CHAPTER

8

Physical Health Hazards in Industry

Introduction

The first link in the chain of disease or hazard transmission is a disease agent or hazardous agent. Other two links in the chain of disease or hazard transmission are host and environment factors. An agent is defined as a substance, living or non-living, or a force, trangible or intrangible, the excessive presence or relative lack of which may initiate or perpetuate a disease or hazard process. A disease or hazard may have a single agent, a number of independent alternative agents or a complex of two or more factors (agents) whose combined presence is essential for the development of the disease or hazard.

A physical hazard is an agent, factor or circumstance that can cause harm with or without contact. They can be classified as one type of occupational or environmental hazards. Physical hazards include heat, cold, noise, vibration, ionizing radiation, non ionizing radiation, barometric pressure, electromagnetic field. Some control measures are often used to mitigate physical hazards. Physical hazards are a common source of injuries in many industries. They are perhaps unavoidable in certain industries, such as construction and mining, but over time people have developed safety methods and procedures to manage the risks of physical danger in the workplace.

INDUSTRIAL HEAT AND COLD EXPOSURE

Thermal Balance

The human body follows the law of conservation of energy expressed in the equation:

$$M \pm (R + C + K) - E = \pm S$$

where M is the heat generated by metabolism (always positive); R is the radiant heat exchange with the environment (negative for loss from the body); C is the convective heat exchange with the surrounding fluid (negative for loss); K is the conductive heat exchange with any contacting solid (negative for loss); E is the evaporative heat loss resulting from the evaporation of liquid from the body or its clothing (almost always negative or zero); and S is the heat stored (positive) or lost (negative) by the body, and thus reflected in a change in mean body temperature. Basal metabolic heat generally remains relatively constant in health; little bit is diminished in hypothermia, endocrine disorders such as hypothyroidism, certain drug intoxications, etc. It rises following a meal (the specific dynamic effect), and in some endocrine and some other disorders. However, the greatest variation in metabolism is seen in the additional heat generation resulting from physical activity. Depending on the intensity of that activity, total metabolic heat production can vary from less than 80 W when asleep, to over 500 W during short-duration intense exercise. Examples of some

thermal balance: (1) *Sedentary work*: In sedentary work, M will be approximately 100 W. For S = 0, thermal equilibrium, most of that 100 W will be lost in C, with a small loss in E as a result of insensible perspiration (here, M = 100 and S = 0). (2) *Exercise*: In this condition, when M rises to 250 W or more, thermal equilibrium can only be maintained by overt sweating to increase E substantially, to 200 W or more, as C will not increase much. This requires the effective evaporation of upwards of 300 ml of sweat per hour, comfortably within human capability (here, M > 250 and S = 0). (3) *Exercise in occlusive clothing*: Workers who are required to perform physical exercise inside occlusive clothing can find that R, C, K and E all fall close to 0 W, so that all the heat that they generate, perhaps 200 W or more, will be stored at S. This results in a rapid rise in body temperature, perhaps as high as 10°C per hour, and rapid onset of heat illness (here, M = 250 and S = 250). (4) *Cooling when wearing wet clothing*: Inadequate protective clothing can result in excessive evaporative loss too. With a sedentary M of about 100 W, someone whose clothing is soaked might evaporate as much as a litre of water every hour, which would make E become about 700 W. S then becomes very negative and their body cools in spite of shivering attempting to increase M. They will become hypothermic unless their evaporative loss is stopped, perhaps by surrounding them with a plastic bag to act as a water vapour barrier (here, M = 100 and S = –700).

HEAT

Introduction

The character of the thermal environment is determined not only by its total thermal energy content but also by the flow of thermal energy as a result of temperature differences. Heat-transfer analysis is a specialist field, but the environmental heat load can be approximately by a set of simple measurement.

The ramification of human heat exposure is legion: They embrace social relationship, physical and mental well-being, and, ultimately, human productivity. This chapter addresses a major facet, namely physical work in heat: It considers the thermal environment, man's capabilities and limitations during work in heat, strategies to resolve the problems associated with heat exposure, practical procedures to protect workers, and medical recommendations.

Operation involving high air temperatures, radiant heat sources, high humidity, direct physical contact with hot objects or strenuous physical activities has a high potential for inducing heat stress in employees engaged in such operations. Such places include: Iron and steel foundries, nonferrous foundries, brick-firing and ceramic plants, glass products facilities, rubber products factories, electrical utilities (particularly boiler rooms), bakeries, confectioneries, commercial kitchens, laundries, food canneries, chemical plants, mining sites, smelters and steam tunnels. Outdoor operations conducted in hot weather, such as construction, refining, asbestos removal and hazardous waste site activities, especially those that require workers to wear semi-permeable or impermeable protective clothing, are also likely to cause heat stress among exposed workers.

Five main medical disorders can result from excessive exposure to hot environments and they are heat stroke, heat exhaustion, heat cramps, heat syncope, and skin disorders.

A stable internal body temperature requires maintenance of a balance between heat production and loss, which the hypothalamus regulates by triggering changes in thirst, muscle tone, vascular tone, and sweat gland function. Production and evaporation of sweat are a major mechanism of heat removal (however, sweating causes loss of body water and sodium). The transfer of heat from the skin to surrounding gas or liquid (convection) or between two solids in direct contact

(conduction) also may occur, but this decreases in efficiency as ambient temperature increases. The passive transfer of heat via infrared rays from a warmer to a cooler object (radiation) account for 65% of body heat under normal conditions. Radiant heat loss also decreases as temperature increases up to 37.2°C (99°F), at which point heat transfer reverses. At normal temperatures, evaporation accounts for approximately 20% of body heat loss, but at excessive temperature, 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 output, and stroke volume while the heart rate decreases.

Health conditions that inhibit sweat production or evaporation and increase susceptibility to heat injury include obesity, skin disease, decrease cutaneous blood flow, dehydration, hypotension, cardiac disease resulting in reduced cardiac output, use of alcohol or medications that inhibit sweating, reduce cutaneous blood flow, or cause dehydration (e.g. atropine, antipsychotics, tricyclic antidepressants, diuretics, laxatives, antihistamines, monoamine oxidase inhibitors, vasoconstrictors, and beta-blockers), and use of drugs that increase muscle activity and thereby increase the generation of body heat [e.g. phencyclidine (PCP), lysergic acid diethylamide (LSD), amphetamines, cocaine, and lithium carbonate]. Infection, cancer, malnutrition, thyroid dysfunction and other medical conditions characterized by debilitation and poor physical condition can reduce the effectiveness of the sweating mechanism and circulatory response to heat. Age and sex also affect susceptibility to heat injury. Older people do not acclimatize as easily as because of their reduced sweating efficiency, and women generally generate more internal heat than men when performing the same task.

Heat Balance Equation

Heat exchange takes place between body and the environment by convection, radiation, conduction and evaporation. Since the contact area between the skin and solid objects is usually very small, conduction is negligible and it is discounted except in the case of body cooling garments. For normal body function, heat exchange between the body and its environment should be balanced.

The exchange of heat between the body and its environment is described by the heat balance equation. All living organisms generate heat, and it is, therefore, necessary to incorporate metabolic heat into the equation. The consumption of oxygen 1 L/min corresponds to 4825 C/min, 20,197 kJ/min, or 337 W. The equation is:

$$H \pm K \pm C \pm R - E = 0$$

The equation representing steady state thermal balance where K represents conduction, C represents convection, R represents radiation and E represents evaporation. The symbol M is often used to denote total energy liberation in the body. To obtain the value for heat production, it is necessary to subtract mechanical work rate (W). The term M – W may, therefore replace H. In practice, the heat balance equation can be 'manipulated', with due cognizance of physiological limitations, to describe various combinations of metabolic and environmental conditions under which thermal equilibrium exists or could be achieved. This approach presumes a comprehensive knowledge of all of the relevant variables and assumptions. Within the scope of occupational health, this approach falls in the province of the specialist biophysicist or bioengineer.

Physiological Responses and Adaptation to Work in Heat

Human thermoregulation remains the subject of intensive research. It is a practical importance to appreciate that it is necessary to postulate a 'set-point hypothesis' for the control of body temperature but, rather, that body temperature will be regulated (within limits) at the lowest level consonant with the maintenance of homeostasis. Although thermal balance can be achieved solely by physical means, at least in theory, physiologic control is invoked whenever thermal balance is challenged. In essence, physiologic thermoregulation is achieved through three main factors: (1) An elevation in metabolic rate to counter heat loss during cold exposure, (2) vasomotor adjustments that either facilitate (dermal vasodilatation) or restrict (dermal vasoconstriction) heat loss from the body, and (3) sweating, which promotes evaporative heat loss.

Comfort Zones

Comfortable thermal conditions are those under which a person can maintain normal balance between production and loss of heat, at normal body temperature and without sweating. Comfort zones evaluation is done with two factors: (1) Effective temperature (ET) or corrected effective temperature (CET): Effective Temperature (ET) is an arbitrary index which combines into a single value of the effect of temperature, humidity and movement of the internal air on the sensation of warmth or cold felt by the human body. Corrected Effective Temperature (CET) is an improvement over the effectiveness temperature index. Instead of the dry bulb temperature, the reading of the globe thermometer is used to allow for radiant heat. That is, the CET scales deal with all the four factors namely air temperature, air velocity, relative humidity and mean radiant heat. Whenever a source of radiation is present, it is preferable to measure CET.

Zone	*Corrected effective temperature (CET)*
Pleasant and cool	69°F (20°C)
Comfortable and cool	69–76°F (20–25°C)
Comfortable	77–80°F (25–27°C)
Hot and uncomfortable	81–82°F (27–28°C)
Extremely hot	83°F+ (28°C+)
Intolerable hot	86°F+ (30°C+)

Predicted four-hour sweat rate (P_4SR): P_4SR can be obtained from any combination of dry and wet bulb temperature of the air, mean radiant air temperature, and air velocity, under different work intensities. McArdle and associates have put P_4SR value of 3 as upper limit of comfort zone.

Zone	*P_4SR*
Comfort zone	1–3 Litres
Just tolerable	3–4.5 Litres
Intolerable	4.5 Litres+

Heat Stress

Heat stress is the amount of heat that is to be eliminated from human body to remain the body in thermal equilibrium and measured as metabolic heat load and heat loss or gain through the process of convection, conduction, radiation and evaporation. The equation of store (S) heat of the body due to heat stress is:

$$M + (R + C + K) - E = S$$

The American Conference of Governmental Industrial Hygienists (in 1992) states that workers should not be permitted to work when their deep body temperature exceeds 30°C (100.4°F). Heat is a measure of energy in terms of quantity. A calorie is the amount of heat required to raise 1 gram of water 1°C (based on a standard temperature of 16.5 to 17.5°C).

Conduction (K) is the transfer of heat between materials that contact each other. Heat passes from the warmer material to the cooler material. For example, a worker's skin can transfer heat to a contacting surface if that surface is cooler and vice versa.

Convection (C) is the transfer of heat in a moving fluid. Air flowing past the body can cool the body if the air temperature is cool. On the other hand, air that exceeds 35°C (95°F) can increase the heat load on the body.

Radiation (R) is the transfer of heat energy through space. A worker whose body temperature is greater than the temperature of the surrounding surfaces radiates heat to these surfaces. Hot surfaces and infrared light sources radiate heat that can increase the body's heat load.

Evaporation (E) cooling takes place when sweat evaporates from the skin. High humidity reduces the rate of evaporation and thus reduces the effectiveness of the body's primary cooling mechanism.

Metabolic heat (M) is always positive which a byproduct of the body's activity.

Globe temperature is the temperature inside a blackened, hallow, and thin copper glob.

Natural wet bulb (NWB) temperature is measured by exposing a wet sensor, such as a wet cotton wick fitted over the bulb of a thermometer, to the effects of evaporation and convection. The term natural refers to the movement of air around the sensor.

Dry bulb (DB) temperature is measured by a thermal sensor, such as an ordinary mercury-in-glass thermometer, that is shielded from direct radiant energy sources.

Causal Factors of Heat Stress

1. Age, weight, degree of physical fitness, degree of acclimatization, metabolism, use of alcohol or drugs, and a variety of medical conditions such as hypertension all effect a person's sensitivity to heat to develop heat stress. However, even the type of clothing worn must be considered. Prior heat injury predisposes an individual to additional injury.
2. It is difficult to predict just who will be affected and when, because individual susceptibility varies. In addition, environmental factors including air temperature, radiant heat, air movement conduction, and relative humidity, all affect an individual's response to heat.

Heat Stress Criteria

The most practical and accurate criteria of physiological heat stress are body core (rectal) temperature and heart rate. Qualitatively both show a positive relationship to increasing work rates and heart loads (including combination therefore), although the respective response pattern may differ significantly. For example, during prolonged work (54 W for 4 hours) in hot humid conditions (33.2°C dry-bulb, 31.7°C wet-bulb), the increase in heart rate relative to rectal temperature for unacclimatized persons exceeds the value for acclimatized persons. This implies that while dual upper limits certainly enhance safety precautions, care should be exercised to ensure that they are realistically adjusted to one another for set conditions.

For general industry, there is a consensus that deep body temperature should not be permitted to rise above 38°C, and accordingly this standard has been built into, perhaps, the most universally accepted heat stress index—the WBGT index. While the choice of 38°C may seem to be too conservative, especially to exercise physiologists, it should be borne in mind that it must serve for a considerable cross-section of the workforce, workers who differ in age, sex, inherent work capacity, and fitness. On the other hand, for a youthful, healthy, all-male group in the South Africa mining industry, this value may be set at a much higher level, 39.5°C. This value is based on an analysis of Wyndham and co-workers' findings, which clearly indicate that sweat production and physiologic heat conductance become 'saturated' when rectal temperature approaches 39.5°C; for example, sweat rate and conductance are at a maximum. Obviously, an upper-limit rectal temperature criterion of 39.5°C can only be applied under conditions of strict individual supervision, where rectal temperature and heart rate are

constantly monitored; it should under no circumstances be incorporated into 'indices' based on environmental conditions and eliminated work rates.

Heart rate is not only related to work load but also reflects the influences of other factors, such as the environmental heat load. For sustained work (e.g. an 8-hours shift), there is general consensus that work rate should not tax more than 40% of an individual's maximum work capacity (Vo_2MAX). For untrained men aged 20 to 60 years, an analysis of Shephard's data reveals an average Vo_2MAX of 3.01 L/min (range 2.27 to 3.90 L/min). This suggests that, an average, sustained work should not be performed at rates exceeding an oxygen consumption of 1.2 L/min, with a range of 0.9 to 1.6 L/min. Corresponding heart rates are of the order of 110 to 130 beats/min, the implication being that 110 represents an upper limit. In as much as Vo_2MAX is systematically eroded with increasing heat loads, it follows that lower work rates are indicated to conform to this limit.

General relation between work rate, heart beat rate and oxygen consumption

Category	*O_2 consumption (L/min)*	*Heart beat rate (beat/min)*
Light	0.5–1.0	75–100
Moderate	1.0–1.5	100–125
Heavy	1.5–2.0	125–150
Very heavy	2.0–2.5	150–175
Extremely heavy	2.5	175

With training and heat acclimatization, when the individual is well capable of sustained work at much higher fractions of Vo_2MAX, these restrictions are no longer relevant and rectal temperatures approaching 38.5°C and heart rates of up to 135 beats/min are well-tolerated.

From the above it is evident that considerable benefit could be derived in any given work situation, without sacrificing safety, from individual monitoring. Unfortunately, in most instances, individual monitoring is impractical, and consequently, indices of the environmental heat load that correlate most closely with the physiologic response to work in heat are applied. Of these, the WBGT index is internationally recognized, especially where physical work is involved, while the effective temperature (ET) or corrected effective temperature (CET) is intended as an index of comfort. However, irrespective of the particular index or its degree of sophistication, they all suffer a common shortcoming: Metabolic rate is either omitted or estimated with no consideration of individual reaction. Of necessity, therefore, such indices are inaccurate and have to err on the side of conservatism.

Heat Stress Investigation Guidelines

These guidelines for evaluating employee heat stress approximate those found in the 1992–93 ACGIH publication, Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices.

A. *Employer and employee interviews*: The inspector will review the OSHA 200 Log and, if possible, the OSHA 101 forms for indications of prior heat stress problems. Following are some questions for employer interviews: What type of action, if any, has the employer taken to prevent heat stress problems? What are the potential sources of heat? What employee complaints have been made? Following are some questions for employee interviews: Which heat stress problems have been experienced? What type of action has the employee taken to minimize heat stress? What is the employer's involvement, i.e. does employee training include information of heat stress?

