SPORT MEDICINE MANUAL

9

ENVIRONMENTAL FACTORS

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9

A. Altitude

With increasing altitude, there is a decrease in barometric pressure.
The percentage of oxygen in the atmosphere remains the same (20.93%), however the partial pressure of oxygen decreases which reduces the oxygen content in the blood (SaO_2). The hypoxia associated with high altitude triggers a number of physiological adaptations which may enhance performance dependent on exposure time and elevation. Physiological adjustments to altitude include:

- increase in pulmonary ventilation
- increase in cardiac output
- diuresis
- haemoconcentration, initially due to a decrease in plasma volume
- increase in circulating red blood cells and haemoglobin via erythropoiesis after 2-3 days which is sustained as long as the individual remains at altitude
- increase in erythrocyte 2, 3-diphosphoglycerate (DPG) facilitates removal of oxygen from haemoglobin at the tissue level (see Figure 10.1)
- increase in myoglobin, facilitating muscle uptake of oxygen from haemoglobin
- increased size and number of mitochondria (increased oxidative enzymes)

Metabolically, hypoxia stimulates hyperventilation and this decreases arterial carbon dioxide, resulting in respiratory alkalosis and subsequent increased renal excretion of bicarbonate in an attempt to restore normal pH.

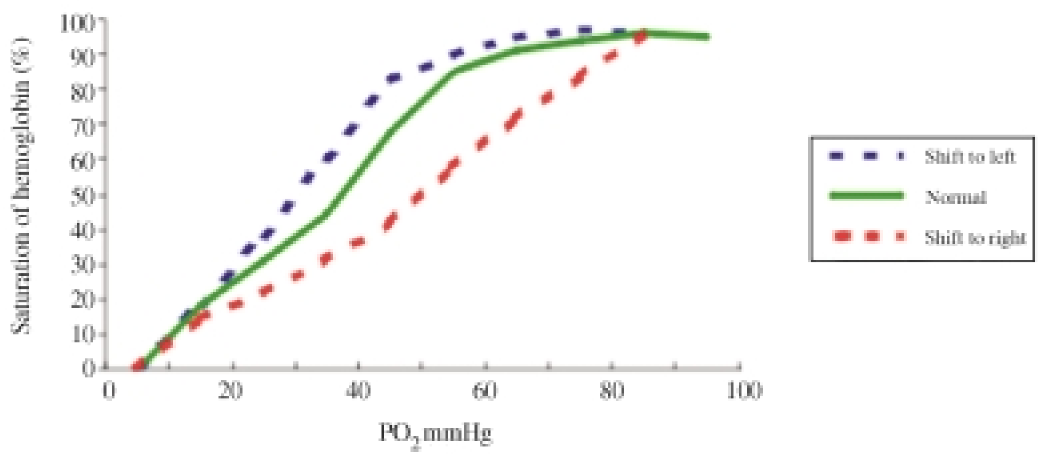

Figure 9.1 Rightward shift in the oxyhaemoglobin dissociation curve is characteristic of the response to hypoxia. (DPG = 2, 3-diphosphoglycerate). Adapted from Luce, J. (1979). Respiratory Adaption and Maladaptation to Altitude, The Physician and Sport Medicine, 7, (6), p.58.

Athletic Performance at Altitude

Altitude is known for its impact on athletic performance and athlete health. Athletes tend to seek altitude training as a means of adding a greater stress (hypoxic environment) on the cardio-pulmonary and metabolic systems. Physiological adaptations to this stress are believed to be transferable for a period of time for use at lower elevations.

It is generally accepted that athletic events lasting less than two minutes will be minimally affected by altitude. Some may even be aided by increased altitude, eg. Bob Beamon's world record long jump at the 1968 Olympics held in Mexico City at an altitude of 2,240 metres. It is the longer events, greater than two minutes, where there is a definite impairment of athletic performance.

The effect of altitude on performance is greatest during the first few days upon arrival, prior to physiological adaptation taking place. The maximum oxygen intake ($\dot{V}O_2$ max) is lowest at this time. The amount of decline in aerobic capacity is proportional to the increase in altitude (see Figure 9.2).

