

## CONTENTS

1 Editorial

2 Disease  
Diagnosis

11 Interpretation

14 Troubleshooting

15 Bouquet

16 Tulip News

## Editorial

**Heat stroke** or **heatstroke**, also known as **sun-stroke**, is a severe heat illness that results in a body temperature greater than 40.0 °C (104.0 °F), along with red skin, headache, dizziness, and confusion. Sweating is generally present in exertional heatstroke, but not in classic heatstroke. The start of heat stroke can be sudden or gradual. Heatstroke is a life-threatening condition due to the potential for multi-organ dysfunction, with typical complications including seizures, rhabdomyolysis, or kidney failure.

Heat stroke occurs because of high external temperatures and/or physical exertion. It usually occurs under preventable prolonged exposure to extreme environmental or exertional heat. However, certain health conditions can increase the risk of heat stroke, and patients, especially children, with certain genetic predispositions are vulnerable to heatstroke under relatively mild conditions.

Preventive measures include drinking sufficient fluids and avoiding excessive heat. Treatment is by rapid physical cooling of the body and supportive care. Recommended methods include spraying the person with water and using a fan, putting the person in ice water, or giving cold intravenous fluids. Adding ice packs around a person is beneficial but does not by itself achieve the fastest possible cooling. Complete clinical, diagnostic, preventive and therapeutic aspects related to HEAT STROKE are presented lucidly under the section – “**DISEASE DIAGNOSIS**”. In this worldwide global warming we request you to be safe and take all precautionary care.

**UNDERSTANDING** portion highlights PERITONITIS and its important clinic diagnostic elements.

TB diagnostics often poses a challenge, so **TROUBLESHOOTING** makes your job easier manifold.

Cool down yourself with a hearty laugh while you go through **BOUQUET**.

Good luck and happy reading.



## DISEASE DIAGNOSIS

### HEAT STROKE



#### Practice Essentials

Heat illness may be viewed as a continuum of illnesses relating to the body's inability to cope with heat. It includes minor illnesses, such as heat edema, heat rash (ie, prickly heat), heat cramps, heat tetany, as well as heat syncope and heat exhaustion. Heat stroke is the most severe heat-related illness and is defined as a body temperature higher than 40°C (104°F) associated with neurologic dysfunction. **Two forms of heat stroke exist.** Exertional heat stroke (EHS) generally occurs in young individuals who engage in strenuous physical activity for a prolonged period in a hot environment. Classic nonexertional heat stroke (NEHS) more commonly affects sedentary elderly individuals, persons who are chronically ill, and very young persons. Classic NEHS occurs during environmental heat waves and is more common in areas that do not typically experience periods of prolonged hot weather. Both types of heat stroke are associated with high morbidity and mortality, especially when cooling therapy is delayed. **With the influence of global warming, it is predicted that** the incidence of heat stroke cases and fatalities will also become more prevalent. Behavioral responses are important in the management of temperature elevations and may provide clues to preventing the development of heat stroke.

#### Symptoms

Heat stroke is defined as hyperthermia exceeding 40°C (104°F) associated with an altered sensorium. However, when a patient is allowed to cool down prior to measurement of the temperature (as may occur during transportation in a cool ambulance or evaluation in an emergency department), the measured temperature may be lower than 41°C, making the temperature criterion relative. Anhidrosis, or lack of sweating, has been cited as a feature of heat stroke, but some patients with heat stroke present with profuse sweating. Because of variable presentations, a high index of suspicion is needed to avoid delays in diagnosis and treatment.

#### Diagnostics

Computerized tomography scans may be helpful in ruling out CNS injury in patients with altered mental status. **Chest radiographs may show atelectasis,** pneumonia, pulmonary infarction, or pulmonary edema. **On electrocardiography, sinus tachycardia of 130-140 beats per minute** and nonspecific and ischemic ST-T wave abnormalities are common. In

addition, a number of conduction abnormalities (eg, right bundle branch block, prolonged QT interval) may be noted.

#### Cooling methods

The optimal method of rapidly cooling patients has been a matter of debate for some time. A 2013 guideline from the Wilderness Medical Society recommends ice-water immersion as a superior method for rapidly lowering core body temperature below the critical levels normally found in heat stroke patients. However, each method has its own theoretical advantages and disadvantages. **Ice-water immersion or an equivalent method** has the advantage of rapidly reducing core body temperature. Because of its high thermal conductivity, ice water can reduce core body temperature to less than 39°C in approximately 20-40 minutes. **The disadvantages of ice-water immersion include** the fact that it may be extremely uncomfortable for patients who are awake. In addition, in theory it can cause subcutaneous vasoconstriction, preventing the transfer of heat via conduction. Ice water also increases shivering, which in turn increases internal heat production. Other criticisms include difficulty monitoring and resuscitating patients while using this method. **Evaporative heat loss, although perhaps less effective than immersion techniques,** poses fewer practical difficulties. Evaporative body heat loss may be accomplished by removing all of the patient's clothes and intermittently spraying the patient's body with tepid water while a powerful fan blows across the body, allowing the heat to evaporate. **A number of other cooling techniques have been suggested,** but none has proven superior to or equal to cold-water immersion or evaporative techniques. These include peritoneal, thoracic, rectal, and gastric lavage with ice water; cold intravenous fluids; cold humidified oxygen; cooling blankets; and wet towels. **Cardiopulmonary bypass has been suggested for use in the most severe cases.** However, this requires highly trained personnel and sophisticated equipment.

See the images below.



Sample display of equipment useful for noninvasive cooling techniques. Clockwise from top: ice pack and water, air-cooling blanket, Foley catheter, and intravenous fluids.



A sample display of equipment useful for cooling via gastric lavage. Clockwise from top: ice water, nasogastric tube, endotracheal tube, and lavage bag.



Sample display of equipment useful for cooling via peritoneal lavage. Clockwise from top: iced water, peritoneal catheter, and saline fluid.

### Surgical care

Compartment syndrome must be suspected in all patients who exhibit rhabdomyolysis and muscle edema and tenderness. Intramuscular compartment pressure measurements must be performed when compartment syndrome is suspected, and fasciotomy must be performed when the intramuscular pressure exceeds 50 mm Hg. Fasciotomy also should be considered when intracompartmental pressures are 30-50 mm Hg, especially when they show no tendency to decrease in 6 hours and in patients who are hypotensive.

### Consultations

Consider consultation with a nephrologist as soon as renal failure occurs. Consultation with a surgeon is indicated when compartment syndrome is suspected. Consider consultation with a liver transplant

service for patients with persistent fulminant liver failure.

### Diet and activity

Patients may resume oral feeding when mental status, swallowing, and gastrointestinal tract function are normal. **During the initial phase of therapy, neuromuscular blockade with muscular paralysis** should be considered for patients who are not cooling adequately. Depolarizing agents (eg, succinylcholine) and inhaled anesthetics should be avoided because of the risk of malignant hyperthermia. Patients may resume activity when their temperature has stabilized.

### Long-term monitoring

Long-term outpatient therapy may be required when chronic renal failure develops and when irreversible damage to the CNS, lungs, heart, and liver occurs.

### Prevention

Heat stroke is a preventable illness, and education is the single most important tool for its prevention. **Recognition of host risk factors and modification of behavior** (eg, limiting alcohol and drug intake, avoiding use of medications and drugs that interfere with heat dissipation) and physical activity also can prevent heat stroke. **Cooling during exercise offers a similar benefit to exercise performance** in hot environments as does the application of precooling, and both methods improve exercise performance with and without physiological alterations. A study performed in 2015 is the first to show that cooling of the neck region during exercise can improve repeated sprint performance in hot environments without altering hormonal or physiological responses, in particular to repeated sprinting or a soccer-specific intermittent treadmill protocol. Additionally, it has been suggested that regular carbohydrate ingestion during exertion in the subclinical stages of exertional heat stroke may play a role in preventing or mitigating heat stroke symptoms, though this assertion has not been verified in actual exertional heat stroke patients.

