ATMOSPHERIC VARIATIONS

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HEAT

Four factors influence the interchange of heat between man and his environment. These are 1) air temperature, 2) air velocity, 3) moisture content of the air, and 4) radiant temperature. The industrial heat problem is one in which a combination of these factors produces a working environment which may be uncomfortable or even hazardous because of imbalance of metabolic heat production and heat loss.

The fundamental thermodynamic processes involved in heat exchange between the body and its environment may be described by the basic equation of heat balance:

$$\Delta S = M - E \pm R \pm C \qquad (1)$$

where M = rate of metabolism; ΔS = change in body heat content; E = heat loss through evaporation; R = heat loss or gain by radiation; and C = heat loss or gain through convection. Under conditions of thermal equilibrium, this becomes:

$$M = E \pm R \pm C \qquad (2)$$

Under these conditions, since equilibrium means no change in body heat content, the heat generated within the body by metabolism is completely dissipated to the environment and $\Delta S = 0$.

For purposes of temperature determination, the body can be divided into two regions, the deep region or the core, and the superficial region which is made up largely of the skin and subcutaneous tissues. The thermo-regulatory mechanisms of the body are directed at maintaining a uniform core temperature (about 37.0±0.5°C/98.6±1.0°F) while the temperature of the superficial tissues may vary within a relatively wide range according to the amount of heat received from or lost to the environment. The mean weighted skin temperature may vary within the range of 29 to 36°C/84 to 97°F.

TRANSFER MECHANISMS

When heat loss fails to keep pace with heat gain, the core temperature begins to rise. At this point certain physiologic mechanisms come into play in an attempt to increase heat loss from the body. First, there is dilation of the blood vessels of the skin and subcutaneous tissues with diversion of a large part of cardiac output to these superficial regions. There is a concomitant increase in circulating blood volume brought

about by contraction of the spleen and by dilution of the circulating blood with fluids drawn from other tissues. Cardiac output is also increased. All these circulatory adjustments enhance heat transport from the body core to the surface. Concomitantly the sweat glands become active, spreading fluid over the skin which removes the heat from the skin surface by evaporation. Under these conditions the equation (2) may be modified:

$$E = M \pm R \pm C \qquad (3)$$

to indicate that evaporative cooling must balance metabolic plus environmental heat load to maintain thermal equilibrium. If this fails, heat storage begins with the strain of increased body temperature occurring. Unchecked, this can lead to heat stroke which is often fatal, and is always more or less debilitating.

In general, industrial heat exposures may be classified as either hot-dry or as warm-moist. In the former the moisture content of the air is not excessive, so evaporative cooling is not impeded. The difficulties in hot-dry situations arise when the body absorbs more heat by radiation or convection or both than the cooling power of the sweat which man can produce and evaporate, that is: $M+R+C>E$. The human sweat producing capacity may be as high as 2 liters per hour, but over an 8-hour period a sweat rate of 1 liter per hour is considered to be the maximum which a healthy acclimatized worker can maintain day by day. Warm-moist environments may occur during the summer in areas where the outdoor air has a high moisture content or in plants where large amounts of moisture are released from the industrial processes involved, while air and radiant temperatures may be moderate. Here, the heat load from radiation and convection may not be great, but the high humidity inhibits heat loss from the body through evaporation of sweat and the same imbalance may occur.

It is apparent from the foregoing that an ordinary room thermometer (which corresponds to a dry bulb thermometer in scientific terminology) will not describe the total heat load imposed upon the worker by his job because it reacts only to the air temperature and thus informs us only about convective heat change. The globe thermometer is most widely used for assessing the radiant heat load; the wet bulb thermometer, for assessing the humidity of the air; and the anemometer, for wind velocity measurement. The metabolic heat generated within the body can be assessed by using energy requirement tables published in the literature. For more accurate determination of metabolism the oxygen consumption has to be measured.