B. *Walk-around inspection*: During the walk-around inspection, the investigator will determine building and operation characteristics; determine whether engineering controls are functioning properly; verify information obtained from the employer and employee

interviews; and perform temperature measurements and make other determinations to identify potential sources of heat stress. Investigators may wish to discuss any operations that have the potential to cause heat stress with engineers and other knowledgeable personnel. The walk-around inspection should cover all affected areas. Heat source, such as furnaces, ovens, and boilers, and relative heat load per employee should be noted.

C. *Workload assessment*: Under conditions of high temperature and heavy workload, one should determine the workload category of each job. Workload category is determined by averaging metabolic rates for the tasks and then ranking them:
   1. Light work: Up to 200 kcal/hour
   2. Medium work: 200–350 kcal/hour
   3. Heavy work: 350–500 kcal/hour

Cool rest area: Where heat conditions in the rest area are different from those in the work area, the metabolic rate (M) should be calculated using a time-weighed average, as follows:

$$\text{Average } M = \frac{(M_1)(t_1) + (M_2) + t(t_2) \ldots . (M_n) + (t_n)}{(t_1) + (t_2) \ldots . (t_n)}$$

where M = metabolic rate
t = time in minutes

In some cases, a videotape is helpful in evaluating work practices and metabolic load.

Activity examples:

- Light hand-work: Writing, hand knitting.
- Heavy hand-work: Typewriting.
- Heavy work with one arm: Hammering in nails.
- Light work with two arms: Filing metal, planning wood, raking the garden.
- Moderate work with the body: Cleaning of floor, beating a carpet.
- Heavy work with the body: Railroad track laying, digging, barking trees.

Sample calculation: Assembly line work using a heavy hand tool

- Walking along 2.0 kcal/min
- Intermediate value between heavy work with two arms and light work with the body 3.0 kcal/min
- Add for basal metabolism 1.0 kcal/min

Total 6.0 kcal/min

Assessment of work: Body position and movement:

- Add for basal metabolism 1.0 kcal/min
- Sitting 0.3 kcal/min
- Standing 0.6 kcal.min
- Walking 2.0–3.0 kcal/min
- Walking uphill: add 0.8 kcal/min every meter (yard) rise

Type of	*Type*	*Average kcal/min*	*Range kcal/min*
Hand work	Light	0.4	0.2–1.2
	Heavy	0.9	
Work:			
One arm	Light	1.0	0.7–2.5
	Heavy	1.7	
Both arm	Light	1.5	1.0–3.5
	Heavy	2.5	
Whole body	Light	3.5	2.5–15.0
	Moderate	5.0	
	Heavy	7.0	
	Very heavy	9.0	

Heat Stress Sampling Methods

1. *Body temperature measurements:* Instruments are available to estimate deep body temperature by measuring the temperature in the ear canal or on the skin or oral temperature or rectal temperature.
2. *Environmental measurement:* Environmental heat measurements should be made at, or as close as possible to, the specific work area where the worker is exposed. When a worker is not continuously exposed in a single hot area but moves between two or more areas having different levels of environmental heat, or when the environmental heat varies substantially at a single hot area, environmental heat exposures should be measured for each area and for each level

of environmental heat to which employees are exposed.

3. *Wet bulb globe temperature index:* ISO 7243 prescribes a standard method for the estimation of heat stress using three temperature measurements: Those of a standard dry bulb, a wet bulb and one inside a blackened globe of 150 mm diameter. The three temperatures are combined into the WBGT index using the following equation:

$$\text{Outdoor WBGT} = 0.7T_{\text{wet bulb}} + 0.2T_{\text{globe}} + 0.1T_{\text{dry bulb}}$$

Under this ISO standard, it is permissible to ignore the dry bulb temperature if there is little difference between it and that of the globe (e.g. indoors with title radiant heat load), in which case the equation becomes:

$$\text{Indoor WBGT} = 0.7\text{T}_{\text{wet bulb}} + 0.3\text{T}_{\text{globe}}$$

Wet bulb globe temperature (WBGT) should be calculated using the appropriate formula. The WBGT for continuous all-day or several hour exposures should be averaged over a 60-minute period. Intermittent exposures should be averaged over a 120 minute period. These averages should be calculated using the following formula:

$$\text{Average WBGT} = \frac{(WBGT_1)(t_1) + (WBGT_2)(t_2) + ... + (WBGT_n)(t_n)}{(t_1) + (t_2) + ... + (t_n)}$$

Portable heat stress meters or monitors are used to measure heat conditions. These instruments can calculate both the indoor and outdoor WBGT index according to established ACGIH Threshold Limit Value equations. With this information and information on the type of work being performed, heat stress meters can determine how long a person can safely work or remain in a particular hot environment.

These TLVs are based on the assumption that nearly all acclimatized, fully clothed workers with adequate water and salt intake should be able to function effectively under the given working conditions without exceeding a deep body temperature of 38°C (100.4°F). They are also based on the assumption that the WBGT of the resting place is the same or very close to that of the workplace. Where the WBGT of the work area is different from that of the rest area, a time-weighted average should be used. These TLVs apply to physically fit and acclimatized individuals wearing light summer clothing. If heavier clothing that impedes sweat or has a higher insulation value is required, the permissible heat exposure TLVs must be reduced by the correction as per the ACGIH 1992–93 Threshold Limits Values for Chemical Substances and Physical Agents and Biological Exposure Indices (1992).

WBGT correction factors in °C

Clothing type	*Clo value*	*WBGT correction*
Summer lightweight working clothing	0.6	0
Cotton overalls	1.0	–2
Winter work clothing	1.4	–4
Water barrier, permeable	1.2	–6

Clo: insulation value of clothing. One clo = 5.55 kcal/hr of heat exchange by radiation and convection for (Degree) °C difference in temperature between the skin and the adjusted dry bulb temperature.

Permissible heat exposure threshold limit value (ACGIH)

Work/Rest regimen	*Workload (WBGT)*		
	Light	*Moderate*	*Heavy*
Continuous work	30.0°C (86°F)	26.7°C (80°F)	25.0°C (77°F)
75% Work, 25% rest, each hour	30.6°C (87°F)	28.0°C (82°F)	25.9°C (78°F)
50% Work, 50% rest, each hour	31.4°C (89°F)	29.4°C (85°F)	27.9°C (82°F)
25% Work, 75% rest, each hour	32.20°C (90°F)	31.1°C (88°F)	30.0°C (86°F)

Work rate categories				
Category	*Metabolic rate*			
	O_2L/min	*kcal/hr*	*kJ/hr*	*W/hr*
Light	≤0.60	≤200	≤840	≤233
Moderate	0.70–1.0	201–350	841–1465	234–407
Severe	>1.0	>350	>1465	>407

Other thermal stress indices: (a) Effective temperature (ET): Effective temperature is an index that combines into a single value of the subjective thermal sensation resulting from air temperature (or globe temperature when radiant heat exceeds air temperature by more than 1°C), humidity, air movement. The index does not take into account metabolic rates other than for light or sedentary work, and its use is, therefore, limited to an index of comfort or as a guide to analyze productivity. In summer, the maximum number of people should be comfortable at an ET of 21.7°C (range: 18–26°C), in winter at 20°C (range: 15–23°C). These values are subject to variation in different geographic regions. Although useful, the index does have a number of shortcomings: (1) It gives insufficient weight to the detrimental effect of air movement below 0.5 m/sec in hot, humid conditions; (2) it exaggerates the effect of high dry-bulb temperature at air movements in the range of 0.5 to 1.5 m/sec during physical work, and it underestimates the harmful effects of air movement in excess of 1.5 m/sec at dry-bulb temperature of 49°C and higher; and 3 environmental conditions inducing the same physiologic stress, in terms of rectal temperature, heart rate, sweat rate, and tolerance, do not constitute the same ET, especially in severe heat stress. ET remains a useful measurement technique in mines and other places where humidity is high and radiant heat is low. When ET is corrected by adding radiant heat where applicable, it is known as corrected effective temperature (CET). (b) The heat stress index (HSI): HSI was developed by Belding and Hatch in 1965. It represents the percentage of heat storage capacity of an average man. HSI are as follows:

HSI	*Interpretation*
0	No thermal stress
10–30	Moderate-to-mild heat strain
40–60	Severe heat strain
70–90	Very severe heat strain
100	Upper limit of heat tolerance

Although the HSI considers all environmental factors and work rate, it is not completely satisfactory for determining an individual worker's heat stress and is also difficult to use. (c) Psychrometric chart: In many situations, radiant heat loads are fairly small in comparison to the heat exchange by convection and evaporation. The psychrometric chart (ironically, derived from the Greek psychron meaning cold) is a clear graphical way of understanding the effect of changing humidity and temperatures alone. Use of the chart requires a measurement of the water content of the air and the dry bulb temperature. Limits may be defined on the chart appropriate to a given group of individuals for specified activities and clothing, thus allowing the user to make recommendation as to how their physiological requirements may be met. The chart may also be used to understand the relative effects of lowering air temperature and humidity when trying to control an environment. (d) P_4SR: A potentially very attractive approach to the assessment of heat stress is to attempt to relate it to the amount of evaporative heat loss required for thermal balance; this was originally incorporated into a 'predicted 4-hour sweat rate' (P_4SR), but has more recently evolved into the required sweat rate and 'predicted heat strain', as defined in ISO 7933. Measurement essentially consists of a semi-empirical solution of the equation of heat balance, to arrive at an estimate of the sweat rate required for the maintenance of thermal equilibrium. It is thus considerably more complex than the WBGT index and more suited to experimental investigations rather than routine monitoring. The latest studies confirmed its efficacy in predicting average

and limits to exposure, although modifications have been required to the calculation of the evaporative efficacy of sweating. (e) More modern thermal stress may need to be assessed by more complex methods than the simple WBGT index, as the transfer of heat by each physical mode is usually more sensitive to subtle changes. Detail accounts of the 'predicted mean vote' (PMV) and 'predicted percentage of dissatisfied' (PPD) indices are given in ISO 7730, based on measurement of air temperature, mean radiant temperature, humidity, air velocity, metabolic rate and clothing insulation. Indeed, in any situation in which clothed individuals are being assessed, reference will need to be made to the estimation of the thermal insulation and assistance to evaporative heat loss imposed by clothing; ISO 9920 provides a sound practical basis for this.

Assessment of Heat Strain

'Stress' is the force applied to an object/subject, whilst 'strain' is the response within the object/subject to the applied stress.

Heat strain can be defined as the physiological and pathological changes of the body due to heat stress. The principal measurement of heat strain, like that of cold, is core body temperature. However, core temperature can rise rapidly in the heat and the fact that rectal temperature changes later and more slowly than other sites make it less suitable for safety purposes. Accordingly, it is more common to estimate deep body temperature in the heat from a tympanic membrane or auditory canal reading; this has the added advantage that this site should be a good indicator of the temperature of the brain, which is not only the central temperature controller, but is also one of the more critical organs in heat illness. Unfortunately, the otherwise convenient infrared tympanic membrane thermometers that have become so popular in general clinical practice are again a potential source of error, and should not be trusted: A thermistor or thermocouple needs to be placed just short of the tympanic membrane, and the auditory meatus occluded and carefully insulated if the temperature recorded is to be a reliable estimate of core temperature.

Three other variables are commonly recorded in heat studies: Heart rate is the simplest of all to measure, loss of body mass due to sweating is also very easily determined, whilst skin temperature is sometimes of value if judiciously interpreted.

Heart rate is well-correlated with heat strain, provided that changes due to exercise are taken into consideration. It is often used, in conjunction with aural temperature, in establishing criteria for the withdrawal of people exposed to heat loads. Simple estimate of body weight loss is a useful measure of fluid loss in laboratory conditions. Skin temperature can prove misleading. As they fall if there is good evaporation from the skin, but climb close to core if evaporation is limited or absent. These methods are described in ISO 9886.

Heat Disorders and Health Effects

1. **Heat exhaustion:** Heat exhaustion may be seen in military personnel during fitness training and moderately well-trained athlete competing in a marathon during the warmer months in a temperate country as well as in groups of manual workers with moderate environmental heat loads. Heat exhaustion is attributed to an inability of the circulation to meet simultaneously the demands of thermoregulation (i.e. by affecting a vast flow of blood to the skin) and those of vital organs such as the brain and active skeletal muscle. Chronic heat exhaustion, in contrast to the common acute version, may have its origin in salt depletion, for example, dietary imbalance.

   Heat exhaustion is more likely to occur during the un-acclimatized state and in individuals with some form of circulatory insufficiency. Obviously, the condition is aggravated by dehydration. However, it

may also occur completely independently of dehydration as a result of an improper redistribution of the circulation. Irrespective of the precise origin, it follows that the trigger mechanism is an effective reduction in circulating blood volume. The first sign of impending heat exhaustion is usually hyperventilation, in someone who appears ill at ease with the exercise that they are performing. Classical signs and symptoms from the disturbance of the body's acid–base balance and the calcium–phosphate ratio in the blood flow, with dizziness, nausea, paraesthesia in the peripheries and around the mouth, progressing to confusion, collapse, vomiting and even seizures. Further investigation may reveal high heart rates and low arterial blood pressure, the signs of circulatory shock. Core temperature at this stage is below 40°C, and usually between 37°C and 39°C. Early cases respond rapidly to rest in a recumbent position, re-breathing from a paper bag to restored end-tidal CO_2 levels and simple cooling. Fluids may also be beneficial and should always be given by appropriate means. If these patients are allowed or even encountered to continue exercising, they rapidly develop frank heat stroke.

2. **Heat cramps:** Heat cramps are usually caused by performing hard physical labour in a hot environment. There are painful and spasmodic contractions of the skeletal muscles. These cramps have been attributed to an electrolyte imbalance caused by sweating. It is important to understand that cramps can be caused by both too much and too little salt. Cramps appear to be caused by the lack of water replenishment. Because sweat is a hypotonic solution (±0.3% NaCl), excess salt can build-up in the body if the water lost through sweating is not replaced. Thirst cannot be relied on as a guide to the need for water; instead, water must be taken every 15–20 minutes in hot environments. Classical salt depletion, due to salt loss in sweat while on a low salt diet, with adequate water intake, is now rarely seen. The classical accompaniment of cramp (e.g. Miner's cramp) only occurs in association with hard exercise. Plasma sodium only falls in more severe cases, with milder cases showing a fall in extracellular fluid volume with normal plasma sodium. In the worst cases, vigorous treatment with oral or intravenous saline may be required. Subacute and other variants of this condition sometime attributed to different combination of water and electrolyte depletion. They are best treated by cooling, rest, rehydration and the restoration of a normal diet. The blind administration of salt, even with the copious quantities of fluid that are required, is potentially dangerous and should be avoided. Recent studies have shown that drinking commercially available carbohydrate–electrolyte replacement liquids is effective in minimizing physiological disturbances during recovery.

   One possible danger is that of hyperkalaemia; many young people often rehydrate following exercise in the heat using some form of orange juice, or a 'spot rehydration' fluid, which may be high in potassium. They should be advised to ensure that their rehydration fluid is well diluted with water.