### Pathophysiology

Despite wide variations in ambient temperatures, humans and other mammals can maintain a constant body temperature by balancing heat gain with heat loss. When heat gain overwhelms the body's mechanisms of heat loss, the body temperature rises, potentially leading to heat stroke. Excessive heat denatures proteins, destabilizes phospholipids and lipoproteins, and liquefies membrane lipids, leading to cardiovascular collapse, multiorgan failure, and, ultimately, death. **The exact temperature at which cardiovascular collapse occurs varies among individuals because coexisting disease, drugs, and other factors may contribute to or delay organ dysfunction.** Full recovery has been observed in patients with temperatures as high as 46°C, and death has occurred in patients with much lower temperatures. Temperatures exceeding 106°F or 41.1°C generally are catastrophic and require immediate aggressive therapy. **Heat may be acquired by a number of different mechanisms.** At rest, basal metabolic processes produce approximately 100 kcal of heat per hour or 1 kcal/kg/h. These reactions can raise the body temperature by 1.1°C/h if the heat-dissipating mechanisms are nonfunctional. Strenuous physical activity can increase heat production more than 10-fold, to levels exceeding 1000 kcal/h. Similarly, fever, shivering, tremors, convulsions, thyrotoxicosis, sepsis, sympathomimetic drugs, and many other conditions can increase heat production, thereby increasing body temperature. **The body also can acquire heat from the environment** through some of the same mechanisms involved in heat dissipation, including conduction, convection, and radiation. These mechanisms occur at the level of the skin and require a properly functioning skin surface, sweat glands, and

autonomic nervous system, but they also may be manipulated by behavioral responses. **Conduction refers to the transfer of heat between 2 surfaces with differing temperatures** that are in direct contact. Convection refers to the transfer of heat between the body's surface and a gas or fluid with a differing temperature. Radiation refers to the transfer of heat in the form of electromagnetic waves between the body and its surroundings. The efficacy of radiation as a means of heat transfer depends on the position of the sun, the season, clouds, and other factors. For example, during summer, lying down in the sun can result in a heat gain of up to 150 kcal/h. **Under normal physiologic conditions, heat gain is counteracted by a commensurate heat loss.** This is orchestrated by the hypothalamus, which functions as a thermostat, guiding the body through mechanisms of heat production or heat dissipation, thereby maintaining the body temperature at a constant physiologic range. **In a simplified model, thermosensors located in the skin, muscles, and spinal cord** send information regarding the core body temperature to the anterior hypothalamus, where the information is processed and appropriate physiologic and behavioral responses are generated. Physiologic responses to heat include an increase in cardiac output and blood flow to the skin (as much as 8 L/min), which is the major heat-dissipating organ; dilatation of the peripheral venous system; and stimulation of the eccrine sweat glands to produce more sweat. **As the major heat-dissipating organ, the skin transfers heat** to the environment through conduction, convection, radiation, and evaporation. Radiation is the most important mechanism of heat transfer at rest in temperate climates, accounting for 65% of heat dissipation, and it can be modulated by clothing. At high ambient temperatures, conduction becomes the least important of the 4 mechanisms, while evaporation, which refers to the conversion of a liquid to a gaseous phase, becomes the most effective mechanism of heat loss. **The efficacy of evaporation as a mechanism of heat loss depends** on the condition of the skin and sweat glands, the function of the lung, ambient temperature, humidity, air movement, and whether or not the person is acclimated to the high temperatures. For example, evaporation does not occur when the ambient humidity exceeds 75% and is less effective in individuals who are not acclimated. Nonacclimated individuals can only produce 1 L of sweat per hour, which only dispels 580 kcal of heat per hour, whereas acclimated individuals can produce 2-3 L of sweat per hour and can dissipate as much as 1740 kcal of heat per hour through evaporation. Acclimatization to hot environments usually occurs over 7-10 days and enables individuals to reduce the threshold at which sweating begins, increase sweat production, and increase the capacity of the sweat glands to reabsorb sweat sodium, thereby increasing the efficiency of heat dissipation. **When heat gain exceeds heat loss, the body temperature rises.** Classic heat stroke occurs in individuals who lack the capacity to modulate the environment (eg, infants, elderly individuals, individuals who are chronically ill). Furthermore, elderly persons and patients with diminished cardiovascular reserves are unable to generate and cope with the physiologic responses to heat stress and, therefore, are at risk of heat stroke. Patients with skin diseases and those taking medications that interfere with sweating also are at increased risk for heat stroke because they are unable to dissipate heat adequately. Additionally, the redistribution of blood flow to the periphery, coupled with the loss of fluids and electrolytes in sweat, place a tremendous burden on the heart, which ultimately may fail to maintain an adequate cardiac output, leading to additional morbidity and mortality. **Factors that interfere with heat dissipation include an inadequate intravascular volume,** cardiovascular dysfunction, and abnormal skin. Additionally, high ambient temperatures, high ambient humidity, and many drugs can

interfere with heat dissipation, resulting in a major heat illness. Similarly, hypothalamic dysfunction negatively affects temperature regulation, predisposing to temperature rise and heat illness. **On a cellular level, heat directly influences the body by interfering with cellular processes** along with denaturing proteins and cellular membranes. In turn, an array of inflammatory cytokines, interleukins and heat shock proteins (HSPs) are produced. In particular, HSP-70 allows the cell to endure the stress of its environment. Intense heat stress that is uncompensated leads to apoptosis and cell death. **On a microvascular level, heat stroke resembles sepsis and involves** inflammation, translocation of lipopolysaccharides from the gut, and activates the coagulation cascade. Certain preexisting factors, such as age, genetic makeup, and the nonacclimated individual, may allow progression from heat stress to heat stroke, systemic inflammatory response syndrome (SIRS), multiorgan dysfunction syndrome (MODS), and ultimately death. Progression to heat stroke may occur through thermoregulatory failure, an amplified acute-phase response, and alterations in the expression of HSPs. **The Wet Bulb Globe Temperature (WBGT) is an index used to gauge ambient conditions** that can place an individual at risk for heat illness. This environmental heat stress index, used by the American College of Sports Medicine, is calculated using three parameters: temperature, humidity, and radiant heat. There is low risk if the WBGT is less than 65°F, moderate risk if it is 65-73°F, high risk if 73-82°F, and very high risk if above 82°F.

### Etiology

The etiology of heat stroke may involve any of the following:

- Increased heat production
- Decreased heat loss
- Reduced ability to acclimatize
- Reduced behavioral responsiveness

### Increased heat production

Increased metabolism can result from any of the following:

- Infections
- Sepsis
- Encephalitis
- Stimulant drugs
- Thyroid storm
- Drug withdrawal

Increased muscular activity may involve any of the following:

- Exercise
- Convulsions
- Tetanus
- Strychnine poisoning
- Sympathomimetics
- Drug withdrawal
- Thyroid storm

Moderate physical exercise, convulsions, and shivering can double heat production and result in temperature elevations that generally are self-limited and resolve with discontinuation of the activity. **Strenuous exercise and status epilepticus can increase heat production 10-fold** and, when uninterrupted, can overwhelm the body's heat-dissipating mechanisms, leading to dangerous rises in body temperature. **Stimulant drugs, including cocaine and amphetamines, can generate** excessive amounts of heat by increasing metabolism and motor activity through the stimulatory effects of dopamine, serotonin, and norepinephrine. The development of heat stroke in individuals intoxicated with stimulants is multifactorial and may involve a complex interaction between dopamine and serotonin in the hypothalamus and the brainstem. **Neuroleptic**

agents also may elevate body temperature by increasing muscle activity, but, occasionally, these agents may cause neuroleptic malignant syndrome (NMS). NMS is an idiosyncratic reaction characterized by hyperthermia, altered mental status, muscle rigidity, and autonomic instability and appears to be due to excessive contraction of muscles. Certain drugs, such as inhaled volatile anesthetics and succinylcholine, may result in malignant hyperthermia. In contrast to heat stroke, malignant hyperthermia is believed to be induced by a decreased ability of the sarcoplasmic reticulum to retain calcium, resulting in sustained muscle contraction.

