Emergent medical

Sudden death (athletes)

The prevention of sudden death by athletes, defined as natural, unexpected, death within one hour of the onset of symptoms and subsequent cardiac arrest, remains a challenge. First, the prevalence of any single, associated, condition is very low, probably less than 0.3%. Second, the sensitivity and specificity of tests commonly used to screen leaves much to be desired. Wider definitions of sudden death are also in use but not usually applied to the athletic situation.

The single most important predictor is exercise fainting or near fainting, which should be a warning requiring explanation and investigation.

While almost all of the causes relate to congenital or acquired cardiovascular disease, an exception is commotio cordis, in which the heart is structurally normal but a potentially fatal loss of normal rhythm occurs because of the accident of timing of a blow to the chest. Survival is about 15% and time to CPR and defibrillation may determine survival.

Incidence

Sudden death occurs in approximately one in 200,000 athletes annually. The victim is usually male and, in the US, two-thirds occurs in football and basketball. This reflects the popularity of these sports and the number of athletes involved in them and elsewhere in the world, soccer is most commonly associated. From statistics derived from NCAA data, one death per 25,000 collegiate athletes over a five-year period was attributable to medical causes.

A series of observational studies have reported on death rates in athletes for causes other than trauma. Van Camp reported rates of approximately 7:1,000,000 and 1:1,000,000 for male and female high school and colleges students respectively, during organized athletic activity. Philips reported rates of 1:735,000 US Air Force recruits aged 17 to 28 during training. The sudden death rate amongst Rhode Island joggers was reported as 1:280,000 for those under 30 years of age, by Ragosta, and 1:7,260 for those 30–65 years according to Thompson. Finally, Maron reported rates of 1:50,000 race finishers in marathons, where the mean age was 37.

Age

Age 35 serves as a watershed as to the likely cause of sudden death. Before age 35, congenital abnormalities of the heart and blood vessels predominate. These are usually asymptomatic prior to the fatal event although not invariably so. Congenital cardiovascular problems are reported to occur disproportionately in African-American athletes.

After age 35, acquired coronary artery disease predominates (80%), and this is true regardless of the athlete's former level of fitness.
Causes

- Hypertrophic cardiomyopathy 26.4% Genetically determined
- Commotio cordis 19.9% Structurally normal heart
- Coronary artery anomalies 13.7% Exact mechanisms unknown - some association with other congenital cvs abnormalities
- Left ventricular hypertrophy of undetermined origin 7.5% Probable variant of hypertrophic cardiomyopathy
- Myocarditis 5.2%
- Ruptured aortic aneurysm (Marfan syndrome) 3.2% Genetically determined
- Arrhythmogenic right ventricular cardiomyopathy 2.8% Genetically determined
- Tunned coronary artery 2.8%
- Aortic valve stenosis 2.6%
- Atherosclerotic coronary artery disease 2.6% Mainly acquired

Screening

Screening is problematic because of low prevalence and indifferent performance of various tests that have been used. Nevertheless, sudden death attracts much public and legislator attention because of its visible and tragic nature. As an example, the Texas Legislature appropriated funds for a pilot study of state-wide screening in 2007.

The study employed a combination of questionnaire, examination, and electrocardiography, in all of the students and echocardiography in a majority selected because of abnormal findings in the preceding three. 35% of the student population studied were flagged as potentially at risk but there were many false positive results with actual disease being confirmed in only 1.23%. Further, a substantial number of that 1.23% declined follow-up evaluation. It should be stressed that this is a single pilot program and original research, but it is indicative of the problems associated with screening and concordant with other experience.
**Genetics**

*Cardiomyopathy*

Cardiomyopathies are generally inherited as autosomal dominants although recessive forms have been described, and dilated cardiomyopathy can also be inherited in a X linked pattern. Consequently, in addition to tragedy involving an athlete who succumbs, there are wider implications. Within many families of index cases, more than 300 causative mutations have been identified although there is no clear understanding of how these mutations (which affect the same myosin molecule) can lead to the dramatically different clinical characteristics and outcomes associated with hypertrophic cardiomyopathy(HCM) and dilated cardiomyopathy(DCM).

Since HCM, as an example, is typically an autosomal dominant trait, children of an HCM parent have a 50% chance of inheriting the mutation. In individuals without a family history, the most common cause of the disease is a 'de novo' mutation of the gene that produces the β-myosin heavy chain. Not all mutations, it should be noted, have the same potential for a disastrous outcome. For example, troponin T mutations are associated with a 50% mortality before the age of 40, while β-myosin mutations are less threatening.

*Heritable connective tissue disease.* On the other hand, heritable connective tissue diseases are rare, each disorder estimated at one to ten per 100,000 of which *Marfan syndrome* is the most common.

It is carried by the FBN1 gene on chromosome 15, which encodes the connective protein fibrillin-1, inherited as a dominant trait. This protein is essential for synthesis and maintenance of elastic fibers. Since these fibers are particularly abundant in the aorta, ligaments and the ciliary zonules of the eye, these areas are among the worst affected. Everyone has a pair of FBN1 genes and because transmission is dominant, those who have inherited one affected FBN1 gene from either parent will have Marfan syndrome. Although it is most frequently inherited as an 'autosomal dominant', there is no family history in 25% of cases.

Recruiting practices aimed at attracting athletes who are unusually tall or who have an unusually wide arm span (phenotypic characteristics of Marfan Syndrome) have the potential to alter the expected prevalence of the syndrome within certain sports.

**Testing**

Once a disease causing mutation has been identified in an index case, the main task is genetic identification of carriers within a pedigree, a process known as "cascade testing." Family members with the same mutation may show different severities of disease, a phenomenon known as variable penetrance. As a result, some may remain asymptomatic, with little lifelong evidence of disease. Nevertheless, their children remain at risk of inheriting the disorder and potentially being more severely affected.
Notable athletes who have succumbed

- Pheidippides
- Hank Gathers
- Reggie Lewis
- Marc Vivien Foe
- Pete Maravich
- Flo Hyman
- Jim Fixx
- Sergei Grinkov
- Darryl Kile
- Miklós Fehér

References
Hyperthermia

Hyperthermia is elevated body temperature due to failed thermoregulation that occurs when a body produces or absorbs more heat than it dissipates. Extreme temperature elevation then becomes a medical emergency requiring immediate treatment to prevent disability or death.

The most common causes include heat stroke and adverse reactions to drugs. The former is an acute temperature elevation caused by exposure to excessive heat, or combination of heat and humidity, that overwhelms the heat-regulating mechanisms. The latter is a relatively rare side effect of many drugs, particularly those that affect the central nervous system. Malignant hyperthermia is a rare complication of some types of general anesthesia.

Hyperthermia can also be deliberately induced using drugs or medical devices and may be used in the treatment of some kinds of cancer and other conditions, most commonly in conjunction with radiotherapy.\[4\] Hyperthermia differs from fever in that the body's temperature set point remains unchanged. The opposite is hypothermia, which occurs when the temperature drops below that required to maintain normal metabolism.

Classification

Hyperthermia is defined as a temperature greater than 37.5–38.3 °C (99.5–100.9 °F), depending on the reference used, that occurs without a change in the body's temperature set point.

The normal human body temperature in health can be as high as 37.7 °C (99.9 °F) in the late afternoon. Hyperthermia requires an elevation from the temperature that would otherwise be expected. Such elevations range from mild to extreme; body temperatures above 40 °C (104 °F) can be life-threatening.
Signs and symptoms

Hot, dry, skin is typical as blood vessels dilate in an attempt to increase heat loss. An inability to cool the body through perspiration may cause the skin to feel dry.

Other signs and symptoms vary. Accompanying dehydration can produce nausea, vomiting, headaches, and low blood pressure and the latter can lead to fainting or dizziness, especially if the standing position is assumed quickly.

In severe heat stroke, there may be confused, hostile, or seemingly intoxicated behavior. Heart rate and respiration rate will increase (tachycardia and tachypnea) as blood pressure drops and the heart attempts to maintain adequate circulation. The decrease in blood pressure can then cause blood vessels to contract reflexly, resulting in a pale or bluish skin color in advanced cases. Young children, in particular, may have seizures. Eventually, organ failure, unconsciousness and death will result.

Causes

Heat stroke

Heat stroke occurs when thermoregulation is overwhelmed by a combination of excessive metabolic production of heat (exertion), excessive environmental heat, and insufficient or impaired heat loss, resulting in an abnormally high body temperature. In severe cases, temperatures can exceed 40 °C (104 °F). Heat stroke may be non-exertional (classic) or exertional.

Significant physical exertion in hot conditions can generate heat beyond the ability to cool, because, in addition to the heat, humidity of the environment may reduce the efficiency of the body's normal cooling mechanisms. Heat loss mechanisms are limited to vasodilation of skin vessels and increased rate of sweating. Vasodilation dissipates heat by convection and sweating by evaporation. However, thermoregulation can be assisted with shade or fans. Other factors, such as insufficient water intake, consuming alcohol, or lack of air conditioning, can worsen the problem.