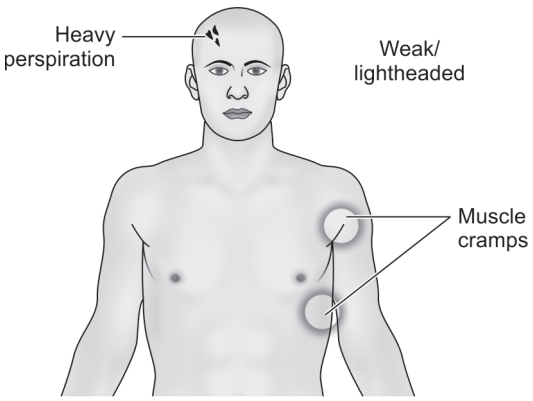


Fig. 8.1: Heat cramps

3. *Skin disorders: Prickly heat or miliaria is an acute, inflammatory disease of the skin.* Perhaps the most common of the 'mild' heat disorders is prickly heat or miliaria rubra. It occurs, especially in the summer, in the tropics, and following prolonged sweating. The pathology is well known: Sweat gland ducts become blocked by a plug of keratinized cells, and the accumulating sweat is forced through the wall of the duct into the surrounding tissues; an inflammatory reaction ensues due to irritants and infection. There are three forms of miliaria: Miliaria crystallina, miliaria rubra and miliaria profunda. As the site of duct obstruction becomes deeper in the skin, the severity increases and presentation varies like vesicles, erythema, desquamation, macules. There are three possible causes are listed: (1) excessive wasting away of the natural skin oil, (2) infection, and (3) electrolyte imbalance and the effect thereof on sweat composition. Prickly heat is manifested as red papules and usually appears in areas where the clothing is restricted which give rise to a prickling sensation. Treatment is symptomatic and preventive, but topical applications of corticosteroids may be used in some cases. Environmental controls and good personal hygiene are most effective. *Erythema ab igne* is characterized by the appearance of hyperkeratotic nodules following direct contact with heat that is insufficient to cause a burn. *Intertrigo* results from excessive sweating and often is seen in obese individuals. Skin in the body folds (the groin and axillas) is erythematous and macerated. *Heat articaria* (cholinergic articaria) can be localized or generalized and is characterized by the presence of wheals with surrounding erythema (hives). Treatment for these disorders consists of reduction or removal of heat exposure, reduction of sweating, and control of symptoms. Antihistamines may help to relieve pruritus in patients with urticaria. Corticosteroids are not beneficial.

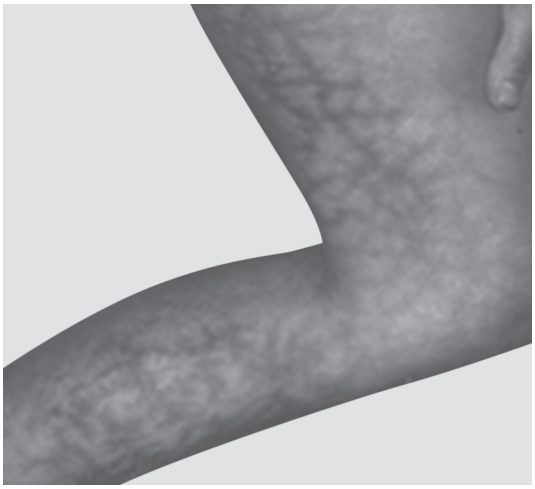


Fig. 8.2: Erythema ab-igne

4. *Heat syncope:* It is also known as heat collapse (fainting). This is the common ill-effect of heat. In its milder form, the person standing in the sun becomes pale, his blood pressure falls and he collapses suddenly. This reaction is similar to that of heat exhaustion and does not affect the body's heat balance; therefore, there is practically no rise in body temperature. The condition results from pooling blood in lower limb due to dilatation of blood vessels (vasodilatation), with the result that the amount of blood returning to the heart is reduced, which in turn is responsible for lowering of blood pressure and lack of blood supply to the brain. The onset of heat syncope is rapid and unpredictable. This condition is quite common among soldiers when they are standing for parades in the sun. Treatment is quite simple. The patient should be made to lie in the shade with the head slightly down; recovery usually comes within 5 to 10 minutes. To prevent heat syncope, the worker should gradually become acclimatized to the hot environment.

5. *Heat fatigue:* A factor that predisposes an individual to heat fatigue is lack of acclimatization. The use of programme of acclimatization and training for work in hot environments is advisable. The signs and symptoms of heat fatigue include impaired performance of skilled sensorimotor, mental, or vigilance jobs. There is no treatment for heat fatigue except to remove the heat stress before a more serious heat-related condition develops.
6. *Heat stroke:* Heat stroke is regarded as one of the few true medical emergencies, and if effective treatment is not instituted promptly, it carries a mortality rate of up to 80%. Earlier heat stroke was defined as a "disorder of thermoregulation characterized by the total absence of sweating, body temperature in excess of 41.1°C (106°F) and severe disturbances of brain function". A major objection of such definition is that sweating cessation is not always a cardinal sign, since heat stroke may occur not only from failure of the thermoregulatory system, as a result of impaired central nervous system function, but also from overloading its capacity. By current definition, heat stroke represents a condition in which elevated body temperatures are causally related to tissue damage, often of an irreversible nature. Although many tissues are damaged in heat stroke, the patient's outcome depends mainly on the degree of injury to the nervous system, kidneys and liver, the latter two organs being damaged almost irreversibly. The accurate diagnosis of heat stroke, by implication, therefore, rests solely on parameters of tissue damage, the most practical being the assay of tissue enzymes in serum.

Those working in very hot surroundings, particularly when evaporative cooling is ineffective due to high humidity, may undergo a rapid rise in core temperature, to the point at which thermoregulation fails. The primary signs and symptoms of heat stroke are: Confusion, delirium, irrational behavior. The classic victim of heat stroke has stopped sweating and is dry to the touch. They may still maintain peripheral circulation and thus be red, or may have undergone circulatory collapse, in which case their skin colour is not diagnostic. They are very hot, semiconscious or comatose, and prone to convulsions, cardiac and respiratory arrest. The onset of this dramatic illness may have been very rapid, although many will have shown evidence of earlier heat exhaustion.

An elevation of serum enzyme levels is a consistent finding in heat stroke of especial prognostic significance is the elevation of aspartate aminotransferase (AST) in that it is regarded as an indicator of the severity of tissue damage, while an elevation in creatine kinase (CK) in cerebrospinal fluid may provide a good index of neurologic damage. Furthermore, AST and lactate dehydrogenase (LD) levels are almost invariably increased in heat stroke. Diagnostic procedures regarding heat stroke call for serum enzyme assays of LD, CK, AST, and alanine aminotransferase (ALT) on admission and 48 hours following admission.

Cooling is best achieved in the field by stripping the patient in the shade, drenching with tepid water and fanning if there is little natural wind. Cold water or ice is less useful in adults because of the intense vasoconstriction which it can precipitate, which limits heat transfer from the core to the skin; however, there are recent claims that ice-water baths may still produce the most rapid fall in core temperature even in adults, as they do in infants with their much greater surface area to volume ratio. Alternating litres of isotonic dextrose and saline intravenously are the best means of fluid administration, but cannot be a substitute for immediate and effective cooling. The precise quantity given may be based on an estimate of fluid loss, but in unconscious cases it is probably best to give the first litre over 15 minutes or less, thereafter reducing the rate of infusion according to

clinical indications and the perceived risk of hypervolaemia leading to pulmonary oedema. Such fluid should not be warmed, but neither should they be cooled below 15°C. Elevation of the legs to assist venous return, administration of oxygen and possibly sedative, are of value. However, vasodilators and platelet inhibitors, such as aspirin, should be avoided. Rapid but careful evacuation to an intensive care unit is essential, where urinary output, blood biochemistry and direct measurement of the central venous pressure can be monitored closely. Cooling is normally discontinued when the rectal or aural temperature has reached 38ºC. The rectal or aural temperature should be monitored continuously, both to monitor the efficacy of hyperthermia treatment and to guard against the development of clinically significant hypothermia, which can occur if cooling is continued too long. Further treatment is supportive and directed towards the many potential complications of hyperthermia. Hypovolaemia, hyperkalaemia, rhabdomyolysis, hypocalcaemia and bleeding diathesis may require intensive supportive treatment. The patient should be kept in bed for several days until the temperature control becomes stable. If core temperature rises to 45ºC, irreversible heat denaturation of protein causes multiple organ failure or disseminated intravascular coagulation. If it is not immediately fatal, renal dialysis may be necessary during recovery.

The host factors reported to increase risk of heatstroke are: Unacclimatization, obesity, lack of physical fitness, fatigue, lack of sleep, dehydration, febrile illness, acute and convalescent infections, reactions to immunizations, conditions affecting sweating, skin disorders (heat rash, sunburn), consumption of alcohol, drug (barbiturate), past history of heat injury, past history of living in climatic areas with more atmospheric cooling power, chronic diseases (diabetes, cardiovascular diseases), lesions of the hypothalamus, brainstem, and cervical part of spinal cord, following certain surgical operations, recent intake of food, sustain output of muscular metabolic heat, and increased susceptibility due to biological variability.

The Effect of Heat on Performance and Productivity

Reconciling of subjective and objective is not easier for the possible effects of heat than were noted for cold. Ramsey has reviewed more than 150 studies that attempted to find differences in 'perceptual motor performance' in the heat and commented on the remarkable lack of objective evidence of dominant effects. It appears that there is no significant detectable impairment in the performance of the great majority of tasks until the level of heat stress reaches a WBGT index of approximately 30ºC. This is broadly in accordance with the criteria originally laid down by NIOSH in their original recommended exposure limit (REL). Paradoxically, when raised 14 years later, NIOSH omitted this REL because of lack of supporting evidence.

A few studies cast interesting and potentially different light on the area. There is much stronger support for adverse effects in circumstances in which core temperature has risen; when core temperature is 38°C or greater a prominent effect is an increase in irritability independent of the rise in subjective discomfort.

Perhaps the most useful tool (beyond tenuous attempts to relate accident statistics to heat stress or strain) in the practical examination of performance effects is the expression of subjective judgment scales. ISO 10551 lays down a standard approach to these, which allows inclusion of comfort, acceptability and tolerance in the assessment of thermal environment.

The competitive demands for adequate perfusion of active skeletal muscle and the skin impose a limit on work capacity, overall performance, and, ultimately, productivity. Irrespective of the complexity of the task, human performance declines significantly in the effective temperature (ET) range of 27° to

30°C. For strenuous physical work in gold mines, the decline is 5% at 29°C, 10% at 30°C, 17% at 31.5°C, and 30% at 32.5°C ET. This study highlights the progressive decline in performance associated with increasing wet-bulb temperatures.

Control Measures

Ventilation, air cooling, fans, shielding, and insulation are the five major types of engineering controls used to reduce heat stress in hot work environments. Heat reduction can also be achieved by using power assists and tools that reduce the physical demands placed on a worker.

However, for this approach to be successful, the metabolic effort required for the worker to use or operate these devices must be less than the effort required without them. Another method is to reduce the effort necessary to operate power assistants. The worker should be allowed to take frequent rest breaks in a cooler environment.

Acclimatization: The human body can adapt to heat exposure to some extent. This physiological adaptation is called acclimatization. After a period of acclimatization, the same activity will produce fewer cardiovascular demands. The worker will sweat more efficiently (causing better evaporative cooling), and thus will more easily be able to maintain normal body temperature. Cardiovascular changes sustain peripheral vasodilatation and the blood flow required by the active sweat gland. Salt of the body is retained by the kidneys and salt content of the sweat starts to decline, so that increased sweating does not result in severe sodium deficiency. This is achieved by an early peak in rennin, followed by an increased in aldosterone levels.

The ability to perform work that is easily done in cool environments is impaired because the initial demands of thermoregulation and skeletal muscle activity are competitive, and the resulting strain is manifested in high heart rates. If these

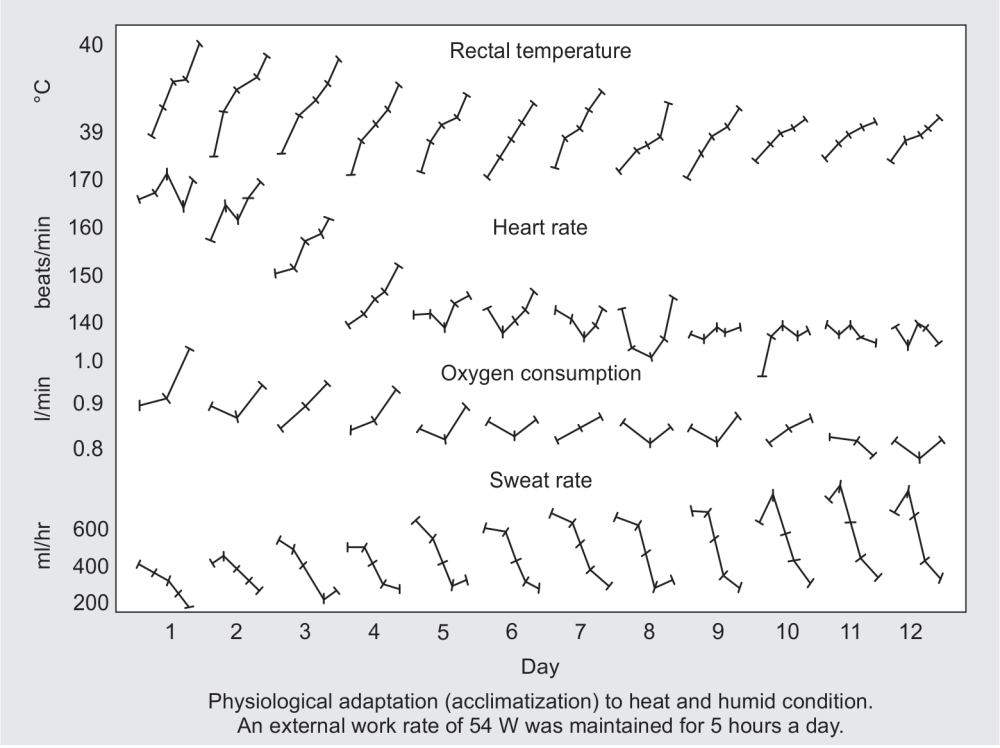


Fig. 8.3: Acclimatization

conditions (work in heat) last for several days, there is a gradual return of the ability to work with little or no discomfort—heat acclimatization has taken place.

De-acclimatization can occur surprisingly rapidly, after just a few days of withdrawal from the heat stress. Those who spend a period working in air-conditioned comfort, or who return to more temperate climates for a holiday, therefore need to undergo supervised re-acclimatization on their return to heat exposure. This introduces a dilemma in choosing optimal living and working conditions in hot climates; while many find it more comfortable and sleep better in conditioned accommodation, more constant exposure to heat stress will maintain a better level of acclimatization.

A proper designed and applied acclimatization programme decreases the risk of heat-related illness. Such a programme basically involves exposing employees to work in a hot environment for progressively longer periods. NIOSH (1986) says that, for workers who have had previous experience in jobs where heat levels are high enough to produce heat stress, the regimen should be 50% exposure on day one, 60% on day two, 80% on day three and 100% on day four. For new workers who will be similarly exposed, the regimen should be 20% one day one, with a 20% increase in exposure each additional day.

Fluid replacement: Cool (50–60°F) water or any cool liquid (except alcoholic beverages) should be made available to workers to encourage them to drink small amounts frequently, e.g. one cup every 20 minutes. Ample supply of liquid should be placed closed to the work area. Although some commercial replacement drinks contain salt, this is not necessary for acclimatized individuals because most people add enough salt to their summer diets.

The aim of drinking during exposure to heat stress should be to replace fluid and electrolyte losses in sweat. Even when acclimatized and fit, few humans are capable of losing more than 2 litres an hour in sweat, so drinking should never exceed that rate. Over a typical extended working day, a ceiling of 10–12 liters of fluid intake is advisable.

Fluid replacement strategies are primarily designed to counter dehydration (and possibly electrolyte loss) induced by profuse, sustained sweating. Although the fundamental approach of replacing exactly what is being lost at the same rate is quite simple in theory, the maintenance of an optimum state of hydration often presents practical problems:

1. Drinking water according to the dictates of thirst is insufficient to prevent a voluntary dehydration up to 1% of body mass.
2. Upon drinking water, a person's thirst is alleviated well before the fluid deficit is recovered, the attenuation of drinking subjectively being attributed to stomach fullness.
3. Once dehydration is set in, the subsequent rate of water absorption from the gut will be reduced as a result of compensatory splanchnic vasoconstriction. This reflex, by which a redistribution of the cardiac output is achieved, occurs at dehydration levels approximating 1.5% of body mass.
4. Sweat is hypotonic fluid with an electrolyte content varying between 0.1 to 0.3 g%. The most important constituent is sodium chloride (salt), which accounts for about 80% of the tonicity of sweat. On a balanced diet, or in the short term (hours), the threat to continued wellbeing does not reside in salt depletion but, rather, in dehydration.

These considerations suggest that optimum hydration is best achieved by drinking relatively small amounts of water at relatively short intervals.