#### Decreased heat loss

Reduced sweating can result from any of the following:

- Dermatologic diseases
- Drugs
- Burns

Reduced central nervous system (CNS) responses may result from the following:

- Advanced age
- Young age (toddlers and infants)
- Alcohol
- Barbiturates
- Other sedatives

Reduced cardiovascular reserve may result from the following:

- Advanced age
- Beta-blockers
- Calcium channel blockers
- Diuretics
- Cardiovascular drugs - Interfere with the cardiovascular responses to heat and, therefore, can interfere with heat loss

Drugs that can result in decreased heat loss include the following:

- Anticholinergics
- Neuroleptics
- Antihistamines

Exogenous factors that can decrease heat loss include the following:

- High ambient temperatures
- High ambient humidity

#### Reduced ability to acclimatize

Persons at the extremes of age (ie, toddlers and young children, the elderly) may be less able to generate adequate physiologic responses to heat stress. Diuretic use and hypokalemia can also impair accommodation to heat stress.

#### Reduced behavioral responsiveness

Infants, patients who are bedridden, and patients who are chronically ill are at risk for heat stroke because they are unable to control their environment and water intake. To compound matters, comorbidities and polypharmacy in the elderly can compromise their recovery.

### Epidemiology

#### Frequency

Heat stroke is uncommon in subtropical climates. The condition is recognized increasingly in countries that experience heat waves rarely (eg, Japan), and it commonly affects people who undertake a pilgrimage to Mecca, especially pilgrims who come from a cold environment. In 1998, one of the worst heat waves to strike India in 50 years resulted in more than 2600 deaths in 10 weeks. Unofficial reports described the number of deaths as almost double that figure.

#### Racial and sexual disparities in incidence

With the same risk factors and under the same environmental conditions, heat stroke affects all races equally. However, because of

differences in social advantages, the annual death rate due to environmental conditions is more than three times higher in Blacks than in Whites. Factors investigated as influencing this disparity include housing and neighborhood characteristics (eg, urban heat islands, crime and safety), using air conditioning (more common among whites, particularly central air conditioning), opening windows, using fans, using cooler public spaces, and social isolation. With the same risk factors and under the same environmental conditions, heat stroke affects both sexes equally. However, because males are more likely to work outside and in hot conditions, the annual death rate from heat stroke is two times higher in men than in women. The CDC reports that the rate of heat-related mortality tends to be higher in males.

#### Age-related disparities in incidence

Infants, children, and elderly persons have a higher incidence of heat stroke than young, healthy adults. Infants and children are at risk for heat illness due to inefficient sweating, a higher metabolic rate, and their inability to care for themselves and control their environment. Elderly persons also are at increased risk for heat-related illnesses because of their limited cardiovascular reserves, preexisting illness, and use of many medications that may affect their volume status or sweating ability. In addition, elderly people who are unable to care for themselves are at increased risk for heat stroke, presumably because of their inability to control their environment. The CDC reports that the rate of heat-related mortality tends to be higher in persons aged 65 years and older. Exertional heat stroke (EHS) is a leading cause of injury and death in high school athletes; approximately two-thirds of such cases occur in August and involve football players, often those who are obese or overweight. Lack of acclimatization is a major risk factor for EHS in young adult.

### Prognosis

Indicators of poor prognosis during acute episodes include the following:

- Initial temperature measurement higher than 41°C (106°F) or a temperature higher than 42°C (108°F) or a temperature persisting above 39°C (102°F) despite aggressive cooling measures
- Coma duration longer than 2 hours
- Severe pulmonary edema
- Delayed or prolonged hypotension
- Lactic acidosis in patients with classic heat stroke
- Acute kidney injury and hyperkalemia
- Aminotransferase levels greater than 1000 IU/L during the first 24 hours

Morbidity and mortality from heat stroke are related to the duration of the temperature elevation. When therapy is delayed, the mortality rate may be as high as 80%; however, with early diagnosis and immediate cooling, the mortality rate can be reduced to 10%. Mortality is highest among the elderly population, patients with preexisting disease, those confined to a bed, and those who are socially isolated.

### Patient Education

Education is the single most important tool for the prevention of heat stroke. The media, public education, public health programs, and athlete safety programs can play a pivotal role in increasing the public's awareness of the dangers of heat during heat waves and advising the public on methods of remaining cool. Similarly, drinking fluids on schedule (and not based only on thirst), frequent cooling breaks, and frequent visits to air-conditioned places are very important because even short stays in an air-conditioned environment may drastically reduce the incidence of heat stroke. Recognition of host risk factors and

**modification of behavior** (eg, limiting alcohol and drug intake and the use of medications and drugs that interfere with heat dissipation) and physical activity also will prevent heat stroke.

## Clinical Presentation

### History

Clinically, 2 forms of heat stroke are differentiated: classic, or nonexertional, heat stroke (NEHS) and exertional heat stroke (EHS). NEHS, which occurs during environmental heat waves, is more common in the very young and the elderly and should be suspected in children, elderly persons, and chronically ill individuals who present with an altered sensorium. NEHS occurs because of failure of the body's heat dissipating mechanisms. **On the other hand, EHS affects young, healthy individuals** who engage in strenuous physical activity, and EHS should be suspected in all such individuals who exhibit bizarre, irrational behavior or experience syncope. EHS results from increased heat production, which overwhelms the body's ability to dissipate heat.

### Exertional heat stroke

EHS is characterized by hyperthermia, diaphoresis, and an altered sensorium, which may manifest suddenly during extreme physical exertion in a hot environment. **A number of symptoms** (eg, abdominal and muscular cramping, nausea, vomiting, diarrhea, headache, dizziness, dyspnea, weakness) commonly precede the heat stroke and may remain unrecognized. Syncope and loss of consciousness also are observed commonly before the development of EHS. **EHS commonly is observed in young, healthy individuals** (eg, athletes, firefighters, military personnel) who, while engaging in strenuous physical activity, overwhelm their thermoregulatory system and become hyperthermic. Because their ability to sweat remains intact, patients with EHS are able to cool down after cessation of physical activity and may present for medical attention with temperatures well below 41°C. Despite education and preventive measures, EHS is still a leading cause of disability and death in high school athletes, particularly football players. **Risk factors that increase the likelihood of heat-related illnesses include** a preceding viral infection, dehydration, fatigue, obesity, lack of sleep, poor physical fitness, and lack of acclimatization. Although lack of acclimatization is a risk factor for heat stroke, EHS also can occur in acclimatized individuals who are subjected to moderately intense exercise. EHS also may occur because of increased motor activity due to drug use, such as cocaine and amphetamines, and as a complication of status epilepticus.

### Nonexertional heat stroke

NEHS is characterized by hyperthermia, anhidrosis, and an altered sensorium, which develop suddenly after a period of prolonged elevations in ambient temperatures (ie, heat waves). Core body temperatures greater than 41°C are diagnostic, although heat stroke may occur with lower core body temperatures. **Numerous central nervous system (CNS) symptoms**, ranging from minor irritability to delusions, irrational behavior, hallucinations, and coma have been described. Other possible CNS symptoms include seizures, cranial nerve abnormalities, cerebellar dysfunction, and opisthotonos. **Anhidrosis due to cessation of sweating** is a late occurrence in heat stroke and may not be present when patients are examined. **Patients with NEHS initially may exhibit a hyperdynamic circulatory state**, but, in severe cases, hypodynamic states may be noted. **Classic heat stroke most commonly occurs during episodes of prolonged elevations in ambient temperatures.** It affects people who are unable to control their environment and water intake (eg, infants, elderly persons, individuals who are chronically ill), people with reduced cardiovascular reserve (eg, elderly persons, patients with chronic cardiovascular illnesses), and

people with impaired sweating (eg, from skin disease or ingestion of anticholinergic or psychiatric drugs). In addition, infants have an immature thermoregulatory system, and elderly persons have impaired perception of changes in body and ambient temperatures and a decreased capacity to sweat.

## Physical Examination

### Vital signs

**Temperature:** Typically, the patient's temperature exceeds 41°C. However, in the presence of sweating, evaporating mechanisms, and the initiation of cooling methods, body temperatures lower than 41°C are common.

