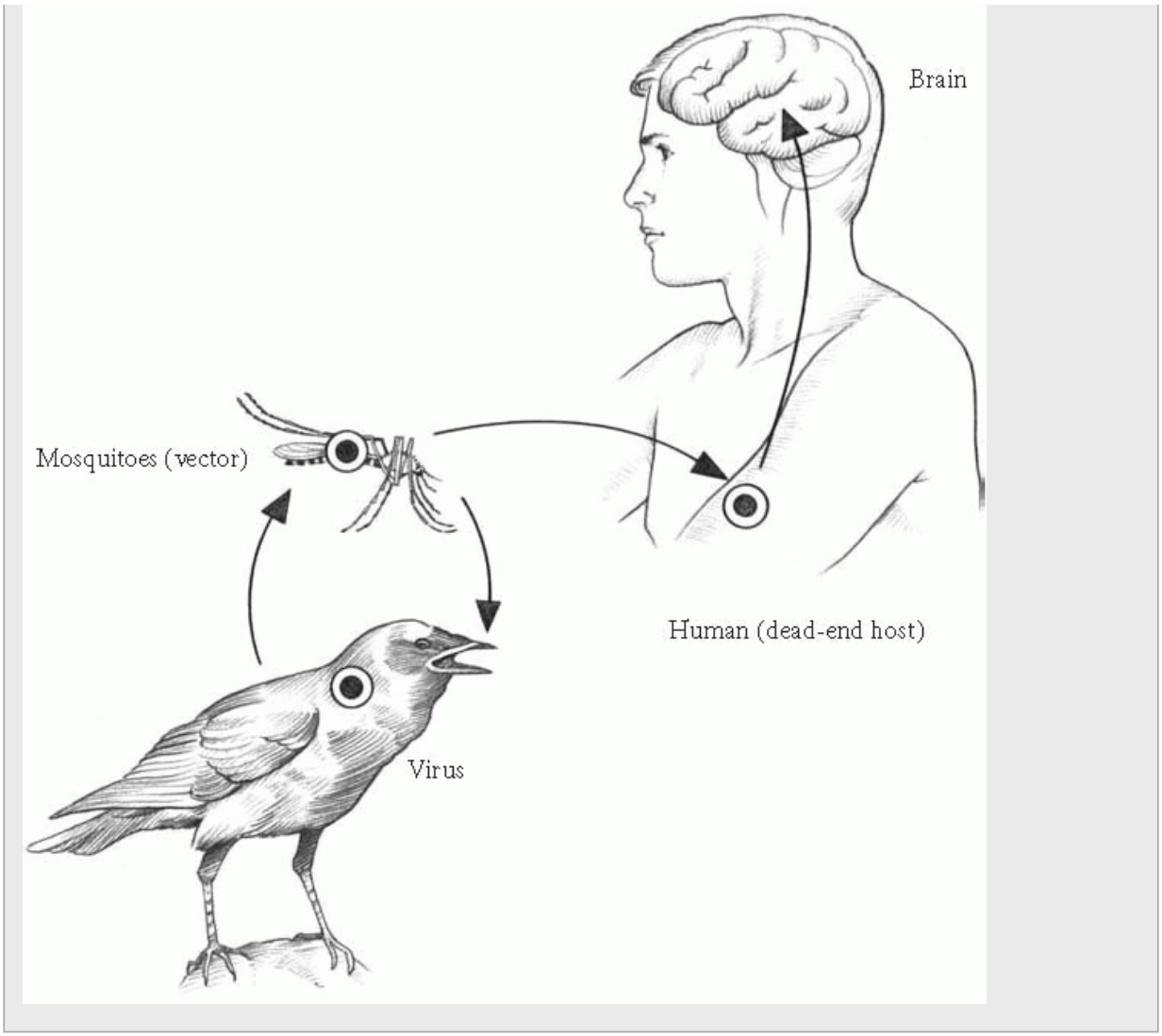
Causes and incidence

WNV is transmitted to humans by the bite of a mosquito (primarily the *Culex* species) infected with the virus. It's considered the primary vector for WNV and the source of the August 1999 outbreak in New York, New Jersey, and Connecticut. Mosquitoes become infected by feeding on birds contaminated with the West Nile virus and then transmitting it to humans and animals during a blood meal or "bite." (See *Transmission routes of West Nile virus.*)

Ticks have been found infected with WNV in Africa and Asia only. The role of ticks in the transmission and maintenance of the virus remains uncertain, and to date they aren't considered vectors for WNV in the United States.

TRANSMISSION ROUTES OF WEST NILE VIRUS

Birds are the reservoir of the West Nile virus. They harbor the virus but are unable to spread it. Mosquitoes serve as the vectors, spreading it from bird to bird and from birds to people. Humans are believed to be "dead-end hosts" because the virus can live and cause illness in humans, but it isn't believed that a feeding mosquito can acquire the virus from an infected person.



The Centers for Disease Control and Prevention has reported that there is no evidence that a person can contract the virus from handling live or dead infected birds. However, avoid barehanded contact when handling dead animals, including birds, and use gloves or double plastic bags to dispose of a carcass. Report the finding to the local health department.

Complications

- Tremors
- Convulsions
- Paralysis
- Coma
- Death (rare)

Signs and symptoms

Mild infections of the virus are more common and include fever, headache, and body aches, usually accompanied by a skin rash and swollen lymph glands. Severe infections can be manifested by headache, high fever, neck stiffness, stupor, disorientation, coma, tremors, occasional convulsions, paralysis and, rarely, death.

The incubation period for West Nile encephalitis is anywhere from 5 to 15 days after exposure. Most patients who are bitten by an infected mosquito won't develop symptoms. It's estimated that only 1 in 300 people who are bitten by an infected mosquito will actually get sick.

PREVENTION

PREVENTING WEST NILE VIRUS

To reduce your patient's risk for becoming infected with West Nile encephalitis, advise him to do the following:

- Stay indoors at dawn, dusk, and in the early evening.
- Wear long-sleeved shirts and long pants whenever he is outdoors.
- Apply insect repellent sparingly to exposed skin. (Effective repellents contain 20% to 30% DEET [N,N-diethyltoluamide]. DEET in high concentrations [greater than 30%] can cause adverse effects, particularly in children; avoid products containing more than 30% DEET.)
- Avoid applying repellant to the hands of children; they may irritate the eyes and mouth. Insect repellants shouldn't be applied to children younger than age 3 years.
- Spray clothing with DEET-containing repellents because mosquitoes may bite through thin clothing.
- Read and follow the manufacturer's directions for insecticide and insect repellent use.

Diagnosis

The immunoglobulin (Ig) M antibody capture enzyme-linked immunosorbent assay (MAC-ELISA) is the test of choice for rapid definitive diagnosis. The major advantage of MAC-ELISA laboratory analysis is the high probability of accurate

diagnosis of WNV infection when performed with acute serum or cerebrospinal fluid specimens obtained while the patient is still hospitalized.

A new diagnostic test, the WNV MAC-ELISA, was recently approved by the Food and Drug Administration. This test detects levels of IgM antibodies in a patient's serum and is intended for use in patients with clinical symptoms consistent with viral encephalitis.

Other conditions to consider include St. Louis encephalitis, which is symptomatically similar.

Encephalitis can be caused by numerous viral and bacterial infections; all data must be examined to determine a definitive diagnosis.

Treatment

There is no specific therapy utilized to treat West Nile encephalitis and no known cure. Treatment is generally aimed at controlling the specific symptoms. Supportive care, such as I.V. fluids, fever control, and respiratory support, is rendered when necessary.

There is no vaccine present to prevent the transmission of West Nile encephalitis. Research trials are underway to determine if ribavirin, an antiviral drug, may be helpful.

Special considerations

- Obtain an extensive history of the patient's whereabouts within the past 2 to 3 weeks (especially around bodies of water, such as lakes and ponds), the presence of dead birds, and recent mosquito bites acquired.
- Perform a comprehensive physical assessment and report signs of fever, headache, lymphadenopathy, and a maculopapular rash.
- Perform a complete neurological examination and report any signs of confusion, lethargy, weakness, or slurred speech.
- Maintain adequate hydration with I.V. fluids.
- Monitor strict intake and output.
- Use fever control methods, such as cooling blankets and acetaminophen as ordered.
- Provide respiratory support measures when applicable.

- West Nile encephalitis isn't transmitted from person to person, but use standard precautions when handling body fluids and blood.

- Report any suspected cases of West Nile encephalitis to the applicable state health department.
- Teach the patient ways to reduce his risk of becoming infected with West Nile encephalitis. (See *Preventing West Nile virus.*)

RICKETTSIA

Rocky Mountain spotted fever

Rocky Mountain spotted fever (RMSF) is a febrile, rash-producing illness caused by *Rickettsia rickettsii*. The disease is transmitted to humans by a tick bite.

RMSF is fatal in about 5% of patients. Mortality rises when treatment is delayed and in older patients.

Causes and incidence

R. rickettsii is transmitted to a human or small animal by the prolonged bite (4 to 6 hours) of an adult tick—the wood tick (*Dermacentor andersoni*) in the west and by the dog tick (*Dermacentor variabilis*) in the east. Occasionally, it's acquired through inhalation (it can occur in laboratory settings where aerosolization of blood and specimens may occur) or through the contact of abraded skin with tick excreta or tissue juices. (This explains why people shouldn't crush ticks between their fingers when removing them from other people and animals.) In most tick-infested areas, 1% to 5% of the ticks harbor *R. rickettsii*.

Endemic throughout the continental United States, RMSF is particularly prevalent in the southeast and southwest. Because RMSF is associated with outdoor activities, such as camping and backpacking, the incidence of this illness is usually higher in the spring and summer. Epidemiologic surveillance reports for RMSF indicate that the incidence is also higher in children ages 5 to 9, men and boys, and whites.

Complications

- Lobar pneumonia
- Pneumonitis
- Otitis media
- Parotitis
- Disseminated intravascular coagulation (DIC)
- Shock
- Renal failure

- Meningoencephalitis
- Hepatic injury

Signs and symptoms

The incubation period is usually about 7 days, but it can range from 2 to 14 days. Generally, the shorter the incubation time, the more severe the infection. Signs and symptoms, which usually begin abruptly, include a persistent temperature of 102° to 104° F (38.9° to 40° C); a generalized, excruciating headache; nausea and vomiting; and aching in the bones, muscles, joints, and back. In addition, the tongue is covered with a thick white coating that gradually turns brown as the fever persists and rises.