Heat produced by the body and the environmental heat together determine the total heat load. Therefore, if work is to be performed under hot environmental conditions,

the work load category of each job shall be established and the heat exposure limit pertinent to the workload evaluated against the applicable standard in order to protect the worker against exposure beyond the permissible limit. The workload category may be established by ranking each job into light, medium and heavy categories on the basis of type of operation. Where the workload is ranked into one of said three categories, that is, (1) light work (up to 200 kcal/hr), for example, sitting or standing to control machine, performing light hand or arm work, (2) moderate work (200 to 350 kcal/hr): for example, walking about with moderate lifting and pushing, and (3) heavy work (350 to 500 kcal/hr), for example, pick and shovel work.

Workers should be encouraged to salt their food abundantly during the hot season and particularly during hot spells. If the workers are unacclimatized, salted drinking water shall be made available in a concentration of 0.1% (1g NaCl to 1.0 liter). The added salt shall be completely dissolved before the water is distributed, and the water shall be kept reasonably cool.

Engineering Controls

1. General ventilation: General ventilation is used to dilute hot air with cooler air (generally cooler air that is brought in from the outside). This technique clearly works better in cooler climates than in hot ones. A permanently installed ventilation system usually handles large area or entire buildings. Portable or local exhaust systems may be more effective or practical in smaller areas.
2. Air treatment/air cooling: It differs from ventilation because it reduces the temperature of the air by removing heat (and sometime humidity) from the air.
3. Air conditioning: It is a method of air cooling, but it is expensive to install and operate. The alternative to air conditioning is the use of chillers to circulate cool water through heat exchangers over which air from the ventilation system is then passed; chillers are more efficient in cooler climates or in dry climates where evaporative cooling can be used.
4. Local air cooling: It can be effective in reducing air temperature in specific areas. Two methods have been used successfully in industrial settings. One type, cool rooms, can be used to enclose a specific workplace or to offer a recovery area near hot jobs. The second type is a portable blower with built-in air chiller. The main advantage of a blower, aside from portability, is minimal set-up time.
5. Another way to reduce heat stress is to increase the air flow or convection using fans in the work area (as long as the air temperature is less than the worker's skin temperature). Changes in air speed can help workers stay cooler by increasing both the convection heat exchange (the exchange between the skin surface and the surrounding air) and the rate of evaporation. Because this method does not actually cool the air, any increases in air speed must impact the worker directly to be effective. If the dry bulb temperature is higher than 35°C (95°F), the hot air passing over the skin can actually make the worker hotter. When the temperature is more than 35°C (95°F) and the air is dry, evaporative cooling may be improved by air movement, although this improvement will be offset by the convective heat. When the temperature exceeds 35°C (95°F) and the relative humidity is 100%, air movement will make the worker hotter. Increases in air speed have no effect on the body temperature of workers wearing vapour-barrier clothing.
6. Heat conduction: These methods include insulating the hot surface that generates the heat and changing the surface itself.
7. Simple engineering controls, such as shields, can be used to reduce radiant heat, i.e. heat coming from hot surfaces within the worker's line of sight. Surface

that exceed 35°C (95°F) are sources of infrared radiation that can add to the worker's heat load. Flat black surfaces absorb heat more than smooth, polished ones. Having cooler surfaces surrounding the worker assists in cooling because the worker's body radiates heat toward them.

With some sources of radiation, such as heating pipes, it is possible to use both insulation and surface modifications to achieve a substantial reduction in radiant heat. Instead of reducing radiation from the source, shielding can be used to interrupt the path between the source and the worker. Polished surfaces make the best barriers, although special glass or metal mesh surfaces can be used if visibility is a problem.

Shields should be located so that they do not interfere with air flow, unless they are also being used to reduce convective heating. The effective surface of the shield should be kept clean to maintain its effectiveness.

Administrative Controls and Work Practices

1. Training is the key to good work practice. Unless all employees understand the reasons for using new, or changing old, work practices, the chances of such a programme succeeding are greatly reduced.
2. Hot jobs should be scheduled for the cooler part of the day, and routine maintenance and repair work in hot areas should be scheduled for the cooler seasons of the year.
3. NIOSH (1986) states that a good heat stress training programme should include at least the following components:
   a. Knowledge of the hazards of heat stress;
   b. Recognition of predisposing factors, danger signs, and symptoms;
   c. Awareness of first-aid procedures for, and the potential health effects of heat stroke;
   d. Employee responsibilities in avoiding heat stress;
   e. Dangers of using drugs, including therapeutic ones, and alcohol in hot work environments;
   f. Use of protective clothing and equipment; and
   g. Purpose and coverage of environmental and medical surveillance programme and the advantages of worker participation in such programme.

Worker Monitoring Programme

1. Every worker who works in extraordinary conditions that increase the risk of heat stress should be personally monitored. These conditions include wearing semipermeable or impermeable clothing when the temperature exceeds 21°C (69.8°F), working with extreme metabolic loads (greater than 500 kcal/hour).
2. Personal monitoring can be done by checking the heart rate, recovery heart rate, oral temperature, or extent of body water loss.
3. To check the heart rate, count the radial pulse for 30 seconds at the beginning of the rest period. If the heart rate exceeds 110 beats per minutes, shorten the next work period by one-third and maintain the same rest period.
4. The recovery heart rate can be checked by comparing the pulse rate taken at 30 seconds (P_1) with the pulse rate taken at 2.5 minutes (P_3) after the rest break starts. The two pulse rates can be interpreted as mentioned in heart rate recovery criteria.
5. Oral temperature can be checked with a clinical thermometer after work but before the employee drinks water. If the oral temperature taken under the tongue exceeds 37.6°C, shorten the next work cycle by one third.
6. Body water loss can be measured by weighing the worker on a scale at the beginning and end of each work day. The

Heart rate recovery criteria		
Heart rate recovery	*P_3 pattern*	*Difference between P_1 and P_3*
Satisfactory recovery	<90	–
High recovery (conditions may require further study)	90	10
No recovery (may indicate too much stress)	>90	<10

worker's weight loss should not exceed 1.5% of total body weight in a work day. If a weight loss exceeding this amount is observed, fluid intake should increase.

Other Administrative Controls

The following administrative controls can be used to reduce heat stress:

1. Reduce the physical demands of work, e.g. excessive lifting or digging with heavy objects.
2. Provide recovery areas, e.g. airconditioned enclosures and rooms.
3. Use shifts, e.g. early morning, cool part of the day, or night work.
4. Use intermittent rest periods with water breaks.
5. Use relief workers.
6. Use worker pacing.
7. Assign extra workers and limit worker occupancy or the number of workers present, especially in confined or enclosed spaces.
8. Pre-employment medical examination with physical fitness test.

Personal Protective Equipment

A. Reflective clothing: It can vary from aprons and jackets to suits that completely enclose the worker from neck to feet can stop the skin from absorbing radiant heat. However, since most reflective clothing does not allow air exchange through the garment, the reduction of radiant heat must more than offset the corresponding loss in evaporative cooling. For this reason, reflective clothing should be worn as loosely as possible. In situations where radiant heat is high, auxiliary cooling systems can be used under the reflective clothing.

B. Auxiliary body cooling: (1) Ice vest: Commercially available ice vest, though heavy, may accommodate as many as 72 ice packets, which are usually filled with water. Carbon dioxide (dry ice) can also be used as a coolant. The cooling offered by ice packets lasts only 2 to 4 hours in moderate-to-heavy heat loads, and frequent replacement is necessary. However, ice vest does not encumber the worker and thus permit maximum mobility. Cooling with ice is also relatively inexpensive. (2) Wetted clothing: It is another simple and inexpensive personal cooling technique. It is effective when reflective or other impermeable protective clothing is worn. The clothing may be wetted terry cloth coveralls or wetted two-piece, whole-body cotton suits. This approach to auxiliary cooling can be quite effective under conditions of high temperature and low humidity, where evaporation from the wetted garment is not restricted. (3) Water-cooled garments: These are ranged from a hood, which cools only the head, to vests and 'long johns', which offer partial or complete body cooling. Use of this equipment requires a battery-driven circulating pump, liquid-ice coolant, and a container. Although this system has the advantage of allowing wearer mobility, the weight of the components limits the amount of ice that can be carried and thus reduces the effective use time. The heat transfer rate in liquid cooling systems may limit their use to low-activity jobs; even in such jobs, their service time is only about 20 minutes per pound of cooling ice. To keep outside heat from melting the ice, an outer insulating jacket should be an

integral part of these systems. (4) Circulating air: It is the most highly effective, as well as the most complicated, personal cooling system. By directing compressed air around the body from a supplied air system, both evaporative and convective cooling is improved. The greatest advantage occurs when circulating air is used with impermeable garment or double cotton overalls. One type, used when respiratory protection is also necessary, forces exhaust air from a supplied-air hood (bubble hood) around the neck and down inside an impermeable suit. The air then escapes through openings in the suit. Air can also be supplied directly to the suit without using a hood in three ways: by a single inlet, by a distribution tree, or by a perforated vest. In addition, a vertex tube can be used to reduce the temperature of circulating air. The cooled air from this tube can be introduced either under the clothing or into a bubble hood. The use of a vertex tube separates the air stream into a hot and cold stream; these tubes also can be used to supply heat in cold climates. Circulating air, however, is noisy and requires a constants source of compressed air supplied through an attached air hose. The problem with this system is the limited mobility of workers whose suits are attached to an air hose. Another is that of getting air to the work area itself. These systems should therefore be used in work areas where the workers are not required to move around much or to climb. Another concern with these systems is that they can lead to dehydration. The cool, dry air feels comfortable and the worker may not realize that it is important to drink liquids frequently.

(C) Respirator usage: The weight of a self-contained breathing apparatus (SCBA) increases stress on a worker, and this stress contributes to overall heat stress. Chemical protective clothing such as totally encapsulating chemical protection suits will also add to the heat stress problem.

Medical Evaluation of Workers in Hot Jobs

Employees assigned to work in hot jobs should be medically screened at the following times:

1. Pre-employment medical examination: A good occupational medical history should be obtained. Personal history like smoking and drug or alcohol use should be taken. Cardiovascular, respiratory, gastrointestinal, skin, liver and biliary, and renal and urinary systems need to be reviewed. The musculoskeletal systemic review is considered an important component of the overall review, overlapping general fitness status. Psychological (and behavioral) disorders should be taken into consideration, including past history of mental illness. The sensory organs, including vision, hearing, etc. have a bearing on the individual's total health picture. Physical examinations and physical fitness should be done.
2. Periodical medical examination: It is recommended that employees in hot jobs below the age of 45 years undergo medical examinations every two or three years subject to the medical history, clinical judgment, and any unforeseen job conditions. At 45 and thereafter, employees previously selected for work in hot areas at the highest level should receive annual periodical examinations. In assessing the worker's capacity of continue on the same job, the medical examiner might consider and request physiologic monitoring on the job to especially observe recovery heart rates and oral temperature responses.
3. Return-to-work examination after absence of greater than 7 calendar days due to the illness and injury.
4. Special examination whenever considered medically necessary by plant

management on an individual basis or on a physician's recommendation.

Evaluation of each employee for work in hot areas must be on an individual basis, and the final decision as to suitability should be made by the examining physician only after analysis of various types of information, such as:

1. Medical history: (a) General medical history and system review, (b) occupational history with emphasis on any past heat disorders or illness and (c) history of work performance in heat stress jobs.
2. Physical condition: (a) Physical fitness, (b) age, (c) excessive weight, (d) pregnancy and (f) other clinical findings.
3. Diseases (especially chronic): (a) Those interfering with oxygen uptake and/or exchange, particularly atherosclerotic heart disease and chronic pulmonary disease, either restrictive or obstructive, (b) other type of significant acquired or congenital heart disease, (c) circulatory disease, (d) hepatic disease, (e) drug or alcohol intake habits, (f) diseases causing increased body metabolism, (g) renal disease, (h) any medical problems requiring sodium intake restriction, (i) mental illness and psychological disorders and (j) impairment of verbal communication ability.
4. Medications: (a) Hypotensive agents, (b) diabetics, (c) belladonna alkaloids, (d) sedatives and (e) tranquilizers, antidepressants, amphetamines.
5. Other factors: (a) Type of work (light, moderate, heavy), (b) intensity of heat exposure (low, intermediate or high level) and (c) presence of adequate engineering controls, work practices such as rest periods, training and education, personal protective measures and work clothes.
6. In unusual cases, a more thorough determination of heat stress responses may have to be made. In such instances, factors to be considered would include: (a) Job evaluation at the work site, which should involve such factors as heart rate and body core temperature determinations and other physiological responses and their variation in response to the job and heat stress and (b) physical examination with ancillary tests.

Precautions for Some Other Medical Conditions

1. *Myocardial infarction:* A history of myocardial infarction within the past year is sufficient cause for rejection from work under hot condition.
2. *Renal impairment*: Any evidence of renal impairment must also be investigated to determine the underlying basis before a decision can be taken.
3. *Hearing impairment*: A procedure must be devised for the deaf worker who is unable to communicate in order to enable that person to express any deterioration of fitness or the feeling of well-being during work.
4. *Anaemia*: In men, haemoglobin less than 13 g may be considered for up to moderate work in a hot environment; in women, haemoglobin under 12 g may preclude preplacement in a hot job.
5. *Sickle cell*: The presence of sickle cell trait does not limit participation in strenuous physical activity. Since sudden death is most frequently caused by coronary artery disease or other congenital cardiac abnormalities, imputing any undue risk of sudden death to persons with sickle cell trait appear unjustified.
6. *Toxic substances and other factors*: Heat exposure on the job may increase the hazard to some toxic substances (e.g. carbon monoxide) or increase the workers' susceptibility to other physical stresses, such as work at high or low barometric pressure. Dehydration from uncompensated loss of water and electrolytes may impair the tolerance for toxic agents.

Work practice for hot jobs as recommended by the Standard Advisory Committee on Heat Stress of OSHA

Compulsory work practices for all hot jobs	*Special work practices for hot job*	*Work practices required for extreme heat exposure only*
Adequate water supply, acclimatization, first-aid training, training of workers for health and safety procedures and work practices, in case of heat illness, the WBGT (wet bulb globe temperature) must be assessed on the site	Engineering controls, work–rest regimen, additional acclimatization, adaptive work scheduling, protective clothing and/or equipment, freedom to interrupt work during extreme discomfort	Duration of exposure time regulated by experienced workers' judgment (freedom to interrupt work during extreme discomfort), pre-placement and periodical medical examination (also required in any hot job if work load is heavy), observation by trained supervisor, protective clothing (mandatory)

7. *Reproductive effects*: There are possible effects on reproductive functions resulting from high environmental temperature, particularly with respect to embryonic or fetal development. It has been reported that hyperthermia in human pregnancy may cause harm to the developing nervous system in the early pregnancy. Some men in their hot jobs are concern about their sexual potency. Heat stress may be the partial cause for their apparent impotency.

Measurement of Wet Bulb Globe Temperature

Measurement is often required of those environmental factors that most nearly correlate with deep body temperature and other physiological responses to heat. At the present time, the wet bulb globe temperature index (WBGT) is the most used technique to measure these environmental factors. WBGT index is by far the most widely used throughout the world. It was developed in a US Navy investigation into heat casualties during training as an approximation to the more cumbersome corrected effective temperature (CET), modified to account for the solar absorptivity of green military clothing (ILO, 1998). The determination of WBGT requires the use of a black globe thermometer, a natural wet-bulb thermometer and a dry bulb thermometer (ACGIH, 2004).

WBGT values are calculated by the following equations:

Indoor or outdoor wet bulb globe temperature index:

Indoor or outdoor with no solar load:

$$WBGT = 0.7\ NWB + 0.3\ GT$$

Outdoor with solar load:

$$WBGT = 0.7\ NWB + 0.2\ GT + 0.1\ DB$$

where WBGT = Wet bulb globe temperature
NWB = Natural wet bulb temperature
DB = Dry bulb (air) temperature
GT = Globe thermometer temperature

The determination of WBGT requires the use of a black globe thermometer, a natural (static) wet bulb thermometer, and a dry-bulb thermometer. The measurement of environmental factors shall be performed as follows:

1. The range of the dry and the natural wet bulb temperature should be –5°C to +50°C. The dry-bulb thermometer must be shielded from the sun and the other radiant surfaces of the environment without restricting the airflow around the bulb. The wick of the natural wet bulb thermometer should be kept wet with distilled water for at least one-half hour before the temperature reading is made. It is not enough to immerse the other end of the wick into a reservoir of distilled

water and wait until the whole wick becomes wet by capillarity. The wick must be wetted by direct application of water from a syringe one-half hour before each reading. The wick must cover the bulb of the thermometer and an equal length of additional wick must cover the stem above the bulb. The wick should always be clean, and new wicks should be washed before using.