**Pulse:** Tachycardia to rates exceeding 130 beats per minute is common.

**Blood pressure:** Patients commonly are normotensive, with a wide pulse pressure; however, hypotension is common and may result from a number of factors, including vasodilation of the cutaneous vessels, pooling of the blood in the venous system, and dehydration. Hypotension also may be due to myocardial damage and may signal cardiovascular collapse. Blood pressure will usually correct with normalization of the body temperature.

### Central nervous system

Symptoms of CNS dysfunction are present universally in persons with heat stroke. Symptoms may range from irritability to coma. **Patients may present with delirium, confusion, delusions, convulsions, hallucinations, ataxia, tremors, dysarthria, and other cerebellar findings**, as well as cranial nerve abnormalities and tonic and dystonic contractions of the muscles. Seizures may occur. Patients also may exhibit decerebrate posturing, decorticate posturing, or they may be limp. **Coma also may be caused by electrolyte abnormalities**, hypoglycemia, hepatic encephalopathy, uremic encephalopathy, and acute structural abnormalities, such as intracerebral hemorrhage due to trauma or coagulation disorders. **Cerebral edema and herniation also may occur** during the course of heat stroke. However, even severe cerebral edema arising from heat stroke may be reversed with extensive supportive measures.

### Eyes

Examination of the eyes may reveal nystagmus and oculozytic episodes due to cerebellar injury. The pupils may be fixed, dilated, pinpoint, or normal.

### Cardiovascular

Heat stress places a tremendous burden on the heart. Patients with preexisting myocardial dysfunction do not tolerate heat stress for prolonged periods. **Patients commonly exhibit a hyperdynamic state**, with tachycardia, low systemic vascular resistance, and a high cardiac index. However, a hypodynamic state, with a high systemic vascular resistance and a low cardiac index, may occur in patients with preexisting cardiovascular disease and low intravascular volume. A hypodynamic state also may signal cardiovascular collapse. **The central venous pressure generally is within the reference range** or elevated unless the patient is severely volume depleted. **High-output cardiac failure** and low-output cardiac failure may occur.

### Pulmonary

Patients with heat stroke commonly exhibit tachypnea and hyperventilation caused by direct CNS stimulation, acidosis, or hypoxia. Hypoxia and cyanosis may be due to a number of processes, including atelectasis, pulmonary infarction, aspiration pneumonia, and pulmonary edema.

### Gastrointestinal

Gastrointestinal hemorrhage and intestinal infarction are complications

that can occur in patients with heat stroke.

### Hepatic

Patients commonly exhibit evidence of hepatic injury, including jaundice and elevated liver enzymes. **Rarely, fulminant hepatic failure occurs,** accompanied by encephalopathy, hypoglycemia, and disseminated intravascular coagulation (DIC) and bleeding.

### Musculoskeletal

Muscle tenderness and cramping are common; rhabdomyolysis is a common complication of EHS. The patient's muscles may be rigid or limp.

### Renal

Acute kidney injury is a common complication of heat stroke and may be due to hypovolemia, low cardiac output, and myoglobinuria (from rhabdomyolysis). Patients may exhibit oliguria and a change in the color of urine.

### Complications

Heat stroke is a multisystem insult that potentially can affect almost every organ system. **The central nervous system (CNS) is especially sensitive to the damaging effects of hyperthermia.** Widespread cell death occurs but is more evident in the region of the cerebellum (Purkinje cells). Heat stroke–related long-term CNS sequelae include cerebellar deficits, dementia, hemiplegia, quadriplegia, and personality changes. **In one study, rhabdomyolysis was observed in almost all patients with EHS and in as many as 86% of patients with NEHS.** Compartment syndrome is observed most commonly in patients with severe rhabdomyolysis and in patients who are immobilized. **Acute kidney injury may occur** in as many as 25-30% of patients who have heat stroke (especially EHS). **Acute liver failure due to centrilobular hepatic necrosis and cholestasis** generally occurs in the first 48 hours, but it can peak as long as 2 weeks after the onset of heat stroke. In rare instances, liver failure may be complicated by a fulminant course requiring liver transplantation. Patients who survive generally have a complete return of hepatic function. **DIC is a rare complication and carries a poor prognosis when it occurs.** Electron microscopy studies have shown that direct thermal injury to the vascular endothelium is the primary trigger of platelet aggregation and, possibly, DIC. **ARDS may be due to direct thermal injury to the lung,** or it may complicate liver failure, infection, or aspiration. When associated with liver failure, the patient's prognosis is much worse.

### Diagnostic Considerations

Other problems to be considered include the following:

- Sepsis
- Diabetic ketoacidosis
- Closed head trauma
- Malignant hyperthermia
- Encephalitis
- Cerebral malaria
- Cerebral hemorrhage
- Amphetamine and cocaine toxicity
- Strychnine poisoning

Differential Diagnoses

- Cocaine Toxicity
- Delirium
- Delirium Tremens (Dts)
- Hepatic Encephalopathy
- Hyperthyroidism and Thyrotoxicosis
- Hyponatremia

- Meningitis
- Neuroleptic Malignant Syndrome
- Salicylate Toxicity
- Tetanus
- Uremic Encephalopathy

### Laboratory Studies

#### Arterial blood gas testing

Arterial blood gas analysis may reveal respiratory alkalosis due to direct central nervous system (CNS) stimulation and metabolic acidosis due to lactic acidosis. Hypoxia may be due to pulmonary atelectasis, aspiration pneumonia, or pulmonary edema.

#### Lactic acidosis

Lactic acidosis commonly occurs following exertional heat stroke (EHS) but may signal a poor prognosis in patients with classic heat stroke.

#### Glucose

Hypoglycemia may occur in patients with EHS and in patients with fulminant hepatic failure.

#### Electrolytes

##### Sodium

Hypernatremia due to reduced fluid intake and dehydration commonly is observed early in the course of disease but may be due to diabetes insipidus. Hyponatremia is observed in patients using hypotonic solutions, such as free water, and in patients using diuretics. It also may be due to excessive sweat sodium losses.

##### Potassium

Hypokalemia is common in the early phases of heat stroke, and deficits of 500 mEq are not unusual. However, with increasing muscle damage, hyperkalemia may be observed.

##### Other

Hypophosphatemia secondary to phosphaturia and hyperphosphatemia secondary to rhabdomyolysis, hypocalcemia secondary to increased calcium binding in damaged muscle, and hypomagnesemia also are observed commonly.

#### Hepatic function tests

Hepatic injury is a consistent finding in patients with heat stroke. Aminotransferase (aspartate aminotransferase [AST] and alanine aminotransferase [ALT]) levels commonly rise to the tens of thousands during the early phases of heat stroke and peak at 48 hours, but they may take as long as 2 weeks to peak.

Jaundice may be striking and may be noted 36-72 hours after the onset of liver failure.

#### Muscle function tests

Creatinine kinase (CK), lactate dehydrogenase (LDH), aldolase, and myoglobin commonly are released from muscles when muscle necrosis occurs.

CK levels exceeding 100,000 IU/mL are common in patients with EHS. Elevations in myoglobin may not be noted despite muscle necrosis because myoglobin is metabolized rapidly by the liver and excreted rapidly by the kidneys.

#### Complete blood cell count

Elevated white blood cell counts commonly are observed in patients with heat stroke, and levels as high as 40,000/ $\mu$ L have been reported. Platelet levels may be low.

#### Renal function tests

Elevations in serum uric acid levels, blood urea nitrogen, and serum creatinine are common in patients whose course is complicated by renal failure.

## Urinalysis

Remember that urinary benzidine dipsticks do not differentiate between blood, hemoglobin, and myoglobin. Urine dipstick analyses that are positive for blood must be followed by a microscopic urinalysis to determine the presence or absence of red blood cells. Proteinuria also is common.

## Cerebrospinal fluid analysis

Cerebrospinal fluid (CSF) cell counts may show a nonspecific pleocytosis, and CSF protein levels may be elevated as high as 150 mg/dL.

## Other

Myoglobin causes a reddish brown discoloration of the urine but does not affect the color of plasma. This is in contrast to hemoglobin, which causes discoloration of both plasma and urine.

## Management



Stay hydrated to prevent dehydration. Avoid caffeine or alcohol as this will dehydrate.