The principles of physics involved include:

- Newton's law of cooling which states that dry heat loss is proportional to temperature difference between the human body (shell) and surroundings;
- Stefan-Boltzmann law which states that the higher the temperature of an object, the more it radiates, and the energy radiating from an object and received by the human body is proportional to temperature difference between object and skin.

Non-exertional heat stroke mostly affects the young and elderly. In the elderly in particular, it can be precipitated by medications such as anticholinergic drugs, antihistamines, and diuretics that reduce vasodilation, sweating, and other heat-loss mechanisms. In this situation, the body's tolerance for high environmental temperature may be insufficient, even at rest.

Heat waves in the United States are followed by a rise in the death rate and these 'classical hyperthermia' deaths involve the elderly and infirm. This is partly because thermoregulation involves cardiovascular, respiratory and renal systems which may be inadequate for the additional stress because of the existing burden of aging and disease, further compromised by medications. During the July 1995 heat wave in Chicago, there were at least 700 heat-related deaths. The strongest risk factors were being confined to bed, and living alone, while the risk was reduced for those with working air conditioners and those with access to transportation. Even then, reported deaths may be underestimates as diagnosis can be misclassified as stroke or heart attack.
Drugs
Some drugs cause excessive internal heat production. The rate of drug-induced hyperthermia is higher where use of these drugs is higher.

- Many psychotropic medications, such as selective serotonin reuptake inhibitors (SSRIs), monoamine oxidase inhibitors (MAOIs), and tricyclic antidepressants, can cause hyperthermia. Serotonin syndrome is a rare adverse reaction to overdose of these medications or the use of several simultaneously. Similarly, neuroleptic malignant syndrome is an uncommon reaction to neuroleptic agents. These syndromes are differentiated by other associated symptoms, such as tremor in serotonin syndrome and "lead-pipe" muscle rigidity in neuroleptic malignant syndrome.
- Various stimulant drugs, including amphetamines, cocaine, PCP, LSD, and MDMA can produce hyperthermia as an adverse effect.
- Malignant hyperthermia is a rare reaction to common anesthetic agents (such as halothane) or the paralytic agent succinylcholine. Those who suffer this reaction, which is potentially fatal, have a genetic predisposition.
- The use of anticholinergics, more specifically muscarinic antagonists are thought to cause mild hyperthermic episodes due to its parasympatholytic effects. The sympathetic nervous system a.k.a the "Fight or Flight Response" dominates by raising catecholamine levels by the blocked action of the Rest and Digest System.

Personal protective equipment
Those working in industry, the military and first responders, may be required to wear personal protective equipment (PPE) against hazards such as chemical agents, gases, fire, small arms and even Improvised Explosive Devices (IEDs). PPE includes a range of hazmat suits, firefighting turnout gear, body armor and bomb suits, amongst others. Depending on design, the wearer may be encapsulated in a microclimate, due to an increase in thermal resistance and decrease in vapor permeability. As physical work is performed, the body's natural thermoregulation (i.e., sweating) becomes ineffective. This is compounded by increased work rates, high ambient temperature and humidity levels, and direct exposure to the sun. The net effect is that desired protection from some environmental threats inadvertently increases the threat of heat stress.
Other causes of hyperthermia include thyrotoxicosis and an adrenal gland tumor, called pheochromocytoma, both of which can cause increased heat production. Damage to the central nervous system, from brain hemorrhage, status epilepticus, and other kinds of injury to the hypothalamus can also cause hyperthermia.

Pathophysiology
A fever occurs when the core temperature is set higher, through the action of the pre-optic region of the anterior hypothalamus. For example, in response to a bacterial or viral infection, certain white blood cells within the blood will release pyrogens which have a direct effect on the anterior hypothalamus, causing body temperature to rise, much like raising the temperature setting on a thermostat.

In contrast, hyperthermia occurs when the body temperature rises without a change in the heat control centers.

Some of the gastrointestinal symptoms of acute exertional heat stroke, such as vomiting, diarrhea, and gastrointestinal bleeding, may be caused by barrier dysfunction and subsequent endotoxemia. Ultraendurance athletes have been found to have significantly increased plasma endotoxin levels. Endotoxin stimulates many inflammatory cytokines, which in turn may cause multiorgan dysfunction. Experimentally, monkeys treated with oral antibiotics prior to induction of heat stroke do not become endotoxemic.[5]

There is scientific support for the concept of a temperature set point - that is, maintenance of an optimal temperature for the metabolic processes that life depends on. Nervous activity in the preoptic-anterior hypothalamus of the brain triggers heat losing (sweating, etc.) or heat generating (shivering and muscle contraction, etc.) activities through stimulation of the autonomic nervous system. The pre-optic anterior hypothalamus has been shown to contain warm sensitive, cool sensitive, and temperature insensitive neurons, to determine the body's temperature setpoint. As the temperature that these neurons are exposed to rises above 37 °C, the rate of electrical discharge of the warm-sensitive neurons increases progressively. Cold-sensitive neurons increase their rate of electrical discharge progressively below 37 °C.

Diagnosis
Hyperthermia is generally diagnosed by the combination of unexpectedly high body temperature and a history that supports hyperthermia instead of a fever. Most commonly this means that the elevated temperature has occurred in a hot, humid environment (heat stroke) or in someone taking a drug for which hyperthermia is a known side effect (drug-induced hyperthermia). The presence of signs and symptoms related to hyperthermia syndromes, such as extrapyramidal symptoms characteristic of neuroleptic malignant syndrome, and the absence of signs and symptoms more commonly related to infection-related fevers, are also considered in making the diagnosis.

If fever-reducing drugs lower the body temperature, even if the temperature does not return entirely to normal, then hyperthermia is excluded.
Hyperthermia

Prevention

When ambient temperature is excessive, humans and many animals cool themselves below ambient by evaporative cooling of sweat (or other aqueous liquid; saliva in dogs, for example); this helps prevent potentially fatal hyperthermia. The effectiveness of evaporative cooling depends upon humidity. Wet-bulb temperature, which takes humidity into account, or more complex calculated quantities such as wet-bulb globe temperature (WBGT), which also takes solar radiation into account, give useful indications of the degree of heat stress and are used by several agencies as the basis for heat-stress prevention guidelines. (Wet-bulb temperature is essentially the lowest skin temperature attainable by evaporative cooling at a given ambient temperature and humidity.)

A sustained wet-bulb temperature exceeding 35 °C is likely to be fatal even to fit and healthy people unclothed in the shade next to a fan; at this temperature, environmental heat gain instead of loss occurs. As of 2012 wet-bulb temperatures only very rarely exceeded 30 °C anywhere, although significant global warming may change this.

In cases of heat stress caused by physical exertion, hot environments, or protective equipment, prevention or mitigation by frequent rest breaks, careful hydration, and monitoring body temperature should be attempted. However, in situations demanding one is exposed to a hot environment for a prolonged period or must wear protective equipment, a personal cooling system is required as a matter of health and safety. There is a variety of active or passive personal cooling systems; these can be categorized by their power sources and whether they are person- or vehicle-mounted.

Because of the broad variety of operating conditions, these devices must meet specific requirements concerning their rate and duration of cooling, their power source, and their adherence to health and safety regulations. Among other criteria are the user's need for physical mobility and autonomy. For example, active-liquid systems operate by chilling water and circulating it through a garment; the skin surface area is thereby cooled through conduction. This type of system has proven successful in certain military, law enforcement, and industrial applications. Bomb-disposal technicians wearing special suits to protect against improvised explosive devices (IEDs) use a small, ice-based chiller unit that is strapped to one leg; a liquid-circulating garment, usually a vest, is worn over the torso to maintain a safe core body temperature. By contrast, soldiers traveling in combat vehicles can face microclimate temperatures in excess of 65 °C and require a multiple-user, vehicle-powered cooling system with rapid connection capabilities. Requirements for hazmat teams, the medical community, and workers in heavy industry vary further.