- Approximately 3% decrease in aerobic capacity for every 300m elevation gain above 1,800m
- 12-15% decrease in aerobic capacity at 3,000 m
- 20-25% decrease in aerobic capacity at 4,000m
- 50% decrease in aerobic capacity at 5,000m

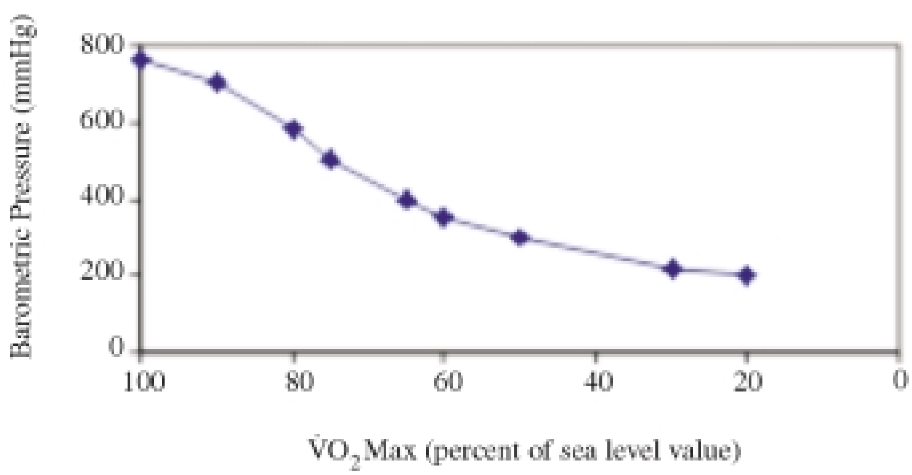

Figure 9.2 $\dot{V}O_2$ max initially decreases by approximately 3% for each 300m gain in altitude. Note: The rate of decrease gets more severe at higher elevations. $\dot{V}O_2$ max on the top of Mt. Everest (Pbar = 248 mmHg) has been estimated to be as low as 7 to 10 ml/kg/min.

This altitude intolerance is made worse by external factors, such as illness (most commonly viral infections), jet lag, dehydration and poor sleep. There is also great individual variation in the effect of altitude on athletic performance.

The benefit of high altitude training for low altitude competition results from the temporary persistence of an increase in haemoglobin and maximal pulmonary ventilation, enhanced cardiovascular efficiency, and enhanced tolerance of anaerobic work.

Athlete's Health at Altitude

There are a number of medical complications associated with high altitude. The extent to which these illnesses may occur in unacclimatized persons is dependent on both the rate of ascent to altitude and individual medical history.

Table 9.1 Features of the Different Forms of Altitude Illnesses. (Adapted from Hart, LE & Sutton, JR. (1987). Environmental Considerations for Exercise, Cardiology Clinics, 5(2), p. 254 and Auerbach, PS & Geehr, EC. (1995). Management of Wilderness Medicine and Environmental Emergencies, published by Mosby, 1995 pgs 1-29).

Forms of Altitude Illness
Altitude Mountain Sickness (AMS) -symptoms include headache, insomnia, nausea, vomiting, malaise and lassitude -onset is dependent on elevation gain and rate of ascent -symptoms are accentuated by physical exercise -unpleasant but not serious -descent brings dramatic relief
Chronic Mountain Polycythemia (Monge's Disease) -pulmonary hypertension, polycythemia, mental deterioration -decent to sea level ensures recovery
High Altitude Pulmonary Edema (HAPE) -develops as dyspnea and dry cough but later progresses to cough with pink frothy sputum -indicative of severe hypoxemia, cyanosis -serious, rapid descent mandatory or death ensues -oxygen administration may be useful -some investigators recommend diuretic therapy (furosemide, 40 mg intravenously)
High Altitude Cerebral Edema (HACE) -severe headache, mental confusion, hallucinations, ataxia, weakness, and coma -rare but very serious -descent to lower altitude mandatory -Dexamethasone 4 - 8 mg by mouth every 6 hours -Nifedipene 10 mg by mouth every 6 hours
Miscellaneous Conditions -high altitude retinal hemorrhage (HARH) -common above 5,000m, usually asypmtomatic, resolves without treatment -circulation problems such as throbophlebitis and embolism -sickle cell crises -persons with sickle cell trait may develop crises at even moderate altitude

Treatment and Prevention

Case History - Altitude

A 22-year-old male 10 km runner arrived at a meet two days prior to a competition held at 2,300 metres above sea level. That night he slept poorly and awoke with a mild headache and fatigue. He trained, but rapidly became breathless and fatigued. He terminated his session after 20 minutes. The following evening he was nauseated, lacked an appetite, and had an increased headache. A poor night's sleep resulted in his feeling even worse the day of competition. He started the race but dropped out due to a poor performance after falling off the pace dramatically at 6 km.

After returning home, a disappointed and down-hearted athlete and coach presented to your office for an evaluation and explanation. The history and physical were otherwise unremarkable.

Discussion

This case illustrates the usual symptoms associated with mild altitude illness - acute mountain sickness. The treatment strategies for acute mountain sickness, chronic mountain sickness and high altitude retinal haemorrhage include descent, rest, analgesia for the headaches, and the use of acetazolamide.

