1.3. INJURIES DUE TO PHYSICAL FACTORS HAZARDS

1.3.1. HYPOTHERMIA (COLD INJURY)

Cold injuries are classified as systemic or localised.

Factors influencing the risk for these injuries include the atmospheric temperature, humidity, wind velocity and health status of the persons.

The risk is increased if the person is elderly; is intoxicated with drugs or alcohol; is receiving medications such as barbiturates, phenothiazines, or reserpine; or has adrenal insufficiency, diabetes, myxedema etc.

SYSTEMIC HYPOTHERMIA

PATHOGENESIS

Systemic hypothermia is reduction of the body's core temperature below 35^0C. Hypothermia can occur at air temperatures up to 18.3^0C.

When the body is exposed to cold environments, it has two types of normal physiologic reactions:

1. constriction of superficial blood vessels in the skin and subcutaneous tisssues, resulting in heat conservation;
2. increase in metabolic heat production through voluntary movement and by shivering;
3. In cases of systemic hypothermia, physiologic functions are diminished.

Physical examination often reveals diminished neurologic reflexes, slow mental and muscular reactions, weak or nonpalpable pulse, arrhythmia, low blood pressure, and increased blood viscosity.

Core temperature should be taken with a thermometer or thermocouple capable of measuring temperatures as low as 28^0C, and esophageal or deep rectal measurement (15 cm) is best.

PREVENTION

Hypothermia can be prevented by wearing clothing specially designed to resist wind and rain but also to allow water vapor generated by perspiration to escape.

The exposed persons should be cautioned to avoid smoking and drug or alcohol use.

HYPOTHERMIA OF THE EXTREMITIES

The cheeks, nose, earlobes, fingers, toes, hands, and feet are the areas most likely to develop ice crystals within the tissue, resulting in localised hypothermic injury. As skin temperature falls beloy 25^0C tissue metabolism slows, although oxygen demand increases it work continues. There may be tissue damage at 15^0C due to ischemia and thrombosis and at - 3^0C due to actual freezing of the tissue.

Chilblains, also called acute pernio, consist of erythematous, pruritic skin lesions due to inflammation as a result of cold or dampness with cold.

With prolonged exposure, this condition can progress to chronic pernio or "blue toes" characterised by erythematous, edematous, ulcerating lesions of the acral parts of the toes. Scarring, fibrosis, and atrophy can occur.

PREVENTION

Prevention is the same as for systemic hypothermia (see above).

TREATMENT

Treatment includes elevating the extremities, gradually rewarming them by exposure to air at room temperature, and protecting pressure sites from trauma.

1.3.2. DISORDERS DUE TO HEAT

A stable internal body temperature requires maintenance of a balance between heat production and loss, which the hypothalamus regulates by triggering changes in muscle tone, vascular tone and sweat gland function. Production and evaporation of sweat are a major mechanism of heat removal.

The transfer of heat from the skin to surrounding gas or liquid (convection) may also occur, but this decreases in efficiency as temperature increases.

The passive transfer of heat via infrared rays for 65% of body heat loss under normal conditions.

Radiant heat loss also decreases as temperature increases up to 37.2^{0}C, at which point heat transfer reverses.

At normal temperatures evaporation accounts for about 20% of body heat loss, but at excessive temperatures it becomes the most important means for heat dissipation.

It, too, is limited as humidity increases and is ineffective at 100% relative humidity.

The scheduled and regulated exposure to heated environments of increasing intensity and duration (acclimatization) allows the body to adjust to heat by beginning to sweat at lower body temperatures, increasing the quantity of sweat produced, reducing the salt content of sweat, and increasing the plasma volume, cardiac out put, and stroke volume while the heart rate decreases.

HEAT STROKE

Heat stroke is a life-threatening medical emergency due to thermal regulatory failure manifested by cerebral dysfunction with altered mental status, hyperpyrexia, abnormal vital signs, and, usually, hot, dry skin. Heat stroke becomes imminent as the core (rectal) temperature approaches 41.4^{0}C.

It is most apt to occur following excessive exposure to heat; it occurs in one or two forms: "classic" or "exertional".

The classic form occurs under conditions of extreme heat among those with compromised heat dissipation capability (elderly individuals, infants, and chronically ill or debilitated patients).

Exertional heat stroke results from strenuous exertion in hot environments, often in unclimatized individuals.

Morbidity and mortality can results from cerebral, cardiovascular, hepatic, or renal damage.

CLINICAL FINDINGS

Thermal regulatory failure is characterised by dizziness, weakness, nausea, vomiting, confusion, delirium, and visual disturbances; changes in mental status are its hallmark. Convulsions, collapse, or unconsciousness may occur. The skin is hot and initially covered with perspiration; later it dries. Blood pressure may be slightly elevated but becomes hypotensive. Core temperatures usually exceed 41^{0}C. As with heat exhaustion, hyperventilation can occur and lead to respiratory alkalosis and compensatory metabolic acidosis. There may, also be abnormal bleeding, renal failure, or arrhythmias.

HEAT CRAMPS

Heat cramps result from sodium depletion caused by replacement of sweat losses with water alone.