Initially, the skin may simply appear flushed. Between days 2 and 5, eruptions begin around the wrists, ankles, or forehead; within 2 days, they cover the entire body, including the scalp, palms, and soles. The rash consists of erythematous macules 1 to 5 mm in diameter that blanch on pressure; if untreated, the rash may become petechial and maculopapular. By the third week, the skin peels off and may become gangrenous over the elbows, fingers, and toes.

The pulse is strong initially, but it gradually becomes rapid (possibly reaching 150 beats/minute) and thready.

ALERT

A rapid pulse rate and hypotension (systolic pressure less than 90 mm Hg) herald imminent death from complete vascular collapse.

Other signs and symptoms include a bronchial cough, a rapid respiratory rate (as high as 60 breaths/minute), anorexia, constipation, abdominal pain, hepatomegaly, splenomegaly, insomnia, restlessness and, in extreme cases, delirium. Urine output falls to half of the normal level or less, is dark in color, and contains albumin.

Diagnosis

CONFIRMING DIAGNOSIS

Diagnosis is usually based on a history of tick bite or travel to a tick-infested area and a positive complement fixation test (which shows a fourfold increase in convalescent antibody titer compared with acute titers). Blood cultures or skin biopsy at the

rash site should be performed to isolate the organism and confirm the diagnosis.

Another common but less reliable antibody test is the Weil-Felix reaction, which also shows a fourfold increase between the acute and convalescent sera titer levels. Increased titers usually develop after 10 to 14 days and persist for several months.

Additional recommended laboratory tests consist of a platelet count for thrombocytopenia (12,000 to 150,000/µl) and a white blood cell count (elevated to 11,000 to 33,000/µl) during the second week of illness.

Treatment

Treatment requires careful removal of the tick and administration of antibiotics, such as chloramphenicol or tetracycline (preferably doxycycline), until 3 days after the fever subsides. Treatment also includes symptomatic measures and, in DIC, heparin and platelet transfusion.

Special considerations

- Carefully monitor the patient's intake and output. Watch closely for decreased urine output—a possible indicator of renal failure.
- Be alert for signs of dehydration, such as poor skin turgor and dry mouth.
- Administer antipyretics as ordered, and provide tepid sponge baths to reduce fever.
- Monitor vital signs, and watch for profound hypotension and shock.
- Be prepared to administer oxygen therapy and assisted ventilation if pulmonary complications develop.
- Turn the patient frequently to prevent complications of immobility, such as pressure ulcers and pneumonia.
- Pay attention to the patient's nutritional needs; vomiting may indicate a need for parenteral nutrition or for scheduling frequent small meals.
- Instruct the patient to report any recurrent symptoms to the physician at once so that treatment measures may resume immediately.

PREVENTION

- *Advise the patient to avoid tick-infested areas (woods, meadows, streams, and canyons) if possible.*
- *Teach the patient ways to reduce his risk of becoming infected with Rocky Mountain spotted fever:*

– Encourage him to inspect his entire body (including his scalp) every 3 to 4 hours for attached ticks.

– Remind him to wear protective clothing, such as a long-sleeved shirt, pants securely tucked into laced boots, and a protective head covering such as a cap.

– Advise him to apply insect repellant to exposed skin as well as to clothing.

- Offer printed and illustrated instructions, if available, to teach the patient and his family members or other caregivers how to correctly and safely remove a tick. Demonstrate how to use tweezers or forceps and apply steady traction to release the entire tick without leaving its mouth parts in the skin.
- After the patient removes the tick, caution him not to handle it or its fragments.
- Finally, instruct the patient to clean his skin with alcohol at the point of attachment.
- Report all cases to the appropriate health department.

PROTOZOA

Pneumocystis carinii (jiroveci) pneumonia

Because of its association with human immunodeficiency virus (HIV) infection, *Pneumocystis carinii* (jiroveci) pneumonia (PCP), an opportunistic infection, has increased in incidence since the 1980s. Before the advent of PCP preventative, this disease was the first clue in about 60% of patients that HIV infection was present.

PCP was the leading cause of death in these patients. Preventive therapy with cotrimoxazole in HIV patients with low immune function has prevented PCP from higher mortality rates. Disseminated infection doesn't occur.

PCP is also associated with other immunocompromising conditions, including organ transplantation, leukemia, lymphoma, and steroid use.

Causes and incidence

P. carinii (jiroveci), the cause of PCP, usually is classified as a protozoan, although some investigators consider it more closely related to fungi. The organism exists as a saprophyte in the lungs of humans and various animals as part of the normal

flora in most healthy people. It becomes an aggressive pathogen in the immunocompromised patient. Impaired cell-mediated (T-cell) immunity is thought to be more important than impaired humoral (B-cell) immunity in predisposing the patient to PCP, but the immune defects involved are poorly understood. *P. carinii* becomes activated in immunocompromised patients when the CD4+ T-cell count falls below 200/µl.

P. carinii (jiroveci) invades the lungs bilaterally and multiplies extracellularly. As the infestation grows, alveoli fill with organisms and exudate, impairing gas exchange. The alveoli hypertrophy and thicken progressively, eventually leading to extensive consolidation.

The primary transmission route seems to be air, although the organism is already present in most people. The incubation period probably lasts for 4 to 8 weeks.

Complications

- Pulmonary insufficiency
- Death

Signs and symptoms

The patient typically has a history of an immunocompromising condition (such as HIV infection, leukemia, or lymphoma) or procedure (such as organ transplantation).

PCP begins insidiously with increasing shortness of breath and a nonproductive cough. Anorexia, generalized fatigue, and weight loss may follow. Although the patient may have hypoxemia and hypercapnia, he may not exhibit significant symptoms. He may, however, have a low-grade, intermittent fever.

Other signs and symptoms include tachypnea, dyspnea, accessory muscle use for breathing, crackles (in about one-third of patients), marked pallor, and decreased breath sounds (in advanced pneumonia). Cyanosis may appear with acute illness; pulmonary consolidation develops later.

Diagnosis

CONFIRMING DIAGNOSIS

Histologic studies confirm P. carinii *(jiroveci) in all patients. Fiber-optic bronchoscopy remains the most commonly used study to confirm PCP. Invasive procedures, such as transbronchial biopsy and open-lung biopsy, are performed less commonly.*

In patients with HIV infection, initial examination of a first-morning sputum specimen (induced by inhaling an ultrasonically dispersed saline mist) may be

sufficient; however, this technique usually is ineffective in patients without HIV infection.

Chest X-rays may show slowly progressing, fluffy infiltrates and, occasionally, nodular lesions or a spontaneous pneumothorax, but these findings must be differentiated from findings in other types of pneumonia or acute respiratory distress syndrome.

Gallium scan may show increased uptake over the lungs even when the chest X-ray appears relatively normal. Arterial blood gas (ABG) studies detect hypoxia and an increased A-a gradient.

Treatment

PCP may respond to drug therapy with cotrimoxazole. Other agents used to treat PCP include pentamidine, trimethoprimdapsone, clindamycin, primaquine, and atovaquone. Corticosteroids are frequently used as well. However, because of immune system impairment, many patients with PCP, who also have HIV, experience severe adverse reactions to drug therapy.

Supportive measures, such as oxygen therapy, mechanical ventilation, adequate nutrition, and fluid balance, are important

adjunctive therapies. Oral morphine sulfate solution may reduce the respiratory rate and anxiety, thereby enhancing oxygenation.

Special considerations

- Implement standard precautions to prevent contagion.
- Frequently assess the patient's respiratory status, and monitor ABG levels every 4 hours.
- Administer oxygen therapy as ordered. Encourage the patient to ambulate as well as to perform deep-breathing exercises and incentive spirometry to facilitate effective gas exchange.
- Administer antipyretics as ordered, to relieve fever.
- Monitor the patient's intake and output and daily weight to evaluate fluid balance. Replace fluids as ordered.
- Provide diversionary activities and coordinate health care team activities to allow adequate rest periods between procedures.
- Teach the patient energy conservation techniques.
- Supply nutritional supplements as needed. Encourage the patient to eat a high-calorie, protein-rich diet. Offer small, frequent meals if the patient cannot

tolerate large amounts of food.

- Reduce anxiety by providing a relaxing environment, eliminating excessive environmental stimuli, and allowing ample time for meals.
- Give emotional support and help the patient identify and use meaningful support systems.
- Instruct the patient about the medication regimen, especially about the adverse effects.
- Emphasize the importance of continuing chemoprophylaxis for those patients at high risk of developing PCP.
- If the patient will require oxygen therapy at home, explain that an oxygen concentrator may be most effective.
- Because this infection is usually associated with acquired immunodeficiency syndrome (AIDS), provide the patient with resources and support organizations for both AIDS and HIV.

Malaria

Malaria, an acute infectious disease, is caused by protozoa of the genus *Plasmodium: P. falciparum, P. vivax, P. malariae,* and *P. ovale,* all of which are transmitted to humans by mosquito vectors. Falciparum malaria is the most severe form of the disease. When treated, malaria is rarely fatal; untreated, it's fatal in 10% of victims, usually as a result of complications such as disseminated intravascular coagulation (DIC).

Untreated primary attacks last from a week to a month, or longer. Relapses are common and can recur sporadically for several years. Susceptibility to the disease is universal.