Acetazolamide is a carbonic anhydrase inhibitor which results in increased renal excretion of bicarbonate thus creating metabolic acidosis. This stimulates the medullary chemoreceptors to stimulate hyperventilation, thus increasing SaO_2. The usefulness of acetazolamide in altitude sickness stems from the impression that those susceptible have a blunted respiratory response to the hypoxia of altitude. Acetazoiamide results in an increase in this respiratory response.

Life threatening high altitude pulmonary edema should be treated aggressively with rapid descent, oxygen, and the possible use of intravenous furosemide or another diuretic. The treatment for high altitude cerebral edema includes rapid descent, oxygen, intravenous glucocorticosteroids and possible intravenous furosemide or another diuretic. It is obligatory to start treatment and descend to a lower altitude as soon as the initial symptoms of these entities become manifest. To minimize the significance of the symptoms or to develop a "wait-and-see" attitude is to court death.

Of course, the preferred avenue of approach with altitude illnesses is prevention. This includes:

a. gradual acclimatization:
- start acclimatization at less than 3,000 m
- ensure little exertion for the first 24 hours
- gradually increase volume and intensity of training during initial 2-3 days at altitude
- if symptoms of acute mountain sickness result, then either rest or descend
- if symptoms of high altitude pulmonary edema result, treat with descent and oxygen
- for optimum athletic performance, 3-6 weeks of exposure at altitude is required; this helps to minimize the effects of altitude but does not eliminate it
- due to the great individual variability in response to altitude, individualization of the training and/or acclimatization programme is appropriate

b. maintain hydration
c. minimize alcohol use
d. minimize use of sedation at night
e. consider using acetazolamide, 250 mg three times a day or 750 mg at bedtime, appreciating that this may give a false sense of security and in no way replaces gradual acclimatization

Athletes with the sickle cell trait are at increased risk with altitude. Hypoxemia can result in a sickle cell crisis with its many manifestations including splenic infarction. This would be unusual but possible, based on the high altitude training camps that are not uncommon in certain sports.

Our 22-year-old 10 km runner, in preparation for competition at altitude, had two four-week training camps at the same altitude as the site of the competition separated by training at sea level. He arrived at the site four weeks prior to competition. He took acetazolamide, 250 mg three times a day for the first week, with minimal headache, sleep disturbance and lethargy. He was able to train at a high intensity and duration and placed tenth in the 10 km race.

It appears that there is little value in training at altitudes greater than 3,000 m and indeed this elevation may be too high. At this altitude, severe physiological stress inhibits the ability to work at race pace for reasonable periods of time. An athlete may detrain if left too long at such an altitude. It appears one must go to an altitude of at least 1,800 m in order to achieve strong enough hypoxic stress to result in significant physiological adaptation.

B. Heat Injury

Case History - Heat Injury

A female marathon runner, racing at the Olympic Games, collapsed just before the finish line. The weather was hot (28° C), sunny, humid and windless, and the runner was from a European country with a mean daily high temperature, at that time of year, of 20° C. She had flown into the Olympic site two days prior to competition. With the lure of the medals, she had bypassed the water stations throughout the run. At the 32 km mark, she was noted to have slowed down her pace and was somewhat disoriented, with an unsteady gait. She was sweating profusely. During the last 1.5 kilometres, she stopped frequently and was significantly ataxic, non-communicative and hostile to the medical attendants who tried to assist her. At the finish she collapsed. Her last kilometre had taken 15 min to complete.

Discussion

The diagnosis is heat injury. Notwithstanding the frustration and nuisance of musculoskeletal injuries, running is a safe sport. There is, though, one life-threatening condition that all endurance runners risk - heat injury.

Human metabolism is 20-30% efficient, with some 70-80% of all the energy produced as heat. With activity and strenuous muscle contraction, this heat production can increase 15-20 times over the basal rate. This could result in a 1° C increase in core body temperature every 5 min unless there is an efficient thermoregulatory system. The amount of heat stored within the body is related to a number of factors, as shown in Winlow's heat balance equation:

Heat stored	=	Metabolic heat produced	±	Heat G/L by radiation	±	Heat G/L by convection	±	Heat G/L by conduction	-	evaporative heat loss

(Note: G/L = gained/lost)

In the cold, the body predominantly relies on radiation, convection and conduction to dissipate heat. With warmer ambient temperatures, the body relies predominantly on radiation and evaporative cooling. With muscle activity and heat production, as the core body temperature rises, there is a reflex

9 - Environmental Factors

vasodilation of the cutaneous blood vessels mediated through the sympathetic nervous system. This results in a three-fold increase in blood flow with a subsequent increase in sweating and evaporation. This mechanism works very capably as long as there is adequate body fluid to allow for sweating. Without replenishing this supply through drinking, dehydration will ensue with a decrease in circulating blood volume.