2. A globe thermometer, consisting of a 15 cm (6-inch) in diameter hollow copper sphere painted on the outside with a matte black finish, or equivalent, must be used. The bulb or sensor of the thermometer (range –5°C to +100°C with an accuracy of ±5°C) must be fixed in the centre of the sphere. The globe thermometer should be exposed at least 25 minutes before it is read.
3. A stand should be used to suspend the three thermometers so that they do not restrict free air flow around the bulbs and the wet-bulb and globe thermometer are not shaded.
4. It is permissible to use any other type of temperature sensor that gives a reading similar to that of a mercury thermometer under the same conditions.
5. The thermometers must be placed so that the readings are representative of the employee's work or rest areas, as appropriate.

Once, the WBGT has been estimated, employers can estimate workers' metabolic heat load (as per activity example and assessment of work) and use the ACGIH method of determine the appropriate work/rest regimen, clothing and equipment to use to control the heat exposure of workers in their facilities.

Conclusion

Scientific and technological advances in ergonomics/human factors have greatly contributed to the health promotion and comfort of workers in different working environments around the world. Nevertheless, the heat stress is one of the damaging factors to be treated in various industries and jobs.

Human beings live their entire lives within a very small range of internal temperatures. The maximum tolerance limits for living cells range from about 0°C to about 45°C, however, humans can tolerate internal temperatures below 35°C or above 41°C for only very brief periods. To maintain the internal temperature within these limits, people have developed very effective and, in some instances, specialized physiological responses to acute thermal stresses. These responses-designed to facilitate the conservation, production or elimination of body heat-involve the finely controlled coordination of several body systems (ILO, 1998).

The cardiovascular system is under considerable strain when a person is working in hot environment. In such a situation, peripheral vasodilatation requires an increase in blood flow to the skin and working muscles demands increased blood supply. In long run, worker becomes dehydrated; sweat production decreases and core body temperature increases.

In extreme situations, the thermoregulatory system may be unable to cope. If core body temperature rises above 42°C, blood pressure may drop and insufficient blood is pumped to the vital organs including the heart, kidney and brain. Under such a condition, a worker will collapse with heat stroke (Bridger, 1995).

The committee on biological markers of the National Council (NRC) divided biomarkers into three types: Exposure, effect and susceptibility (NRC, 1989). Clearly, a continnum exists between biologic markers of exposure and effect that can be extended to a continuum between source and disease (Lioy, 1990).

COLD INJURY

Introduction

The primary physiological responses to cold exposure are peripheral vasoconstriction, piloerection, and the increase in metabolic heat production by shivering. Skin temperature falls first, a result of local cooling without a corresponding increase in the delivery of heat to the skin by the flow of blood. This fall in skin temperature stimulates peripheral cold receptors and leads to both locally mediated and centrally regulated vasoconstriction, which in turn allows a further fall in skin temperature. If the rest of the body is sufficiently warm, cyclical cold vasodilatation ('cold induced vasodilatation' or the 'hunting reaction') may ensue, with skin temperatures falling below 12°C, rising with the vasodilatation, and then falling again. The mechanism responsible for this phenomenon remains controversial, but extensive experimentation has shown that it is very variable between and within individuals, and absent if the rest of the body is cold. Sustained peripheral vasoconstriction may be accompanied by fluid shifts resulting in a reduction in plasma volume. Shivering first appears as short bursts in a few groups of muscles, becoming continuous and generalized as a rectal temperature of about 35°C is reached. Further cooling or exhaustion results in shivering gradually tailing off, until it is replaced by generalized rigidity and finally the flaccidity of imminent death.

The list of workers potentially exposed to cold and/or cold wet working conditions is long: Airline maintenance crew, cooling room workers, divers, dry-ice workers, fishermen, ice makers, liquefied gas workers, military personnel, miners, refrigerated warehouse workers, snow removal crews, maintain engineers, food processors in chilled environments and farmers, forestry workers as well as all other outdoor workers in temperate winter and colder conditions.

Time limits that have been recommended for working for various temperature (source: NSC Data Sheet 465, Cold room testing of gasoline and diesel engines) are available. Efforts have been made to develop indices for evaluating cold environments such as the indices for evaluating hot environments. The 'wind chill index' probably is the best known and most used of these indices; the 'shiver index' is another. All have their limitations, as do those for heat stress; but under the right conditions, the information they yield can be useful guidelines.

Physiology of Cold Exposure

The dominating metabolic need of the human body exposed to cold is to maintain core body temperature. Specific physiologic responses enable humans to do this during exposure to mild or moderate degree of cold. However, these are limited and can be overwhelmed with subsequent progressive physiologic and anatomic damage. Because these defense mechanisms are limited, we must depend on

Low-temperature time limits

Temperature range	*Maximal daily exposure*
30°F to 0°F (–1°C to –17°C)	No exposure time limit, if the person is properly clothed
0°F to –30°F (–17°C to –34°C)	Total cold-room work time: 4 hours. Alternate 1 hour in and 1 hour out of chamber
–30°F to –70°F (–34°C to –56°C)	Two periods of 30 minutes each, at least 4 hours apart. Total cold-room work time allowed: 1 hour.
–70°F to –100°F (–56°C to –73°C)	Maximal permissible cold-room work time: 5 minutes over an 8-hour working day. For these extreme temperatures, the wearing of a completely enclosed headgear, equipped with a breathing tube running under the clothing and down the leg to preheat the air, is recommended

Shiver index		
Temperature		*Time to shivering (hours)*
°F	°C	
10	–12	6.0
0	–17	5.0
–10	–23	4.0
–20	–28	2.5
–30	–34	2.0
–40	–40	1.5
–70	–56	0.4

Source: Cold and the worker, National Safety News 100(6):98, 1969.

Shivering is the body's attempt to warm itself. This table shows how long a man can stay in subfreezing weather before temperatures in his extremities drop below 55°F (13°C) and he starts to shiver violently. The table is based on a man dressed in heavy Arctic clothing, boots, and mittens for light, sedentary work.

external protective measures to survive and work under very cold conditions.

The conscious detection of cold temperature by humans depends on cold receptors found in the skin. These are cutaneous nerve endings, but they are not anatomically discrete, specialized organs. They function alone when cold is moderate, but pain receptors are triggered as cold becomes more intense, and pain receptors dominate peripheral cutaneous response altogether at very low temperatures. Sensation is lost entirely when tissue freezes, and local anesthesia occurs in the frozen area. This peripheral nervous response is fed to the hypothalamus, which maintains central control of core body temperature. The hypothalamus response to cold is directly affected by blood temperature and a multiplicity of other signals, in addition to those primary signals sent by the peripheral cold receptors. The hypothalamus, when stimulated by signals of a cold environment, responds with feedback and triggers available body defensive mechanisms to cold. Hypothalamic control is lost, however, when core body temperature falls below a minimum point.

Body defenses capable of being triggered are either increased (i.e. heat production resulting from increased metabolic activity) or decreased (i.e. heat loss resulting from reduced peripheral blood flow). Superficial vasoconstriction occurs as temperature of the air in contact with the skin falls, and heat loss is then reduced. As heat loss continues, shivering begins, and decreased metabolic rate is generated by this intense muscular activity. The superficial circulation in the limbs is quite responsive to cold, and the regulation of core body temperature is both protected by and dependent upon the heat balance of the extremities. Core body temperature must be maintained for the limbs to be warmed, and increased metabolic heat resulting from exercise results in increased warming of the extremities.

Body Heat Loss in Cold Environment

Bodily heat losses in cold environments by conduction, convection and radiation. When protective coverings are wet, heat may also be lost by evaporation. Therefore, evaporation loss via the skin is limited to situations in which clothing becomes wet from sweat or from outside moisture in a cold environment. Conductive loss will occur when there is contact with a cold surface. However, loss of heat by convection will occur as warmed air around the body rises. There is very efficient forced convective heat loss when there is wind. This effect is known as the 'wind chill factor'. Radiant heat loss occurs when surrounding surfaces are cold. Human skin absorbs heat from surroundings of higher temperature and loses heat to surroundings of lower temperature. Consequently, the most dangerous and rapid heat loss takes place when clothing is wet, wind is high, and surroundings are cold, or when the human body is immersed in cold water.

Assessment of Cold Stress

The dominant components of cold stress are the temperature and nature (air or water) of the fluid surrounding the body. Wind speed, and to a lesser extent water movement, can

also be important. A simple thermometer and a wind speed indicator are the main measuring instruments needed. Anemometers need not be complex: One of the cup types is best for outside use, whilst vane and hot wire systems are preferred in enclosed spaces. Inexpensive hand-held anemometers are popular in outdoor leisure activities and generally sufficiently accurate for these purposes. However, they often present derived measures such as 'wind chill equivalent temperature' that should be treated with suspicion. The measurement of radiant heat exchange is more difficult to measure accurately, although it is generally small except under a clear night sky.

Evaporative loss is even harder to assess and is small in cold-dry systems. However, if the clothing is wet, evaporative losses alone can exceed 700 W, exceeding the total of other modes of heat loss; simple techniques such as repeated weighing to estimate water loss can then only give a crude overestimate. Although convection losses in water are much greater than in air of the same temperature, partial immersion presents a particularly complex situation. Subtle differences in behavior and conditions can result in large changes in a heat loss, as the body switches from convective cooling in water, through evaporative cooling of wet surfaces in air, to dry convection cooling. The delayed evaporative cooling of sweat-soaked garments after exercise is also difficult to quantify, but of potentially great significance in those working hard in cold condition.

Siple and Passell were the first to attempt to incorporate dry bulb air temperature and wind speed into a single figure, now commonly referred to as 'wind chill equivalent temperature'.

Assessment of Cold Strain

Any fall in core temperature below 35°C, the accepted threshold for hypothermia, is the best guide to a serious degree of general cold strain. Rectal measurement, at least 10 cm and preferably 15 cm beyond the anal sphincter, is the most reliable in cold conditions, though tympanic, oesophageal, gastric, vaginal, deep arterial and deep venous sites can each be used in different circumstances; detailed discussion is given in ISO 9886. All require careful calibration of probes and measures to prevent the transmission of infections, such as HIV. Oral temperature is never reliable in the cold, as it is depressed by cold saliva, and infrared tympanic techniques are likely to be dangerously misleading. Skin temperatures can give warning of lesser degrees of general cold strain, and are necessary measures of local cold strain, particularly when there is a risk of cold injury. Techniques are based on thermistors or thermocouples, or infrared emission measurement systems for uncovered skin. Electromyography (EMG) and oxygen uptake can be used to assess shivering and total metabolic heat production, although these are of limited value outside the laboratory.

Adaptation and Habituation

Repeated brief exposures to cold produce clear reductions in both the vasoconstrictor and metabolic responses to cold stress. This 'hypothermic' adaptation has an advantage in increasing comfort during cold exposure.

Cold-induced Injuries

These injuries occur as a result of man's inability to properly protect himself from his environment with subsequent lowering of core body temperature. This is followed by local tissue cooling with tissue anoxia and freezing. Cold injuries may be systemic or localized depending upon temperature and the length of exposure. Depending upon temperature and the length of exposure, cold-induced injury may be divided into freezing and nonfreezing injuries.

Freezing Cold Injuries

Local freezing of tissues, the most rapid and dramatic form of cold injury, may occur when

people are exposed to temperature below 0°C without adequate protection or opportunities to return to warm environments when the extremities become chilled.

The areas most commonly affected include fingers and toes, followed by nose, cheeks and ears, and occasionally the male genitalia. Such injuries may be associated with hypothermia, in which case the potentially life-threatening condition must be treated first, or with local or general trauma. The damage is mainly caused by high concentrations of electrolytes left in tissue fluids when most of the water turns to ice. While frozen, the skin is white and hard; on thawing, there is first hyperaemia and then, in severe cases, pallor and a woody feel to the skin as local blood vessels become occluded by red cells. The complications of freezing cold injury are:

1. *Frostnip:* Frostnip is popularly used to describe mild freezing cold injury in which the superficial tissues (that are the only layer affected) recover completely within 30 minutes of starting rewarming; such recovery must include the return of normal sensation. Classically, it involves any combination of the loss of peripheral sensation and the slight freezing of superficial tissues. Typically, one or more finger or toe tips are blanched, the skin is leathery to the touch, but not of wooden feel (which usually indicates deeper freezing) and anaesthetic. The immediate and diagnostic treatment is to rewarm the affected periphery against the warm skin of an understanding colleague; hand and feet may conveniently be placed in an axilla or groin, whilst portions of the nose, cheeks or ears are best rewarmed by firm contact with a hand or fur patch on the back of a mitten. Rubbing, massage and direct heat should be avoided. Provided that recovery is complete within the 30-minute period, no further treatment is necessary. However, those who have not returned to normal should be treated for superficial frostbite.
2. S*uperficial frostbite:* More severe freezing injury that would not recover so rapidly is usually termed 'frostbite'. If confined to the skin and most peripheral layers of tissues, it can usually be qualified as being 'superficial'. Initial appearances may be indistinguishable from frostnip, but in the hours and days following thawing, gross discolouration may occur, with haemorrhagic patches and blisters with fluid varying from clear serous to the frankly haemorrhagic. Later still, small and superficial areas of dry gangrene may develop, with skin peeling and nail loss. The standard treatment for superficial frostbite remains conservative. Rapid warming in a stirred waterbath at 38–41°C should be prolonged and thorough, with good analgesic cover. Care must be taken to ensure that patches do not remain partially defrosted on removal from the waterbath. For some, rewarming is an exquisitely painful process that may even merit titration with intravenous morphine, but non-narcotic analgesics normally suffice. Infection must be prevented with liberal use of topical antibacterials, such as those based on chlorhexidine gluconate in combination with twice daily 'whirlpool' baths at 40°C. Tetanus prophylaxis is also required in most cases. Systemic antibiotics are normally only considered when there is active infection or deeper tissue is involved.
3. *Deep and complicated frostbite:* Freezing of muscles and other deeper tissues, including their blood vessels, is much rarer and more serious. Whole limbs may freeze solid and victims are invariably hypothermic. Rapid rewarming should only be attempted in hospital under full biochemical control, as massive release of potassium from damaged cells can otherwise cause sudden death. Surgical decompression of tissue spaces may be needed before rewarming, to limit the rise

of tissue pressures which results from the volume expansion accompanying the melting of ice crystals. The amount of tissue likely to be dead or dying demands the utmost care in preventing tetanus, gas gangrene and resistant infections.

Cyclical freezing, thawing and refreezing is even more destructive of cells, and can result in the very worst end result (*freeze–thaw–refreeze injury*). Early and radical amputation, at two to three months following injury, may be preferred in these cases, as it might in established infections.

This form of cold injury (frostbite) may be divided into four pathologic phases:

1. *Prefreeze phase*: This is secondary to chilling, prior to ice crystal formation. Changes are caused by vasospsticity and transendothelial plasma leakage. Tissue temperatures range from 38 to 50°F (3 to 10°C). Cutaneous sensation is generally absent.
2. *Freeze–thaw phase*: Here, there is actual ice crystal formation. Tissue temperature drops below the freezing point. The susceptibility of tissue to freezing varies. Endothelium, nerve tissue, and bone marrow are more sensitive than muscle, bone, or cartilage.
3. *Vascular stasis phase*: This involves blood vessels changes. There is spasticity and dilatation with plasma leakage, stasis coagulation, and shunting.
4. *Late ischaemic phase*: This results from thrombosis and arteriovenous (A-V) shunting, ischaemia, gangrene, and autonomic dysfunction.

Long-term Sequelae of Freezing Injury

The most common long-term sequelae of freezing injury include cold sensitization, chronic pain, residual neurological defects that rarely may involve loss of proprioception rather than touch, joint pain and stiffness, hyperhidrosis, skin and nail abnormalities. Radiographic examination may reveal characteristic appearances of 'frostbite arthritis', but long-term changes in gait may be more proximal joints, particularly in the lower limb. Although hyperhidrosis may lead to chronic fungal infection, the skin is often dry and scaly, and may crack painfully. Fungal infections of the nails may respond little to treatment and, together with disturbances of nail growth, can result in thickening and frank onychogryphosis.