When outdoors, wear lightweight, loose-fitting, light-colored clothing and a wide-brimmed hat. Use sunscreen with an SPF of 30 or more.



Take it easy during the hottest times of the day & avoid strenuous activity.

Do not sit in a parked car in warm or hot weather & lock your car so that children cannot get inside.



On hot days >33°C (91°F) stay inside in air conditioning if available.



Speak with your pharmacist or doctor about any prescribed medication that may increase your risk of heat-induced illness.



## Medical Care

Heat stroke is a medical emergency and continues to be one of the leading causes of preventable death in sports. Rapid reduction of the core body temperature is the cornerstone of treatment because the duration of hyperthermia is the primary determinant of outcome. Patients diagnosed with exertional heat stroke (EHS) or nonexertional heat stroke (NEHS) should be admitted to the hospital for at least 48 hours to monitor for complications. **Once heat stroke is suspected, cooling must begin immediately and must be continued during the patient's resuscitation.** The American College of Sports Medicine recommends that cooling be initiated at the scene, before transporting the patient to an emergency department for further evaluation and treatment. Despite extensive education and training, delays are still reported due to trepidation by athletic trainers to accurately diagnose and rapidly initiate treatment for EHS. **Controversy still exists over what therapeutic modality is most effective in the treatment of heat stroke.** However, the basic premise of rapidly lowering the core temperature to about 39°C (to avoid overshooting and rebound hyperthermia) remains the primary goal. Rehydration therapy alone is insufficient for heat stroke patients and should be combined with active cooling. **According to 1 study, oral temperature assessments consistently reflected inaccurate core body temperatures, which delayed the diagnosis and ultimate treatment of patients with heat stroke.** Rectal temperatures are still the preferred method of accurately obtaining core body temperatures. **Some studies have shown that promptly reducing the exposure time to excessive heat can dramatically improve long-term outcomes and decrease irreversible injury.** If treatment is initiated within this so-called golden hour and is aggressive enough to rapidly reduce the core body temperature, complications (including multisystem organ failure) may be averted and the patient may have a much better prognosis. **In a review of 19 clinical trials and observational studies involving 556 patients,** the conduction method of cooling was found to be more efficacious in young, active adults with EHS. Unfortunately, this review did not identify a preferred treatment found for NEHS, or a temperature endpoint to prevent overcooling. **Removal of restrictive clothing and spraying water on the body,** covering the patient with ice water-soaked sheets, or placing ice packs in the axillae and groin may reduce the patient's temperature significantly. Patients who are unable to protect their airway should be intubated. Patients who are awake and responsive should receive supplemental oxygen. **Intravenous lines may be placed in anticipation of fluid resuscitation** and for the infusion of dextrose and thiamine if indicated. Hypoglycemia is a common occurrence in patients with EHS and may be a manifestation of liver failure; therefore, infusion of dextrose 50% in water solution (D50W) should be considered in all patients with heat stroke. **Intensive care personnel must pay meticulous attention to the airway,** reduce the temperature, limit the production of heat, optimize circulation, and monitor for and treat complications. Interventions to enable monitoring include the following:

- Insert a thermistor probe or temperature-sensing Foley catheter to monitor temperature continuously
- Insert a nasogastric tube to monitor for gastrointestinal bleeding and fluid losses
- Place a Foley catheter to monitor urine output and/or monitor body temperature

The goal of treatment is to reduce the temperature by at least 0.2°C/min to approximately 39°C. A flexible indwelling thermistor rectally or an esophageal probe can be placed to monitor core body temperature during treatment; alternatively, a more modern method is to use a

temperature-sensing Foley catheter. Because thermal instability may persist for a few days after the onset of heat stroke, the temperature must be monitored continuously until it is stable.

#### Pharmacologic measures

Antipyretics (eg, acetaminophen, aspirin, other nonsteroidal anti-inflammatory drugs) have no role in the treatment of heat stroke because antipyretics interrupt the change in the hypothalamic set point caused by pyrogens; they are not expected to work on a healthy hypothalamus that has been overloaded, as in the case of heat stroke. In this situation, antipyretics actually may be harmful in patients who develop hepatic, hematologic, and renal complications because they may aggravate bleeding tendencies. **Dantrolene has been studied as a possible pharmacologic option** in the treatment of hyperthermia and heat stroke. To date, however, it has not proved efficacious in clinical trials. **Immediate administration of benzodiazepines is indicated in patients** with agitation and shivering, to stop excessive production of heat. In addition, benzodiazepines are the sedatives of choice in patients with sympathomimetic-induced delirium as well as alcohol and sedative drug withdrawals. **Neuroleptics (eg, chlorpromazine), which were the mainstays of therapy** in the past, are best avoided because of their deleterious adverse effects, including lowering of the seizure threshold, interference with thermoregulation, anticholinergic properties, hypotension, hepatotoxicity, and other adverse effects. **Benzodiazepines and, if necessary, barbiturates are the recommended agents** for treatment of patients who are having convulsions. Barbiturates may be used despite their theoretical impedance of sweat production. **Phenytoin is not effective in controlling convulsions in this situation.** Patients whose convulsions are refractory to benzodiazepines and barbiturates should be paralyzed and provided mechanical ventilation. Electroencephalographic monitoring is recommended in all such patients, and anticonvulsant medications should be adjusted accordingly.

#### Fluid resuscitation



Recommendations on the administration of intravenous fluids for circulatory support differ among patient populations and depend on the presence of hypovolemia, preexisting medical conditions, and preexisting cardiovascular disease. **While patients with heat stroke invariably are volume depleted,** cooling alone may improve hypotension and cardiac function by allowing blood to redistribute centrally. Aggressive fluid resuscitation generally is not recommended because it may lead to pulmonary edema. Cor pulmonale also is a common finding in patients with heat stroke. **When pulse rate, blood pressure, and urine output do not provide adequate hemodynamic information,** fluid

administration should be guided by more invasive hemodynamic parameters, such as central venous pressure (CVP), pulmonary capillary wedge pressure, systemic vascular resistance index (SVRI), and cardiac index (CI) measurements. Patients who exhibit a hyperdynamic state (ie, high CI, low SVRI) generally respond to cooling and do not require large amounts of intravenous crystalloid infusions.

**Hypotensive patients who exhibit a hypodynamic response** (ie, high CVP, low CI) historically have been treated with low-dose isoproterenol; however, its arrhythmogenicity has raised questions about its continued use. Dobutamine, which is less arrhythmogenic than isoproterenol and more cardio selective, may be the inotrope of choice in these patients. Alpha-adrenergic drugs generally are contraindicated because they cause vasoconstriction and may interfere with heat loss.

#### Rhabdomyolysis

The occurrence of rhabdomyolysis may be heralded by the development of dark, tea-colored urine and tender edematous muscles. Rhabdomyolysis releases large amounts of myoglobin, which can precipitate in the kidneys and result in acute kidney injury (AKI). Renal failure especially is common in patients who develop hypotension or shock during the course of their disease and may occur in as many as 25-30% of patients with EHS. **Treatment of rhabdomyolysis involves infusion of large amounts of intravenous fluids** (fluid requirements may be as high as 10 L), alkalization of the urine, and infusion of mannitol. Fluid administration is best guided by invasive hemodynamic parameters, and urine output should be maintained at 3 mL/kg/h to minimize the risk of renal failure. **Alkalinization of the urine (to a pH of 7.5-8.0) prevents the precipitation of myoglobin** in the renal tubules and may control acidosis and hyperkalemia in acute massive muscle necrosis. Mannitol may improve renal blood flow and glomerular filtration rate, increase urine output, and prevent fluid accumulation in the interstitial compartment (through its osmotic action). Mannitol also is a free radical scavenger and, therefore, may reduce damage caused by free radicals. Once renal failure occurs, dialysis is the only effective therapeutic modality for rhabdomyolysis.