Causes and incidence

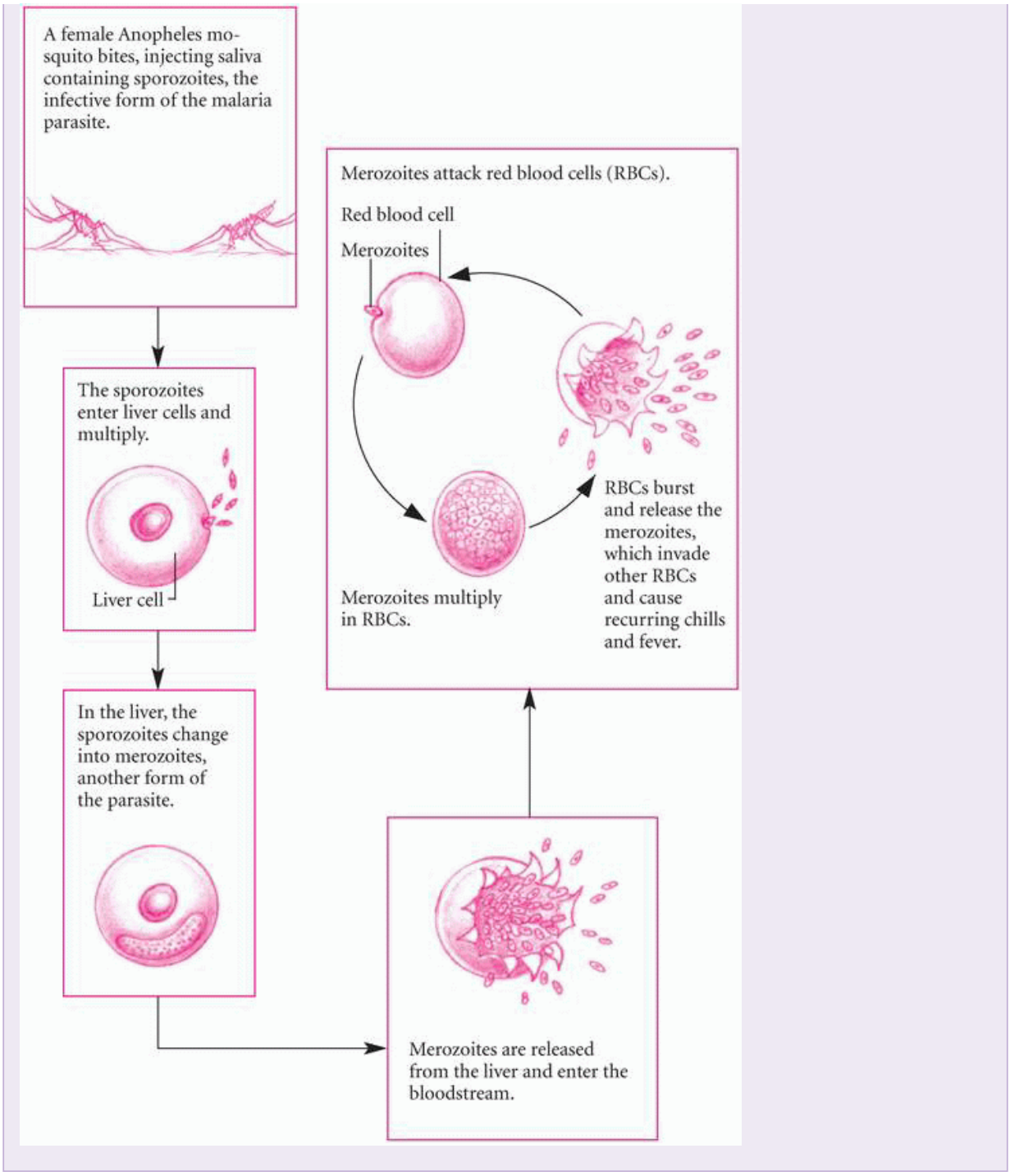
Malaria literally means “bad air” and for centuries was thought to result from the inhalation of swamp vapors. It's now known that malaria is transmitted by the bite of female *Anopheles* mosquitoes, which abound in humid, swampy areas. When an infected mosquito bites, it injects *Plasmodium* sporozoites into the wound. The infective sporozoites migrate by blood circulation to parenchymal cells of the liver; there they form cystlike structures containing thousands of merozoites.

Upon release, each merozoite invades an erythrocyte and feeds on hemoglobin. Eventually, the erythrocyte ruptures, releasing heme (malaria pigment), cell debris, and more merozoites, which, unless destroyed by phagocytes, enter other erythrocytes. (See *What happens in malaria.*) At this point, the infected person becomes a reservoir of malaria who infects any mosquito that feeds on him, thus beginning a new cycle of transmission. Hepatic parasites (*P. vivax, P. ovale,* and *P.*

malariae) may persist for years in the liver. These parasites are responsible for the chronic carrier state. Because blood transfusions and street-drug paraphernalia can also spread malaria, drug addicts have a higher incidence of the disease. Malaria is a worldwide health problem that continues to impede the development of many countries.

Malaria is a tropical and subtropical disease. It's most prevalent in Asia, Africa, and Latin America. The Centers for Disease Control and Prevention (CDC) estimates 300 to 500 million cases occur each year, with more than 1 million resulting in death. It's the greatest disease hazard for travelers in warm climates.

PATHOPHYSIOLOGY
WHAT HAPPENS IN MALARIA



Complications

- Renal failure
- Liver failure

- Heart failure
- Pulmonary edema
- DIC
- Seizures
- Hypoglycemia
- Splenic rupture
- Cerebral dysfunction
- Death

Signs and symptoms

After an incubation period of 12 to 30 days, malaria produces chills, fever, cough, fatigue, headache, and myalgia, interspersed with periods of well-being (the hallmark of the benign form of malaria). Acute attacks (paroxysms) occur when erythrocytes rupture. There are three stages:

- *cold stage*, lasting 1 to 2 hours, ranging from chills to extreme shaking
- *hot stage*, lasting 3 to 4 hours, characterized by a high fever (up to 107° F [41.7° C])
- *wet stage*, lasting 2 to 4 hours and characterized by profuse sweating.

Paroxysms occur every 48 to 72 hours when malaria is caused by *P. malariae* and every 42 to 50 hours when malaria is caused by *P. vivax* or *P. ovale*. All three types have low levels of parasitosis and are self-limiting as a result of early acquired immunity.

P. vivax and *P. ovale* also produce hepatosplenomegaly. Hemolytic anemia is present in all but the mildest infections.

The most severe form of malaria, which causes the most morbidity and mortality, is caused by *P. falciparum*. This species produces persistent high fever, orthostatic hypotension, and red blood cell (RBC) sludging that leads to capillary obstruction at various sites. Signs and symptoms of obstruction include:

- *cerebral*—hemiplegia, seizures, delirium, and coma
- *pulmonary* — coughing and hemoptysis
- *splanchnic* — vomiting, abdominal pain, diarrhea, and melena
- *renal* — oliguria, anuria, and uremia.

During blackwater fever (a complication of *P. falciparum* infection), massive intravascular hemolysis causes jaundice, hemoglobinuria, a tender and enlarged

spleen, acute renal failure, and uremia. This complication is fatal in about 20% of patients.

Diagnosis

CONFIRMING DIAGNOSIS

A history showing travel to endemic areas, recent blood transfusion, or drug abuse in a person with high fever of unknown origin strongly suggests malaria. However, because symptoms of malaria mimic other diseases, unequivocal diagnosis depends on laboratory identification of the parasites in RBCs of peripheral blood smears.

The CDC can identify donors responsible for transfusion malaria through indirect fluorescent serum antibody tests. These tests are unreliable in the acute phase because antibodies can be undetectable for 2 weeks after onset.

Supplementary laboratory values that support this diagnosis include decreased hemoglobin levels, normal to decreased leukocyte count (as low as 3,000/µl), and protein and leukocytes in urine sediment. In falciparum malaria, serum values reflect DIC: reduced number of platelets (20,000 to 50,000/µl); prolonged prothrombin time (18 to 20 seconds); prolonged partial thromboplastin time (60 to 100 seconds); and decreased plasma fibrinogen.

Treatment

Malaria is best treated with oral chloroquine in all forms except chloroquineresistant *P. falciparum*. Symptoms and parasitemia decrease within 24 hours after such therapy begins, and the patient usually recovers within 3 to 4 days. If the patient is comatose or vomiting frequently, chloroquine is given I.M.

Malaria caused by *P. falciparum*, which is resistant to chloroquine, requires treatment with oral quinine given concurrently with pyrimethamine and a sulfonamide such as sulfadiazine. Mefloquine can also be used for resistant *P. falciparum*. Relapses require the same treatment, or quinine alone, followed by tetracycline.

SPECIAL CONSIDERATIONS FOR ANTIMALARIAL DRUGS
Chloroquine

- Perform baseline and periodic ophthalmologic examinations, and report blurred vision, increased sensitivity to light, and muscle weakness to the physician.

- Consult with the physician about altering therapy if muscle weakness appears in a patient on long-term therapy.
- Monitor the patient for tinnitus and other signs of ototoxicity, such as nerve deafness and vertigo.
- Caution the patient to avoid excessive exposure to the sun to prevent exacerbating drug-induced dermatoses.

Primaquine

- Give with meals or antacids.
- Halt administration if you observe a sudden fall in hemoglobin concentration or in erythrocyte or leukocyte count or marked darkening of the urine, suggesting impending hemolytic reaction.

Pyrimethamine

- Administer with meals to minimize GI distress.
- Check blood counts (including platelets) twice per week. If signs of folic or folinic acid deficiency develop, reduce or discontinue dosage while the patient receives parenteral folinic acid until blood counts become normal.

Quinine

- Use with caution in patients with cardiovascular conditions. Discontinue dosage if you see any signs of idiosyncrasy or toxicity, such as headache, epigastric distress, diarrhea, rashes, and pruritus, in a mild reaction; or delirium, seizures, blindness, cardiovascular collapse, asthma, hemolytic anemia, and granulocytosis, in a severe reaction.
- Monitor blood pressure frequently while administering quinine I.V. infusion. Rapid administration causes marked hypotension.

The only drug effective against the hepatic stage of the disease that's available in the United States is primaquine. This drug can induce hemolytic anemia, especially in patients with glucose-6-phosphate dehydrogenase deficiency. (See *Special considerations for antimalarial drugs.*)

For travelers spending less than 3 weeks in areas where malaria exists, weekly prophylaxis includes oral chloroquine beginning 2 weeks before the trip and ending 6 weeks after it. (See *How to prevent malaria,* page 1022.) Any traveler who develops an acute febrile illness should seek prompt medical attention, regardless of the prophylaxis taken.

Special considerations

- Obtain a detailed patient history, noting any recent travel, foreign residence, blood transfusion, or drug addiction. Record symptom pattern, fever, type of malaria, and any systemic signs.
- Assess the patient on admission and daily thereafter for fatigue, fever, orthostatic hypotension, disorientation, myalgia, and arthralgia. Enforce bed rest during periods of acute illness.
- Protect the patient from secondary bacterial infection by following proper hand-washing and sterile techniques.
- Protect yourself by wearing gloves when handling blood or body fluids.
- Activate safety devices, and use safety syringes in practice.
- Discard needles and syringes in an impervious container designated for incineration.
- Handle bed linens according to standard precautions.
- To reduce fever, administer antipyretics as ordered. Document onset, duration, and symptoms before and after episodes.
- Fluid balance is fragile, so keep a strict record of intake and output. Monitor I.V. fluids closely. Avoid fluid overload (especially in *P. falciparum*), because it can lead to pulmonary edema and aggravate cerebral symptoms. Observe blood chemistry levels for hyponatremia and increased blood urea nitrogen, creatinine, and bilirubin levels. Monitor urine output hourly, and maintain it at 40 to 60 ml/hour for an adult and at 15 to 30 ml/hour for a child. Immediately report any decrease in urine output or the onset of hematuria as a possible sign of renal failure; be prepared to perform peritoneal dialysis for uremia caused by renal failure.

