Occupational Health Hazards

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Occupational health hazards

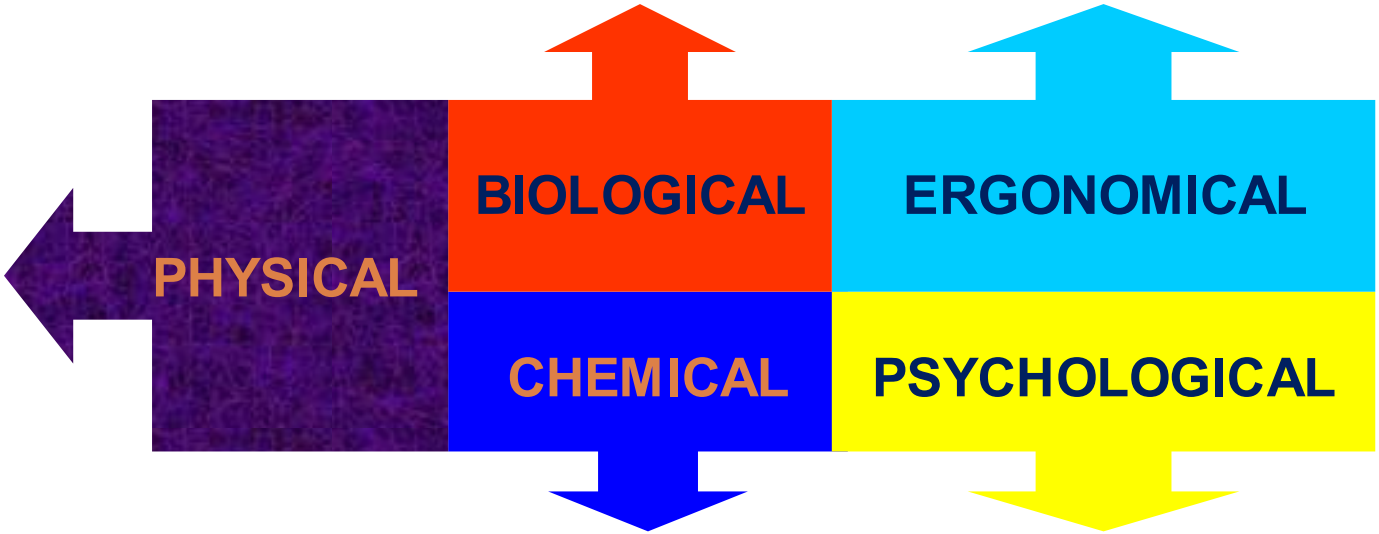
1. Physical
2. Chemical
3. Biological
4. Mechanical
5. Psychosocial
6. Ergonomics

Occupational Hazards

- Bacteria
- Fungi
- Insects
- Viruses

- Manual handling
- Repetitive movement
- Restrictive working area

- Noise
- Vibration
- Radiation
- Lighting
- Heat
- Cold
- Fibres
- Dusts



- Fumes
- Gases
- Aerosols
- Mists
- Liquids
- Vapours

- Job stress
- Job monotony
- Unsociable hours
- Job organization

1. Physical hazard

Temperature: *A. Heat*
B. Cold
C. Noise
D. Vibration
E. Atmospheric pressure
F. Radiation
G. Illumination
H. Electricity

Temperature:-
Heat &cold

Workers who labor under adverse environmental conditions may suffer serious physical injury from the effects of extreme elevation or depression of body temperature. Hyperthermia in particular has been associated with many occupational death. Environmental temperature extremes may be constant and predictable, like those that prevail in an underground mine, foundry, or ice making plant, or can vary with the season in outdoor occupations such as surface mining, roofing, farming, or construction.

A. Heat injuries.

1. Heat cramps

Heat cramps may be a symptom among forge and foundry workers, metal caster, iron and glass workers and miners, as well as those who work out of doors in hot climates.

Symptoms.

Heat cramps are extremely painful muscle contractions that occure in well-acclimated, physically fit persons as a consequence of sodium depletion following intense use of the involved muscle.Sometimes there are prodromal symptoms such as headache and dizziness.

The condition is a form of water intoxication, brought about because the workman drinks a lot of water in response to the sweating experienced in the hot environment, which in turn, leads to adecrease in the plasma osmolarity.

Treatment.

Heat cramps are adequately treated by rest and oral administration of salt-containing fluids. Men who work regularly in a hot environment gradually become acclimatized and it is found that the concentration of these ions in the sweat falls. This phenomenon explains why heat cramps are not more common.

2. Heat syncope.

Heat sycope is a potential problem for workers who must stand for long periods in a hot environment. It is a consequence of venous pooling in persons un acclimatized to heat, and is usually observed in the absence of any substantial exertion. Perior to loss of consciousness, the pulse rate is significantaly elevated but the core temperature is not. Orthostatic pulse and blood pressure changes are evident, which improve with acclimatization. Its occurrence may endanger workers operating at heights where balance and station are critical or those who operate machinery.

3. Heat exhaustion.

Heat exhaustion occurs during exercise in the heat, resulting in collapse or inability to continue work. Heat exhaustion is associated with a depletion of both salt and water. The concentration of the body fluids is not altered greatly but there is a diminution in blood volume which accounts for the symptoms. Over a period of few days the patient complains of weakness, fatigue and headache with perhaps anorexia and vomiting. If the vomiting is persistent, circulatory collapse may follow. The disease is rarely fatal. When seen by the doctor, the patient will have signs of peripheral circulatory failure with pallor, hypotension and profuse sweating.