If all these measurements are performed, it is possible to calculate the required evaporation (E_{req}) for maintaining heat equilibrium in a given work environment. The nomograms of the Belding-Hatch heat stress index (HSI) make this calculation relatively simple. Furthermore, the HSI permits the estimation of the maximum evaporative capacity of the ambient air (E_{max}). It is the ratio $\frac{E_{req}}{E_{max}} \times 100$ which gives us the HSI

value, indicative of the stressfulness of a hot job. There are other simpler heat stress indices, however, which can be used for the purpose of describing the environmental heat load. The Corrected Effective Temperature (CET) can be assessed by the use of a single nomogram and it combines the values of air temperature, humidity, and wind velocity into one number which is related to the human comfort feeling. The Wet Bulb Globe Temperature index (WBGT) is a simplified version of CET. Instead of a nomogram an equation can be used:

$$WBGT = 0.7\ NWB + 0.3\ GT \quad \text{(for indoors)} \quad (4)$$

$$WBGT = 0.7\ NWB + 0.2\ GT + 0.1\ DB \quad \text{(for outdoors)} \quad (5)$$

where NWB = natural wet bulb temperature
GT = globe temperature and
DB = dry bulb temperature.

Thus for estimating the WBGT index there is no need for wind velocity measurements which further simplifies this methodology.

ACCLIMATIZATION

Acclimatization is essential if man is to work in hot environments. This process of adaptation is characterized by the worker's ability to perform with less increase in core temperature and heart rate and less salt loss, due to a lower concentration of sodium chloride in the sweat. The greatest portion of adaptive changes in acclimatization to heat occurs within the first week. Nonadaptable individuals often abandon hot jobs within that time span. Acclimatization to heat can, however, be lost almost as rapidly as it is acquired.

The human sense of thirst is not an adequate regulator of fluid replacement during heat exposure. If workers are sweating profusely and do not replace their fluid and salt loss systematically, most of them will end up each work day in a dehydrated state. The amount of water loss considered to be still compatible with good health and high degree of fitness, provided that the water content of the body is restored by the start of the next work day, is 1.5% of the total body weight.

HARMFUL EFFECTS

Prolonged exposure to excessive heat may cause increased irritability, lassitude, decrease in morale, increased anxiety, and inability to concentrate. The results are mirrored by a general decrease in the efficiency of production and in the quality of a finished product.

The physical disabilities caused by excessive heat exposure are, in order of increasing severity, heat rash, heat cramps, heat exhaustion, and heat stroke.

Heat rash (prickly heat) may be caused by unrelieved exposure to hot and humid air as may occur in warm-moist climatic zones. The orifices of the sweat ducts become plugged due to the swelling of the moist keratin layer of the skin which leads to inflammation of the glands.

There are tiny red vesicles visible in the affected skin area and, if the affected area is extensive, sweating can be substantially impaired. As a consequence heat rash not only is a nuisance because of the discomfort it causes but also can greatly diminish the workers' capacity to tolerate heat.

Heat cramps may occur after prolonged exposure to heat with profuse perspiration and inadequate replacement of salt. The signs and symptoms of heat cramps consist of spasm and pain in the muscles of the abdomen and extremities. Albuminuria may be a transient finding.

Heat exhaustion may result from physical exertion in a hot environment when vasomotor control and cardiac output are inadequate to meet the increased demand placed upon them by peripheral vasodilatation or the plasma volume is reduced by dehydration. Signs and symptoms of heat exhaustion may include palor, lassitude, dizziness, syncope, profuse sweating, and cool moist skin. There may or may not be a mild hyperthermia, observable by rectal measurement.

Heat stroke is a serious medical condition. An important predisposing factor is excessive physical exertion. Signs and symptoms may include dizziness, nausea, severe headache, hot dry skin because of cessation of sweating, very high body temperature (usually 106°F and rising), confusion, collapse, delirium, and coma. Often circulation is also compromised to the point of shock. If cooling of the victim's body is not started immediately, irreversible damage to vital organs may develop, leading to death.

Some studies performed in Europe and in South America showed evidence that workers employed for prolonged time in hot industry have a higher morbidity rate from cardiovascular diseases.

RECOMMENDED LIMITS

Higher heat exposures than shown in Table 11 are permissible if the workers have been undergoing medical surveillance and it has been established that they are more tolerant to work in heat than the average worker. Workers should not be permitted to continue their usual work routine when their deep body temperature exceeds 38.0°C.