PREVENTION

HOW TO PREVENT MALARIA

Advise patients on these actions that should be taken to help prevent malaria:

- Drain, fill, and eliminate breeding areas of the *Anopheles* mosquito.
- Install screens in living and sleeping quarters in endemic areas.
- Use a residual insecticide on clothing and skin to prevent mosquito bites.

- Seek treatment for known cases.
- Question blood donors about a history of malaria or possible exposure to malaria.

They *may* give blood if:

– they haven't taken any antimalarial drugs and are asymptomatic after 6 months outside an endemic area

– they were asymptomatic after treatment for malaria more than 3 years ago

– they were asymptomatic after receiving malaria prophylaxis more than 3 years ago.

- Seek preventive drug therapy before traveling to an endemic area. Agents include mefloquine, doxycycline, chloroquine, hydroxychloroquine, or malarone (a combination of atovaquone and proguanil). They're usually started 2 weeks before visiting the endemic area and continue for 6 weeks after leaving the area.

- Slowly administer packed RBCs or whole blood while checking for crackles, tachycardia, and shortness of breath.
- If humidified oxygen is ordered, note the patient's response, particularly any changes in rate or character of respirations, or any improvement in mucous membrane color.
- Watch for and immediately report signs of internal bleeding, such as tachycardia, hypotension, and pallor.
- Encourage frequent coughing and deep breathing, especially if the patient is on bed rest or has pulmonary complications. Record the amount and color of sputum.
- Watch for adverse effects of drug therapy, and take measures to relieve them.
- If the patient is comatose, make frequent, gentle changes in his position, and give passive range-of-motion exercises every 3 to 4 hours. If the patient is unconscious or disoriented, use restraints as needed, and keep an airway available as appropriate.
- Provide emotional support and reassurance, especially in critical illness. Explain the procedures and treatment to the patient and his family. Suggest that other family members be tested for malaria. Emphasize the need for follow-up care to check the effectiveness of treatment and to manage residual problems.
- Report all cases of malaria to local public health authorities.

Amebiasis

Amebiasis, also known as *amebic dysentery,* is an acute or chronic protozoal infection caused by *Entamoeba histolytica*. This infection produces varying degrees of illness, from no symptoms at all or mild diarrhea to fulminant dysentery. Extraintestinal amebiasis can induce hepatic abscess and infections of the lungs, pleural cavity, pericardium, peritoneum and, rarely, the brain.

The prognosis is generally good, although complications — such as ameboma, intestinal stricture, hemorrhage or perforation, intussusception, or abscess — increase mortality. Brain abscess, a rare complication, is usually fatal.

Causes and incidence

E. histolytica exists in two forms: a cyst (which can survive outside the body) and a trophozoite (which can't survive outside the body). Transmission occurs through ingesting feces-contaminated food or water. The ingested cysts pass through the intestine, where digestive secretions break down the cysts and liberate the motile trophozoites within. The trophozoites multiply and either invade and ulcerate the mucosa of the large intestine or simply feed on intestinal bacteria. As the trophozoites are carried slowly toward the rectum, they are encysted and then excreted in feces. Humans are the principal reservoir of infection.

Amebiasis occurs worldwide but is most common in the tropics, subtropics, and other areas with poor sanitation and health practices. Incidence in the United States averages between 1% and 3% but may be higher among homosexuals and institutionalized people, in whom fecal-oral contamination is common.

Complications

- Chronic diarrhea
- Abdominal pain
- Extraintestinal abscesses
- Ameboma
- Megacolon
- Intussusception
- Intestinal stricture
- Intestinal hemorrhage
- Intestinal perforation

Signs and symptoms

The clinical effects of amebiasis vary with the severity of the infestation. *Acute amebic dysentery* causes a sudden high temperature of 104° to 105° F (40° to 40.6° C) accompanied by chills and abdominal cramping; profuse, bloody, mucoid diarrhea with tenesmus; and diffuse abdominal tenderness due to extensive rectosigmoid ulcers.

Chronic amebic dysentery produces intermittent diarrhea that lasts for 1 to 4 weeks and recurs several times a year. Such diarrhea produces 4 to 8 (or, in severe diarrhea, up to 18) foul-smelling mucus- and blood-tinged stools daily in a patient with a mild fever, vague abdominal cramps, possible weight loss, tenderness over the cecum and ascending colon and, occasionally, hepatomegaly. Amebic granuloma (ameboma), commonly mistaken for cancer, can be a complication of the chronic infection. Amebic granuloma produces blood and mucus in the stool and, when granulomatous tissue covers the entire circumference of the bowel, causes partial or complete obstruction.

Parasitic and bacterial invasion of the appendix may produce typical signs of subacute appendicitis (abdominal pain and tenderness). Occasionally, *E. histolytica* perforates the intestinal wall and spreads to the liver. When it perforates the liver and diaphragm, it spreads to the lungs, pleural cavity, peritoneum and, rarely, the brain.

Diagnosis

CONFIRMING DIAGNOSIS

Isolating E. histolytica *(cysts and trophozoites) in fresh feces or aspirates from abscesses, ulcers, or tissue confirms acute amebic dysentery.*

Diagnosis must distinguish between cancer and ameboma with X-rays, sigmoidoscopy, stool examination for amebae, and cecum palpation. In patients with amebiasis, exploratory surgery is hazardous; it can lead to peritonitis, perforation, and pericecal abscess.

Other laboratory tests that support the diagnosis of amebiasis include:

- indirect hemagglutination test — positive with current or previous infection
- complement fixation — usually positive only during active disease
- barium studies — rule out nonamebic causes of diarrhea, such as polyps and cancer
- sigmoidoscopy — detects rectosigmoid ulceration; a biopsy may be helpful.

Patients with amebiasis shouldn't have preparatory enemas because these may remove exudates and destroy the trophozoites, thus interfering with test results.

Treatment

Drugs used to treat amebic dysentery include metronidazole, an amebicide at intestinal and extraintestinal sites; emetine hydrochloride, also an amebicide at intestinal and extraintestinal sites, including the liver and lungs; iodoquinol (diiodohydroxyquin), an effective amebicide for asymptomatic carriers; chloroquine, for liver abscesses, not intestinal infections; and tetracycline (in combination with emetine hydrochloride, metronidazole, or paromomycin), which supports the antiamebic effect by destroying intestinal bacteria on which the amebae normally feed.

When nausea and vomiting are present, I.V. therapy may be necessary until medications are tolerated by mouth.

Special considerations

- Tell patients with amebiasis to avoid drinking alcohol when taking metronidazole. The combination may cause nausea, vomiting, and headache.
- Antidiarrheals aren't prescribed and can make the condition worse.
- After treatment, stools should be rechecked to make sure the infection has been cleared.

Giardiasis

Giardiasis (also called *Giardia enteritis* or *lambliasis*) is an infection of the small bowel caused by the symmetrical flagellate protozoan *Giardia lamblia*. A mild infection may not produce intestinal symptoms. In untreated giardiasis, symptoms wax and wane; with treatment, recovery is complete.

Causes and incidence

G. lamblia has two stages: the cystic stage and the trophozoite stage. Ingestion of *G. lamblia* cysts in fecally contaminated water or the fecal-oral transfer of cysts by an infected person results in giardiasis. Giardiasis may be transmitted through sexual contact (direct or indirect fecal-oral contact). When cysts enter the small bowel, they become trophozoites and attach themselves with their sucking disks to the bowel's epithelial surface. After this, the trophozoites encyst again, travel down the colon, and are excreted. Unformed feces that pass quickly through the intestine may contain trophozoites as well as cysts.

Giardiasis occurs worldwide but is most common in developing countries and other areas where sanitation and hygiene are poor. In the United States, giardiasis is most common in travelers who have recently returned from endemic areas, and in campers who drink unpurified water from contaminated streams. Probably because of frequent hand-to-mouth activity, children are more likely to become infected with *G. lamblia* than adults. Hypogammaglobulinemia also appears to predispose people to this disorder. Giardiasis doesn't confer immunity, so reinfections may occur.

Complications

- Malabsorption
- Dehydration
- Lactose intolerance

Signs and symptoms

Attachment of *G. lamblia* to the intestinal lumen causes superficial mucosal invasion and destruction, inflammation, and irritation. All of these destructive effects decrease food transit time through the small intestine and result in malabsorption. Such malabsorption produces chronic GI complaints — such as abdominal cramps — and pale, loose, greasy, malodorous, and frequent stools (from 2 to 10 daily) with concurrent nausea. Stools may contain mucus but not pus or blood. Chronic giardiasis may produce fatigue and weight loss in addition to these typical signs and symptoms.

Diagnosis

Suspect giardiasis when travelers to endemic areas or campers who may have drunk unpurified water develop symptoms.

CONFIRMING DIAGNOSIS

Actual diagnosis requires laboratory examination of a fresh stool specimen for cysts or examination of duodenal aspirate for trophozoites. An antibody test of the stool for giardiasis is also very effective in diagnosis. A small bowel biopsy shows Giardia.

A barium X-ray of the small bowel may show mucosal edema and barium segmentation.

Diagnosis must also rule out other causes of diarrhea and malabsorption.

Treatment

Giardiasis responds readily to a 10-day course of metronidazole or a 7-day course of oral quinacrine and furazolidone. Severe diarrhea may require parenteral fluid replacement to prevent dehydration if oral fluid intake is inadequate.

Special considerations

- Inform the patient receiving metronidazole of the possible adverse effects of this drug: commonly, headache, anorexia, and nausea and, less commonly, vomiting, diarrhea, and abdominal cramps. Warn against drinking alcoholic beverages, which may provoke a disulfiram-like reaction. If the patient is a woman, ask if she's pregnant because metronidazole is contraindicated during pregnancy.
- When talking to family members and other suspected contacts, emphasize the importance of stool examinations for *G. lamblia* cysts.
- If hospitalization is required, use standard precautions. A child or an incontinent adult requires a private room. Pay strict attention to hand hygiene, particularly after handling feces. Quickly dispose of fecal material. (Normal sewage systems can remove and process infected feces adequately.)
- Teach good personal hygiene, particularly proper hand-washing technique.
- To help prevent giardiasis, warn travelers to endemic areas not to drink water or eat uncooked and unpeeled fruits or vegetables (they may have been rinsed in contaminated water). Prophylactic drug therapy isn't recommended. Advise campers to purify all stream water before drinking it.
- Report epidemic situations to the public health authorities.