At a certain level of dehydration, a more basic reflex, cutaneous vasoconstriction, dominates in order to maintain adequate blood supply to the brain, kidneys and other essential organs. This explains the frequently encountered cold, clammy, pale extremities of the hyperthermic athlete. With continued activity, the core body temperature will increase. Above 40.5° C rectal temperature, there is a breakdown of the body's thermoregulatory mechanism with or without sweat production, and continued elevation of core body temperature. Loss of avenues for heat dissipation causes the exercising athlete to become a "heat sink" potentiating the risk of hyperthermic crisis.

The risk of heat injury is dependent on both environmental and athlete-dependent factors. The environmental factors include:

- high temperature
- high relative humidity, which results in decreased evaporation (see Figure 9.3)
- increased solar radiation
- absence of wind

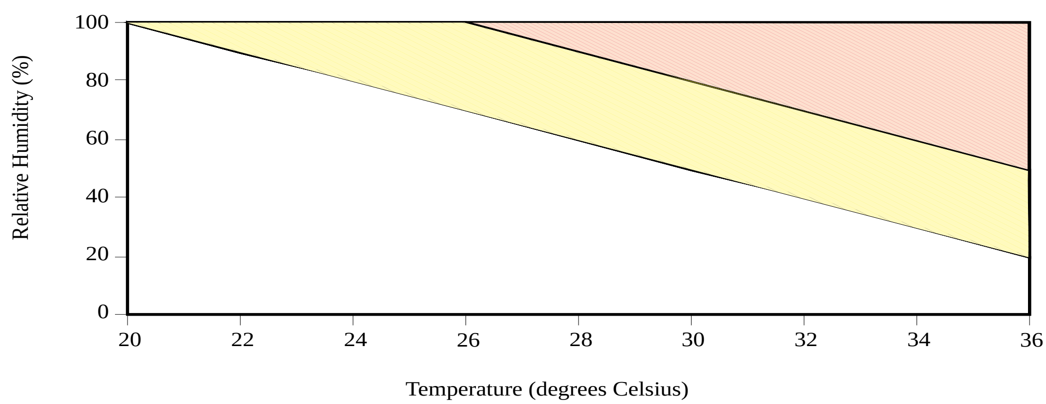

Figure 9.3 Approximate temperature-humidity relationship that potentiates serious hyperthermia during exercise. The shaded zone is that in which heat stroke fatalities have occurred in athletic activities. The darker the shading (yellow to orange), the more likely that cases of heat exhaustion and heat stroke will occur. (From Vinger, P & Hoerner, E (Eds). Hyperthermia, Hypothermia, and Drowning, Sports Injuries, PSG Pub. Co. Littleton, Mass. p. 2B7.)

The athlete-dependent factors contributing to heat injury are numerous and include:

- illness: fever, infection, recent vomiting and/or diarrhea
- injury
- lack of heat acclimatization (which takes 4-8 days of moderate exercise at race temperature to improve evaporation)
- sunburn
- excessive sun block (which may reduce evaporative cooling)
- fatigue
- excessive clothing, dark clothing, rubberized clothing that prevents heat dissipation
- dehydration from lack of fluid intake before and during the race increase in race pace (see Figure 9.4)

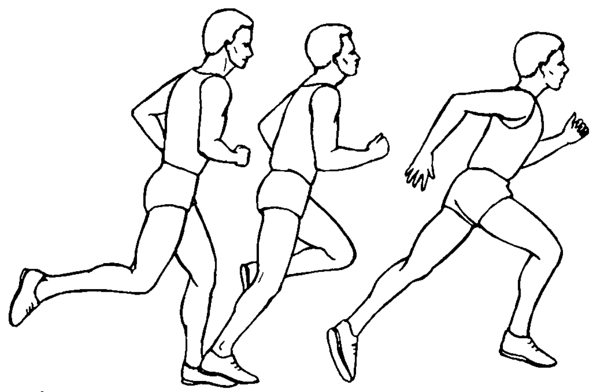

Figure 9.4 Relationship between heat production and race pace.

Other risk factors that are related to the individual but not the elite athlete include obesity, a low level of fitness, alcohol abuse and age (either less than 15 or greater than 40 years).

The clinical presentation of heat injury is based on severity, and can be classified as heat cramps, heat exhaustion or heat stroke.

Heat Cramps

Heat cramps are the most common type of heat injury manifested by muscle cramping that is not relieved by stretching.

Heat Exhaustion

Heat exhaustion is a moderate form of heat injury manifested by:

- headache
- tingling arms, legs and back
- fatigue
- muscle cramps
- chills or shivering
- profuse sweating
- rapid, weak pulse
- pale, moist, cool skin, rectal temperature less than 40° C
- hypotension

Heat Stroke

Heat stroke is the severe, acute, life-threatening heat injury manifested by:

- headache
- convulsions
- loss of consciousness, coma
- rapid, full pulse
- hot, red skin with or without sweating
- rectal temperature greater than 40° C
- ataxia

The taking of rectal temperatures in all endurance athletes with an altered level of consciousness must be emphasized or the diagnosis of heat injury may be missed. Heat stroke is a true medical emergency requiring definitive care. There is often an overlap of symptoms and signs with heat injury where the major distinguishing feature is an elevated core temperature over 40° C.