More usual sequelae include recurrent ulceration and breakdown of old scars, which may in turn lead to an increased risk of local skin cancers. It is not clear whether this is specifically related to the frostbite or is common to other causes of recurrent ulceration. Although rare, complete local loss of proprioception is a neurological abnormality that is characteristic of old cold injury. In the hands, this can make dressing very hard, while in the feet it may threaten mobility.

Nonfreezing Cold Injury

Longer exposures to less severe temperatures, particularly when coupled with other conditions liable to cause circulatory stasis, can result in injuries that appear generally mild in comparison with frostbite. A wide variety of terms have been applied to nonfreezing cold injury (NFCI) since it was first described in the 18th century, including trench foot, immersion foot, shelter limb and Flanders foot, depending on the circumstances in which the variant was described. It often coexists with freezing injury, but in spite of its apparent innocence during the acute phase, NFCI frequently results in more severe long-term sequelae. In common with freezing injuries, NFCI is overtly more frequent in certain ethnic groups, most notably Africans and Caribbeans, even when their antecedents migrated to colder areas, such as northern Europe. The great majority of cases of NFCI affect the feet alone, although recent series in the UK claim that a quarter of cases are found to have suffered NFCI of the hands as well as

the feet. There also appear to be relationships with other similar conditions: Long immersion in luke-warm water results in 'paddy foot', which is clinically indistinguishable from NFCI. The pathology of NFCI remains obscures; factors that have been proposed include the direct effect of cooling on nerves, prolonged ischaemia during cold exposure and the liberation of reactive oxygen compounds during reperfusion. Some or all of these may be important in the evolution of vibration injuries, complex regional pain syndrome (formerly known as reflex sympathetic dystrophy) and other similar syndromes.

Presentation of NFCI and its Management

Clinical appearances have been divided into four stages:

1. *Injury*: In which the foot is very cold, ischaemic and numb.
2. *Rewarming:* In which the foot becomes mottled blue-pink and exquisitely painful.
3. *Hyperaemia:* In which the pulses are full and bounding, but there is slow capillary refill, there is marked swelling, some degree of anaesthesia and paraesthesia, and severe pain (primarily nocturnal).
4. *Recovery:* In which the foot gradually returns towards normal, with residual sequelae including cold sensitization.

Stage 1 is seen throughout the period in which the injury is actually occurring, ranging from minutes to days in duration. It is therefore usually only witnessed by the patient, who may provide the diagnostic description of numbness or other sensory impairment. Stage 2 is seen fleetingly during rewarming, typically lasting just a few minutes. The great majority of cases present in stage 3, which lasts for several days or weeks. Stage 4 may then supervene for months, years possibly lifelong.

It has been established that rapid rewarming in a waterbath exacerbates both damage and pain. Slow (or, passive) rewarming is therefore preferred. The management of early pain: Bedclothes should be cradled over the feet at night and conventional analgesics are invariably ineffective. A single dose of amitriptyline (25–150 mg) in the evening can be used as it is used in the treatment of other types of neuropathic pain. Patients who do not obtain good relief from amitriptyline alone can be tried on a combination of 10 mg amitriptyline at night together with pregabalin, the latter prescribed in accordance with the recommendations of a current formulary. Experience with sympathetic blocks or sympathectomy is generally very discouraging and they should normally be avoided.

Late consequences are common and may last for life, even after subclinical NFCI. Most prolonged vasoconstrictive response to further cold exposure; when a patient complains of this as symptom, it is termed 'cold sensitivity', but assumes a diagnosis of 'cold sensitization' when supported by physical findings. A cold stimulus as innocuous as a 2-minute immersion in water at 15°C may precipitate a cold, vasoconstricted foot for four or more hours afterwards.

The cause of cold sensitization is ill understood. The cause may lie in a vascular endothelial abnormality (and NFCI is known to result in endothelial injury) or the devascularization which is known to arise when local blood flow is chronically reduced. No effective treatment is known, although exposure to heat may be ameliorative. This can be accomplished by living in a tropical country for some years, or possibly by bathing the affected parts in water at 40°C for at least 30 minutes every day. Severe cases may enjoy slow and slight benefit from sustained release nifedipine but with some side effects. Because of the risk of long-term alteration in gait and resulting overuse injury, early and expert assessment of gait should be considered in those who have sustained NFCI of the feet.

Chilblain (Pernio)

This condition usually results from chronic intermittent exposure to high humidity and ambient temperatures above freezing. It is less severe than trench foot and is most commonly seen in women. It is characterized by localized erythema, cyanosis, plaques, and nodules. Recovery is the rule; however, the victim is prone to recurrences with subsequent lesser exposures. Therefore, workers who have had this condition should be warned and carefully protected from future exposure.

Hypothermia

Hypothermia may be defined as lowering of the core body temperature to or below 95°F (35°C). At this level, clinical signs begin to appear. These include vasoconstriction, pallor, incoordination, muscular weakness, and dulling of cerebration. As core temperature continues to fall, breathing becomes shallow, shivering diminishes, and consciousness is lost at 86.6–86°F (32–30°C). At this point, the skin may show a blue mottling, and breathing is barely perceptible. At lower temperatures, ventricular fibrillation is a potential danger. 'Primary' hypothermic fatalities are usually considered 'violent' and are classified as accidental, homicidal or suicidal. 'Secondary' hypothermia deaths are often looked upon as natural complications of systemic disorders (like sepsis, carcinoma). The victim's history may suggest the presence of hypothermia. Major trauma, immersion, overdose, and cerebrovascular accidents may be the other causes of hypothermia. Immersion in cold water reduces core temperature very rapidly. Water has 20 times the capacity to extract heat compare with air. Sudden immersion leads to hyperventilation with additional heat loss. Immersion may chill the deeper tissues without surface tissue freezing. It can also result in rapid loss of consciousness. Those working out on or at the edge of cold bodies of water (i.e. marine workers of all kinds) are at special risk of hypothermia.

Field treatment of cold injury is the art of the possible. Since cold, stiff and cyanotic victims with fixed and dilated pupils have been revived, the dictum for field personnel should be "no one is dead until warm and dead". Attempt should be made to rewarm every victim. This can be done actively or passively. All victims of each type of cold injury should be moved to hospital facilities as soon as possible. The patient must be kept warm during transport.

Evaluation and Control of Cold Injury

For exposed skin, continuous exposure should not be permitted when the air speed and temperature results in an equivalent chill temperature –32°C (–25°F), superficial or deep local tissue freezing will occur only at temperature below –1°C regardless of wind speed.

At air temperatures of 2°C (35.6°F) or less it is imperative that workers who become immerse in water or whose clothing becomes wet be immediately provided a change of clothing and be treated for hypothermia.

Special protection of the hands is required to maintain manual dexterity for the prevention of the accidents:

1. If fine work is to be performed with bare hands for more than 10 to 20 minutes in an environment below 16°C (60°F), special provisions should be established for keeping the workers' hand warm, for this purpose, warm air jets, radiant heaters (fuel burner or electric radiator), or contact warm plates may be utilized. Metal handles of tools and control bars shall be covered by thermal insulating material at temperatures below –1°C (30°F).
2. If the temperature falls below 16°C (60°F) for sedentary, 4°C (40°F) for light, –7°C (20°F) for moderate work and fine manual dexterity is not required, then gloves should be used by the workers.

To prevent contact frostbite, the workers should wear anticontact gloves:

1. When cold surface below –7°C (20°F) are within reach, a warning should be given to each worker by his supervisor to prevent inadvertent contact by bare skin.
2. If the temperature is –17.5°C (0°F) or less, the hands should be protected by mittens. Machine controls and tools for use in cold conditions should be designed so that they can be handled without removing the mittens.

Provision for additional total body protection is required if work is performed in an environment at or below 4°C (40°F). The workers shall wear cold protective clothing appropriate for the level of cold and physical activity:

1. If the air velocity at the job site is increased by wind, draft, or artificial ventilating equipment, the cooling effect of the wind shall be reduced shielding the work area, or by wearing an easily movable outer windbreak layer garment.
2. If only light work is involved and if the clothing on the worker may become wet on the job site, the outer layer of the clothing in use may be of a type impermeable to water. With more severe work under such conditions the outer layer should be water repellent, and the outerwear should be changed as it becomes wetted. The outer garment must include provisions for easy ventilation in order to prevent wetting the inner layers by sweat. If work is done at normal temperatures or in a hot environment before entering the cold area, the employee shall make sure that his clothing is not wet as a consequence of sweating. If his clothing is wet, the employee shall change into dry clothes before entering the cold area. The workers shall change shocks and any removable felt insoles at regular daily intervals or use vapor barrier boots. The optimal frequency of change shall be determined empirically and will vary individually and according to the type of shoe worn and how much the individual's feet sweat.
3. If extremities, ears, toes and nose, cannot be protected sufficiently to prevent sensation of excessive cold or frostbite by hand wear, footwear, and face masks, these protective items shall be supplied in auxiliary heated versions.
4. If the available clothing does not give adequate protection to prevent hypothermia or frostbite, work shall be modified or suspended until adequate clothing is made available or until weather conditions improve.
5. Workers handling evaporative liquid (gasoline, alcohol or cleaning fluids) at air temperatures below 4°C (40°F) shall take special precautions to avoid soaking of clothing or gloves with the liquids because of the added danger of cold injury due to evaporative cooling. Special note should be taken of the particularly acute effects of splashes of 'cryogenic fluids' or those liquids with a boiling point only just above ambient temperature.

Work–Warming Regimen

If work is performed continuously in the cold at an equivalent chill temperature (ECT) or below –7°C (20°F), heated warming shelters (tents, cabins, rest rooms, etc.) shall be made available nearby and the workers should be encouraged to use these shelters at regular intervals, the frequency depending on the severity of the environmental exposure. The onset of heavy shivering, frostnip, the feeling of excessive fatigue, drowsiness, irritability, or euphoria, is indications for immediate return to the shelter. When entering the heated shelter, the outer layer of the clothing shall be removed and the remainder of the clothing loosened to permit sweat evaporation or a change of dry work clothing provided. A change of dry work clothing shall be provided

as necessary to prevent workers from returning to their work with wet clothing. Dehydration or the loss of body fluids occurs insidiously in the cold environment and may increase the susceptibility of the worker to cold injury due to a significant change in blood flow to the extremities. Warm sweet drinks and soups should be provided at the worksite to provide caloric intake and fluid volume. The intake of coffee should be limited because of a diuretic and circulatory effect.

For work practices at or below –12°C (10°F) equivalent chill temperature (ECT) the following shall apply:

1. The worker shall be under constant protective observation (supervision).
2. The work rate should not be so high as to cause heavy sweating that will result in wet clothing; if heavy work must be done, rest periods must be taken in heated shelters and opportunity for changing into dry clothing shall be provided.
3. New employees shall not be required to work full-time in cold in the first days until they become accustomed to the working conditions and required protective clothing.
4. The weight and bulkiness of clothing shall be included in estimating the required work performance and weights to be lifted by the worker.
5. The worker shall be arranged in such a way that sitting still or standing still for long periods is minimized. Unprotected metal chair seats shall not be used.
6. The worker shall be instructed in safety and health procedures. The training programme shall include as a minimum instruction in:
   a. Proper re-warming procedures and appropriate first-aid treatment.
   b. Proper clothing practices.
   c. Proper eating and drinking habits.
   d. Recognition of impending frostbite,
   e. Recognition of signs and symptoms of impending hypothermia or excessive cooling of the body even when shivering does not occur.
   f. Safe work practices.

Special Workplace Recommendations

Special design requirements for refrigerator rooms including the following:

1. In refrigerator rooms, the air velocity should be minimized as much as possible and should not exceed 1 m/sec (20 fpm) at the job site. This can be achieved by properly designed air distribution systems.
2. Special wind protective clothing shall be provided based on existing air velocities to which workers are exposed.

Special caution shall be exercised when working with toxic substances and when workers are exposed to vibration. Cold exposure may require reduced limits.

Eye protection for workers employed out-of-doors in a snow and/or ice-covered terrain shall be supplied. Special safety goggles to protect against ultraviolet light or glare (which can produce temporary conjunctivitis and/or temporary loss of vision) and blowing ice crystals are required when there is an expanse of snow coverage causing a potential eye exposure hazard.

Workplace Monitoring

1. Suitable thermometry should be arranged in any workplace where the environmental temperature is below 16°C (60°F) to enable overall compliance with the requirements of the TLV to be maintained.
2. Whenever the air temperature at a workplace falls below –1°C (30°F), the dry bulb temperature should be measured and recorded at least every 4 hours.
3. In indoor workplaces, the wind speed should also be recorded at least every 4 hours whenever the rate of air movement exceeds 2 meters per second (5 mph).

4. In outdoor work situations, the wind speed should be measured and recorded together with the air temperature whenever the air temperature is below –1°C (30°F).
5. The equivalent chill temperature (ECT) shall be obtained.

Summary

Cold injuries are classified as systemic or localized and as freezing and nonfreezing. The factors influencing the risk for these injuries include the atmospheric or water temperature, humidity, wind velocity, duration of exposure, type of protective equipment or clothing, type of work being performed and associated energy expenditure, and age and health status of the worker. The risk of hypothermia increases with age and also is increased if the employee is intoxicated with drug or alcohol; is receiving medication such as barbiturates, antipsychotics, or reserpine; or smokes; or has adrenal insufficiency, diabetes, myxedema, neurologic disease affecting hypothalamic or pituitary function or causing peripheral sensory impairment, peripheral vascular disease, or cardiovascular disease causing diminished cardiac output.

When the body is exposed to cold environments, it has two types of normal physiological reactions: (1) Constriction of superficial blood vessels in the skin and subcutaneous tissue, resulting in heat conservation, and (2) increase in metabolic heat production through voluntary movement and by shivering. In case of systemic hypothermia, cellular and physiologic functions are diminished. Oxygen consumption is decreased by approximately 7% per degree Celsius, myocardial repolarisation is slowed, and ventricular fibrillation is a major hazard.

There are many persons, even in temperate and tropical climates, who work in cold. Pathophysiological changes occur when bodily defense mechanisms are overwhelmed by cold. These cold-induced injuries require careful treatment. Special protective clothing is needed for those who work at low temperatures. Health and safety education is a must for these workers. It should include special instruction in avoidance of injury due to cold exposure. Person with cardiovascular, peripheral vascular, or asthmatic respiratory disease, and those with prior history of pernio (chilblain) are at special risk during cold work and should not be placed in these jobs.

INDUSTRIAL NOISE

Introduction

Noise is one of the most common occupational hazards in workplace. The National Institute for Occupational Safety and Health (NIOSH) estimates that 30 million workers in the United States are exposed to hazardous noise. Industrial noise is the most pervasive of all industrial pollutions. It involves most of the industries and causes severe hearing loss in every country of the world. Exposure to high levels of noise may cause hearing loss, create physical and psychological stress, reduce productivity, interfere with communication, and contribute to accidents and injuries by making it difficult to hear warning signals. Occupational noise-induced loss is considered one of the most common occupational disorders in industrialized countries.

The first consideration in determining the probability of a cause effect relationship between an occupational hearing loss and excessive noise at the workplace is to establish: (a) that a hearing loss dose, in fact, exist and (b) that the particular manifestations of the hearing loss appear to be the result of exposure to excessive noise at workplace. Occupational hearing loss can be defined as the loss of hearing in one or both ears as the result of one's employment. The lost may be the result of hazardous noise exposure or due to acoustic trauma. The early signs of occupational hearing loss include: (a) Difficulty in understanding spoken words in a noisy environment, (b) need to be near or look at the person speaking to help understand

words, (c) familiar sounds are muffled, (d) complaints that people do not speak clearly, and (e) ringing noises in the ears.

The application of a mechanical force to an elastic medium, such as air, results in the displacement of the particles or molecules that constitute that medium. The energy in the displacing force effectively overcomes the mass or inertia of the molecules. This displacement is resisted by a force that tends to return the molecule or particle to its resting position that is the elasticity. In effect, the displacing force sets up an oscillation or vibration in that medium, which is essentially sound. This oscillation would continue indefinitely if it were not for the frictional resistance that is also inherent to a vibrating system. As a result of this frictional resistance, the energy in the initial displacing force is dissipated as heat and the vibration ceases. The transfer of kinetic energy between vibrating molecules, however, leads to a propagation of the sound through the medium. This generates alternating areas of condensation (with a rise in ambient pressure) and rarefaction (with a fall in ambient pressure) within the medium. There is no net movement of the medium as such, but rather the movement of changes in pressure in the medium, which is a sound wave and takes the form of a sine wave when the pressure changes are plotted against time. A sound wave has two fundamental properties: Its intensity (amplitude of the peak of the wave form), correlating subjectively with loudness, and its frequency, which has the subjective correlate of pitch.