Toxoplasmosis

Toxoplasmosis, one of the most common infectious diseases, is caused by the protozoa *Toxoplasma gondii*. Distributed worldwide, it's less common in cold or hot, arid climates and at high elevations. It usually causes localized infection but may produce significant generalized infection, especially in neonates and patients who are immunodeficient. Congenital toxoplasmosis, characterized by lesions in the central nervous system, may result in stillbirth or serious birth defects. For this reason, pregnant women are advised to avoid cleaning cat litter boxes because fecal-oral contamination from infected cats transmits toxoplasmosis.

Causes and incidence

T. gondii exists in trophozoite forms in the acute stages of infection and in cystic forms (tissue cysts and oocysts) in the latent stages. In addition to possible

fecaloral transmission from infected cats, ingestion of tissue cysts in raw or uncooked meat (heating, drying, or freezing destroys these cysts) can also transmit toxoplasmosis. However, toxoplasmosis also occurs in vegetarians who aren't exposed to cats, so other means of transmission may exist. Congenital toxoplasmosis follows transplacental transmission from a chronically infected mother or one who acquired toxoplasmosis shortly before or during pregnancy.

Complications

- Encephalitis
- Myocarditis
- Pneumonitis
- Hepatitis
- Polymyosis

Signs and symptoms

Toxoplasmosis acquired in the first trimester of pregnancy commonly results in stillbirth. About one-third of infants who survive have congenital toxoplasmosis. The later in pregnancy that maternal infection occurs, the greater the risk of congenital infection in the infant. Obvious signs of congenital toxoplasmosis include retinochoroiditis, hydrocephalus or microcephalus, cerebral calcification, seizures, lymphadenopathy, fever, hepatosplenomegaly, jaundice, and rash. Other defects, which may become apparent months or years later, include strabismus, blindness, epilepsy, and mental retardation. (See *Ocular toxoplasmosis,* page 1026.)

OCULAR TOXOPLASMOSIS

Ocular toxoplasmosis (active retinochoroiditis), characterized by focal necrotizing retinitis, accounts for about 25% of all cases of granulomatous uveitis. It's usually the result of congenital infection, but may not appear until adolescence or young adulthood, when infection is reactivated. Symptoms include blurred vision, scotoma, pain, photophobia, and impairment or loss of central vision. Vision improves as inflammation subsides but usually without recovery of lost visual acuity. Ocular toxoplasmosis may subside after treatment with prednisone.

Acquired toxoplasmosis may cause localized (mild lymphatic) or generalized (fulminating, disseminated) infection. Localized infection produces fever and a

mononucleosis-like syndrome (malaise, myalgia, headache, fatigue, and sore throat) and lymphadenopathy. Generalized infection produces encephalitis, fever, headache, vomiting, delirium, seizures, and a diffuse maculopapular rash (except on the palms, soles, and scalp). Generalized infection may lead to myocarditis, pneumonitis, hepatitis, and polymyositis.

Diagnosis

CONFIRMING DIAGNOSIS

Identification of T. gondii *in an appropriate tissue specimen confirms the diagnosis of toxoplasmosis.*

Serologic tests may be useful and, in patients with toxoplasmosis encephalitis, computed tomography scans and magnetic resonance imaging disclose lesions.

Treatment

Treatment of acute disease consists of drug therapy with sulfonamides, pyrimethamine, folinic acid, clindamycin, or cotrimoxazole. In patients who also have acquired immunodeficiency syndrome, treatment continues indefinitely. No safe, effective treatment exists for chronic toxoplasmosis or toxoplasmosis occurring in the first trimester of pregnancy.

Special considerations

When caring for patients with toxoplasmosis, monitor drug therapy carefully and emphasize thorough patient teaching to prevent complications and control spread of the disease.

- Because sulfonamides cause blood dyscrasias and pyrimethamine depresses bone marrow, closely monitor the patient's hematologic values. Also emphasize the importance of regularly scheduled follow-up care.
- Teach all patients to wash their hands after working with soil (because it may be contaminated with cat oocysts); to cook meat thoroughly and freeze it promptly if it isn't for immediate use; to change cat litter daily (cat oocysts don't become infective until 1 to 4 days after excretion); to cover children's sandboxes; and to keep flies away from food (flies transport oocysts).
- Advise all pregnant women to avoid cleaning and handling of cat litter boxes. If this can't be avoided, advise them to wear gloves.
- Patients who are receiving immunosuppressants are very susceptible to toxoplasmosis. Warn them of the risks and suggest having all cats that go outdoors tested for toxoplasmosis.
- Report all cases of toxoplasmosis to your local public health department.

Cryptosporidiosis

Cryptosporidiosis is an intestinal infection that typically results in acute, self-limited diarrhea. However, in immunocompromised patients—who contract it more often —cryptosporidiosis causes chronic, severe, and lifethreatening symptoms.

Those at greatest risk for contracting cryptosporidiosis include:

- Patients with hypogammaglobulinemia
- Patients receiving immunosuppressants for cancer therapy or organ transplantation
- Malnourished children

Cryptosporidiosis is especially prevalent in patients with acquired immunodeficiency syndrome.

In addition to immunocompromised patients, travelers to foreign countries, medical personnel caring for patients with the disease, and children are at particular risk. It's spread easily in day-care centers and among household contacts and medical providers. Contaminated water, such as in a swimming pool, is a frequent source of infection. This disease occurs worldwide.

Causes and incidence

Cryptosporidiosis is caused by the protozoan *Cryptosporidium*. These small spherules inhabit the microvillus border of the intestinal epithelium. There, the protozoa shed infected oocysts into the intestinal lumen, where they pass into the stool.

These oocysts are particularly hardy, resisting destruction by routine water chlorination. This increases the risk of infection spreading through contact with contaminated water. The disease can also be transmitted via contaminated food and person toperson contact.

Complications

- Severe fluid and electrolyte depletion
- Malnutrition
- Rectal excoriation and breakdown
- Papillary stenosis
- Sclerosing cholangitis

- Cholecystitis

Signs and symptoms

Although asymptomatic infections can occur in both healthy and immunocompromised patients, the typical patient with cryptosporidiosis develops symptoms after an incubation period of about 7 days. (The incubation period may be shorter in an immunocompromised patient.) The patient initially complains of watery, nonbloody diarrhea. He may also report abdominal pain, anorexia, nausea, fever, and weight loss. In the 10% of patients who develop biliary tract involvement, right upper abdominal pain may be severe. Signs and symptoms usually subside within 2 weeks but may recur sporadically for months to years.

The history of an immunocompromised patient typically reveals a more gradual onset of symptoms. Such a patient may also develop more severe diarrhea with daily fluid losses as high as 20 L.

For all patients, auscultation of the abdomen may reveal hyperactive bowel sounds. Palpation may reveal abdominal tenderness.

Diagnosis

Cryptosporidiosis often goes undetected as the cause of profuse diarrhea because an acid-fast stain needed to detect the organism isn't routinely used. However, the acid-fast stain as well as microscopic examination of stool samples reveals the presence of oocysts. If too few oocysts are excreted to show up readily under microscopic examination, Sheather's cover-slip flotation method can make detection easier by concentrating the oocysts.

The infecting organisms can also be detected by light and electron microscopy at the apical surfaces of intestinal epithelium obtained through biopsies of the small bowel. Although serologic tests exist, their value in diagnosing acute or chronic infections in immunocompromised patients hasn't been determined.

With biliary tract involvement, studies may reveal an elevated alkaline phosphatase level, gallbladder wall thickening, and dilated bile ducts.

Treatment

Although no treatment currently exists to eradicate the infecting organism, paromomycin may be partially effective for some patients with human immunodeficiency virus.

Treatment for cryptosporidiosis consists mainly of supportive measures to control symptoms. Such measures include fluid replacement to prevent dehydration as well as administration of analgesics to relieve pain and antidiarrheal and antiperistaltic agents to control diarrhea.

Special considerations

- Closely monitor the patient's fluid and electrolyte balance.
- Encourage an adequate intake of fluids, especially those rich in electrolytes.
- Monitor the patient's intake and output, and weigh him daily to evaluate the need for fluid replacement. Watch him closely for signs of dehydration and provide fluid replacement as indicated.
- Administer analgesics, antidiarrheal and antiperistaltic agents, and antibiotics as indicated. Watch the patient for signs of adverse reactions as well as therapeutic effects.
- Apply perirectal protective cream to prevent excoriation and skin breakdown.
- Encourage frequent small meals to help prevent nausea.
- Teach the patient about his medications. Make sure he understands how to take the drugs and what adverse reactions to watch for. Stress the importance of calling his health care provider immediately if he develops an adverse reaction.
- Teach the patient and his family to recognize the signs and symptoms of dehydration, including weight loss, poor skin turgor, oliguria, irritability, and dry flushed skin. Tell them to report such findings to the physician.
- Teach the patient and his family about good personal hygiene, especially proper hand-washing technique. Explain to them how to safely handle potentially infectious material, such as soiled bed sheets.
- Advise the patient's family members and close contacts to have their stools tested.

HELMINTHS

Trichinosis

Trichinosis (also known as *trichiniasis* or *trichinellosis*) is an infection caused by larvae of the intestinal roundworm *Trichinella spiralis*. It occurs worldwide, especially in populations that eat pork or bear meat. Trichinosis may produce multiple symptoms; respiratory, central nervous system (CNS), and cardiovascular complications; and, rarely, death.

Causes and incidence

Transmission is through ingestion of uncooked or undercooked meat that contains *T. spiralis* cysts. Such cysts are found primarily in swine, less commonly in dogs,

cats, bears, foxes, wolves, and marine animals. These cysts result from the animals' ingestion of similarly contaminated flesh. In swine, such infection results from eating table scraps or raw garbage.

After gastric juices free the worm from the cyst capsule, it reaches sexual maturity in a few days. The female roundworm burrows into the intestinal mucosa and reproduces. Larvae are then transported through the lymphatic system and the bloodstream. They become embedded as cysts in striated muscle, especially in the diaphragm, chest, arms, and legs. Human-to-human transmission doesn't take place.