Treatment

The treatment of heat cramps involves:

- fluid replacement
- ice massage
- continued static stretching of the muscle

The treatment of heat exhaustion and heat stroke is as follows:

a. Discontinue activity. This is something that cannot be left to the discretion of the athlete, for a symptom of heat injury is impairment of judgment.

b. Place the athlete in a cool, shaded area.

c. Maximize external cooling:

- remove insulating clothing
- pack ice bags around the major blood vessels to the limbs, neck, axillary and groin regions of the athlete and place ice water-wetted towels over the athlete, which are replaced as they are warmed up
- use fans to increase convection
- rapid cool water immersion if possible

d. Rehydrate by:

- intravenous fluid, dextrose and saline, as there is a strong association between heat injury and hypoglycemia in the athlete
- fluid by mouth if conscious

e. Monitor vital signs and clinical status frequently, including consciousness and urinary output.

Aggressive treatment should be continued until the rectal temperature is below 39.0° C. If the athlete has heat stroke, then monitoring has to be long enough to ensure that he/she has normal renal function and no evidence of rhabdomyolysis and myoglobinuria.

Our marathon runner's rectal temperature was 40.5° C. She was treated with one litre of dextrose and saline rapidly infused, followed by a further litre infused over the next hour. She was alert and oriented with stable vital and clinical signs within an hour. After continued monitoring, fluid infusion and oral fluids, she was released from the medical area after six hours. Other than a sensation of undue fatigue, she had no recollection of the last half of the race or being taken to the medical tent. She passed urine after four hours. She was advised on the prevention of heat injury in order to decrease the likelihood of a recurrence.

Prevention

Prevention is better than treatment and this can occur as follows:

a. Race organization

- avoid scheduling endurance events during the hottest times of day; use the early morning or late afternoon hours instead
- have a course with optimal shade
- ensure fluid availability at the start and every 2-3 km; encourage fluid intake prior to and during the event (see Unit 13, section A6 - Hydration)
- ensure that race officials are aware of signs of heat injury
- make athletes aware of heat injuries with a heat stress warning prior to the start of the race
- alert the local hospital and ambulance service
- have adequate on-site medical facilities and transport for immediate assessment and treatment of heat injuries

b. Athlete factors

- allow 4-8 days in the race environment prior to competition to permit natural adjustments to your thermoregulatory mechanism
- wear light-weight, loose clothing that facilitates evaporation
- optimize hydration - aim at 500 ml of fluid before a race and 250 ml every 15-20 min; ideally, the fluid is dilute and cool
- educate the athlete to be aware of heat injury with symptoms such as undue fatigue, weakness, feeling unwell, decreased performance, and cramps that are not relieved by stretching

Disabled spinal cord athletes participating in endurance events are even more at risk for heat injury because of deficient sweating below the level of the spinal cord injury. Thus, there is full reliance on evaporative heat loss from the arms and trunk. With heightened awareness and optimal attention to prevention, heat injury to the disabled athlete can be minimized.

The American College of Sports Medicine position statement on prevention of thermal injuries, which is extremely thorough with considerable practical information, can be received from the college, or obtained from their web site at the address: http://www.msse.org

C. Cold Injury: Hypothermia and Frostbite

Case History - Cold Injury: Hypothermia and Frostbite

A 24-year-old male cross-country skier collapsed after finishing a 50 km race. The air mean temperature was -15° C. A gusting 20 km per hour wind was a factor over much of the exposed areas of the course. Over the last 5 km the skier was noted to be faltering, his pace slowing dramatically.

At the finish, he was shivering violently and complaining of being very cold and tired. His speech was slurred and he was stumbling as he was led to the medical area. His vital signs were: blood pressure 100/60 mmHg; heart rate 105 b/min; rectal temperature 33.5° C.

Discussion

The diagnosis is mild hypothermia.

The thermoregulatory mechanism strives to maintain a core body temperature from 36.5° C to 37.5° C. To ensure conservation of heat and maintain core temperature, cold sensitive nerve endings located in the skin trigger reflex vasoconstriction throughout the periphery. This mechanism functions to insulate the core. If this reflex is not adequate and there is a fall in core body temperature, then shivering occurs.