Table 11. Permissible heat exposure threshold limit values.

	Work load*		
Work-Rest regimen	Light	Moderate	Heavy
Continuous work	30.0	26.7	25.0
75% Work 25% Rest, Each hour	30.6	28.0	25.9
50% Work 50% Rest, Each hour	31.4	29.4	27.9
25% Work 75% Rest, Each hour	32.2	31.1	30.0

*Values are given in °C WBGT

POTENTIAL OCCUPATIONAL EXPOSURES

Animal rendering workers
Bakers
Boiler heaters
Cannery workers
Chemical plant operators working near hot containers and furnaces
Cleaners
Coke oven operators
Cooks
Foundry workers
Glass manufacturing workers
Kiln workers
Miners in deep mines
Outdoor workers during hot weather
Sailors passing hot climatic zones
Shipyard workers when cleaning cargo holds
Smelter workers
Steel and metal forgers
Textile manufacturing workers (weaving, dyeing)
Tire (rubber) manufacturing workers

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COLD

For the body to maintain thermal homeostasis in a cold environment, certain physiologic mechanisms come into play which tend to limit heat loss and increase heat production. The first mechanism is one of peripheral vasoconstriction, especially in the extremities, resulting in a marked drop in skin temperature. Body heat loss to the environment is thereby diminished. The most severe strain of this mechanism of heat conservation is chilling of the extremities so that if activity is restricted, the toes and fingers may approach freezing temperatures very rapidly.

Long before that, and in fact when their temperature drops below 15°C, the hands and fingers become insensitive, and the probability of malfunction and accidents increases.

In general, cooling stress is proportional to the total thermal gradient between the skin and the environment since this gradient determines the rate of heat loss from the body by radiation and convection. Loss of heat through the mechanism of the evaporation of perspiration is not significant at environmental temperatures lower than about 15° to 20°C. When vasoconstriction is no longer adequate to maintain body heat balance, muscular hypertonus and shivering become important mechanisms for increasing body temperature by causing metabolic heat production to increase to several times the resting rate. Not only shivering, but general physical activity acts to increase metabolic heat. With proper insulation from clothing to minimize heat loss through even a large thermal gradient, a satisfactory microclimate may be maintained with only exposed body surfaces (as the face and the digits of hands and feet) liable to excessive chilling and frostbite. However, if the garments become wet either from contact with water or due to sweating during intensive physical work, their cold insulating property will be greatly diminished.

HARMFUL EFFECTS

Frostbite occurs when there is actual freezing of the tissues with the attendant mechanical disruption of cell structure. Theoretically, the freezing point of the skin is −1°C; however, with increasing wind velocity, heat loss is greater and frostbite will occur more rapidly. Once started, freezing progresses rapidly. For example, if the wind velocity reaches 20 mph, exposed flesh will freeze within about one minute at −10°C. Furthermore, if the skin comes in direct contact with objects whose surface temperature is below freezing point, frostbite may develop in spite of warm environmental temperatures. The first warning of frostbite is often a sharp, pricking sensation. However, cold itself produces numbness and anesthesia which may permit serious freezing to develop without the warning of acute discomfort. Injury produced by frostbite may range from simple superficial injury with redness of the skin, transient anesthesia and superficial bullae to deep tissue freezing with persisting ischemia, thrombosis, deep cyanosis, and gangrene.

Trench foot or immersion foot may be caused by long continuous exposure to cold without freezing, combined with persistent dampness or actual immersion in water. This condition is due to persistent local tissue anoxia, combined with mild or severe cold with resultant injury to the capillary walls. Edema, tingling, itching, and severe pain occur and may be followed by blistering, superficial skin necrosis, and ulceration.

General hypothermia is an extreme acute problem resulting from prolonged cold exposure and heat loss. If an individual becomes fatigued during physical activity, he will be more prone to heat loss, and as exhaustion approaches, the vasoconstrictor mechanism is overpowered; then sudden vasodilatation occurs with resultant rapid loss of heat, and critical

cooling ensues. Sedative drugs and alcohol increase the danger of hypothermia.