Trichinosis, though common worldwide, is seldom seen in the United States because of regulations regarding animal feed and meat processing.

Complications

- Encephalitis
- Myocarditis
- Pneumonia
- Respiratory failure

Signs and symptoms

In the United States, trichinosis is usually mild and seldom produces symptoms. When symptoms do occur, they vary with the stage and degree of infection:

- *Stage 1* (invasion) occurs 1 week after ingestion. Release of larvae and reproduction of adult *T. spiralis* may cause anorexia, nausea, vomiting, diarrhea, abdominal pain, and cramps.
- *Stage 2* (dissemination) occurs 7 to 10 days after ingestion. *T. spiralis* penetrates the intestinal mucosa and begins to migrate to striated muscle. Signs and symptoms include edema, especially of the eyelids or face; muscle pain, particularly in extremities; and, occasionally, itching and burning skin, sweating, skin lesions, a temperature of 102° to 104° F (38.9° to 40° C), and delirium. In severe respiratory, cardiovascular, or CNS infections, palpitations and lethargy can occur.
- *Stage 3* (encystment) occurs during convalescence, generally 1 week later. *T. spiralis* larvae invade muscle fiber and become encysted.

Diagnosis

A history of ingestion of raw or improperly cooked pork or pork products, with typical clinical features, suggests trichinosis, but infection may be difficult to prove. Stools may contain mature worms and larvae during the invasion stage. Skeletal muscle biopsies can show encysted larvae 10 days after ingestion; if available, analyses of contaminated meat also show larvae.

Skin testing may show a positive histamine-like reactivity 15 minutes after intradermal injection of the antigen (within 17 to 20 days after ingestion). However, such a result may remain positive for up to 5 years after exposure. Elevated acute and convalescent antibody titers (determined by flocculation tests 3 to 4 weeks after infection) confirm this diagnosis.

Other abnormal results include elevated aspartate aminotransferase, alanine aminotransferase, creatine kinase, and lactate dehydrogenase levels during the acute stages and an elevated eosinophil count (up to 15,000/µl). A normal or increased cerebrospinal fluid lymphocyte level (to 300/µl) and increased protein levels indicate CNS involvement.

Treatment

Mebendazole effectively combats this parasite during the intestinal stage; severe infection (especially CNS invasion) may warrant glucocorticoids to combat possible inflammation. There's no treatment for trichinosis once it's in the muscles, but analgesics may be used for muscle pain.

Special considerations

- Question the patient about recent ingestion of pork products and the methods used to store and cook them.
- Reduce fever with alcohol rubs, tepid baths, cooling blankets, or antipyretics; relieve muscle pain with analgesics, enforced bed rest, and proper body alignment.
- To prevent pressure ulcers, frequently reposition the patient, and gently massage bony prominences.
- Explain the importance of bed rest. Sudden death from cardiac involvement may occur in a patient with moderate to severe infection who has resumed activity too soon. Warn the patient to continue bed rest into the convalescent stage to avoid a serious relapse and possible death.

PREVENTION

To help prevent trichinosis take these actions:

- *Educate the public about proper cooking and storing methods not only for pork and pork products but also for meat from*

other carnivores. To kill trichinae, internal meat temperatures should reach 150 °F (65.6°C) and meat color should change from pink to gray unless the meat has been cured or frozen for at least 10 days at low temperatures.

- *Warn travelers to foreign countries or to poor areas in the United States to avoid eating pork; swine in these areas are commonly fed raw garbage.*
- *Report all cases of trichinosis to local public health authorities.*

Hookworm disease

Hookworm disease, also called *uncinariasis* or *ground itch,* is an infection of the upper intestine caused by *Ancylostoma duodenale* (found in the eastern hemisphere) or *Necator americanus* (in the western hemisphere). Sandy soil, high humidity, a warm climate, and failure to wear shoes all favor its transmission. In the United States, hookworm disease is most common in the southeast. Although this disease can cause cardiopulmonary complications, it's rarely fatal, except in debilitated people and infants younger than age 1.

Causes and incidence

Both forms of hookworm disease are transmitted to humans through direct skin penetration (usually in the foot) by hookworm larvae in soil contaminated with feces containing hookworm ova. These ova develop into infectious larvae in 1 to 3 days. Larvae travel through the lymphatics to the pulmonary capillaries, where they penetrate alveoli and move up the bronchial tree to the trachea and epiglottis, where they're swallowed and enter the GI tract. When they reach the small intestine, they mature, attach to the jejunal mucosa, and suck blood, oxygen, and glucose from the intestinal wall. These mature worms then deposit ova, which are excreted in the stool, starting the cycle anew. Hookworm larvae mature in approximately 5 to 6 weeks.

Hookworm disease, affecting billions of people worldwide, is most common in moist tropical and subtropical regions. There's little risk of aquiring hookworm disease in the United States because of advances in sanitization and waste control.

Complications

- Cardiomegaly
- Heart failure

- Massive edema

Signs and symptoms

Most cases of hookworm disease produce few symptoms and may be overlooked until worms are passed in the stool. The earliest signs and symptoms include irritation, pruritus, and edema at the site of entry, which are sometimes accompanied by secondary bacterial infection with pustule formation.

When the larvae reach the lungs, they may cause pneumonitis and hemorrhage with fever, sore throat, crackles, and cough. Finally, intestinal infection may cause fatigue, nausea, weight loss, dizziness, melena, and uncontrolled diarrhea.

In severe and chronic infection, anemia from blood loss may lead to cardiomegaly (a result of increased oxygen demands), heart failure, and generalized massive edema.

Diagnosis

CONFIRMING DIAGNOSIS

Identification of hookworm ova in the stool confirms the diagnosis. Anemia suggests severe chronic infection.

Treatment

Treatment of hookworm infection includes administering mebendazole or albendazole, and providing an iron-rich diet or iron supplements to prevent or correct anemia.

Special considerations

- Obtain a complete history, with special attention to travel or residency in endemic areas. Note the sequence and onset of symptoms. Interview the patient's family and other close contacts to see if they have symptoms.
- Carefully assess the patient, noting signs of entry, lymphedema, and respiratory status.
- Perform meticulous hand hygiene after every patient contact.
- For severe anemia, administer oxygen, if ordered, at low to moderate flow. Be sure the oxygen is humidified because the patient may already have upper airway irritation from the parasites. Encourage coughing and deep breathing to stimulate removal of blood or secretions from involved lung areas and to prevent secondary infection. Plan your care to allow frequent rest periods

because the patient may tire easily. If anemia causes immobility, reposition the patient often to prevent skin breakdown.

- Closely monitor intake and output. Note the frequency of diarrhea and the quantity of stools. Dispose of feces promptly, and wear gloves when doing so.
- To help assess nutritional status, weigh the patient daily. To combat malnutrition, emphasize the importance of good nutrition, with particular attention to foods high in iron and protein. If the patient receives iron supplements, explain that they will darken stools. Administer anthelmintics on an empty stomach, but without a purgative.
- To help prevent reinfection, educate the patient in proper hand-washing technique and sanitary disposal of feces. Tell him to wear shoes in endemic areas.

Ascariasis

Ascariasis, also known as *roundworm infection,* is caused by *Ascaris lumbricoides*. It's the most common type of intestinal worm infection, occurring worldwide. Most patients recover without treatment, but complications can occur when adult worms move into certain organs and multiply, resulting in blockage of the intestine.

Causes and incidence

A. lumbricoides is a large roundworm resembling an earthworm. It's transmitted to humans by ingestion of soil contaminated

with human feces that harbor *A. lumbricoides* ova. Such ingestion may occur directly (by eating contaminated soil) or indirectly (by eating poorly washed raw vegetables grown in contaminated soil).

Ascariasis never passes directly from person to person. After ingestion, *A. lumbricoides* ova hatch and release larvae, which penetrate the intestinal wall and reach the lungs through the bloodstream. After about 10 days in pulmonary capillaries and alveoli, the larvae migrate to the bronchioles, bronchi, trachea, and epiglottis. There they are swallowed and return to the intestine to mature into worms.

Ascariasis is most common in tropical areas with poor sanitation and in Asia, where farmers use human feces as fertilizer. In the United States, it's more prevalent in the south, particularly among people ages 4 to 12.

Complications

- Biliary or intestinal obstruction
- Pulmonary disease

Signs and symptoms

Ascariasis produces two phases: early pulmonary and prolonged intestinal. Mild intestinal infection may cause only vague stomach discomfort. The first clue may be vomiting a worm or passing a worm in the stool. Severe infection, however, causes stomach pain, vomiting, restlessness, disturbed sleep and, in extreme cases, intestinal obstruction. Larvae migrating by the lymphatic and the circulatory systems cause symptoms that vary; for instance, when they invade the lungs, pneumonitis may result.

Diagnosis

CONFIRMING DIAGNOSIS

The key to diagnosis is identifying ova in the stool or adult worms, which may be passed rectally or by mouth.

When migrating larvae invade alveoli, other conclusive tests include X-rays that show characteristic bronchovascular markings: infiltrates, patchy areas of pneumonitis, and widening of hilar shadows. In a patient with ascariasis, these findings usually accompany a complete blood count that shows eosinophilia.

Treatment

Drug therapy, the primary treatment, consists of albendazole or mebendazole to kill intestinal parasitic worms, permitting peristalsis to expel them. No specific treatment exists for migratory infection because anthelmintics affect only mature worms.

In intestinal obstruction, nasogastric (NG) suctioning controls vomiting. If there's a blockage caused by a large number of worms, a paralyzing vermifuge (such as pyrantel pamoate or piperazine) can make the worms relax and pass through the intestine to relieve obstruction. However, ascariasis may necessitate surgery if the paralyzed worms result in intestinal blockage.

Special considerations

- Although isolation is unnecessary, properly dispose of feces and soiled linen, and carefully wash your hands after patient contact.
- If the patient is receiving NG suction, be sure to give him good mouth care.
- Teach the patient to prevent reinfection with good hand washing, especially before eating and after defecation.

Taeniasis

Taeniasis, also called *tapeworm disease* or *cestodiasis,* is a parasitic infestation by *Taenia saginata* (beef tapeworm), *Taenia solium* (pork tapeworm), *Diphyllobothrium latum* (fish tapeworm), or *Hymenolepis nana* (dwarf tapeworm). Taeniasis is usually a chronic, benign intestinal disease; however, infestation with *T. solium* may cause dangerous systemic and central nervous system symptoms if larvae invade the brain and striated muscle of vital organs.