Shivering, or involuntary muscle contraction generates heat (shivering thermogenesis). With continued cooling, shivering decreases or stops, mental confusion or poor judgment ensues, and there is a decrease in motor function. With continued cooling, there is a collapse of the thermoregulatory mechanisms of the hypothalamus responsible for heat conservation. Metabolic heat production (non-shivering thermogenesis) can no longer offset the rate at which heat is being lost to the environment. Once this occurs, it is just a matter of time before there is loss of consciousness and a gradual decrease in vital signs until death.

Hypothermia

Table 9.2 Hypothermia can be classified as mild, moderate, or severe, based on rectal temperature.

Category	Rectal Temperature	Signs/Symptoms
Mild Hypothermia	33-35°C	Symptoms: shivering, very cold, hunger, lethargy, confusion, muscle spasms, and difficulty with motor tasks Signs: shivering, decreased race pace, slurred speech, and slow reflexes
Moderate Hypothermia	30-33°C	Signs: may not be shivering, semi-conscious with confused actions and irrational behavior, extremely tired, irritable and depressed, poor judgement, loss of memory, disoriented, ataxic gait, muscle stiffness, slurred speech, slow pulse and respiration
Severe Hypothermia	less than 30°C	Signs: loss of consciousness, pupils dilated, slow or absent pulse, and hypotensive

There are different strategies of re-warming based on the different levels of hypothermia. The greatest benefit of the classification system is that it facilitates treatment. The primary objective is to arrest any further cooling by removal from the cold stress, removal of wet clothing and provide proper insulation.

Treatment

Mild hypothermia. If there is good insulation, no fatigue, and the individual can exercise, then passive external re-warming using their own heat production will usually suffice. If the individual is too cool to shiver, then treatment is as per moderate hypothermia.

Moderate hypothermia. One cannot rely on endogenous heat production in this instance. There is a need for active external re-warming which can be via:

- warm shower or bath with temperature around 40° C
- a specially-designed wrap for the trunk that has internal piping allowing warm water to be circulated through it, or warm objects such as hot water bottles placed on the truncal core, axilla and neck
- inhalation of heat via the breathing of heated (43-44° C) water-saturated air or oxygen
- body contact

Warm fluid is worthwhile if the individual will not choke or gag while drinking. This fluid is used not so much for any heat gained, but for rehydration and improvement of their fluid status and circulating blood volume.

Individuals suffering with mild hypothermia should be handled with care. Increased blood flow from the extremities may result in cooling the heart and triggering ventricular fibrillation.

Severe hypothermia. Individuals with rectal temperature less than 30° C require active core re-warming. The periphery (arms and legs) can be left cold but should be insulated with dry clothing. With an exogenous heat source, one should actively heat the chest, neck and head to increase the core body temperature more directly. Transport patient to a medical facility for definitive care where treatment may include peritoneal lavage and airway re-warming via endotracheal tube.

Occasionally, these individuals may appear dead, with undetectable vital signs, but the gradual warming makes them highly resuscitable. This has led to the slogan, "You're never dead when you're cold and dead. You're only dead when you're warm and dead". If the individual has no vital signs, they should be transported to a medical facility. One must not re-warm the individual to greater than 30-32° C. Continual cardiopulmonary resuscitation (CPR) is required. In transport, one must handle the victim carefully because of the predisposition to cardiac arrhythmias.

Our cross country skier was treated with warm blankets and fluids and was encouraged to keep moving around to generate more heat. After 15 minutes, the shivering stopped and after 30 minutes, with a rectal temperature of 35.5° C, he was released from the medical area much improved.

Prevention

There are several things that can be done to prevent or delay the occurrence of hypothermia.

a. Race organizers:

- avoid courses that are exposed to high winds
- cancel the event if race time temperature is less than -20° C
- ensure that detailed, accurate information on weather conditions is provided to the athletes

b. Athletes:

- allow about 10 days for acclimatization to the cold
- optimize muscle glycogen content to minimize fatigue within the race, thus allowing high metabolic rate and endogenous heat production
- hydrate before and during the race to minimize dehydration that can result in fatigue, decreased race pace and decreased heat production
- dress appropriately with layers such as polypropylene so as to draw the sweat away from the skin and allow evaporation
- be mindful of the wind chill factor
- control race pace to minimize exhaustion and fatigue that will lead to decreased endogenous heat production

The disabled wheelchair athlete who is paralyzed has impaired ability to thermoregulate below the level of the spinal injury. Great care needs to be taken to minimize their increased risk of hypothermia.