Vascular abnormalities may be either precipitated or aggravated by cold exposures. These include chilblain (pernio), Raynaud's disease, acrocyanosis, and thromboangiitis obliterans. Workers suffering from these ailments should take special precautions to avoid chilling. Some people develop hypersensitivity reactions when exposed to cold.

RECOMMENDED LIMITS

Cold stress indices have been developed for estimating the significance of cold environments for human welfare and efficiency. Those relating insulating effect of clothing and the convective heat loss of cold air movement (wind chill) are probably most useful in predicting the impact of cold outdoor exposure.

POTENTIAL OCCUPATIONAL EXPOSURES

Occupations with potential exposure include:

Cooling room workers
Divers
Dry ice workers
Firemen
Fishermen
Ice makers
Liquified gas workers
Out-of-door workers during cold weather
Packing house workers
Refrigerated warehouse workers
Refrigeration workers

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HYPERBARIC ENVIRONMENTS

Air pressures in excess of those found at sea level (hyperbaric) are encountered in both terrestrial and aquatic environments. Sea level pressure equals 14.7 pounds per square inch, or one atmosphere absolute (ata). Occupational exposures occur in caisson or tunneling operations, where a compressed gas environment is used to exclude water or mud and to provide support for structures. Pressures encountered in such

operations range from less than 2 ata to more than 4 ata. Similarly, hyperbaric environments are encountered by divers operating underwater, whether by holding the breath while diving, breathing from a self-contained underwater breathing apparatus (SCUBA), or by breathing gas mixtures supplied by compression from the surface. While commercial divers routinely dive to depths greater than 100 meters, even in breath-holding dives to 30 meters, pressures encountered can be considerable (each 10-meter increase in sea water depth is equivalent to an increase of 1 atmosphere pressure).

PRIMARY PRESSURE PHENOMENA

Man can withstand large pressures above normal, providing air has free access to all surfaces of the body including lungs, sinuses, and the middle ear. Unequal distribution of pressure can result in barotrauma, probably the most common occupational disease of those who work in high pressure environments. Barotrauma refers to tissue damage resulting from expansion or contraction of gas spaces found within or adjacent to the body, and can occur either during compression (descent) or during decompression (ascent).

The teeth, sinuses, and ears are frequently affected by such pressure differentials. For example, gas spaces which may be present adjacent to tooth roots or fillings may be compressed during descent. Fluid or tissue forced into these spaces may cause pain either during descent or ascent. Sinus blockage, comparatively rare in divers, is probably due to occlusion of the sinus aperture by inflamed nasal mucosa which prevents equalization of pressures.

Middle ear barotrauma (aerotitis media) occurs commonly among divers. Blockage of the eustachian tube as a result of inflammation or by failure of the diver to clear the ears, creates a negative middle ear pressure during compression, with progressive inward deformation of the tympanic membrane, with possible rupture. Forceful Valsalva maneuvers under these conditions can also result in round window rupture with inner ear damage.

The lungs themselves may be subject to squeeze if the chest is compressed to a volume smaller than the residual volume of the lung, the amount of air left in the lungs following forced expiration. Lung squeeze is occasionally seen in unprotected swimmers who dive by simply holding the breath. The effect of the squeeze is to force blood and tissue fluids into the respiratory passages and alveoli. Considerable lung damage may result.

SECONDARY PRESSURE PHENOMENA

In addition to the mechanical effects there are well known problems of toxicity from the gases of air at elevated partial pressures. Also some normally toxic gases such as carbon monoxide are probably more toxic at elevated partial pressure. These phenomena have to do with molecular rather than with bulk gas characteristics.

Narcotic action of nitrogen: At 4 atmospheres of pressure or more,

the gaseous nitrogen in normal air induces a narcotic action evidenced by decreased ability to work, mood changes, and frequently, a mild to marked euphoria. The responses are similar to those associated with alcoholic intoxication. The exact cause of this cerebral disturbance is unknown. It may be noted, however, that nitrogen is highly soluble in fat, the ratio of its solubility in fat to its solubility in water being about five to one. According to the Meyer-Overton hypothesis, a gas having such a relatively high solubility ratio may act as a narcotic.