<table>
<thead>
<tr>
<th>Hyperthermia Prevention</th>
<th>Cost per day</th>
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<tbody>
<tr>
<td>Keep water on head (such as by using a commercial, wetted sweat band)</td>
<td>0</td>
</tr>
<tr>
<td>Keep cool water on head</td>
<td>+</td>
</tr>
<tr>
<td>Provide shade, or move to a shaded area</td>
<td>+</td>
</tr>
<tr>
<td>Dress as if for running a 26.2-mile marathon</td>
<td>+</td>
</tr>
<tr>
<td>Stay sweaty or keep the patient wet with water</td>
<td>0</td>
</tr>
<tr>
<td>Increase air movement (e.g., use fans or move the person / patient into an area with better air current)</td>
<td>+</td>
</tr>
<tr>
<td>Use a water mister, as used by some restaurants on their patios, or by sporting venues</td>
<td>+</td>
</tr>
<tr>
<td>Lower the air temperature (e.g., open windows, operate mechanical refrigeration, and / or let the air flow over or through ice)</td>
<td>+</td>
</tr>
<tr>
<td>Place the person / patient in a tub of water, pool, or other body of water</td>
<td>+</td>
</tr>
</tbody>
</table>
Hyperthermia

Treatment

The underlying cause must be removed. Mild hyperthermia caused by exertion on a hot day may be adequately treated through self-care measures, such as increased water consumption and resting in a cool place. Hyperthermia that results from drug exposure requires prompt cessation of that drug, and occasionally the use of other drugs as counter measures. Fever-reducing drugs such as paracetamol and aspirin have value in treating hyperthermia.

When body temperature is significantly elevated, mechanical cooling methods are used to remove heat and to restore the body's ability to regulate its own temperatures. Passive cooling techniques, such as resting in a cool, shady area and removing clothing can be applied immediately. Active cooling methods, such as sponging the head, neck, and trunk with cool water, remove heat from the body and thereby speed the body's return to normal temperatures. Drinking water and turning a fan or dehumidifying air conditioning unit on the affected person may improve the effectiveness of the body's evaporative cooling mechanisms (sweating).

Sitting in a bathtub of tepid or cool water (immersion method) can remove a significant amount of heat in a relatively short period of time. It is thought by some that immersion in very cold water is counterproductive, as it causes vasoconstriction in the skin and thereby prevents heat from escaping the body core. However one British analysis of various studies stated "supporters of other cooling methods suggest that iced water immersion may cause peripheral vasoconstriction and therefore slow cooling, although this has never been proven experimentally. Indeed, a recent study using normal volunteers has shown that cooling rates were fastest when the coldest water was used".[8]

In exertional heat stroke, studies have shown that although there are practical limitations, cool water immersion is the most effective cooling technique and the biggest predictor of outcome is degree and duration of hyperthermia. No superior cooling method has been found for nonexertional heat stroke.[9]

When the body temperature reaches about 40 °C, or if the affected person is unconscious or showing signs of confusion, hyperthermia is considered a medical emergency that requires treatment in a proper medical facility. In a hospital, more aggressive cooling measures are available, including intravenous hydration, gastric lavage with iced saline, and even hemodialysis to cool the blood.

Epidemiology

The frequency of environmental hyperthermia can vary significantly from year to year depending on factors such as heat waves. Statistically, outdoor workers, including agricultural workers, are at increased risk of experiencing heat stress and the resulting negative health effects. Between 1992 and 2006, 68 crop workers died from heat stroke, representing a rate 20 times that of US civilian workers overall.

References

**External links**

- CDC Emergency Preparedness and Response: Extreme Heat (http://emergency.cdc.gov/disasters/extremeheat/)
- CDC - NIOSH Workplace Safety and Health Topics: Heat Stress (http://www.cdc.gov/niosh/topics/heatstress/)
- CDC - NIOSH Preventing Heat-related Illness or Death of Outdoor Workers (http://www.cdc.gov/niosh/docs/wp-solutions/2013-143/)
- Excessive Heat Events Guidebook, from the United States' Environmental Protection Agency (EPA) (http://www.epa.gov/hiri/about/pdf/EHEguide_final.pdf)
- Physiological Responses to Exercise in the Heat (http://books.nap.edu/openbook.php?record_id=2094&page=55)—Chapter 3 of *Nutritional Needs in Hot Environments* by the Institute of Medicine of the U.S. National Academies (of Science) (N.B.: entire book is available in HTML format via this link)
Red eye (medicine)

For other uses, see Red eye (disambiguation).

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<td><strong>Classification and external resources</strong></td>
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Subconjunctival hemorrhage causing red coloration as result of ruptured blood vessel in the eye.

<table>
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<th>ICD-10</th>
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</table>

In medicine, red eye is a non-specific term to describe an eye that appears red due to illness or injury. The term usually refers to injection and prominence of the superficial blood vessels of the conjunctiva, or sclera, which may be caused by disorders of these or adjacent structures. Conjunctivitis and subconjunctival hemorrhage are two of the less serious but more common causes.

Management includes assessing whether emergency action (including referral) is needed, or whether treatment can be accomplished without additional resources.

Slit lamp examination is invaluable in diagnosis but initial assessment can be performed using a careful history, testing vision (visual acuity), and carrying out a penlight examination.

**Differential diagnosis**

Of the many causes, conjunctivitis is the most common. Others include:

*Usually nonurgent*

- blepharitis - a usually chronic inflammation of the eyelids with scaling, sometimes resolving spontaneously
- subconjunctival hemorrhage - a sometimes dramatic, but usually harmless, bleeding underneath the conjunctiva most often from spontaneous rupture of the small, fragile blood vessels, commonly from a cough or sneeze
- inflamed pterygium - a benign, triangular, horizontal growth of the conjunctiva, arising from the inner side, at the level of contact of the upper and lower eyelids, associated with exposure to sunlight, low humidity and dust. It may be more common in occupations such as farming and welding.
- inflamed pinguecula [4] - a yellow-white deposit close to the junction between the cornea and sclera, on the conjunctiva. It is most prevalent in tropical climates with much UV exposure. Although harmless, it can occasionally become inflamed.
- dry eye syndrome - caused by either decreased tear production or increased tear film evaporation which may lead to irritation and redness
• airborne contaminants or irritants
• drug use including cannabis[^5]

**Usually urgent**
• acute glaucoma - implies injury to the optic nerve with the potential for irreversible vision loss which may be permanent unless treated quickly, as a result of increased pressure within the eyeball. Not all forms of glaucoma are acute, and not all are associated with increased 'intra-ocular' pressure.
• injury
• keratitis - a potentially serious inflammation

or injury to the cornea (window), often associated with significant pain, light intolerance, and deterioration in vision. Numerous causes include virus infection. Injury from contact lenses can lead to keratitis.
• iritis - together with the ciliary body and choroid, the iris makes up the uvea, part of the middle, pigmented, structures of the eye. Inflammation of this layer (uveitis) requires urgent control and is estimated to be responsible for 10% of blindness in the United States.
• scleritis - a serious inflammatory condition, often painful, that can result in permanent vision loss, and without an identifiable cause in half of those presenting with it. About 30-40% have an underlying systemic autoimmune condition.
• episcleritis - most often a mild, inflammatory disorder of the 'white' of the eye unassociated with eye complications in contrast to scleritis, and responding to topical medications such as anti-inflammatory drops.
• tick borne illnesses like Rocky Mountain spotted fever[^6] - the eye is not primarily involved, but the presence of conjunctivitis, along with fever and rash, may help with the diagnosis in appropriate circumstances.

**Diagnostic approach**

Particular signs and symptoms may indicate that the cause is serious and requires immediate attention. Six such signs are:
• reduced visual acuity
• ciliary flush (circumcorneal injection)
• corneal abnormalities including edema or opacities
• corneal staining
• abnormal pupil size
• abnormal intraocular pressure

**Visual acuity**
A reduction in visual acuity in a 'red eye' is indicative of serious ocular disease, such as keratitis, iridocyclitis, and glaucoma, and never occurs in simple conjunctivitis without accompanying corneal involvement.

**Ciliary flush**
Ciliary flush is usually present in eyes with corneal inflammation, iridocyclitis or acute glaucoma, though not simple conjunctivitis. A ciliary flush is a ring of red or violet spreading out from around the cornea of the eye.

**Corneal abnormalities**
The cornea requires to be transparent to transmit light to the retina. Because of injury, infection or inflammation, an
area of opacity may develop which can be seen with a penlight or ophthalmoscope. In rare instances, this opacity is congenital. In some, there is a family history of corneal growth disorders which may be progressive with age. Much more commonly, misuse of contact lenses may be a precipitating factor. Whichever, it is always potentially serious and sometimes necessitates urgent treatment and corneal opacities are the fourth leading cause of blindness. Opacities may be keratic, that is, due to the deposition of inflammatory cells, hazy, usually from corneal edema, or they may be localized in the case of corneal ulcer or keratitis. Corneal epithelial disruptions may be detected with fluorescein staining of the eye, and careful observation with cobalt-blue light. Corneal epithelial disruptions would stain green, which represents some injury of the corneal epithelium. These types of disruptions may be due to corneal inflammations or physical trauma to the cornea, such as a foreign body.

**Pupillary abnormalities**

In an eye with iridocyclitis, (inflammation of both the iris and ciliary body), the involved pupil will be smaller than the uninvolved, due to reflex muscle spasm of the sphincter muscle of the iris. Generally, conjunctivitis does not affect the pupils. With acute angle-closure glaucoma, the pupil is generally fixed in mid-position, oval, and responds sluggishly to light, if at all.