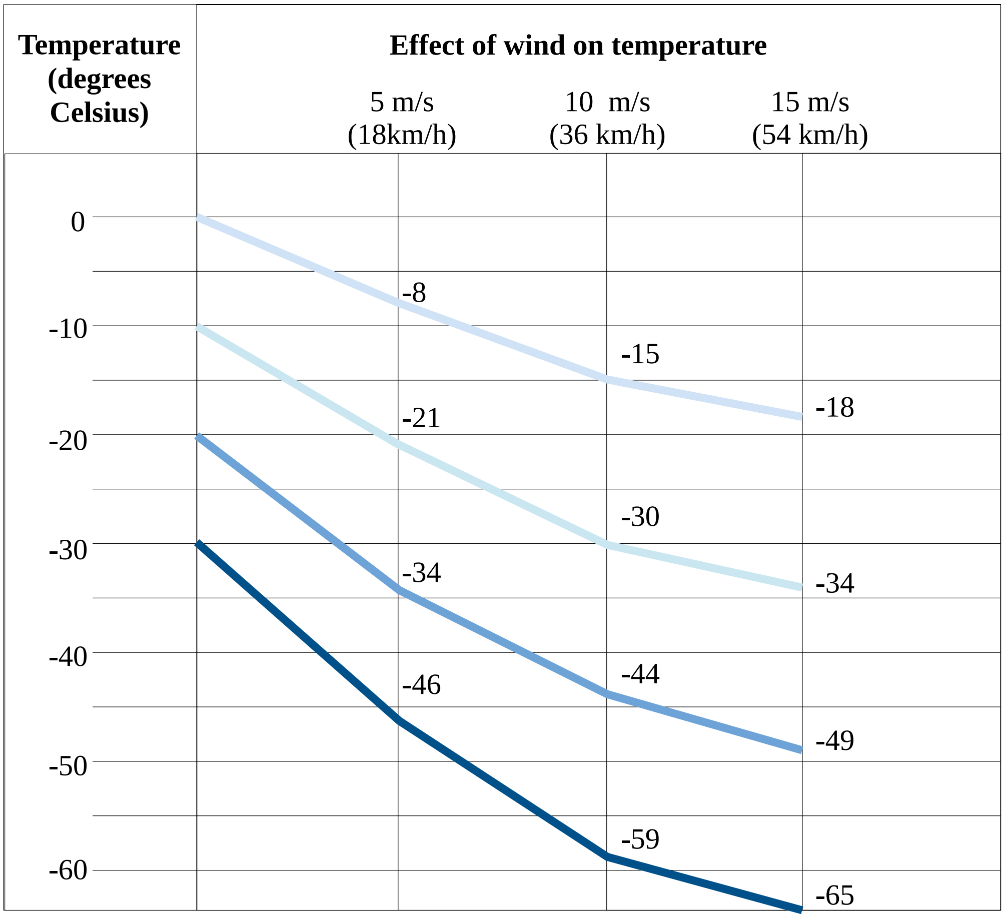

Figure 9.4 Effect of wind on temperature.

Frostbite

A different form of cold injury that can be seen in the athlete with hypothermia is frostbite. Frostbite is a direct consequence of the reflex peripheral vasoconstriction that occurs in response to cold. The local area cools to freezing with resultant tissue destruction. The areas most prone to frostbite are those that are most exposed or with little circulation - toes, fingers, ears, cheeks, nose, chin and neck.

As skin temperature falls, blood vessels vasoconstrict and the skin appears white. If cooling continues, the blood vessels dilate in an attempt to save the local area, which results in redness and burning pain. Further cooling leads to numbness and loss of skin sensation prior to freezing setting in. In the hands, the loose skin over the knuckles does not move freely.

Risk factors for frostbite include:

- low temperature
- high wind
- tight, wet footwear and gloves which result in poor insulative qualities of the material, and restriction of circulation to the extremities
- hypothermia
- previous cold injuries
- dehydration/hypovolemia
- past medical history of Raynaud's syndrome

Table 9.3 Signs of Frostbite.

Superficial Frostbite	**Deep Frostbite**
epidermis frozen	epidermis and dermis frozen
numbness at effected area	numbness at effected area
some pain perception	no pain
tissue pliable	tissue is solid

Treatment for superficial frostbite include:

- field re-warming is safe and effective
- spontaneous warming through cold induced vasodilation
- skin to skin warming
- warm water immersion

Treatment for deep frostbite include:

- do not thaw if there is a chance that the area may re-freeze
- provide definitive care to assure full physiological control
- keep the whole body warm to avoid peripheral vasoconstriction that will perpetuate the frostbite
- thaw rapidly and completely by immersion in warm water (40-42° C)
- protect the thawed area by not breaking blisters using aseptic technique; manage as per burn with dry sterile dressing
- avoid hasty amputations for remarkable recovery can, at times, occur

Prevention includes wearing appropriate clothing and footwear, such as:

- well-insulated mittens (these are preferred to gloves)
- loose-fitting insulated boots with dry, wool socks
- clothes that decrease exposed skin surface (balaclava, etc)
- goggles or glasses to prevent corneal irritation and artificial tear preparations that minimize dehydration which precedes "frostbite"

Spinal cord injured athletes need to have extremely well insulated extremities with frequent checks to ensure that they are not cold or suffering from frostbite.