Causes and incidence

T. saginata, *T. solium*, and *D. latum* are transmitted to humans by ingestion of beef, pork, or fish that contains tapeworm cysts. Gastric acids break down these cysts in the stomach, liberating them to mature. Mature tapeworms fasten to the intestinal wall and produce ova that are passed in the feces. Transmission of *H. nana* is direct from person to person and requires no intermediate host; it completes its life cycle

in the intestine. (See *Common tapeworm infestations*.)

COMMON TAPEWORM INFESTATIONS

Type and source of infection	Incidence	Clinical features
Diphyllobothrium latum *(fish tapeworm)*		
Uncooked or undercooked infected freshwater fish, such as pike, trout, salmon, and turbot	Finland, parts of the former Soviet Union, Japan, Alaska, Australia, the Great Lakes region (United States), Switzerland, Chile, and Argentina	Anemia (hemoglobin level as low as 6 to 8 g)
Hymenolepis nana *(dwarf tapeworm)*		
No intermediate host; parasite passes directly from person to person via ova passed in stool; inadequate hand washing facilitates its spread	Most common tapeworm in humans; particularly prevalent among institutionalized mentally retarded children and in underdeveloped countries	Dependent on patient's nutritional status and number of parasites; commonly no symptoms with mild infestation; with severe infestation, anorexia, diarrhea, restlessness, dizziness, and apathy
Taenia saginata *(beef tapeworm)*		

Uncooked or undercooked infected beef	Worldwide, but prevalent in Europe and East Africa	Crawling sensation in the perianal area caused by worm segments that have been passed rectally; intestinal obstruction and appendicitis due to long worm segments that have twisted in the intestinal lumen
Taenia solium *(pork tapeworm)*		
Uncooked or undercooked infected pork	Highest in Mexico and Latin America; lowest among Muslims and Jews	Seizures, headaches, personality changes; commonly overlooked in adults

Complications

- Dehydration
- Malnutrition

Signs and symptoms

Taeniasis may produce mild symptoms, such as nausea, flatulence, hunger sensations, weight loss, diarrhea, and increased appetite, or no symptoms at all. Occasionally, worm segments may exit through the anus and appear on bed clothes.

Diagnosis

CONFIRMING DIAGNOSIS

Diagnosis of tapeworm infestations requires laboratory observation of tapeworm ova or body segments in feces.

Because ova aren't excreted continuously, confirmation may require multiple specimens. A supporting dietary or travel history aids confirmation.

Treatment

The drug of choice for tapeworm infection is niclosamide, but praziquantel and albendazole can also be used.

Laxative use or induced vomiting are contraindicated because of the danger of autoinfection and systemic disease.

After drug treatment, tapeworm infestation requires a follow-up laboratory examination of stool specimens during the next 3 to 5 weeks to check for any remaining ova or worm segments. Persistent infestation typically requires a second course of medication.

Special considerations

- Obtain a complete history, including recent travel to endemic areas, dietary habits, and physical symptoms.
- Dispose of the patient's excretions carefully. Wear gloves when giving personal care and handling fecal excretions, bedpans, and bed linens; wash your hands thoroughly, and tell the patient to do the same.
- Use enteric precautions. Avoid procedures and drugs that may cause vomiting or gagging. If the patient is a child or is incontinent, he requires a private room. Obtain a list of contacts.
- To prevent reinfection, teach proper hand-hygiene technique and the need to cook meat and fish thoroughly. Stress the need for follow-up evaluations to monitor the success of therapy and to detect possible reinfection.

Enterobiasis

Enterobiasis (also called *pinworm, seatworm,* or *threadworm infection,* or *oxyuriasis*) is a benign intestinal disease caused by the nematode *Enterobius vermicularis*. Found worldwide, even in temperate regions with good sanitation, it's the most prevalent helminthic infection in the United States.

Causes and incidence

Adult pinworms live in the intestine; female worms migrate to the perianal region to deposit their ova. *Direct transmission* occurs when the patient's hands transfer infective eggs from the anus to the mouth. *Indirect transmission* occurs when he comes in contact with contaminated articles, such as linens and clothing.

Enterobiasis infection and reinfection occur most commonly in children between ages 5 and 14 and in certain institutionalized groups because of poor hygiene and frequent hand-to-mouth activity. Crowded living conditions increase the likelihood of it spreading to several members of a family.

Complications

- Salpingitis
- Appendicitis
- Bowel ulceration
- Pelvic granuloma

Signs and symptoms

Asymptomatic enterobiasis is commonly overlooked. However, intense perianal pruritus may occur, especially at night, when the female worm leaves the anus to deposit ova. Pruritus causes irritability, scratching, skin irritation and, sometimes, vaginitis.

Diagnosis

CONFIRMING DIAGNOSIS

A history of anal pruritus suggests enterobiasis; identification of Enterobius *ova recovered from the perianal area with a cellophane tape swab taken before the patient bathes and defecates in the morning confirms it. A stool sample is usually ovaand worm-free because the worms deposit ova outside the intestine and die after return to the anus.*

Treatment

Drug therapy with pyrantel, mebendazole, or albendazole destroys the causative parasites. Effective eradication requires simultaneous treatment of the patient's family members and, in institutions, other patients.

Special considerations

- If the patient receives pyrantel, tell him and his family that this drug colors the stool bright red and may cause vomiting (vomitus will also be red). The tablet form of this drug is coated with aspirin and shouldn't be given to aspirin-sensitive patients.
- To help prevent this disease, tell parents to bathe children daily (showers are preferable to tub baths) and to change underwear and bed linens daily.
- Tell the patient's family not to shake bed linens, to avoid aerosolization of eggs that may be on linens.
- Educate children in proper personal hygiene, and stress the need for proper hand washing after defecation and before handling food. Discourage nail biting. If the child can't stop, suggest that he wear gloves until the infection clears.
- Report *all* outbreaks of enterobiasis to school authorities.

- Tell parents to strictly adhere to the prescribed drug dosage as directed by a physician.

TYPES OF SCHISTOSOMES

Species and incidence	Signs and symptoms	Treatment	Adverse effects
Schistosoma mansoni			
Western hemisphere, particularly Puerto Rico, Lesser Antilles, Brazil, and Venezuela; also Nile delta, Sudan, and central Africa	Irregular fever, malaise, weakness, abdominal distress, weight loss, diarrhea, ascites, hepatosplenomegaly, portal hypertension, fistulas, and intestinal stricture	Praziquantel: 60 mg/kg in three equally divided doses at 4-to 6-hour intervals on the same day	Abdominal discomfort, dizziness, drowsiness, fever, headache, malaise, minimal increase in liver enzyme levels, nausea, and urticaria
Schistosoma japonicum			
Affects men more than women; particularly prevalent among farmers in Japan, China, and the Philippines	Irregular fever, malaise, weakness, abdominal distress, weight loss, diarrhea, ascites, hepatosplenomegaly, portal hypertension, fistulas, and intestinal stricture	Praziquantel: 60 mg/kg in three equally divided doses at 4-to 6-hour intervals on the same day	Abdominal discomfort, dizziness, drowsiness, fever, headache, malaise, minimal increase in liver enzyme levels, nausea, and urticaria
Schistosoma haematobium			
Africa, Cyprus, Greece, India	Terminal hematuria, dysuria, ureteral colic; with secondary infection—colicky pain, intermittent flank pain, vague GI complaints, and total renal failure	Praziquantel: 60 mg/kg in three equally divided doses at 4-to 6-hour intervals on the same day	Abdominal discomfort, dizziness, drowsiness, fever, headache, malaise, minimal increase in liver enzyme levels, nausea, and urticaria

Schistosomiasis

Schistosomiasis, also known as *bilharziasis,* is a slowly progressive disease caused by blood flukes of the class Trematoda. There are three major types: *Schistosoma mansoni* and *S. japonicum* infect the intestinal tract; *S. haematobium* infects the urinary tract. (See *Types of schistosomes*.) The degree of infection determines the intensity of illness. Complications—such as portal hypertension, pulmonary hypertension, heart failure, ascites, hematemesis from ruptured esophageal varices, and renal failure—can be fatal.

Causes and incidence

The mode of transmission is bathing, swimming, wading, or working in water contaminated with *Schistosoma* larvae. These larvae penetrate the skin or mucous membranes and eventually work their way to the liver's venous portal circulation. There, they mature in 1 to 3 months. The adults then migrate to other parts of the body.

The female cercariae lay spiny eggs in blood vessels surrounding the large intestine or bladder. After penetrating the mucosa of these organs, the eggs are excreted in feces or urine. If the eggs hatch in fresh water, the first-stage larvae (miracidia) penetrate freshwater snails, which act as passive intermediate hosts. Cercariae produced in snails escape into water and begin a new life cycle.

Complications

- Portal hypertension
- Hepatosplenomegaly
- Pulmonary hypertension
- Ascites
- Hematemesis
- Heart failure
- Renal failure

Signs and symptoms

Initial signs and symptoms of schistosomiasis depend on the site of infection and the stage of the disease. Initially, a transient, pruritic rash develops at the site of cercariae penetration, along with fever, myalgia, and cough. (See *Schistosomal dermatitis*.) Later signs and symptoms may include hepatomegaly, splenomegaly, and lymphadenopathy. Worm migration and egg deposition may cause such complications as flaccid paralysis, seizures, and skin abscesses.

Diagnosis

CONFIRMING DIAGNOSIS

Typical symptoms and a history of travel to endemic areas suggest the diagnosis; ova in the urine or stool or a mucosal lesion biopsy confirms it.

The white blood cell count shows eosinophilia.

Treatment

The treatment of choice is the anthelmintic drug praziquantel. Between 3 and 6 months after treatment, the patient will need to be examined again. If this checkup detects any living eggs, treatment may be resumed. With acute infection, corticosteroids may be ordered.

SCHISTOSOMAL DERMATITIS

Schistosomal dermatitis, also known as *swimmer's itch* or *clam digger's itch,* affects those who bathe in and camp along freshwater lakes in the eastern and western United States. It's caused by schistosomal cercariae, which are harbored by migratory birds and able to penetrate the skin, causing a pruritic papular rash. Initially mild, the reaction grows more severe with repeated exposure. Treatment consists of 5% copper sulfate solution as an antipruritic and 2% methylene blue as an antibacterial agent.