**Shallow anterior chamber depth** may indicate a predisposition to one form of glaucoma (narrow angle) but requires slit-lamp examination or other special techniques to determine it. In the presence of a "red eye", a shallow anterior chamber may indicate acute glaucoma, which requires immediate attention.

**Abnormal intraocular pressure**

Intraocular pressure should be measured as part of the routine eye examination. It is usually only elevated by iridocyclitis or acute-closure glaucoma, but not by relatively benign conditions. In iritis and traumatic perforating ocular injuries, the intraocular pressure is usually low.

**Important warning symptoms**

Three symptoms in particular require prompt and careful attention:

- reduced visual acuity
- severe ocular pain
- photophobia (light sensitivity)

**Blurry vision**

Blurry vision often indicates serious ocular disease. However, if the blurriness improves with blinking, it suggests ocular surface discharge of some variety. Coloured halos are an indication of corneal edema, and are a warning that acute glaucoma may be present.

**Severe pain**

Those with conjunctivitis may report mild irritation or scratchiness, but never extreme pain, which is an indicator of more serious disease such as keratitis, corneal ulceration, iridocyclitis, or acute glaucoma.

**Photophobia**

Photophobia (intolerance to light) is most characteristic of iritis and injury to the cornea, but may also be present in acute glaucoma (angle closure type).
References

Rhabdomyolysis

Classification and external resources

<table>
<thead>
<tr>
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<td>D012206 [9]</td>
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Urine from a person with rhabdomyolysis showing the characteristic brown discoloration as a result of myoglobinuria.

Rhabdomyolysis /ˈræbdəməˈlɪsəs/ is a condition in which damaged skeletal muscle tissue (Greek: ραβδός rhabdo-stripped μυς myo- muscle) breaks down (Greek: λύσις -lysis) rapidly. Breakdown products of damaged muscle cells are released into the bloodstream; some of these, such as the protein myoglobin, are harmful to the kidneys and may lead to kidney failure. The severity of the symptoms, which may include muscle pains, vomiting and confusion, depends on the extent of muscle damage and whether kidney failure develops. The muscle damage may be caused by physical factors (e.g., crush injury, strenuous exercise), medications, drug abuse, and infections. Some people have a hereditary muscle condition that increases the risk of rhabdomyolysis. The diagnosis is usually made with blood tests and urinalysis. The mainstay of treatment is generous quantities of intravenous fluids, but may include dialysis or hemofiltration in more severe cases.

Rhabdomyolysis and its complications are significant problems for those injured in disasters such as earthquakes and bombings. Relief efforts in areas struck by earthquakes often include medical teams with the skills and equipment to treat survivors with rhabdomyolysis. The disease was first described in the 20th century, and important discoveries as to its mechanism were made during the Blitz of London in 1941. Horses may also suffer from rhabdomyolysis from a variety of causes.
Rhabdomyolysis

Signs and symptoms

The symptoms of rhabdomyolysis depend on the severity of the condition, and whether kidney failure develops. Milder forms of rhabdomyolysis may not cause any muscle symptoms, and the diagnosis is based on abnormal blood tests in the context of other problems. More severe rhabdomyolysis is characterized by muscle pain, tenderness, weakness and swelling of the affected muscles. If the swelling is very rapid, as may happen after someone is released from under a collapsed building, the movement of fluid from the bloodstream into damaged muscle may cause low blood pressure and shock. Other symptoms are nonspecific and result either from the consequences of muscle tissue breakdown or from the condition that originally led to the muscle breakdown. Release of the components of muscle tissue into the bloodstream causes electrolyte disturbances, which can lead to nausea, vomiting, confusion, coma or abnormal heart rate and rhythm. The urine may be dark, often described as "tea-colored", due to the presence of myoglobin. Damage to the kidneys may give rise to decreased or absent urine production, usually 12 to 24 hours after the initial muscle damage.

Swelling of the damaged muscle occasionally leads to compartment syndrome—compression of surrounding tissues, such as nerves and blood vessels, in the same fascial compartment—leading to the loss of blood supply and damage or loss of function in the part(s) of the body supplied by these structures. Symptoms of this complication include pain or reduced sensation in the affected limb. A second recognized complication is disseminated intravascular coagulation (DIC), a severe disruption in blood clotting that may lead to uncontrollable bleeding.

Causes

Any form of muscle damage of sufficient severity can cause rhabdomyolysis. Multiple causes can be present simultaneously in one person. Some people have an underlying muscle condition, usually hereditary in nature, that makes them more prone to rhabdomyolysis.

Common and important causes

<table>
<thead>
<tr>
<th>Type</th>
<th>Causes</th>
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<tbody>
<tr>
<td>Exertional rhabdomyolysis</td>
<td>Extreme physical exercise (particularly when poorly hydrated), delirium tremens (alcohol withdrawal), tetanus, prolonged seizures or status epilepticus</td>
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<tr>
<td>Crush</td>
<td>Crush syndrome, blast injury, car accident, physical torture or abuse, or confinement in a fixed position such as after a stroke, due to alcohol intoxication or in prolonged surgery</td>
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<tr>
<td>Blood supply</td>
<td>Arterial thrombosis (blood clots forming locally) or embolism (clots or other debris from elsewhere in the body), clamping of an artery during surgery</td>
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<td>Metabolism</td>
<td>Hyperglycemic hyperosmolar state, hyper- and hyponatremia (elevated or reduced blood sodium levels), hypokalemia (low potassium levels), hypocalemia (low calcium levels), hypophosphatemia (low phosphate levels), ketoacidosis (e.g., in diabetic ketoacidosis) or hypothyroidism (abnormally low thyroid function)</td>
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<tr>
<td>Body temperature</td>
<td>Hyperthermia (high body temperature) and heat illness, hypothermia (very low body temperature)</td>
</tr>
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### Drugs and toxins

Many medications increase the risk of rhabdomyolysis. The most important ones are:

- **Statins and fibrates**, both used for elevated cholesterol, especially in combination; cerivastatin (Baycol) was withdrawn in 2001 after numerous reports of rhabdomyolysis. Other statins have a small risk of 0.44 cases per 10,000 person-years. Previous chronic kidney disease and hypothyroidism increase the risk of myopathy due to statins. It is also more common in the elderly, those who are severely disabled, and when statins are used in combination with particular other medicines, such as ciclosporin.
- **Antipsychotic medications** may cause neuroleptic malignant syndrome, which can cause severe muscle rigidity with rhabdomyolysis and hyperpyrexia.
- **Neuromuscular blocking agents** used in anesthesia may result in malignant hyperthermia, also associated with rhabdomyolysis.
- **Medications that cause serotonin syndrome**, such as SSRIs.
- **Medications that interfere with potassium levels**, such as diuretics.

Poisons linked to rhabdomyolysis are heavy metals and venom from insects or snakes. Hemlock may cause rhabdomyolysis, either directly or after consuming quail that have fed on it. Hafl disease is rhabdomyolysis after consuming fish; a toxic cause is suspected but has not been proven.

Drugs of abuse, including: alcohol, amphetamine, cocaine, heroin, ketamine, LSD and MDMA (ecstasy)

### Infection

Coxsackie virus, influenza A virus and influenza B virus, Epstein-Barr virus, primary HIV infection, *Plasmodium falciparum* (malaria), herpes viruses, *Legionella pneumophila* and salmonella

### Inflammation

Autoimmune muscle damage: polymyositis, dermatomyositis

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**Genetic predisposition**

Recurrent rhabdomyolysis may result from intrinsic muscle enzyme deficiencies, which are usually inherited and often appear during childhood. Many structural muscle diseases feature episodes of rhabdomyolysis that are triggered by exercise, general anesthesia or any of the other causes of rhabdomyolysis listed above. Inherited muscle disorders and infections together cause the majority of rhabdomyolysis in children.

The following hereditary disorders of the muscle energy supply may cause recurrent and usually exertional rhabdomyolysis:

- **Glycolysis and glycogenolysis defects**: McArdle's disease, phosphofructokinase deficiency, glycogen storage diseases VIII, IX, X and XI
- **Lipid metabolism defects**: carnitine palmitoyltransferase I and II deficiency, deficiency of subtypes of acyl CoA dehydrogenase (LCAD, SCAD, MCAD, VLCAD, 3-hydroxyacyl-coenzyme A dehydrogenase deficiency), thiolase deficiency
- **Mitochondrial myopathies**: deficiency of succinate dehydrogenase, cytochrome c oxidase and coenzyme Q10
- **Others**: glucose-6-phosphate dehydrogenase deficiency, myoadenylate deaminase deficiency and muscular dystrophies
Mechanism

Damage to skeletal muscle may take various forms. Crush injuries and other physical injuries cause damage to muscle cells directly or interfere with their blood supply, while non-physical causes interfere with muscle cell metabolism. When damaged, muscle tissue rapidly fills with fluid from the bloodstream, including sodium ions. The swelling itself may lead to destruction of muscle cells, but those cells that survive are subject to various disruptions that lead to rise in intracellular calcium ions; the accumulation of calcium outside the sarcoplasmic reticulum leads to continuous muscle contraction and depletion of ATP, the main carrier of energy in the cell. ATP depletion can itself lead to uncontrolled calcium influx. The persistent contraction of the muscle cell leads to breakdown of intracellular proteins and disintegration of the cell.