#### Metabolic support

Muscle necrosis may occur so rapidly that hyperkalemia, hypocalcemia, and hyperphosphatemia become significant enough to cause cardiac arrhythmias and require immediate therapy. In the presence of renal failure, hemodialysis may be necessary. **Hypertonic dextrose and sodium bicarbonate may be used to shift potassium** into the intracellular environment while more definitive measures (eg, intestinal potassium binding, dialysis) are prepared. Use of insulin may not be necessary in patients who are not diabetic and may be deleterious for patients with EHS and patients with liver failure, who commonly develop hypoglycemia. **Calcium should be used judiciously because it may precipitate** in and cause additional muscle damage. Use of calcium is reserved for patients with ventricular ectopy, impending convulsions, or electrocardiographic evidence of hyperkalemia. **Various other electrolyte abnormalities have been reported in patients** with heat stroke and must be monitored closely and treated carefully. These abnormalities may be related to solute-altering conditions such as vomiting, diarrhea, and use of diuretics. For example, hypokalemia, which is common in the early phases of heat stroke, may develop in response to respiratory alkalosis, diarrhea, and sweating. Similarly, hyponatremia may be due to sodium losses and/or rehydration with salt-poor solutions (eg, water), and hypernatremia may be due to dehydration.

#### Hepatic injury

Heat stroke commonly leads to severe but reversible hepatic damage.

Hepatic injury is represented by elevations in transaminase levels and bilirubin. During this phase, hypoglycemia, abnormal coagulation, cerebral edema, and death can occur, although rarely. **Prolonged coagulation times also may signal the development of disseminated intravascular coagulation (DIC)**, which, when present, carries a poor patient prognosis. Clinical manifestations can range from abnormal laboratory values to generalized bleeding occurring approximately 48 hours after the initial insult. DIC also may predispose patients to develop acute respiratory distress syndrome (ARDS), which also increases mortality.

Treatment of hepatic failure includes the following:

- Infusion of dextrose solutions to correct hypoglycemia
- Early recognition and treatment of DIC, with replacement of clotting factors, fresh frozen plasma, platelets, and blood
- Meticulous respiratory support

#### **Pulmonary injury**

Pulmonary edema is a common complication of heat stroke and may be due to a number of factors, including fluid overload from aggressive

rehydration, renal failure, congestive heart failure, and ARDS. The latter may develop because of multiple insults, including heat-induced pulmonary damage, aspiration pneumonia, and as a complication of liver failure. ARDS should be treated aggressively, with early mechanical ventilation and positive end-expiratory pressure (PEEP).

#### **Renal injury**

Acute kidney injury (AKI) may occur because of direct thermal injury of the kidney, myoglobinuria, hypotension, and/or shock (acute tubular necrosis). Early manifestations of AKI include oliguria, low-grade proteinuria, and granular casts. **AKI initially is treated with intravenous fluids, diuretics, and correction of associated acid-base and electrolyte abnormalities.** In the setting of rhabdomyolysis, mannitol may be the diuretic of choice because it does not interfere with the acid-base status of the urine, and it may have antioxidant activity. Furosemide may cause tubular acidosis and, therefore, may promote myoglobin deposition within the renal tubules. Once renal failure has set in, hemodialysis is the most effective therapy.

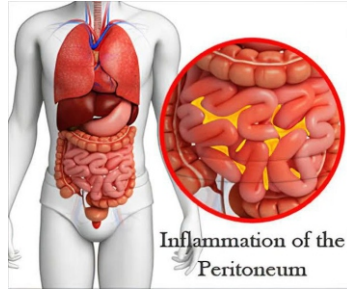
## UNDERSTANDING

### PERITONITIS

(secondary & tertiary – Etiology, D/D & Diagnostic Approach)

#### ETIOLOGY

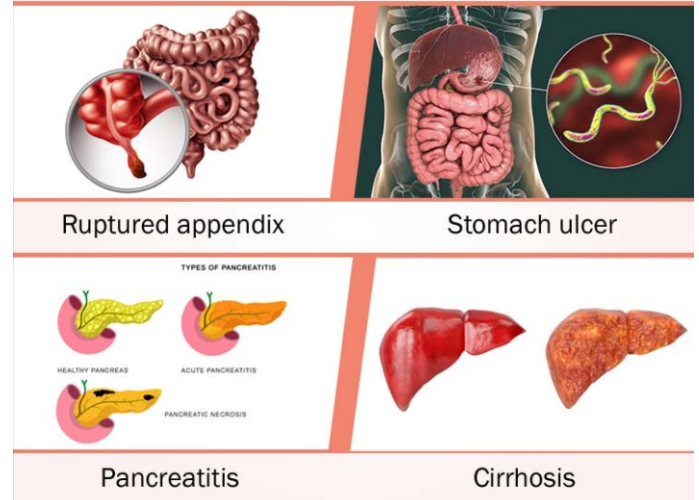
Table 1: Common Causes of Secondary Peritonitis



Source Regions	Causes
Esophagus	Boerhaave syndrome Malignancy Trauma (mostly penetrating) iatrogenic*
Stomach	Peptic ulcer perforation Malignancy (eg, adenocarcinoma, lymphoma, gastrointestinal stromal tumor) Trauma (mostly penetrating) iatrogenic*
Duodenum	Peptic ulcer perforation Trauma (blunt and penetrating) iatrogenic*
Biliary tract	Cholecystitis Stone perforation from gallbladder (ie, gallstone ileus) or common duct Malignancy Choledochal cyst (rare) Trauma (mostly penetrating) iatrogenic*
Pancreas	Pancreatitis (eg, alcohol, drugs, gallstones) Trauma (blunt and penetrating) iatrogenic*
Small bowel	Ischemic bowel Incarcerated hernia (internal and external) Closed loop obstruction Crohn disease Malignancy (rare) Meckel diverticulum Trauma (mostly penetrating)
Large bowel and appendix	Ischemic bowel Diverticulitis Malignancy Ulcerative colitis and Crohn disease Appendicitis Colonic volvulus Trauma (mostly penetrating) iatrogenic
Uterus, salpinx, and ovaries	Pelvic inflammatory disease (eg, salpingo-oophoritis, tubo-ovarian abscess, ovarian cyst) Malignancy (rare) Trauma (uncommon)

\*Iatrogenic trauma to the upper GI tract, including the pancreas and biliary tract and colon, often results from endoscopic procedures; anastomotic dehiscence and inadvertent bowel injury (eg, mechanical, thermal) are common causes of leak in the postoperative period.

### Causes of Peritonitis



Common organisms cultured in secondary peritonitis are presented in Table 2

Table 2. Microbial Flora of Secondary Peritonitis

Type	Organism	Percentage
<b>Aerobic</b>	Gram negative	
	<i>Escherichia coli</i>	60%
	<i>Enterobacter/Klebsiella</i>	26%
	<i>Proteus</i>	22%
	<i>Pseudomonas</i>	8%
Gram positive	Streptococci	28%
	Enterococci	17%
	Staphylococci	7%
	<b>Anaerobic</b>	<i>Bacteroides</i>
	<i>Eubacteria</i>	24%
	<i>Clostridia</i>	17%
	Peptostreptococci	14%
	Peptococci	11%
<b>Fungi</b>	<i>Candida</i>	2%

Other rare, nonsurgical causes of intra-abdominal sepsis include the following:

- *Chlamydia* peritonitis
- Tuberculosis peritonitis
- Acquired immunodeficiency syndrome (AIDS)-associated peritonitis

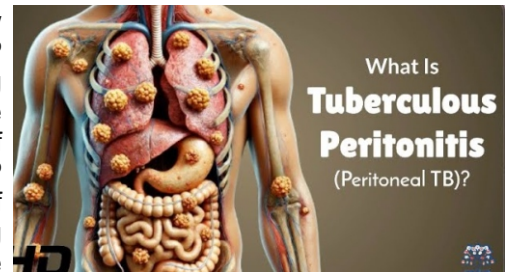
The most common cause of postoperative peritonitis is anastomotic leak, with symptoms generally appearing around postoperative days 5-7. After elective abdominal operations for noninfectious etiologies, the incidence of SP (caused by anastomotic disruption, breakdown of enterotomy closures, or inadvertent bowel injury) should be less than

2%. Operations for inflammatory disease (ie, appendicitis, diverticulitis, cholecystitis) without perforation carry a risk of less than 10% for the development of SP and peritoneal abscess. This risk may rise to greater than 50% in gangrenous bowel disease and visceral perforation. After operations for penetrating abdominal trauma, SP and abscess formation are observed in a small number of patients. Duodenal and pancreatic involvement, as well as colon perforation, gross peritoneal contamination, perioperative shock, and massive transfusion, are factors that increase the risk of infection in these cases. Peritonitis is also a frequent complication and significant limitation of peritoneal dialysis. Peritonitis leads to increased hospitalization and mortality rates.