They are usually characterised by slow and painful muscle contractions and severe muscle spasms that last from 1 to 3 minutes and involve the muscles employed in strenuous work.

The skin is moist and cool, and involved muscle groups feel like hard, stony lumps similar to biliard balls. The temperature may be normal or slightly increased, and blood tests may show low sodium levels and hemoconcentration.

Blood volume is not significantly diminished.

HEAT SYNCOPE

In heat syncope sudden unconsciousness results from cutaneous vasodilatation with consequent systemic and cerebral hypotension.

The skin is cool and moist and the pulse weak.

Systolic blood pressure is usually under 100 mmHg.

Treatment consists of recumbency, cooling, and liquids by mouth. Preexisting medical conditions should be monitored and treated if is necessary.

SKIN DISORDERS DUE TO HEAT

Heat rash is caused by sweat retention due to obstruction of the sweat gland duct.

Erithema ab igne is characterised by the appearance of hyperkeratotic nodules following direct contact with heat that is insufficient to cause a burn.

Intertrigo results from excessive sweating and is often seen in obese individuals.

Skin in the body folds is erythematous and macerated.

1.3.3. ATMOSPHERIC PRESSURE DISORDERS (DYSBARISM)

Sudden shift to an environment of lower ambient pressure, as occurs with rapid ascensions to the surface from deep-sea diving or with loss of cabin pressure while flying at high altitudes, causes decompression sickness. Compression sickness can occur following movement to an environment of higher ambient pressure, but the only common example of this is barotitis.

DECOMPRESSION SICKNESS (CAISSON DISEASE)

Decompression sickness results from mechanical and physiologic effects of expanding gases and bubbles in blood and tissue. When the body is exposed to an environment of higher than athmospheric gas pressure, as in tunneling or diving, it absorbs more of the inhaled gases than it does at sea level. Aided by its fat solubility, nitrogen concentrations increase in tissues, particularly those of the nervous system, bone marrow, and fat. Because the blood supply is poor in the bone marrow and fat, nitrogen enters and leaves these tissues more slowly than oxygen or carbon dioxide does. As the surrounding pressure form gas bubbles if there is insufficient time for its dissolution from tissues. Because oxygen and carbon dioxide have more easily between tissue compartments, their tendency for bubble formation is reduced. Remaining nitrogen gas bubbles are more symptomatic and distructive in less elastic structures of tissues (central nervous system, joints).

Most cases of decompression sickness have occurred after rapid ascension from sea depths in excess of 7000 m.

CLINICAL FINDINGS

There are three types of decompression sickness as described below. The type and severity of symptoms will depend on the age, weight, and physical condition of the pacient, the degree of physical exertion; the depth or altitude before decompression; and the rate and duration of decompression.

TYPE 1. Acute pain, usually around a major joint, may be incapacitating and cause the patient to assume a stoped posture ("the bends"). Pain may begin immediately after decompression or up to 12 hours later.

TYPE 2. Symptoms and signs of central and peripheral nerve damage may include vertigo, "pins and needles", Babinski's sign, paralysis of weakness of the limbs, headache, seizures, vomiting, visual loss or visual field defects, incontinence, impaired speech, tremor, and coma.

TYPE 3. It is characterised by aseptic necrosis of bone (osteonecrosis) which frequently involves the head or shaft of the humerus and less often the lower and of the femur and the tibial head.

COMPRESSION SICKNESS

When athmospheric pressure is increased, internal gases become compressed, usually with little effect.

The only common form of compression sickness is **barotitis**. This can occur with descent of an aircraft from a high altitude, which causes a relative vacuum in the middle ear space if the auditory tube is already obstructed due to allergies or upper pain or a foggy feeling in the ears, dizziness, tympanic membrane may appear inflamed and retracted or ruptured.

Barotitis can be prevented in people at risk by avoiding high-pressure exposures or, for short exposures, by using decongestants. Barotitis is usually self-limiting but can be treated with decongestant nose drops, a nasal vasoconstrictor inhaler, or use of Valsalva's maneuver.

1.3.4. INJURIES DUE TO RADIATION

INJURIES DUE TO INFRARED RADIATION

Infrared radiation covers of the electromagnetic spectrum between visible and RF radiation. It has wavelengths between 750 and 3 million nm and is composed of three spectral bands -A, B, C- which begin at 750 nm, 1400 nm, and 3000 nm, respectively.

Infrared radiation is given off from any object having a temperature greater than absolute zero.

Acute, high-intensity exposure to wavelengths shorter than 2000 nm can cause thermal damage to the cornea, iris or lens.

Thermal injury to the skin can also occur, but, it is usually self-limited and results in an acute skin burn with increased pigmentation.

Exposure to infrared radiation has been associated with cataract formation, particularly among glassblowers and furnace workers.

Injuries can be prevented by shielding heat sources, using protective eye and skin wear, and monitoring exposure levels.

INJURIES DUE TO VISIBLE RADIATION

Visible radiation (light) covers the portion of the electromagnetic spectrum between infrared and ultraviolet radiation and the wavelengths between 400 and 750 nm.