Oxygen poisoning: Inhalation of oxygen when its partial pressure exceeds two atmospheres may result in the production of the signs and symptoms of oxygen poisoning. These include tingling of fingers and toes, visual disturbances, acoustic hallucination, confusion, muscle twitching, especially about the face, nausea, and vertigo. The final result of such exposure may be the epileptiform convulsion, which ceases as soon as exposure to high oxygen partial pressures is terminated. This toxic action of oxygen is greatly enhanced by exercise or by the presence of moderate amounts of carbon dioxide. At one atmosphere, about 15 p.s.i., pure oxygen will irritate the throat although symptoms of systemic oxygen poisoning do not occur if the exposure is relatively short.

It should be noted that the greatest hazard in oxygen administration in chambers is the danger of fire. It is also true that in increased environmental pressures an increased partial pressure of oxygen enhances the fire hazard.

Effect of carbon dioxide: Carbon dioxide enhances the toxicity of oxygen and the narcotic effect of nitrogen, and in addition a higher incidence of bends has been reported in association with a rise in the CO_2 pressure. The partial pressure of CO_2 present in the breathing medium in a compressed air environment should not exceed the equivalent of 0.2 percent CO_2 at one atmosphere pressure.

DECOMPRESSION

An opposite effect to lung squeeze, expansion of air in the lungs, may occur during ascent from depths of water or during decompression in a chamber. Air in the lung at a depth of 130 feet is at 5 ata. It will increase in volume five times when decompression to normal atmospheric pressure occurs. If decompression is excessively rapid and sufficient air is not exhaled, some of the pulmonary alveoli will rupture with the formation of one or more of the following: mediastinal emphysema, pneumothorax, or air embolism. The most dangerous of these conditions is the air embolism which occurs when air, expanding in the lung, is forced into the pulmonary blood vessels and then into the left side of the heart. The arterial circulation may quickly carry the air bubbles to the brain to produce a cerebral air embolism, a condition which may be rapidly fatal if not treated promptly by decompression.

A more likely mechanical problem of a too rapidly decreased air pressure is formation of nitrogen bubbles as the gas leaves solution in blood and tissues, a situation comparable to bubble formation in carbonated beverages when the closure is broken. These bubbles of liberated

gas create circulatory impairment and local tissue damage and are responsible for the signs and symptoms of decompression sickness.

The amount of bubble formation that will occur upon decompression depends to a large extent upon 1) the amount of gas dissolved in the tissues, which in turn is dependent upon the degree and duration of exposure to pressure and upon the amount of body fat in which gas can be dissolved; 2) conditions which alter blood flow, including age, temperature, exercise, fright, and post-alcoholic state, especially if these alterations in blood flow occur during or shortly after the decompression process; and 3) the rapidity of decompression from elevated air pressure to the ambient level. The conditions can, but are less likely to, occur upon rapid ascent from ground level to high elevation in high performance aircraft.

Acute Signs and Symptoms

Bends: A relatively common manifestation of decompression sickness is a dull, throbbing type of pain which is gradual in onset, progressive and shifting in character, and frequently felt in the joints or deep in the muscles and bones. When the symptoms of bends occur, they do so in the first four to six hours in 80 percent of the cases, while the remainder will occur within 24 hours. Contributing to variations in susceptibility are such factors as age, obesity, defects to the lungs, heart impairments, temporary ill health, and individual predisposition.

Chokes: This rather specific type of asphyxia occurs less frequently than bends and is thought to be due to the accumulation in the large veins, the right side of the heart, and the pulmonary vessels of quantities of gas eliminated from the arterial circulation and from the extravascular tissues. The earliest evidence of impending chokes is a sensation of substernal distress felt during deep inspiration, especially during inhalation of tobacco smoke which elicits paroxysmal coughing. These attacks of coughing may proceed to loss of consciousness with all of the signs and symptoms of a true shocklike syndrome.