#### Tertiary peritonitis

Tertiary peritonitis (see Table 3, below) develops more frequently in immunocompromised patients and in people with significant preexisting comorbid conditions. Although rarely observed in uncomplicated peritoneal infections, the incidence of tertiary peritonitis in patients requiring ICU admission for severe abdominal infections may be as high as 50-74%. Tuberculous peritonitis (TP) is rare in the United States (< 2% of all causes of peritonitis), but it continues to be a significant problem in developing countries and among patients with human immunodeficiency virus (HIV) infection. The presenting symptoms are often nonspecific and insidious in onset (eg, low-grade fever, anorexia,

weight loss). Many patients with TP have underlying cirrhosis. More than 95% of patients with TP have evidence of ascites on imaging studies, and more than half of these patients have clinically apparent ascites. In most cases, chest radiographic findings in patients with TP peritonitis are abnormal; active pulmonary disease is uncommon (< 30%). Results on Gram stain of ascitic fluid are rarely positive, and culture results may be falsely negative in up to 80% of patients. A peritoneal fluid protein level greater than 2.5 g/dL, a lactate dehydrogenase (LDH) level greater than 90 U/mL, or a predominantly mononuclear cell count of greater than 500 cells/ $\mu$ L should raise suspicion of TP but have limited specificity for the diagnosis. Laparoscopy and visualization of granulomas on peritoneal biopsy specimens, as well as cultures (requires 4-6 wk), may be needed for the definitive diagnosis; however, empiric therapy should begin immediately.



**Table 3. Microbiology of Primary, Secondary, and Tertiary Peritonitis**

Peritonitis (Type)	Etiologic Organisms		Antibiotic Therapy (Suggested)
	Class	Type of Organism	
Primary	Gram-negative	<i>E coli</i> (40%) <i>K pneumoniae</i> (7%) <i>Pseudomonas</i> species (5%) <i>Proteus</i> species (5%) <i>Streptococcus</i> species (15%) <i>Staphylococcus</i> species (3%) Anaerobic species (< 5%)	Third-generation cephalosporin
Secondary	Gram-negative	<i>E coli</i> <i>Enterobacter</i> species <i>Klebsiella</i> species <i>Proteus</i> species	Second-generation cephalosporin Third-generation cephalosporin Penicillins with anaerobic activity Quinolones with anaerobic activity Quinolone and metronidazole Aminoglycoside and metronidazole
	Gram-positive	<i>Streptococcus</i> species <i>Enterococcus</i> species	
	Anaerobic	<i>Bacteroides fragilis</i> Other <i>Bacteroides</i> species <i>Eubacterium</i> species <i>Clostridium</i> species Anaerobic <i>Streptococcus</i> species	
Tertiary	Gram-negative	<i>Enterobacter</i> species <i>Pseudomonas</i> species <i>Enterococcus</i> species	Second-generation cephalosporin Third-generation cephalosporin Penicillins with anaerobic activity Quinolones with anaerobic activity Quinolone and metronidazole Aminoglycoside and metronidazole Carbapenems Triazoles or amphotericin (considered in fungal etiology) (Alter therapy based on culture results.)
	Gram-positive	<i>Staphylococcus</i> species	
	Fungal	<i>Candida</i> species	

## Chemical peritonitis

Chemical (sterile) peritonitis may be caused by irritants such as bile, blood, barium, or other substances or by transmural inflammation of visceral organs (eg, Crohn disease) without bacterial inoculation of the peritoneal cavity. Clinical signs and symptoms are indistinguishable from those of SP or peritoneal abscess, and the diagnostic and therapeutic approach should be the same.

## Peritoneal abscess

Peritoneal abscess describes the formation of an infected fluid collection encapsulated by fibrinous exudate, omentum, and/or adjacent visceral organs. The overwhelming majority of abscesses occur subsequent to SP. Abscess formation may be a complication of surgery. The incidence of abscess formation after abdominal surgery is less than 1-2%, even when the operation is performed for an acute inflammatory process. The risk of abscess increases to 10-30% in cases of preoperative perforation of the hollow viscus, significant fecal contamination of the peritoneal cavity, bowel ischemia, delayed diagnosis and therapy of the initial peritonitis, and the need for reoperation, as well as in the setting of immunosuppression. Abscess formation is the leading cause of persistent infection and development of tertiary peritonitis.

## Differential Diagnosis

### Diagnostic Considerations

Thoracic processes with diaphragmatic irritation (eg, empyema), extraperitoneal processes (eg, pyelonephritis, cystitis, acute urinary retention), and abdominal wall processes (eg, infection, rectus hematoma) may mimic certain signs and symptoms of peritonitis. Always examine the patient for the presence of external hernias to rule out intestinal incarceration. According to Adler and Gasbarra, the following should be considered in the differential diagnosis :

- Chemical irritants (eg, bile, blood, gastric juice, barium, enema or douche contents)
- Chronic peritoneal dialysis
- Chylous Peritonitis
- Eosinophilic peritonitis
- Familial Mediterranean fever
- Fungal infections (eg, histoplasmosis, cryptococcosis, coccidioidomycosis)
- Granulomatous peritonitis (eg, parasitic infestations, sarcoidosis, tumors, Crohn disease, starch granules)
- Gynecologic disorders (*Chlamydia* peritonitis, salpingitis, endometriosis, teratoma, leiomyomatosis, dermoid cyst)
- Human immunodeficiency virus (HIV)-associated peritonitis (from opportunistic organisms)
- Mesothelial hyperplasia and metaplasia
- Neoplasms (eg, primary mesothelioma, secondary carcinomatosis, *Pseudomyxoma peritonei*)
- Parasitic infections (eg, schistosomiasis, ascariasis, enterobiasis, amebiasis, strongyloidiasis)
- Perforated viscus
- Peritoneal encapsulation
- Peritoneal loose bodies and peritoneal cysts
- Peritoneal lymphangiectasis
- Pyelonephritis
- Sclerosing peritonitis
- Splenosis
- Vascular conditions (eg, mesenteric embolus, mesenteric nonocclusive ischemia, ischemic colitis, portal vein thrombosis, mesenteric vein thrombosis)
- Vasculitis (eg, systemic lupus erythematosus, allergic vasculitis

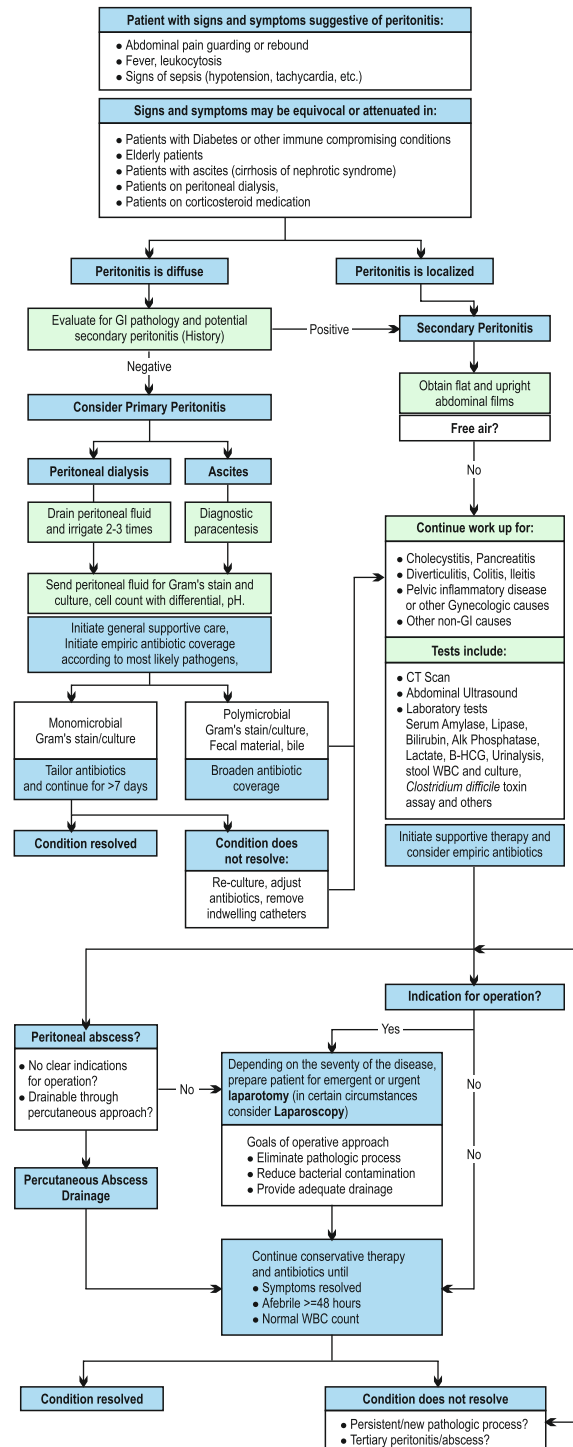
[Henoch-Schönlein purpura], Kohlmeier-Degos disease, polyarteritis nodosa)