D. Air Pollution

Olympic Games have taken place in cities where the possibility of adverse athletic performance due to pollutants has been a concern, eg. in Tokyo (1964), Mexico City (1968), Los Angeles (1984) and Seoul (1988). In each instance, there were major concerns before the event, and few criticisms after.

Types of Pollutants

Air pollutants can be considered primary or secondary. Primary pollutants are directly emitted from the source with little or no chemical change prior to inhalation. These include carbon monoxide (CO), carbon dioxide (CO_2), sulphate (SO_2, SO_4), nitrous oxide (NO, NO_2) and particulate matter - lead, carbon "soot" and ash. Secondary pollutants are formed by chemical reaction of emitted pollutants and natural precursors. They include ozone and peroxyacetylnitrates (formed by reaction between nitrous oxides and hydrocarbons in the presence of light), HNO_2 and H_2SO_4. The predominant source of these pollutants is the combustion of fossil fuels in industry, utilities, business, transportation and domestic use.

It is clear that the healthy young athlete at rest is not at risk from the adverse effects of air pollution. Patients suffering from a cardiac or respiratory problems have cause for concern. Athletes with a history of asthma or hay fever and even those with no previous history of respiratory problems may be affected by the pollution and this will affect their performance.

Adverse Effects

Air pollution may adversely affect the cardiopulmonary functioning of the actively training athlete, via:

- increased respiratory minute volume and therefore increased inhaled pollutants

extent of pollution = ventilation x concentration of pollutant x time

- mouth breathing, therefore bypassing the filtration mechanism of the nasal passage
- increased air flow velocity resulting in the inhalation of pollutants deeper into the lungs
- increased susceptibility to the effect of pollutants due to maximal organ function, eg. heart, lung

The adverse effects of some of the specific primary and secondary pollutants are:

- CO - decreased lung function, decreased work load, decreased tissue release of oxygen, increased cardiac work, and impairment of psycho motor function (eg. perception of time)
- SO_2 - water soluble; at rest, dealt with by nasal passage; with exercise and mouth breathing, there is an increased exposure of the lower respiratory tract, eliciting a known irritant effect
- NO_2 - cough, shortness of breath
- Ozone (O_3) - decreased lung function, congestion, wheezing, headache, laryngeal and tracheal irritation, chest tightness and soreness with deep inspiration
- Peroxyacetylnitrate - visual irritation with eye fatigue and blurred vision

Due to the frequent upper respiratory tract irritation caused by air pollution, it is felt that athletes may be more prone to upper respiratory tract infections with a deleterious effect on morale, training, physiology, and ultimately, performance.

Up to 15% of summer Olympic athletes have asthma which, when managed appropriately, does not interfere in their athletic performance. Air pollution exacerbates asthma and adds a further confounding variable. Endurance athletes who may be most affected by air pollution, include:

- distance running -10 km and marathon
- race walking
- cycling
- soccer
- field hockey
- rowing
- triathlon

Air pollution tends not to be a factor in the winter settings of the Olympic Winter Games.

Management for Athletes

The management of air pollution with regard to athletic performance consists of:

a. Avoidance

- Race organizers, if cognizant of local weather and pollution patterns, can schedule events at sites and times which minimize air pollutants, e.g away from traffic, close to the coast and, if ozone is a factor, early in the morning or late in the afternoon when the level would be lower.
- The major sources of pollutants (cars and industry) should be minimized prior to and during a large-scale athletic event.
- Events can be cancelled if measured air pollutant levels are too high.
- Athletes can train where and when the air pollutant level is low.

b. Acclimatization

- There is a suggestion that exposure to ozone and sulfur dioxide results in a normal rather than decreased lung function, as well as less symptoms; whether this is a practical option is unknown.

c. General reassurance

- If the athlete perceives that the environment in which he/she is competing will hamper performance, then it likely will. Mental preparation and confidence heading into a Olympic event cannot be overstated and part of this preparation involves minimizing the mental impact of the factors over which one has no control; air pollution is clearly one of these factors.

d. Asthmatic athletes

- One has to be prepared to manage a flare-up of asthma due to air pollution in those predisposed. This involves having the appropriate medication available, such as Sodium Cromoglycate taken every 4-6 hours for two days before training and Salbutamol taken 15 minutes before training if there is air pollution.

- Athletes previously not known to be asthmatics may get bronchospasm and symptoms with exposure to air pollution and may require medication, eg. aerosol beta agonist. (Refer to Unit 3, section A2 - Exercise Induced Asthma).

E. References

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For further information, refer to the following web site:
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9 - Environmental Factors