It is differentiated from heat stroke by the presence of a normal mental status and by somewhat lower body temperature, usually not exceeding 105°F.

Treatment

1. Removing the patient to a cool place.
2. Rest in bed.
3. Oral or intravenous administration of sodium-containing fluids.

4. Heat stroke.

Is the most serious manifestation of heat illness. The largest modern experience of heat injury come from studies of miner, military recruits, athlete, and Meca pilgrims.

Analysis of case series of heat stoke reveals identifiable factors that may predict who is most likely to be intolerant of heat. Risk factors for heat stroke include:-

1.sleep deprivation
2.obesity
3.poor physical conditioning
4.lack of acclimatization.
5.dehydration
6.febrile illness
7.skin condition that affect sweating

Clinical manifestation of heat stroke.

The single clinical finding that distinguishes heat stroke from other forms of heat-related illness is; 1.altered mental status caused by heat injury to the brain. Other clinical criteria frequently used to define heat stroke include 2.a temperature greater than 41.1 c (106) and 3.the absence of sweating. The absence of sweating when noted at the onset of heat stroke suggests thermoregulatory failure and occurs in 25% to 50% of healthy patients who suffer exertional heat stroke. Temperature very near 42C the onset of protein coagulation and lipid liquefaction occur. Neurological manifestations range from mild confusion to psychosis, seizures, and coma. Mortality is correlated with the height of the temperature, the duration of temperature elevation, and duration of coma longer than 3 hours. Extensive cerebral edema with gross and microscopic hemorrhage is reported in patients who die soon after the onset of heat stroke. Although complete neurologic recovery is the rule in survivors of heat stroke, deficits may persist. Most patients with exertional heat stroke have tachycardia, and approximately 50% have systolic blood pressure below 100mmHg.

Recent studies suggest a role for elevated endotoxins and tissue cytokines (interleukins, tumor necrosis factor, interferon) in the development of hemodynamic collapse metabolic acidosis, and multiorgan failure in heat stroke.

•Respiratory alkalosis and lactic acidosis are commonly noted in patients with heat stroke.

•Coagulation disturbances are very common in heat stroke.

Thrombocytopenia appears early and has been attributed to the presence of disseminated intravascular coagulation (DIC).

•Renal failure occures in as many as 10% due to precipitation of myoglobin in renal tubules.

•Noncardiogenic pulmonary edema (NCPE) was reported in 23% of 52 consecutive cases of heat stroke observed during the Mecca pilgrimages in 1985.

•Direct thermal injury to the gastrointestinal mucosa commonly leads to diarrhea. Vomiting is also common. Major gastrointestinal hemorrhage occurs.

•Liver is frequently injured, as reflected by elevation of hepatic enzymes, which is noted within hours of the injury. In patients who survive, clinical evidence of liver injury such as jaundice and coagulation disturbances are manifest 2 to 3 days following the thermal insult.

•Heat stroke causes diffuse myocardial injury with nonspecific electrocardiographic ST and T wave changes. Reversible prolongation of the QT interval has been reported in 50% of cases.

Clinical evidence of right-sided heart failure has been observed with elevated central venous pressures and abnormal radionucleotide ventrigulograms.

Management of patients with heat stroke

The most critical steps in the management of heat stroke are the recognition of the possibility of temperature elevation and immediate, on-site initiation of rapid cooling. Rapid cooling by whatever means possible and the aim must be to reduce the temperature to 40°C within one hour, this minimizing the danger of damage to the central nervous system. Because the duration of temperature is critical factor in the development of cellular injury. Cooling must be initiated concurrently with major resuscitative procedure such as management or respiratory failure, or cardiac arrest. Cooling techniques using tape water and fans will be more efficacious than ice water immersion because they minimize the vasoconstriction and shivering and agitation, which do increase heat production, may be effectively controlled by small doses of an intravenous benzodiazepine titrated to effect hypotension should be treated with volume resuscitation and careful monitoring of urine output. Vasopressors with α-adrenergic effects are intraindicated in the management of hypotension owing to their potential to impair cooling. The use of sodium bicarbonate and mannitol infusions to decrease the precipitation of myoglobin has been widely recommended. Treatment may be required for as long as a week before sweating returns and the patient is able to regulate his temperature unaided.

Prevention of heat stroke.

Public education resulting in behavioral changes has had dramatic public health impact in the prevention of heat injury in athletes, military recruits, and the work-place preventive measures, including use of the WBGT (Wet Bulb Globe Temperature) to guide training schedules, close observation of the behavior of military recruits, education about and restriction of the use of ethanol, enforced rests and fluids protocols, and special guidelines for detection and acclimatization of potentially heat-sensitive recruits, have dramatically decreased the incidence of heat stroke in military personnel. American college of sports medicine guidelines has decreased the incidence of heat illness among athletes. Thirst is well known to be an inadequate measure of fluid needs. To avoid dehydration, workers must drink beyond their thirst. Education of workers with regard to the need to:-

a. Drink adequately.
b. Rest periodically in a cool environment.
c. Take off or work at sowler pace when ill.
d. Avoid ethanol prior to and during work is important

Workers and supervisors should also be aware of the meaning of early signs of neurologic impairment, such as irritbillity, confusion, or clumsiness. A ready means of cooling should be available at every work site where heat injury might occur.


Thank you
very much