Paralysis: The most serious complication of decompression sickness is paralysis. Spastic paraplegia or monoplegia involving the lower extremities may follow the formation of bubbles in the blood vessels and tissues of the spinal cord. Immediate and prolonged decompression usually brings about rapid recovery even following paraplegia. Cerebral involvement is very rare.

Chronic Symptoms

Aseptic bone necrosis: The most likely chronic sequela of repeated compressed air exposure is termed aseptic bone necrosis. This condition is thought to be caused by the occlusion of small arteries in the bone by bubbles of nitrogen followed by infarction in the involved area. The sites of predilection for the occurrence of occlusion and necrosis, as seen in this process, are the lower femoral diaphysis, the upper tibial diaphysis, and the head and neck of the humerus and the femur. These lesions are usually multiple and tend to be bilaterally symmetrical.

Aseptic bone necrosis is usually asymptomatic unless joint surfaces are involved, in which case pain may be a symptom. Complete collapse of the affected joint has been known to occur. Healing takes place through an osteocondensing process. This increase in density may appear on roentgenographic examination as a snowcap on the top of the articular surface.

Dysbaric osteonecrosis: A significant incidence of dysbaric osteonecrosis has been recognized in caisson workers in the USA and England. It has also been recognized to occur in Royal Navy divers, among commercial divers working in USA coastal waters, in Japanese breathholding divers, and even in U.S. Air Force pilots. There appears to be a correlation between the disease and the number of decompressions undergone by an individual, frequency of exposure, magnitude of pressure, and frequency of dysbarism-related incidents. It is uncertain whether the disease can be prevented by adherence to recommended decompression schedules.

HYPOBARIC ENVIRONMENTS

Two rather distinct types of occupational exposure to hypobaric environments exist: high altitude and low altitude.

HIGH ALTITUDE SYMPTOMS

Among pilots and air crews engaged in operation of high performance aircraft at extremely high altitudes (in excess of 30,000 feet), the greatest single potential hazard is hypoxia. Deprivation of oxygen at these altitudes results in rapid loss of consciousness. Exposure to these reduced pressures (dysbarism) may also produce symptoms similar to those encountered by rapid decompression in divers. Bends, chokes, neurological disorders, aeroembolism, aerodontalgia, aerotitis, and aerosinusitis have all been described in air crewmen.

Dysbarism may be complicated by a type of neurogenic peripheral circulatory failure or primary decompression shock consisting of any or all of the following manifestations: intense pallor, profuse sweating, faintness and dizziness, nausea, vomiting, and loss of consciousness. These symptoms are usually relieved rapidly by descent from altitude.

OTHER ALTITUDE SYMPTOMS

Potential occupational hazards also exist at much lower altitudes, where the effects of hypoxia are evidenced by impaired judgment and performance, and a general feeling of malaise. Acute mountain sickness (AMS) is considered a definite clinical syndrome characterized by overwhelming depression, severe headache, nausea, vomiting, and loss of appetite. Particularly characteristic is irritability of the subject. Virtually all sojourners develop one or more symptoms, although the severity of such symptoms varies widely among subjects. Peak severity is reached within 48 hours and symptoms disappear over the following 2 to 4 days.

PULMONARY EDEMA

Of considerable concern to the physician is the not infrequent occurrence of high altitude pulmonary edema. This circulatory disturbance appears more frequently in children than adults, and frequently occurs when altitude–acclimatized subjects return from sojourning at sea level. There is a strong tendency for it to recur repeatedly in susceptible subjects. The condition usually begins with progressive cough and dyspnea, and is associated with elevated pulmonary arterial pressures. Treatment with O_2 or return to sea level usually abolishes symptoms rapidly.

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OSCILLATORY VIBRATIONS

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Research and study continue in efforts to verify noise tolerance limits for protecting human hearing. The effects of different vibration conditions for causing pain and injury or illness to individuals exposed are also under study. The following sections offer a broad overview of present findings in relation to recognizing the occupational origin of certain symptoms due to exposure to oscillatory vibrations.

OCCUPATIONAL DISEASES

A Guide to Their Recognition

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