Neutrophil granulocytes—the most abundant type of white blood cell—enter the muscle tissue, producing an inflammatory reaction and releasing reactive oxygen species, particularly after crush injury. Crush syndrome may also cause reperfusion injury when blood flow to decompressed muscle is suddenly restored.

The swollen, inflamed muscle may directly compress structures in the same fascial compartment, causing compartment syndrome. The swelling may also further compromise blood supply into the area. Finally, destroyed muscle cells release potassium ions, phosphate ions, the heme-containing protein myoglobin, the enzyme creatine kinase and uric acid (a breakdown product of purines from DNA) into the blood. Activation of the coagulation system may precipitate disseminated intravascular coagulation. High potassium levels may lead to potentially fatal disruptions in heart rhythm. Phosphate binds to calcium from the circulation, leading to low calcium levels in the blood.

Rhabdomyolysis may cause renal failure by several mechanisms. The most important problem is the accumulation of myoglobin in the kidney tubules. Normally, the blood protein haptoglobin binds circulating myoglobin and other heme-containing substances, but in rhabdomyolysis the quantity of myoglobin exceeds the binding capacity of haptoglobin. Myoglobinuria, the presence of myoglobin in the urine, occurs when the level in plasma exceeds 0.5–1.5 mg/dl; once plasma levels reach 100 mg/dl, the concentration in the urine becomes sufficient for it to be visibly discolored. About 200 grams of muscle needs to be destroyed for visible myoglobinuria to occur. As the kidneys reabsorb more water from the filtrate, myoglobin interacts with Tamm–Horsfall protein in the nephron to form casts (solid aggregates) that obstruct the normal flow of fluid; the condition is worsened further by high levels of uric acid and acidification of the filtrate, which increase cast formation. Iron released from the heme generates reactive oxygen species, damaging the kidney cells. In addition to the myoglobinuria, two other mechanisms contribute to renal impairment: low blood pressure leads to constriction of the blood vessels and therefore a relative lack of blood flow to the kidney, and finally uric acid may form crystals in the tubules of the kidneys, causing obstruction. Together, these processes lead to acute tubular necrosis, the destruction of the cells of tubules. Glomerular filtration rate falls and the kidney is unable to perform its normal excretory functions. This causes disruption of electrolyte regulation, leading to a further rise in potassium levels, and interferes with vitamin D processing, further worsening the low calcium levels.
Rhabdomyolysis

Diagnosis

A diagnosis of rhabdomyolysis may be suspected in anyone who has suffered trauma, crush injury or prolonged immobilization, but it may also be identified at a later stage due to deteriorating kidney function (abnormally raised or increasing creatinine and urea levels, falling urine output) or reddish-brown discoloration of the urine.

General investigations

The most reliable test in the diagnosis of rhabdomyolysis is the level of creatine kinase (CK) in the blood. This enzyme is released by damaged muscle, and levels above 5 times the upper limit of normal (ULN) indicate rhabdomyolysis. Depending on the extent of the rhabdomyolysis, concentrations up to 100,000 U/l are not unusual. CK concentrations rise steadily for 12 hours after the original muscle injury, remain elevated for 1–3 days and then fall gradually. Initial and peak CK levels have a linear relationship with the risk of acute renal failure: the higher the CK, the more likely it is that kidney damage will occur. There is no specific concentration of CK above which renal impairment definitely occurs; concentrations below 20,000 U/l are unlikely to be associated with a risk of renal impairment, unless there are other contributing risk factors. Mild rises without renal impairment are referred to as "hyperCKemia". Myoglobin has a short half-life, and is therefore less useful as a diagnostic test in the later stages. Its detection in blood or urine is associated with a higher risk of renal impairment. Despite this, use of urine myoglobin measurement is not supported by evidence as it lacks specificity and the research studying its utility is of poor quality.

Elevated concentrations of the enzyme lactate dehydrogenase (LDH) may be detected. Other markers of muscle damage, such as aldolase, troponin, carbonic anhydrase type 3 and fatty acid-binding protein (FABP), are mainly used in chronic muscle diseases. The transaminases, enzymes abundant in both liver and muscle tissue, are also usually increased; this can lead to the condition being confused with acute liver injury, at least in the early stages. The incidence of actual acute liver injury is 25% in people with non-traumatic rhabdomyolysis; the mechanism for this is uncertain.

High potassium levels tend to be a feature of severe rhabdomyolysis. Electrocardiography (ECG) may show whether the elevated potassium levels are affecting the conduction system of the heart, as suggested by the presence of T wave changes or broadening of the QRS complex. Low calcium levels may be present in the initial stage due to binding of free calcium to damaged muscle cells.

Urinalysis by urine test strip may reveal a positive result for "blood", even though no red blood cells can be identified on microscopy of the urine; this occurs because the reagent on the test strip reacts with myoglobin. The same phenomenon may happen in conditions that lead to hemolysis, the destruction of red blood cells; in hemolysis the blood serum is also visibly discolored, while in rhabdomyolysis it is normal. If kidney damage has occurred, microscopy of the urine also reveals urinary casts that appear pigmented and granular.

Complications

Compartment syndrome is a clinical diagnosis, i.e., no diagnostic test conclusively proves its presence or absence, but direct measurement of the pressure in a fascial compartment, and the difference between this pressure and the blood pressure, may be used to assess its severity. High pressures in the compartment and a small difference between compartment pressure and blood pressure indicate that the blood supply is likely to be insufficient, and that surgical intervention may be needed.
Disseminated intravascular coagulation, another complication of rhabdomyolysis and other forms of critical illness, may be suspected on the basis of unexpected bleeding or abnormalities in hematological tests, such as a decreasing platelet count or prolongation of the prothrombin time. The diagnosis can be confirmed with standard blood tests for DIC, such as D-dimer.

**Underlying disorders**

If an underlying muscle disease is suspected, for instance if there is no obvious explanation or there have been multiple episodes, it may be necessary to perform further investigations. During an attack, low levels of carnitine in the blood and high levels of acylcarnitine in blood and urine may indicate a lipid metabolism defect, but these abnormalities revert to normal during convalescence. Other tests may be used at that stage to demonstrate these disorders. Disorders of glycolysis can be detected by various means, including the measurement of lactate after exercise; a failure of the lactate to rise may be indicative of a disorder in glycolysis, while an exaggerated response is typical of mitochondrial diseases. Electromyography (EMG) may show particular patterns in specific muscle diseases; for instance, McArdle's disease and phosphofructokinase deficiency show a phenomenon called *cramp-like contracture*. There are genetic tests available for many of the hereditary muscle conditions that predispose to myoglobinuria and rhabdomyolysis.

Muscle biopsy can be useful if an episode of rhabdomyolysis is thought to be the result of an underlying muscle disorder. A biopsy sample taken during an episode is often uninformative, as it will show only evidence of cell death or may appear normal. Taking the sample is therefore delayed for several weeks or months. The histopathological appearance on the biopsy indicates the nature of the underlying disorder. For instance, mitochondrial diseases are characterised by *ragged red fibers*. Biopsy sites may be identified by medical imaging, such as magnetic resonance imaging, as the muscles may not be uniformly affected.

**Treatment**

The main goal of treatment is to treat shock and preserve kidney function. Initially this is done through the administration of generous amounts of intravenous fluids, usually isotonic saline (0.9% weight per volume sodium chloride solution). In victims of crush syndrome, it is recommended to administer intravenous fluids even before they are extracted from collapsed structures. This will ensure sufficient circulating volume to deal with the muscle cell swelling (which typically commences when blood supply is restored), and to prevent the deposition of myoglobin in the kidneys. Amounts of 6 to 12 liters over 24 hours are recommended. The rate of fluid administration may be altered to achieve a high urine output (200–300 ml/h in adults), unless there are other reasons why this might lead to complications, such as a history of heart failure.

While many sources recommend additional intravenous agents to reduce damage to the kidney, most of the evidence supporting this practice comes from animal studies, and is inconsistent and conflicting. Mannitol acts by osmosis to enhance urine production and is thought to prevent myoglobin deposition in the kidney, but its efficacy has not been shown in studies and there is a risk of worsening renal function. The addition of bicarbonate to the intravenous fluids may alleviate acidosis (high acid level of the blood) and make the urine more alkaline to prevent cast formation in the kidneys, but there is limited evidence that it has benefits above saline alone, and it can worsen hypocalcemia by enhancing calcium and phosphate deposition in the tissues. If urine alkalization is used, the pH of the urine is kept at 6.5 or above. Furosemide, a loop diuretic, is often used to ensure sufficient urine production, but evidence that this prevents renal failure is lacking.
Electrolytes

In the initial stages, electrolyte levels are often abnormal and require correction. High potassium levels can be life-threatening, and respond to increased urine production and renal replacement therapy (see below). Temporary measures include the administration of calcium to protect against cardiac complications, insulin or salbutamol to redistribute potassium into cells, and infusions of bicarbonate solution.