## Differential Diagnosis

- Acute Angioedema
- Appendicitis
- Urinary Tract Infection (UTI) and Cystitis (Bladder Infection) in Females
- Whipple Disease.

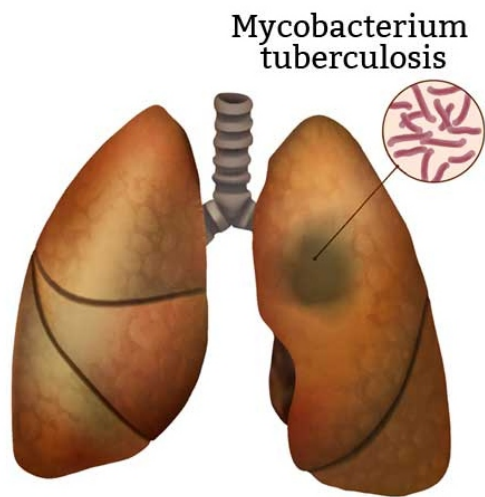
## DIAGNOSTIC APPROACH

Diagnostic and therapeutic approach to peritonitis and peritoneal abscess:



## TROUBLESHOOTING

### TB DIAGNOSIS



Troubleshooting a TB diagnosis involves a multi-faceted approach, starting with initial suspicion based on symptoms and risk factors, followed by a series of tests to confirm or rule out active TB disease. If initial tests are inconclusive, further investigations like sputum cultures and molecular tests (NAATs) can be employed. Chest X-rays are also crucial for identifying abnormalities suggestive of TB.

Here's a more detailed breakdown:

#### 1. Initial Evaluation and Suspicion:

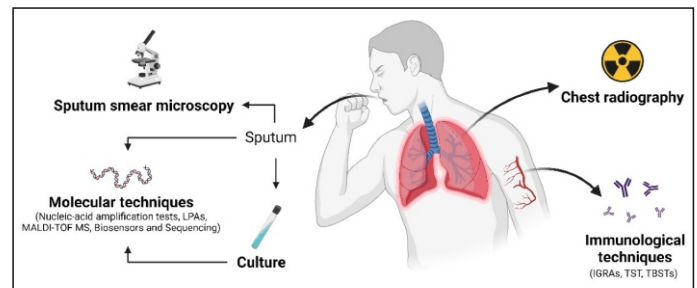
- **Clinical History:**  
Doctors will inquire about symptoms like persistent cough, fever, night sweats, weight loss, and any known TB exposure.
- **Physical Examination:**  
A stethoscope will be used to listen to the lungs, and lymph nodes in the neck will be checked for swelling.
- **Risk Factors:**  
Factors like recent contact with someone with TB, travel to TB-endemic areas, or a history of TB exposure will increase suspicion.

#### 2. Initial Diagnostic Tests:

- **Chest X-ray:**  
This can reveal characteristic features of TB, such as patches in the lungs.
- **TB Skin Test (TST) or IGRA (Interferon-Gamma Release Assay):**  
These tests can indicate exposure to TB bacteria, but not necessarily active disease.
- **Sputum Examination and Culture:**  
If the cough produces sputum, samples are analyzed for the presence of TB bacteria, including both smear microscopy and culture.

#### 3. Further Investigations (if needed):

- **Molecular Tests (NAATs):**  
Rapid tests like PCR can detect TB bacteria in sputum samples more quickly than traditional cultures.
- **Drug Susceptibility Testing:**  
If the TB bacteria are found, tests are conducted to determine if they are resistant to common antibiotics.
- **ADA test :**  
Adenosine deaminase (ADA) testing can aid in the diagnosis of tuberculosis (TB), particularly tuberculous pleural effusion (TPE). Elevated ADA levels in pleural fluid are often associated with TB, with a cut-off value of 40 U/L commonly used for diagnosis.
- **Other Tests:**  
Depending on the individual case, other tests like urine tests, blood tests, or biopsies may be considered.



#### 4. Interpreting Results and Treatment:

- **Positive Results:** Confirm TB infection, either active or latent.
- **Negative Results:** Help rule out active TB, but further investigation may be needed if suspicion remains.
- **Treatment:** Active TB is treated with antibiotics, often for several months. Latent TB is usually treated with antibiotics as well.

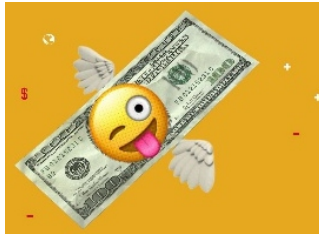
#### 5. Troubleshooting Inconclusive Results:

- **False-Negative Results:**  
If a test is negative despite clinical suspicion, consider alternative diagnoses like other infections, or repeat testing.
- **False-Positive Results:**  
If a test is positive but other factors suggest it's false, consider the possibility of past infection, or other conditions that can cause a false positive.
- **Difficulties Obtaining Samples:**  
In some cases, obtaining appropriate samples from certain areas, like non-reachable sites, can be challenging.

In summary, troubleshooting a TB diagnosis involves a systematic approach, utilizing a combination of clinical evaluation, initial tests, and further investigations when needed. Accurate interpretation of results, along with consideration of the patient's history and clinical picture, is crucial for effective diagnosis and treatment.

## BOUQUET

### In Lighter Vein



Employee: three companies were after me and I need a raise....

Boss: "which companies?"

Employee: Gas, water and electricity company.



My boss calls me "The computer"

Not because of my calculation skills but because I go to sleep when left unattended for 15 minutes.

My boss said I was going to see a big increase on my Payslip this month...

... He increased the font size.



## Wisdom Whispers

When it hurts...Observe.

Life is trying to teach you something



Happiness does not depend on what you have or what you are. It solely relies on what you think.



Be patient. Everything comes to you in the right moment.



## Brain Teasers

1. **What contributes to heat exposure?**
  - a. Temperature and humidity.
  - b. Direct sun.
  - c. Hot surfaces.
  - d. All of the above
2. **When working in the heat, it is best to:**
  - a. Drink 5 to 7 ounces of fluid every 15 to 20 minutes.
  - b. Drink beverages every hour.
  - c. Drink beverages containing caffeine every half hour.
  - d. Wait until you feel thirsty before you drink.
3. **If someone has heat exhaustion, he or she may:**
  - a. Feel weak, dizzy, or nauseous, and have clammy, moist skin.
  - b. Vomit or lose consciousness.
  - c. Need to see a doctor.
  - d. All of the above.
4. **What can you do to control heat exposure?**
  - a. Work as fast and hard as you can to get the job over with.
  - b. Take periodic rest breaks in a cool, shaded area and drink small quantities of water frequently.
  - c. Pace the work and schedule the heaviest jobs during cooler times of the day.
  - d. Both b and c.

# ADA TESTING !!!!

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