A textbook definition of sound is "a rapid vibration of atmospheric pressure caused by some disturbance in air". Sound propagates as a wave of positive pressure disturbances (compressions) and negative pressure disturbances (rarefactions) (Fig. 8.4). Sound can travel through any elastic medium (e.g. air, water, wood, and metal). When air molecules are set to vibrate, the ear perceives the vibrations in pressure as sound.

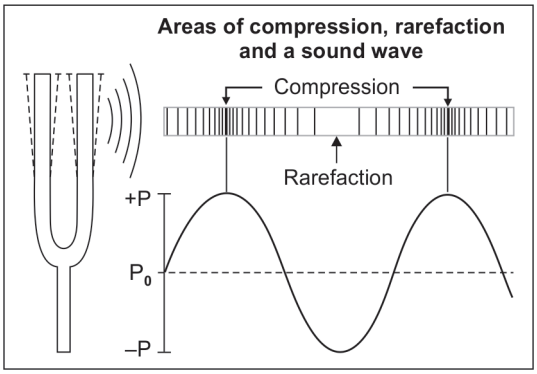


Fig. 8.4: Sound wave (*Ref.* OSHA's manual)

The word *sound* can also be used to mean a physical pressure oscillation (alternate increase and decrease in the normal atmospheric pressure) or the resulting, subjective auditory sensation that occurs when the hearing mechanism is stimulated. The vibrating sound source produces rapid pressure fluctuations that spread outward in the same way ripples do on water after a stone is thrown into it. The result of the movement of the air molecules is a fluctuation in a normal atmospheric pressure, or sound waves. These waves may radiate in all directions from the source and may be reflected and scattered. When the sound source stops vibrating, the sound waves disappear almost instantaneously. The ear is extremely sensitive to these pressure fluctuations, which it converts into auditory sensations in the ear. The term sound is usually applied to that form of energy that produces a sensation of hearing in humans; vibration usually refers to the nonaudible acoustic phenomena that are recognized by the tactile experience of touch or feeling. The sound may be described in terms of three variables: (1) Amplitude or sound level (perceived as intensity or loudness), (2) frequency (perceived as pitch or sharpness) and (3) time pattern (continuous or impulse). Amplitude or sound level is the extent of the pressure fluctuations or the difference between static atmospheric pressure (with no sound present) and the total atmospheric pressure with sound present. The rate at

which the sound pressure fluctuates determines frequency. The pattern of distribution of acoustical energy at the various frequencies is referred to as the *spectrum* of the sound. The wide-band noise is one where the acoustic energy is distributed over a large range of frequencies. Narrow-band noises, with most of their energy confined to a narrow range of frequencies, normally produce a definite pitch sensation. For a true narrow-band noise, only a single octave band will contain a significant amount of energy. The noise caused by a circular saw, planer, or other rotating devices is occasionally of the narrow-band type, but usually there is some spreading of the acoustic energy to several of the octave band. Sound may be described in terms of its pattern of time and level: Continuity, fluctuation, impulsiveness, and intermittency. Continuous sounds are those produced for relatively long periods at a constant level. Continuous noise is normally defined as broad-band noise, of approximately constant level and spectrum. Intermittent sounds are those which are produced for short periods such as ringing of telephone. The impulse type of noise consists of transient pulses, occurring in repetitive or nonrepetitive fashion. The operation of a rivet gun or a pneumatic hammer usually produces repetitive impulse noise. The firing of a gun is an example of nonrepetitive impulse noise.

Physiologically, noise is a complex sound whose characteristics are not easily amenable to analysis and has little or no measurable periodicity. Physiologically, noise is an uninformative signal with variable intensity. Psychologically, any sound that is unpleasant, noxious or unwanted is noise irrespective of its waveform. *Any unwanted sound is noise*. This is the most acceptable definition of noise defined by American National Standard Institute (ANSI). Noise may be classified as continuous (steady-state or fluctuant) or intermittent (impulse or impact). The characteristics of intermittent noise merge into those of continuous noise if the former is repeated very rapidly.

Several key terms describe the qualities of Sound. The basic qualities of sound are:

1. **Wavelength:** The wavelength (λ) is the distance travelled by a sound wave during one second pressure cycle. The wavelength of a sound is usually measured in meters or feet. Wavelength is important for designing engineering controls. For example, a sound-absorbing material will perform most effectively if its thickness is at least one-quarter of the wavelength.
2. **Frequency:** Frequency, *f*, is a measure of the number of vibrations (i.e. sound pressure cycles) that occur per second. It is measures in hertz (Hz), where one Hz is equal to one cycle per second. Sound frequency is perceived as pitch (i.e. how high or low a tone is). The frequency range sensed by the ear varies considerably among individuals. A young person with normal hearing can hear frequencies between approximately 20 Hz and 20,000 Hz. As a person gets older, the highest frequency that he or she can detect tends to decrease. Human speech frequencies are in the range of 250 Hz to 3000 Hz. This is significant because hearing loss in this range will interfere with conversational speech.
3. **Speed:** The speed, at which sound travels, *c*, is determined primarily by the density and the compressibility of the medium through which it is traveling. The speed of sound is typically measured in meters per second or feet per second. Speed increases as the density of the medium increases and its elasticity decreases. For example: (a) In air, the speed of sound is approximately 344 meters per second (1,130 feet per second) at standard temperature and pressure, (b) in liquids and solids, the speed of sound is much higher. The speed of sound is about 1,500 metres per second in water and 5,000 metres per second in steel. The frequency, wave-

length and speed of a sound wave are related by the equation:

$$c = f\lambda$$

where c = speed of sound in metres or feet per second, f = frequency in Hz, and λ = wavelength in metres or feet.

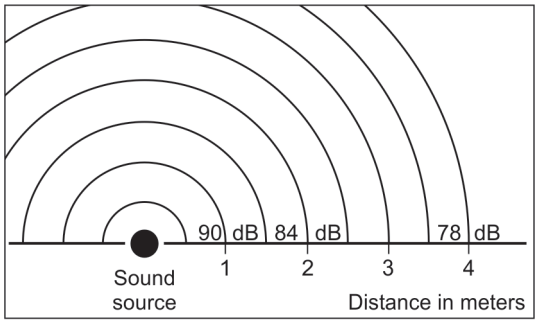
4. **Sound pressure:** The vibrations associated with sound are detected as slight vibrations in pressure. The range of sound pressures perceived as sound is extremely large, beginning with a very weak pressure causing faint sound and increasing to noise so loud that it causes pain. The threshold of hearing is the quietest sound that can typically be heard by a young person with undamaged hearing. This varies somewhat among individuals but is typically in the micropascal range. This reference sound pressure is the standardized threshold of hearing and is defined as 20 micropascals (0.0002 microbars) at 1,000 Hz. The threshold of pain, or the greatest sound pressure that can be perceived without pain, is approximately 10 million times greater than the threshold of hearing. It is therefore, more convenient to use a relative (e.g. logarithmic) scale of sound pressure rather than an absolute scale.
5. **Intensity:** Noise is measured in units of sound pressure called decibels (dB), named after Alexander Graham Bell. The decibel notation is implied any time a 'sound level' or 'sound pressure level' is mentioned. Decibels are measured on a logarithmic scale: A small change in the number of decibels indicates a huge change in the amount of noise and the potential damage to the person's hearing. The decibel scale is convenient because it compresses sound pressures important to human hearing into a manageable scale. By definition, 0 dB is set at the reference sound pressure (20 micropascals at 1,000 Hz). At the upper end of human hearing, noise causes pain, which occurs at sound pressures of about 10 million times that of the threshold of hearing. On the decibel scale, the threshold of pain occurs at 140 dB. This range of 0 dB to 140 dB is not the entire range of sound, but is the range relevant to human hearing. Decibels are logarithmic values, so it is not proper to add them by normal algebraic addition. The decibel is a dimensionless unit; however, the concepts of distance and three-dimensional space are important to understanding how noise spreads through the environment and how it can be controlled. Sound fields and sound power are terms used in describing these concepts.
6. **Sound fields:** Many noise-control problems require a practical knowledge of the relationships between: (a) A sound field (a region in which sound is propagated) and (b) two related concepts: Sound pressure (influenced by the energy, in terms of pressure, emitted from the sound source, the distance from the sound source, and the surrounding environment) and sound power (sound energy emitted from a sound source and not influenced by the surrounding environment).

   Sound fields are categorized as a near field or far field, a distinction that is important to the reliability of the measurement. The near field is the space immediately around the noise source, sometimes defined as within the wavelength of the lowest frequency component (e.g. a little more than 4 feet for a 25 Hz tone, about 1 foot for a 1,000 Hz tone, and less than 7 inches for a 2,000 Hz tone). Sound pressure measurements obtained with standard instruments within the near field are not reliable because small changes in position can result in big differences in the readings. The far field is the space outside the near field, meaning that the far field begins at a point at least one wavelength distance from the noise source. Standard sound

level meters are reliable in this field, but the measurements are influenced by whether the noise is simply originating from a source (free field) or being reflected back from surrounding surfaces (reverberant field).

A free field is a region in which there are no reflected sound waves. In a free field, sound radiates into space from a source uniformly in all directions. The sound pressure produced by the source is the same in every direction at equal distances from the point source. As a principle of physics, the sound pressure level decreases 6 dB each time the distance from the point source is doubled. This is a common way of expressing the inverse-square law in acoustics. Free field conditions are necessary for certain tests, where outdoor measurements are often impractical. Some tests need to be performed in special rooms called free field or anechoic (echo-free) chambers, which have sound-absorbing walls, floors and ceilings that reflect practically no sound.

In spaces defined by walls, however, sound fields are more complex. When sound-reflecting objects such as walls or machinery are introduced into the sound field, the wave picture changes completely. Sound reverberates, reflecting back into the room rather than continuing to spread away from the source. Most industrial operations and many construction tasks occur under these conditions. The net result is a change in the intensity of the sound. The sound pressure does not decrease as rapidly as it would in a free field. In other words, it decreases by less than 6 dB each time the distance from the sound source doubles. Far from the noise source – unless the boundaries are very absorbing – the reflected sound dominates. This region is called the reverberant field. If the sound pressure levels in a reverberant field are uniform throughout the room, and the sound waves travel in all directions with equal probability, the sound is said to be diffuse. In actual practice, however, perfectly free fields and reverberant fields rarely exist – most sound fields are something in between.



If a point source in a free field produces a sound pressure level of 90 dB at a distance of 1 meter, the sound pressure level is 84 dB at 2 meters, 78 dB at 4 meters, and so forth. The principle holds true regardless of the units used to measured distance.

Fig. 8.5: Sound pressure level in a free field

7. **Sound power:** Sound power is an equally concept like sound pressure. Sound power, usually measured in watts, is the amount of energy per unit of time that radiates from the source in the form of an acoustic wave. Generally, sound power cannot be measured directly, but modern instruments make it possible to measure the output at a point that is a known distance from the source.

   Understanding the relationship between sound pressure and sound power is essential to predict what noise problems will be created when particular sound sources are placed in working environments. An important consideration might be how close workers will be working to the source of sound. As a general rule, doubling the sound power increases the noise level by 3 dB.

   A sound power radiates from the source in free space; it is distributed over a spherical surface so that at any given point, there exists a certain sound power per unit area. This is designated as intensity, I,

and is expressed in units of watts per square meter. Sound intensity is heard as loudness, which can be perceived differently depending on the individual and his or her distance from the source and the characteristics of the surrounding space. As the distance from the sound source increases, the sound intensity decreases. The sound power coming from the source remains constant, but the spherical space over which the power is spread increases – so the power is less intense. In other words, the sound power level of the source is independent of the environment. However, the sound pressure level at some distance, *r*, from the source depends on that distance and the sound-absorbing characteristics of the environment.

8. **Filtering:** Most noise is not a pure tone, but rather consists of many frequencies simultaneously emitted from the source. To properly present the total noise of a source, it is usually necessary to break it down into its frequency components. One reason for this is that people react differently to low-frequency and high-frequency sounds. Additionally, for the same sound pressure level, high-frequency noise is much more disturbing and more capable of producing hearing loss than low-frequency noise. Engineering solutions to reduce or control noise are different for low-frequency and high-frequency noise. As a general guideline, low-frequency noise is more difficult to control. Certain instruments that measure sound level can determine the frequency distribution of a sound by passing that sound successively through several different electronic filters that separate the sound into nine octaves on a frequency scale. Two of the most common reasons for filtering a sound include (i) determining its most prevalent frequencies (or octaves) to help engineers better know how to control the sound, and (ii) adjusting the sound level reading using one of several available weighting methods. These weighting methods (e.g. the A-weighted network or scale) are intended to indicate perceived loudness and provide a rating of industrial noise that indicates the impact that particular noise has on human hearing.
9. **Combining and averaging sound levels:** Decibels are measures using a logarithmic scale, which means decibels cannot be added arithmetically. For example, if two noise sources each producing 90 dB right next to each other, the combined noise sound level will be 93 dB, as opposed to 180 dB. The following equation should be used to calculate the sum of sound pressure levels, sound intensity levels or sound power levels:

$$\text{Total } L = 10 \times \log_{10}\left(\sum_1^n 10^{\text{Ln}/10}\right)$$

Often, using this equation to quickly sum sound levels when there is no calculator or computer available is difficult. The following table can be used to estimate a sum of various sound levels:

Difference between two levels	*Amount to add to higher level to find the sum*
0–2 dB	3 dB
3–4 dB	2 dB
5–9 dB	1 dB
10 dB +	0 dB

Example: There are three noise sources immediately adjacent to one another, each producing a sound level of 95 dB. The combined sound level can be found using the table above. The difference between the first two noise sources is 0 dB, which means the sum will be 95 + 3 = 98 dB. The difference between 98 dB and the remaining noise source (95 dB) is 3, which means the sum will be 98 + 2 = 100 dB.

10. **Octave bands (frequency bands):** Octave bands, a type of frequency band, are a convenient way to measure and describe

the various frequencies that are part of a sound. A frequency band is said to be an octave in width when its upper band-edge frequency, f_2, is twice the lower band-edge frequency.

11. **Loudness and weighting networks:** Loudness is the subjective human response to sound. It depends primarily on sound pressure but is also influenced by frequency. Three internationally standardized characteristics are used for sound measurement: Weighting networks A, C, and Z (or 'zero' weighting). The A and C weighting networks are the sound level meter's means of responding to some frequencies more than others. The very low frequencies are discriminated against (attenuated) quite severely by the A-network and hardly attenuated at all by the C-network. Sound levels (dB) measured using these weighting scales are designated by the appropriate letter (i.e. dBA or dBC). The A-weighted sound level measurement is thought to provide a rating of industrial noise that indicates the injurious effects such noise has on human hearing and has been adopted by OSHA in its noise standards. In contrast, the Z-weighted measurement is an unweighted scale (introduced as an international standard in 2003), which provides a flat response across the entire frequency spectrum from 20 Hz to 20,000 Hz. The C-weighted scale is used as an alternative to the Z-weighted measurement (on older sound level meters on which Z-weighting is not an option), particularly for characterizing low-frequency sounds capable of inducing vibrations in buildings or other structures. A previous B-weighted scale is no longer used.

    A-weighted sound level: A person's ability to hear a sound depends greatly on the frequency composition of the sound. People hear sounds most readily when the predominant sound energy occurs at frequencies between 1000 and 6000 Hz. Sounds at frequencies above 10,000 Hz, such as high-pitched hissing, are much more difficult to hear, as are sounds at frequencies below about 100 Hz, a low rumble, for example. To measure sound on a scale that approximates the way it is heard by most people, more emphasis must be given to the frequencies that people hear more easily. The most commonly used scales on sound level meters are the C scale, which gives a flat, equally weighted response across the entire spectrum, and the A scale, which gives less emphasis to low-frequency sounds. The A scale is often used in noise measurements, since it provides more weight to the annoying high frequencies. A sound level meter's A-weighting network is thought to rate most industrial broad-band noises much the same as the human ear does. Because of its simplicity and accuracy in rating hearing hazards, the A-weighted sound level has become the method recommended for measuring noise exposure by the American Conference of Governmental Industrial Hygienists (ACGIH) as well as the US Department of Labour as part of the Occupational Safety and Health regulations.