Calcium levels initially tend to be low, but as the situation improves calcium is released from where it has precipitated with phosphate, and vitamin D production resumes, leading to hypercalcemia (abnormally high calcium levels). This "overshoot" occurs in 20–30% of those people who have developed kidney failure.

Acute renal impairment

Kidney dysfunction typically develops 1–2 days after the initial muscle damage. If supportive treatment is inadequate to manage this, renal replacement therapy (RRT) may be required. RRT removes excess potassium, acid and phosphate that accumulate when the kidneys are unable to function normally and is required until kidney function is regained.

Three main modalities of RRT are available: hemodialysis, continuous hemofiltration and peritoneal dialysis. The former two require access to the bloodstream (a dialysis catheter), while peritoneal dialysis is achieved by instilling fluid into the abdominal cavity and later draining it. Hemodialysis, which is normally done several times a week in chronic kidney disease, is often required on a daily basis in rhabdomyolysis. Its advantage over continuous hemofiltration is that one machine can be used multiple times a day, and that continuous administration of anticoagulant drugs is not necessary. Hemofiltration is more effective at removing large molecules from the bloodstream, such as myoglobin, but this does not seem to confer any particular benefit. Peritoneal dialysis may be difficult to administer in someone with severe abdominal injury, and it may be less effective than the other modalities.

Other complications

Compartment syndrome is treated with surgery to relieve the pressure inside the muscle compartment and reduce the risk of compression on blood vessels and nerves in that area. Fasciotomy is the incision of the affected compartment. Often, multiple incisions are made and left open until the swelling has reduced. At that point, the incisions are closed, often requiring debridement (removal of non-viable tissue) and skin grafting in the process. The need for fasciotomy may be decreased if mannitol is used, as it can relieve muscle swelling directly.

Disseminated intravascular coagulation generally resolves when the underlying causes are treated, but supportive measures are often required. For instance, if the platelet count drops significantly and there is resultant bleeding, platelets may be administered.
**Prognosis**

The prognosis depends on the underlying cause and whether any complications occur. Rhabdomyolysis complicated by acute kidney impairment in patients with traumatic injury may have a mortality rate of 20%. Admission to the intensive care unit is associated with a mortality of 22% in the absence of acute kidney injury, and 59% if renal impairment occurs. Most people who have sustained renal impairment due to rhabdomyolysis fully recover their renal function.

**Epidemiology**

The exact incidence of rhabdomyolysis is difficult to establish, because different definitions have been used. In 1995, hospitals in the U.S. reported 26,000 cases of rhabdomyolysis. Up to 85% of people with major traumatic injuries will experience some degree of rhabdomyolysis. Of those with rhabdomyolysis, 10–50% develop acute kidney injury. The risk is higher in people with a history of illicit drug use, alcohol misuse or trauma when compared to muscle diseases, and it is particularly high if multiple contributing factors occur together. Rhabdomyolysis accounts for 7–10% of all cases of acute kidney injury in the U.S.

Crush injuries are common in major disasters, but especially so in earthquakes. The aftermath of the 1988 Spitak earthquake prompted the establishment, in 1995, of the Renal Disaster Relief Task Force, a working group of the International Society of Nephrology (a worldwide body of kidney experts). Its volunteer doctors and nurses assisted for the first time in the 1999 İzmit earthquake in Turkey, where 462 people received dialysis, with positive results. Treatment units are generally established outside the immediate disaster area, as aftershocks could potentially injure or kill staff and make equipment unusable.

**History**

The Bible may contain an early account of rhabdomyolysis. In Numbers 11:4-6,31–33 [10], the Pentateuch says that the Jews demanded meat while traveling in the desert; God sent quail in response to the complaints, and people ate large quantities of quail meat. A plague then broke out, killing numerous people. Rhabdomyolysis after consuming quail was described in more recent times and called coturnism (after Coturnix, the main quail genus). Migrating quail consume large amounts of hemlock, a known cause of rhabdomyolysis.

In modern times, early reports from the 1908 Messina earthquake and World War I on renal failure after injury were followed by studies by London physicians Eric Bywaters and Desmond Beall, working at the Royal Postgraduate Medical School and the National Institute for Medical Research, on four victims of The Blitz in 1941. [11] Myoglobin was demonstrated in the urine of victims by spectroscopy, and it was noted that the kidneys of victims resembled those of patients who had hemoglobinuria (hemoglobin rather than myoglobin being the cause of the kidney damage). In 1944 Bywaters demonstrated experimentally that the renal failure was mainly caused by myoglobin. Already during the war, teams of doctors traveled to bombed areas to provide medical support, chiefly with intravenous fluids, as dialysis was not yet available. The prognosis of acute renal failure improved markedly when dialysis was added to supportive treatment, which first happened during the 1950–1953 Korean War.
Other animals

In animals, rhabdomyolysis mainly affects horses. Horses can develop a number of muscle disorders, many of which may progress to rhabdomyolysis. Of these, some cause isolated attacks of rhabdomyolysis (e.g., dietary deficiency in vitamin E and selenium, poisoning associated with pasture or agricultural poisons such as organophosphates), while others predispose to exertional rhabdomyolysis (e.g., the hereditary condition equine polysaccharide storage myopathy). 5–10% of thoroughbred horses and some standardbred horses suffer from the condition equine exertional rhabdomyolysis; no specific cause has been identified, but an underlying muscle calcium regulation disorder is suspected.

Rhabdomyolysis affecting horses may also occur in outbreaks; these have been reported in many European countries, and later in Canada, Australia, and the United States. It has been referred to as "atypical myopathy" or "myoglobinuria of unknown etiology". No single cause has yet been found, but various mechanisms have been proposed, and a seasonal pattern has been observed. Very high creatine kinase levels are detected, and mortality from this condition is 89%.

References

Supraventricular tachycardia (SVT) is a condition presenting as a rapid heart rhythm originating at or above the atrioventricular node. Supraventricular tachycardias can be contrasted with the potentially more dangerous ventricular tachycardias—rapid rhythms that originate within the ventricular tissue.

Although "SVT" can be due to any supraventricular cause, the term is most often used to refer to a specific example, paroxysmal supraventricular tachycardia (PSVT), two common types being atrioventricular reciprocating tachycardia and AV nodal reentrant tachycardia. In the older adult population atrial fibrillation becomes a common type of supraventricular arrhythmias—though it is typically considered separately.

In general, SVT is caused by one of two mechanisms: The first is re-entry; the second is automaticity. Re-entry (such as AV nodal reentrant tachycardia and atrioventricular reciprocating tachycardia) often presents with an almost immediate onset with sudden increase in heart rate. A person experiencing this type of PSVT may feel the heart rate accelerate from 60 to 200 beats per minute or more. Typically, when it reverts to normal rhythm, this is also sudden.
Supraventricular tachycardia

The main pumping chamber, the ventricle, is protected (to a certain extent) against excessively high rates arising from the supraventricular areas by a 'gating mechanism' at the atrioventricular node, which allows only a proportion of the fast impulses to pass through to the ventricles. In a condition called Wolff-Parkinson-White Syndrome, a 'bypass tract' avoids this node and its protection and the fast rate may be directly transmitted to the ventricles. This situation has characteristic findings on ECG.

In automatic types of SVT (atrial tachycardia, junctional ectopic tachycardia), there is more typically a gradual increase and decrease in the heart rate. These are due to an area in the heart that generates its own electrical signal.