Special considerations

- To help prevent schistosomiasis, teach people in endemic areas to work for a pure water supply and to avoid swimming or bathing in water that's known to be contaminated or potentially contaminated. If they must enter the water, tell them to wear protective clothing and to dry themselves afterward.

Strongyloidiasis

Strongyloidiasis, also called *threadworm infection,* is a parasitic intestinal infection caused by the helminth *Strongyloides stercoralis*. This worldwide infection is endemic in the tropics and subtropics. Susceptibility to strongyloidiasis is universal. Infection doesn't confer immunity, and people who are immunocompromised may suffer overwhelming disseminated infection. Because the threadworm's reproductive cycle may continue in the untreated host for up to 45 years, autoinfection is highly probable. Most patients with strongyloidiasis recover, but debilitation from protein loss may result in death.

Causes and incidence

Transmission to humans usually occurs through contact with soil that contains infective *S. stercoralis* filariform larvae; such larvae develop from noninfective rhabdoid (rod-shaped) larvae in human feces. The filariform larvae penetrate the human skin, usually at the feet. They migrate by way of the lymphatic system to the bloodstream and the lungs.

Once they enter into pulmonary circulation, the filariform larvae break through the alveoli and migrate upward to the pharynx, where they are swallowed. They then lodge in the small intestine, where they deposit eggs that mature into noninfectious rhabdoid larvae. Next, these larvae migrate into the large intestine and are excreted in feces, starting the cycle again. The threadworm life cycle, which begins with penetration of the skin and ends with excretion of rhabdoid larvae, takes 17 days.

In autoinfection, rhabdoid larvae mature within the intestine to become infective filariform larvae.

Complications

- Malnutrition
- Secondary bacterial infection
- Intestinal perforation

Signs and symptoms

The patient's resistance and the extent of infection determine the severity of symptoms. Some patients have no symptoms, but many develop an erythematous maculopapular rash at the site of penetration that produces swelling and pruritus and that may be confused with an insect bite. As the larvae migrate to the lungs, pulmonary signs develop, including minor hemorrhage, pneumonitis, and pneumonia; later, intestinal infection produces frequent, watery, and bloody diarrhea, accompanied by intermittent abdominal pain.

Severe infection can cause malnutrition from substantial fat and protein loss, anemia, and lesions resembling ulcerative colitis, all of which invite secondary bacterial infection. Ulcerated intestinal mucosa may lead to perforation and, possibly, potentially fatal dissemination, especially in patients with malignancy or immunodeficiency diseases or in those who receive immunosuppressants.

Diagnosis

Diagnosis requires observation of *S. stercoralis* larvae in a fresh stool specimen (2 hours after excretion, rhabdoid larvae look like hookworm larvae). Duodenal aspirations show larvae present in duodenal fluid, and an antigen test that's

positive for *S. stercoralis*. During the pulmonary phase, sputum shows *S. stercoralis;* marked eosinophilia also occurs in disseminated strongyloidiasis.

Treatment

The goal of treatment is to eliminate the larvae with antithelmintics, such as ivermectin or thiabendazole. Patients may need protein replacement, blood transfusions, and I.V. fluids. Retreatment is necessary if *S. stercoralis* remains in stools after therapy.

Special considerations

- Keep accurate intake and output records. Ask the dietary department to provide a high-protein diet. The patient may need tube feedings to increase caloric intake.
- Use standard precautions when handling bedpans or giving perineal care, and dispose of feces promptly.
- Because direct person-to-person transmission doesn't occur, isolation isn't required.
- In pulmonary infection, reposition the patient frequently, encourage coughing and deep breathing, and administer oxygen as ordered.
- To prevent reinfection, teach the patient proper hand-washing technique. Stress the importance of proper hand hygiene before eating and after defecating and of wearing shoes when in endemic areas. Check the patient's family and close contacts for signs of infection. Emphasize the need for follow-up stool examination, continuing for several weeks after treatment.

MISCELLANEOUS INFECTIONS

Ornithosis

Ornithosis (also called *psittacosis* or *parrot fever*) is caused by the gram-negative intracellular parasite *Chlamydia psittaci* and is transmitted by infected birds. This disease occurs worldwide and is mainly associated with occupational exposure to birds (such as poultry farming). With adequate antimicrobial therapy, ornithosis is fatal in fewer than 4% of patients.

Causes and incidence

Psittacine birds (parrots, parakeets, and cockatoos), pigeons, and turkeys may harbor *C. psittaci* in their blood, feathers, tissues, nasal secretions, liver, spleen,

and feces. Transmission to humans occurs primarily through inhalation of dust containing *C. psittaci* from bird droppings; less commonly, through direct contact with infected secretions or body tissues, as in laboratory personnel who work with birds. Person-to-person transmission seldom occurs but usually causes severe ornithosis.

Incidence is higher in women and in people ages 20 to 50.

Signs and symptoms

After an incubation period of 4 to 15 days, onset of symptoms may be insidious or sudden. Clinical effects include chills and a low-grade fever that increases to 103° to 105° F (39.4° to 40.6° C) for 7 to 10 days then, with treatment, declines during the second or third week. Other signs and symptoms include headache, myalgia, sore throat, cough (may be dry, hacking, and nonproductive or may produce blood-tinged sputum), abdominal distention and tenderness, nausea, vomiting, photophobia, decreased pulse rate, slightly increased respiratory rate, secondary purulent lung infection, and a faint macular rash. Severe infection also produces delirium, stupor and, in extensive pulmonary infiltration, cyanosis. Ornithosis may recur but is usually milder.

Diagnosis

Characteristic symptoms and a recent history of exposure to birds suggest ornithosis.

CONFIRMING DIAGNOSIS

Firm diagnosis requires recovery of C. psittaci *from mice, eggs, or tissue culture that has been inoculated with the patient's blood or sputum. Culture of* C. psittaci *can be dangerous for laboratory personnel, making serology the preferred method of confirming diagnosis.*

Comparison of acute and convalescent serum shows a fourfold rise in *Chlamydia* antibody titers. In addition, a patchy lobar infiltrate appears on chest X-rays during the first week of illness.

Treatment

Ornithosis calls for treatment with tetracycline. If the infection is severe, tetracycline may be given via I.V. infusion until the fever subsides. Fever and other symptoms should begin to subside 48 to 72 hours after antibiotic treatment begins, but treatment must continue for 2 weeks after temperature returns to normal. Other antibiotics used to treat ornithosis include doxycycline, erythromycin, and azithromycin.

Special considerations

- Monitor the patient's fluid and electrolyte balance. Give I.V. fluids as needed.
- Carefully monitor the patient's vital signs. Watch for signs of overwhelming infection.
- Reduce fever with tepid alcohol or sponge baths and a hypothermia blanket.
- Observe standard precautions. Instruct the patient to use tissues when he coughs and to dispose of them in a closed plastic bag. Also instruct him to wash his hands afterward.

PREVENTION

Persons who raise birds for sale should feed them tetracycline-treated birdseed and follow regulations on bird importation.

However, routine use of antibiotics in animal and bird feed may contribute to antimicrobial resistance. Infected or possibly infected birds should be segregated from healthy birds, and the structures that housed the infected ones should be disinfected.

- Report all cases of ornithosis to public health authorities.

Toxic shock syndrome

Toxic shock syndrome (TSS) is an acute bacterial infection caused by toxin-producing, penicillin-resistant strains of *Staphylococcus aureus*, such as TSS toxin-1 and staphylococcal enterotoxins B and C. Initially, the disease was thought to primarily affect menstruating women younger than age 30 and was associated with continuous use of tampons during the menstrual period; however, only about 55% of cases are associated with menses. TSS is fatal in 50% of cases.

Causes and incidence

Theoretically, tampons may contribute to development of TSS by introducing *S. aureus* into the vagina during insertion (insertion with fingers instead of the supplied applicator increases the risk) or traumatizing the vaginal mucosa during insertion, thus leading to infection.

When TSS isn't related to menstruation, it appears to be linked to *S. aureus* infections, such as abscesses, osteomyelitis, and postsurgical infections. It's also associated with prior antibiotic use.

Risk factors include recent use of barrier contraceptives (diaphragms or vaginal sponges), childbirth, and surgery.

Signs and symptoms

Typically, TSS produces intense myalgias, fever over 104° F (40° C), vomiting, diarrhea, headache, decreased level of consciousness, rigors, conjunctival hyperemia, and vaginal hyperemia and discharge. Severe hypotension occurs with hypovolemic shock. Within a few hours of onset, a deep red rash develops—especially on the palms and soles—and later desquamates.

Major complications include persistent neuropsychological abnormalities, mild renal failure, rash, and cyanotic arms and legs.

Diagnosis

Diagnosis is based on several criteria: fever, hypotension, rash that peels after 1 to 2 weeks, and at least 3 organs with signs of dysfunction. In some cases, blood cultures may be positive for *S. aureus*. Organs with signs of dysfunction may include:

- GI effects, including vomiting and profuse diarrhea
- muscular effects, with severe myalgias or a fivefold or greater increase in creatine kinase levels
- mucous membrane effects such as frank hyperemia
- renal involvement with elevated blood urea nitrogen or creatinine levels (at least twice the normal levels)
- liver involvement with elevated bilirubin, aspartate aminotransferase, or alanine aminotransferase levels (at least twice the normal levels)
- blood involvement with signs of thrombocytopenia and a platelet count of less than 100,000/µl
- central nervous system effects such as disorientation without focal signs.

Negative results on blood tests for Rocky Mountain spotted fever, leptospirosis, and measles help rule out these disorders.

Treatment

Treatment involves examination and removal of foreign material, such as tampons, vaginal sponges, or nasal packing; and drainage of any identified site of infection such as surgical wounds. Antistaphylococcal antibiotics that are beta-lactamase resistant, such as oxacillin and nafcillin, are given I.V. To reverse shock, expect to replace fluids with saline solution and colloids, as ordered. Blood pressure support

and dialysis may be necessary. In some cases, I.V. immunoglobulin may be required.

Special considerations

- Instruct women to change their tampons frequently and to always wash their hands before and after doing so.
- Monitor the patient's vital signs frequently.

- Administer antibiotics slowly and strictly on time. Be sure to watch for signs of penicillin allergy.
- Check the patient's fluid and electrolyte balance.
- Obtain specimens of vaginal and cervical secretions for culture of *S. aureus,* or other sites of TSS infection.
- Implement standard precautions.

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