**Types**

The following types of supraventricular tachycardias are more precisely classified by their specific site of origin. While each belongs to the broad classification of SVT, the specific term/diagnosis is preferred when possible:

- **Sinoatrial origin:**
  - Sinoatrial nodal reentrant tachycardia (SNRT)

- **Atrial origin:**
  - Ectopic (unifocal) atrial tachycardia (EAT)
  - Multifocal atrial tachycardia (MAT)
  - Atrial fibrillation with rapid ventricular response
  - Atrial flutter with rapid ventricular response
    (Without rapid ventricular response, fibrillation and flutter are usually not classified as SVT)

- **Atrioventricular origin (junctional tachycardia):**
  - AV nodal reentrant tachycardia (AVNRT) or junctional reciprocating tachycardia (JRT)
    - Permanent (or persistent) junctional reciprocating tachycardia (PJRT), a form of JRT that occurs predominantly in infants and children but can occasionally occur in adults
  - AV reciprocating tachycardia (AVRT) – visible or concealed (including Wolff-Parkinson-White syndrome)
  - Junctional ectopic tachycardia (JET)

**Signs and symptoms**

Symptoms can arise suddenly and may resolve without treatment. Stress, exercise, and emotion can all result in a normal or physiological increase in heart rate, but can also, more rarely, precipitate SVT. Episodes can last from a few minutes to one or two days, sometimes persisting until treated. The rapid heart rate reduces the opportunity for the "pump" to fill between beats decreasing cardiac output and as a consequence blood pressure. The following symptoms are typical with a rate of 150–270 or more beats per minute:

- Pounding heart
• Shortness of breath
• Chest pain
• Rapid breathing
• Dizziness
• Loss of consciousness (in only the most serious cases)
• Symptoms of heart failure in infants are more difficult to assess since infants cannot communicate these symptoms to others. Caregivers should watch for lack of interest in feeding, shallow breathing, and lethargy. These symptoms can be very understated and may be accompanied by vomiting and/or a decrease in responsiveness.

Diagnosis

Individual subtypes of SVT can usually be distinguished by the electrical characteristics of the electrocardiogram (ECG).

Most supraventricular tachycardias have a narrow QRS complex, although, occasionally, electrical conduction abnormalities may produce a wide QRS complex that may mimic ventricular tachycardia (VT). In the clinical setting, the distinction between narrow and wide complex tachycardia (supraventricular vs. ventricular) is fundamental since they are treated differently. In addition, ventricular tachycardia can quickly degenerate to ventricular fibrillation and death and merits different consideration. In the less common situation in which a wide-complex tachycardia may actually be supraventricular, a number of algorithms have been devised to assist in distinguishing between them. In general, a history of structural heart disease markedly increases the likelihood that the tachycardia is ventricular in origin.

• Sinus tachycardia is physiologic or “appropriate” when a reasonable stimulus, such as the catecholamine surge associated with fright, stress, or physical activity, provokes the tachycardia. It is identical to a normal sinus rhythm except for its faster rate (>100 beats per minute in adults). In general, it is not considered SVT.

• Sinoatrial node reentrant tachycardia (SANRT) is caused by a reentry circuit localised to the SA node, resulting in a P-wave of normal shape and size (morphology) that falls before a regular, narrow QRS complex. It cannot be distinguished electrocardiographically from sinus tachycardia unless the sudden onset is observed (or recorded on a continuous monitoring device). It may sometimes be distinguished by its prompt response to vagal manoeuvres.

• Ectopic (unifocal) atrial tachycardia arises from an independent focus within the atria, distinguished by a consistent P-wave of abnormal morphology that falls before a narrow, regular QRS complex. It is caused by automaticity, which means that some cardiac muscle cells, which have the primordial ability to generate electrical impulses that is common to all cardiac muscle cells, have established themselves as a ‘rhythm center’ with a natural rate of electrical discharge that is faster than the normal SA node.

• Multifocal atrial tachycardia (MAT) is tachycardia arising from at least three ectopic foci within the atria, distinguished by P-waves of at least three different morphologies that all fall before irregular, narrow QRS
Atrial fibrillation is not, by itself, a tachycardia, but meets the definition when associated with a ventricular response greater than 100 beats per minute. It is characterized as an "irregularly irregular rhythm" both in its atrial and ventricular depolarizations and is distinguished by its fibrillatory atrial waves that, at some point in their chaos, stimulate a response from the ventricles in the form of irregular, narrow QRS complexes.

Atrial flutter is caused by a re-entry rhythm in the atria, with a regular atrial rate often of about 300 beats per minute. On the ECG, this appears as a line of "sawtooth" waves preceding the QRS complex. The AV node will not usually conduct at such a fast rate, and so the P:QRS ratio is usually 2:1 or 4:1 pattern, (though rarely 3:1, and sometimes 1:1 where class IC antiarrhythmic drug are in use). Because the ratio of P to QRS is usually consistent, A-flutter is often regular in comparison to its irregular counterpart, A-fib. Atrial Flutter is also not necessarily a tachycardia unless the AV node permits a ventricular response greater than 100 beats per minute.

AV nodal reentrant tachycardia (AVNRT) involves a reentry circuit forming next to, or within, the AV node. The circuit most often involves two tiny pathways one faster than the other, within the AV node. Because the node is immediately between the atria and ventricle, the re-entry circuit often stimulates both, meaning that a retrogradely (backward) conducted P-wave is buried within or occurs just after the regular, narrow QRS complexes.

Atrioventricular reciprocating tachycardia (AVRT), also results from a reentry circuit, although one physically much larger than AVNRT. One portion of the circuit is usually the AV node, and the other, an abnormal accessory pathway (muscular connection) from the atria to the ventricle. Wolff-Parkinson-White syndrome is a relatively common abnormality with an accessory pathway, the Bundle of Kent crossing the AV valvular ring.

In orthodromic AVRT, atrial impulses are conducted down through the AV node and retrogradely re-enter the atrium via the accessory pathway. A distinguishing characteristic of orthodromic AVRT can therefore be a P-wave that follows each of its regular, narrow QRS complexes, due to retrograde conduction.

In antidromic AVRT, atrial impulses are conducted down through the accessory pathway and re-enter the atrium retrogradely via the AV node. Because the accessory pathway initiates conduction in the ventricles outside of the bundle of His, the QRS complex in antidromic AVRT is often wider than usual, with a delta wave.

Finally, junctional ectopic tachycardia (JET) is a rare tachycardia caused by increased automaticity of the AV node itself initiating frequent heart beats. On the ECG, junctional tachycardia often presents with abnormal morphology P-waves that may fall anywhere in relation to a regular, narrow QRS complex. It is often due to drug toxicity.

**Treatment**

Most SVTs are unpleasant rather than life-threatening, although very fast heart rates can be problematic for those with underlying ischemic heart disease or the elderly. Episodes require treatment when they occur, but interval therapy may also be used to prevent or reduce recurrence. While some treatment modalities can be applied to all SVTs, there are specific therapies available to treat some sub-types. Effective treatment consequently requires knowledge of how and where the arrhythmia is initiated and how it is spread.

SVTs can be classified by whether the AV node is involved in maintaining the rhythm. If so, slowing conduction through the AV node will terminate it. If not, AV nodal blocking maneuvers will not work, although transient AV block is still useful as it may unmask an underlying abnormal rhythm.

AV nodal blocking can be achieved in at least three ways:
**Physical maneuvers**

A number of physical maneuvers increase AV nodal block, principally through activation of the parasympathetic nervous system, conducted to the heart by the vagus nerve. These manipulations are collectively referred to as vagal maneuvers.

The Valsalva maneuver should be the first vagal maneuver tried and works by increasing intra-thoracic pressure and affecting baroreceptors (pressure sensors) within the arch of the aorta. It is carried out by asking the patient to hold his/her breath and try to exhale forcibly as if straining during a bowel movement. Holding the nose and exhaling against the obstruction has a similar effect.

Lying on your back with your legs vertical (normally against a wall) and relaxing with slow steady breaths may end the episode.

There are other vagal maneuvers including: holding one's breath for a few seconds, coughing, plunging the face into cold water, (via the diving reflex), drinking a glass of ice cold water, and standing on one's head. Carotid sinus massage, carried out by firmly pressing the bulb at the top of one of the carotid arteries in the neck, is effective but is often not recommended in the elderly due to the potential risk of stroke in those with atherosclerotic plaque in the carotid arteries.

Reducing coffee, alcohol, or tobacco use or increasing the amount of rest may help to alleviate symptoms. Pressing down gently on the top of closed eyes may also bring heartbeat back to normal rhythm for some people suffering from atrial or supraventricular tachycardia (SVT).

**Medications**

Adenosine, an ultra-short-acting AV nodal blocking agent, is indicated if vagal maneuvers are not effective. If successful, followup therapy with diltiazem, verapamil or metoprolol may be indicated. Adenosine may be safely used during pregnancy.[5]

SVT that does not involve the AV node may respond to other anti-arrhythmic drugs such as sotalol or amiodarone.

**Cardioversion**

If the patient is unstable or other treatments have not been effective, synchronized electrical cardioversion may be used.

**Prevention**

Once an acute arrhythmia has been terminated, ongoing treatment may be indicated to prevent recurrence. However, those that have an isolated episode, or infrequent and minimally symptomatic episodes, usually do not warrant any treatment other than observation.

In general, patients with more frequent or disabling symptoms warrant some form of prevention. A variety of drugs including simple AV nodal blocking agents such as beta-blockers and verapamil, as well as anti-arrhythmics may be used, usually with good effect, although the risks of these therapies need to be weighed against potential benefits.

Radiofrequency ablation has revolutionized the treatment of tachycardia caused by a re-entrant pathway. This is a low-risk procedure that uses a catheter inside the heart to deliver radio frequency energy to locate and destroy the abnormal electrical pathways. Ablation has been shown to be highly effective: around 90% in the case of AVNRT. Similar high rates of success are achieved with AVRT and typical Atrial Flutter.

There is a newer treatment for SVT involving the AV node directly. This treatment is called Cryoablation. SVT involving the AV node is often a contraindication for using radiofrequency ablation due to the small (1%) incidence
of injuring the AV node, requiring a permanent pacemaker. With Cryoablation, a supercooled catheter is used (cooled by nitrous oxide gas), and the tissue is frozen to $-10\, ^\circ\mathrm{C}$. This provides the same result as radiofrequency ablation but does not carry the same risk. If you freeze the tissue and then realize you are in a dangerous spot, you can halt freezing the tissue and allow the tissue to spontaneously rewarm and the tissue is the same as if you never touched it. If after freezing the tissue to $-10\, ^\circ\mathrm{C}$, you get the desired result, then you freeze the tissue down to a temperature of $-73\, ^\circ\mathrm{C}$ and you permanently ablate the tissue.

This therapy has further improved the treatment options for people with AVNRT (and other SVTs with pathways close to the AV node), widening the application of curative ablation to young patients with relatively mild but still troublesome symptoms who would not have accepted the risk of requiring a pacemaker.

### Notable cases

After being successfully diagnosed and treated, Bobby Julich went on to place third in the 1998 Tour de France and win a Bronze Medal in the 2004 Summer Olympics. Women's Olympic volleyball player Tayyiba Haneef-Park underwent an ablation for SVT just two months before competing in the 2008 Summer Olympics. Tony Blair, former PM of the UK, was also operated on for atrial flutter. Anastacia was diagnosed with the disease. Women's Olympic gold medalist swimmers, Rebecca Soni and Dana Vollmer have both had heart surgery to correct SVT. In addition, Neville Fields had corrective surgery for SVT in early 2006. Wrestling manager Paul Bearer's heart attack was attributed to SVT, resulting in his death. Nathan Cohen, New Zealand's two-time world champion and Olympic champion rower, was diagnosed with SVT in 2013 when he was 27 years old.\(^6\)

### References

1. [http://apps.who.int/classifications/icd10/browse/2010/en#/I47.1](http://apps.who.int/classifications/icd10/browse/2010/en#/I47.1)
6. Scott Brady of punk band Brave The Wild ([https://www.facebook.com/BraveTheWild](https://www.facebook.com/BraveTheWild)) suffers from this. He had his first attack on April 9, 2012 while golfing and was hospitalized over night. He was diagnosed April 17, 2014 in Hamilton ON after having an attack walking home from dinner on Mach 16, 2014.

### External links

- [Cardiac Disorders – Open Directory Project](http://www.dmoz.org/Health/Conditions_and_Diseases/Cardiovascular_Disorders/Heart_Disease/Support_Groups/)
- [Supraventricular Tachycardia information](http://heartcenter.seattlechildrens.org/conditions_treated/supraventricular_tachycardia.asp) from Seattle Children's Hospital Heart Center

Movie/Animation of SVT: Video section: The University of Iowa Children’s Hospital ([http://www.uichildrens.org/cardiology/](http://www.uichildrens.org/cardiology/))
Testicular torsion occurs when the spermatic cord (from which the testicle is suspended) twists, cutting off the testicle's blood supply, a condition called ischemia. The principal symptom is rapid onset of testicular pain. The most common underlying cause is a congenital malformation known as a "bell-clapper deformity" wherein the testis is inadequately affixed to the scrotum allowing it to move freely on its axis and susceptible to induced twisting of the cord and its vessels.

The diagnosis should usually be made based on the presenting signs and symptoms. An urgent ultrasound should only be done when the diagnosis is unclear. Irreversible ischemia begins around six hours after onset and emergency diagnosis and treatment is required within this time in order to minimize the risk of testicle loss.

It is most common just after birth and during puberty. It occurs in about 1 in 4,000 to 1 per 25,000 males per year before 25 years of age.
Signs and symptoms

Testicular torsion usually presents with sudden, severe, testicular pain and tenderness involving one testicle. There is often nausea and vomiting due to the pain.

Some of the symptoms are similar to epididymitis though epididymitis may be characterized by discoloration and swelling of the testis, often with fever, while the cremasteric reflex is not affected. Testicular torsion, or more probably impending testicular infarction, can also produce a low-grade fever.

There is often an absent or decreased cremasteric reflex.

Risk factors

A larger testicle either due to normal variation or a tumor increases the risk of torsion.

Congenital

Conditions that allow the testicle to rotate predispose to torsion. A congenital malformation of the processus vaginalis known as the "bell-clapper deformity" accounts for 90% of all cases. In this condition, rather than the testes attaching posteriorly to the inner lining of the scrotum by the mesorchium, the mesorchium terminates early and the testis is free floating in the tunica vaginalis.

Temperature

Torsions are sometimes called "winter syndrome" because they are more frequent in cold conditions, specifically decreasing atmospheric temperature and humidity.

Diagnosis

The diagnosis should usually be made based on the person's history and presenting signs and symptoms. In those cases with a convincing history and physical exam immediate surgery is recommended. A doppler ultrasound should only be obtained in low suspicion cases to rule out torsion.

Clinical exam

Prehn's sign, a classic physical exam finding, has not been reliable in distinguishing torsion from other causes of testicular pain such as epididymitis. In cases of true torsion the cremasteric reflex is typically absent (the twisted cords of the testicle make reflexive responses all but impossible). On physical examination, the testis will be swollen, tender, and high-riding, with an abnormal transverse lie. The individual will not usually have a fever, though nausea is common.

Imaging

A doppler ultrasound scan of the scrotum is nearly 90% accurate at detecting torsion. It is identified by the absence of blood flow in the twisted testicle, which distinguishes the condition from epididymitis.

Radionuclide scanning of the scrotum is the most accurate, diagnostic, imaging technique, but it is not routinely available, particularly with the urgency that might be required. The agent of choice for this purpose is technetium-99m pertechnetate. Initially it provides a radionuclide angiogram, followed by a static image after the radionuclide has perfused the tissue. In the healthy patient, initial images show symmetric flow to the testes, and delayed images show uniformly symmetric activity.
Pathophysiology
Torsion is due to a mechanical twisting process. It is also believed that torsion occurring during fetal development can lead to so-called neonatal torsion or vanishing testis, and is one of the causes of an infant being born with monorchism (one testicle).

Intermittent testicular torsion
A variant is a less serious but chronic condition called intermittent testicular torsion (ITT), characterized by the symptoms of torsion but followed by eventual spontaneous detortion and resolution of pain. Nausea or vomiting may also occur. Though less pressing, such individuals are at significant risk of complete torsion and possible subsequent orchiectomy and the recommended treatment is elective bilateral orchiopexy. Ninety-seven percent of patients who undergo such surgery experience complete relief from their symptoms.

Extravaginal testicular torsion
A torsion which occurs outside of the tunica vaginalis, when the testis and gubernaculum can rotate freely, is termed an extravaginal testicular torsion. This type occurs exclusively in newborns. Neonates experiencing such a torsion present with scrotal swelling, discoloration, and a firm, painless mass in the scrotum. Such testes are usually necrotic from birth and must be removed surgically.

Torsion of the testicular appendix
This type of torsion is the most common cause of acute scrotal pain in boys ages 7–14. Its appearance is similar to that of testicular torsion but the onset of pain is more gradual. Palpation reveals a small firm nodule on the upper portion of the testis which displays a characteristic "blue dot sign." This is the appendix of the testis which has become discolored and is noticeably blue through the skin. Unlike other torsions, however, the cremasteric reflex is still active. Typical treatment involves the use of over-the-counter analgesics and the condition resolves within 2–3 days.

Treatment
With prompt diagnosis and treatment the testicle can often be saved. Typically, when a torsion takes place, the surface of the testicle has rotated towards the midline of the body. Non-surgical correction can sometimes be accomplished by manually rotating the testicle in the opposite direction (i.e., outward, towards the thigh); if this is initially unsuccessful, a forced manual rotation in the other direction may correct the problem. The success rate of manual detorsion is not known with confidence.

Testicular torsion is a surgical emergency that requires immediate intervention to restore the flow of blood. If treated either manually or surgically within six hours, there is a high chance (approx. 90%) of preserving the testicle. At 12 hours the rate decreases to 50%; at 24 hours it drops to 10%, and after 24 hours the rate of preservation approaches 0. About 40% of cases results in loss of the testicle. Common treatment for children is surgically sewing the testicle to the scrotum to prevent future cases.
**Epidemiology**

Torsion is most frequent among adolescents with about 65% of cases presenting between 12 – 18 years of age. It occurs in about 1 in 4,000 to 1 per 25,000 males per year before 25 years of age; but it can occur at any age, including infancy. \(^{p.149}\)

**References**


**External links**