Population at Risk and Save Noise Exposure

The National Institute of Occupational Safety and Health (NIOSH) has stated that millions of workers in the United States have compensable noise-induced hearing loss. According to NIOSH estimates, 14% of the working populations are employed in environments where the noise level exceeds 90 A-weighted decibels (dBA). If individuals work in an environment where the predominant noise level exceeds 90 dBA for a number of months or years, some workers will develop hearing impairments. The number of workers exposed to noise hazards on the job far exceeds the number exposed to any other significant occupational hazard.

It is impossible to establish any clear-cut distinction between 'safe' and 'unsafe' noise exposure/criteria for noise exposure that have been proposed are based generally upon the results of studies involving exposures to continuous noise. Whether these predictions will also hold true for intermittent exposures to both continuous and impulse noise is somewhat controversial. It is generally agreed that after repeated excessive noise exposure, some individuals can incur a hearing loss. There is a considerable difference of opinion among the acoustic experts as to the boundary that separates the harmless from the harmful noises. Because of the normal variation in susceptibility between individuals, it is not possible scientifically to set a realistic standard for exposure of noise that will protect everyone who is exposed. Generally, limits are set with the intension of protecting 90% or more of an exposed population.

Assessing the amount of hearing loss in employees due to noise exposure in the workplace has always been a difficult task. Outside of working hours, workers often engaged in recreational activities in which noise intensity reaches hazardous levels. Generally, the hearing damage due to off-work activities is considered to be negligible when the noise exposure of these activities is low compared with that at the workplace. In such cases, compensation is awarded on the assumption that all the hearing loss due to noise is work-related. The question is how to determine whether the noise exposure from activities such as hunting and motorcycling, which are unrelated to work, is significant.

Another complicating factor is the difficulty, if not the impossibility, of determining what portion of an employee's hearing loss is due to the normal aging process, medications, lifestyle, and disease capable of producing sensorineural pathology. Presbycusis is the loss of hearing that takes place with increasing age. Such a loss occurs even for persons who are ontologically normal and have not been exposed to high occupational noise levels.

Determining that a hazardous level of noise exists at the workplace is one element in providing that a hearing impairment is work-related. The degree of noise exposure can be determined from noise monitoring records, if available. Problems arise when no previous noise measurements have been taken. If present noise levels are below 90 dBA, it is difficult to determine what the earlier exposures were, particularly in cases where the work environment has been modified.

Anatomy and Physiology of Human Ear

Structurally and functionally, the human ear is divided into three parts, namely the outer ear, middle ear and inner ear. Together with the central connections of the ear in the brain, each part plays a role in the process of hearing (Fig. 8.6).

The outer ear: The outer ear or external ear comprises the auricle (pinna) and the external auditory canal. Collectively, these serve to modify incoming sound in two specific ways. First, the combination of the auricle behaving as an ear trumpet, concentrating sound from a large area to the smaller area of the external canal and the natural resonances of the external canal itself serves to increase the sound pressure level at the tympanic membrane by about 10 dB over a frequency range of 2000–7000 Hz. Secondly, the outer ear provides some information about sound localization.

The middle ear: The middle ear consists of tympanic membrane (eardrum) and within the cavity there are three ossicles ('malleus, incus and stapes' or 'hammer, anvil and stirrup') linking the drumhead to the inner ear. The middle ear cavity communicates with the pharynx at the back of the nose by the Eustachian tube and with the mastoid air cells by the aditus and antrum. The fundamental function of the middle ear is mechanically to couple acoustic energy to the cochlea as an acoustic sound pressure transformer. The effect of this, in combination with the effects of the outer ear already described means that up to 50% of the incident sound energy is

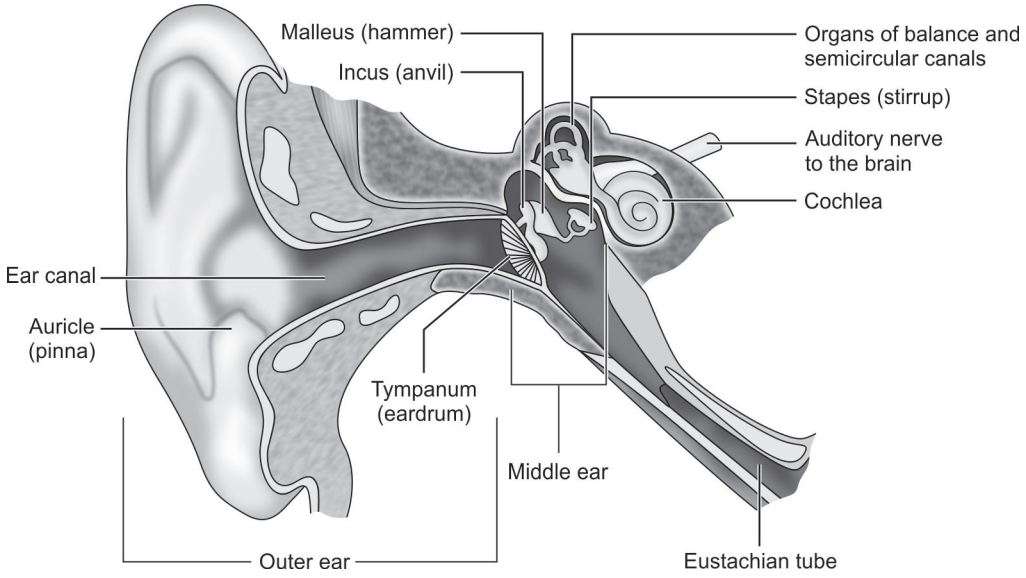


Fig. 8.6: Human ear

transmitted to the inner ear as opposed to the expected 1% in the absence of the transformer. This is achieved by two mechanisms. The leverage of the malleus and incus about their axis of rotation gives a ×1.3 mechanical gains. The difference in the functional surface area between the drumhead and the stapes footplate which sits in the oval window gives rise to a 14-fold hydraulic effect. Combining the two, provides an increase in pressure at the oval window by a factor of around 18.

The inner ear: The inner ear consists of two parts—the osseous labyrinth and the corresponding membranous labyrinth which lies within it. Descriptively, the labyrinth comprises the semicircular canals and vestibule which house the sensory end organs of balance and the cochlea within which is the hearing organ. Lying in the medial wall of the middle ear, the cochlea is shaped like a snail's shell with two or three-quarter turns, 5 mm in height and 9 mm across the base. The central bony axis of the cochlea (modiolus) has a spiral bony lamina projecting from it along its length which is completed by the basilar membrane. On this lies the cochlear duct (membraneous labyrinth) effectively dividing the cochlear lumen into three compartments, the scala vestibule (in continuity with the vestibule, containing perilymph), the scala tympani (in continuity at the apex of the cochlea with the scala vestibule and closed by the round window membrane) and the scala media (containing endolymph), within which is the organ of Corti. The organ of Corti is a specialized area of the lining of the cochlear duct that runs the whole length of the cochlear spiral and is around 35 mm long. The sensory cells of the organ of Corti are arranged in one inner row and three to five outer rows. Microfilaments (stereocilia) from the sensory cell surface have given rise to the descriptive term sensory hair cells. This represents the interface where mechanical energy is converted to electrical energy: The organ of Corti is thus a specialized transducer. From the sensory cells, afferent nerve fibres pass together as the cochlear nerve to the brainstem and ultimately to the auditory cortex of the brain.

Hearing Process

The outer ear collects acoustic energy which the eardrum converts to mechanical energy. The middle ear transfers sound energy from the outer ear to the inner ear. As the eardrum

vibrates, its motion is transferred to the attached malleus (hammer). Because the bones of the ossicular chain are connected to each other, the movements of the malleus are passed on to the incus (anvil), and finally to the stapes (stirrup), which is imbedded in the oval window. The movement of the footplate of the stapes within the oval window transforms this mechanical energy into hydraulic energy, setting the perilymph within the inner ear in motion. The manner in which the hydraulic energy is transformed into electric impulses to be sent by the auditory nerve to the brain is unclear. According to one hypothesis, the hair fibers from the hair cells embedded in the tectorial membrane permit the shearing action of the hair fibers to transfer varying amounts of energy to the hair cells. The hair cells then stimulate the hearing nerve-endings.

Threshold of Hearing

Threshold of hearing for a specified sound is the minimum sound pressure level of the sound that is capable of evoking an auditory sensation. The hearing threshold for an individual is not a sharp boundary but is defined in terms of the probability that a sound will be heard. The threshold depends on the characteristics of the sound, on the manner in which it is presented to the listener, and on the point at which the sound pressure level is measured (for instance, it can be measured at the entrance of the ear canal or in the free field in absence of the listener). To describe a hearing threshold, all of the above factors must be specified. Threshold shifts, expressed in decibels, is the difference between the hearing threshold levels measured before and after noise exposure. If the shift is reversible (i.e. if the ear recovers completely after noise exposure, so that the threshold shift is reduced to zero), the threshold shift is said to be temporary; if the ear does not recover completely, the threshold shift is said to be permanent. If the threshold shift is temporary, the hearing threshold returns to its values measured before exposure. The magnitude of temporary threshold shift depends on the time interval elapsing between the cessation of noise exposure and the measurement of threshold.

Permissible Exposure Limit of Noise: OSHA Standard

Under Occupational Safety and Health Administration (OSHA) standard, workers are not permitted to be exposed to an 8-hours TWA equal to or greater than 90 dBA. OSHA uses a 5 dBA exchange rate, meaning the noise level doubles with each additional 5 dBA. The following table shows how long workers are permitted to be exposed to specific noise levels:

Permissible duration (hours per day)	*Sound level (dBA)*
16	85
8	90
4	95
2	100
1½	102
1	105
½	110
¼ or less	115

To calculate the permissible duration, the following equation is to be used:

$$T = 8/(2^{(L-90)/5})$$

where T is the permissible duration (in hours) and L is the measured sound level (in dBA).

A worker's daily noise exposure typically comes from multiple sources, which have different noise levels of different durations. When adding different noise levels from various noise sources, only noise levels exceeding 80 dBA should be considered. The combined effect of these noise sources can be estimated using the following equation:

$$\text{Sum} = C_1/T_1 + C_2/T_2 + C_3/T_3 .... + C_n/T_n$$

where C_n is the total duration of exposure at a specific noise level, and T_n is the total duration

of noise permitted at that decibel level. If the sum equals or exceeds '1', the combined noise level is greater than the allowable level. If the sum is less than '1', the combined noise level is less than the allowable level.

Schedule XXIV of Indian Factories Act, 1948, operation involving high noise (Model Factories Rules 120 under Section 87):

1. Application: This part of the Schedule shall apply to all operations in any manufacturing process having high noise level.
2. Definition: For the purpose of the schedule:
   a. 'Noise' means any unwanted sound.
   b. 'High noise level' means any noise level measured on the A-weighted scale is 85 dB or more.
   c. 'Decibel' means one-tenth of 'Bel' which is the fundamental division of a logarithmic scale used to express the ratio of two specified or implied quantities, the number of 'Bels' denoting such a ratio being the logarithm to the base of 10 of this ratio. The noise level (or the sound pressure level) corresponds to a reference pressure of 20 × 10 Newton per square meter or 0.0002 dynes per square centimeter which is the threshold of hearing, that is, the lowest sound pressure level necessary to produce the sensation of hearing in average healthy listeners. The decibel in abbreviated form is dB.
   d. 'Frequency' is the rate of pressure variations expressed in cycles per second or hertz.
   e. 'dBA' refers to sound level in decibels as measured on a sound level meter operating on the A-weighting net work with slow meter response.
   f. 'A-weighting' means making graded adjustment in the intensities of sound of various frequencies for the purpose of noise measurement, so that the sound pressure level measured by an instrument reflects the actual response of the human ear to the sound measured.
3. Protection against noise: In every factory, a suitable engineering control or administrative measures shall be taken to ensure, so far is reasonably practicable, that no worker is exposed to sound levels exceeding the maximum permissible noise exposure levels specified in the tables:

Permissible exposure of continuous noise

Total time exposure (continuous short-term exposure)	*Sound pressure level in or a number of dBA per day (in hours)*
8	85
6	87
4	90
3	92
2	95
1½	97
1	100
3/4	102
½	105
¼	110

Note: 1. No exposure in excess of 110 dBA is to be permitted.
2. For any period of exposure falling in between any figure and the next higher or lower figure as indicated in column 1, the permissible sound pressure level is to be determined by extrapolation on a proportionate basis.

Permissible exposure levels of impulsive or impact noise

Peak sound pressure level in dBA	*Permitted number of impulse or impact per day*
140	100
135	315
130	1000
125	3160
120	10000

Note:1. No exposure in excess of 140 dB peak sound pressure level is permitted.
2. For any peak sound pressure level falling in between any figure and the next higher orlower figure as indicated in column 1, the permitted number of impulses or impacts perday is to be determined by extrapolation on a proportionate basis.

2. For the purposes of this Schedule, if the variations in the noise level involve maximum at intervals of one second or less, the noise is to be considered as a continuous one and the criteria given in the table

of continuous noise would apply. In other cases, the noise is to be considered as impulsive or impact noise and the criteria given in would apply.

3. When the daily exposure is composed of two or more periods of noise exposure at different levels their combined effect should be considered, rather than the individual effect of each. The mixed exposure should be considered to exceed the limit value if the sum of the fractions $C_1/T_1 + C_2/T_2 + ... + C_n/T_n$, exceeds unity, where the C_1, C_2, etc. indicate the total time of actual exposure at a specified noise level and T_1, T_2, etc. denote the time of exposure of less than 90 dBA may be ignored in the above calculation.
4. Where it is not possible to reduce the noise exposure to the levels specified in subclause (1) by reasonably practicable engineering control or administrative measures, the noise exposure shall be reduced to the greatest extent feasible by such control measures, and each worker so exposed shall be provided with suitable ear protectors as per relevant national or international standards so as to reduce the exposure to noise to the levels specified in subclause 3(1).
5. The occupier shall provide personal hearing protectors to the workers:
   a. So as to eliminate the risk to hearing or to reduce the risk to as low a level as is reasonably practicable.
   b. After consultation with the employees concerned or their representative.
   c. Ensure the hearing protectors is full and properly fitted, periodically checked for the effectiveness, used and maintained in good working order and repair.
   d. Ensure that workers are given periodical training in the use, care and maintenance of the personal hearing protectors.
5. Where the ear protectors provided in accordance with subparagraph 3(4) and worn by the worker cannot still attenuate the noise reaching near his ear, as determined by subtracting the attenuation value in dBA of the ear protectors concerned from the measured sound pressure level, to a level permissible under continuous or impact noise as the case may be, the noise exposure period shall be suitably reduced to correspond to the permissible noise exposure specified in subpara-graph (1).
6. a. In all cases where the prevailing sound levels exceed the permissible levels specified in subparagraph (1) there shall be administered an effective hearing conservation programme which shall include among other hearing conservation measures, pre-employment and periodical auditory surveys conducted on workers exposed to noise exceeding the permissible levels, and rehabilitation of such workers either by reducing the exposure to the noise levels or by transferring them to places where noise levels are relatively less or by any other suitable means.
   b. Every worker employed in areas where the noise exceeding the maximum permissible exposure levels specified in subclause (1) shall be subjected to any auditory examination by a certifying surgeon within 14 days of his first employment and thereafter, shall be re-examined at least once a every 12 months. Such initial and periodical examination shall include tests which the certifying surgeon may consider appropriate and shall include determination of auditory thresholds of pure tones of 125, 250, 500, 1000, 2000, 4000, 8000 cycles per second.

Instruments for Ambient Noise Measurement

There is a wide variety of instrumentation available for the evaluation of noise exposure,

from very simple equipment to extremely sophisticated equipment used by acoustics engineers and consultants. Noise level measurements for steady-state or continuous noise should be made with a sound level meter set to an A-weighted slow response or with an audio dosimeter of equivalent accuracy. For the measurement of impact noise (such as that from a drop hammer), an impact-noise meter with peak hold capability should be used. Sound level measuring instrument should be calibrated using an acoustic calibrator, preferably before and after the noise measurements. Instruments used for monitoring employee exposures must be carefully checked or calibrated to ensure that the measurements are accurate. Calibration procedures are unique to specific instruments. Employers have the duty to assure that the measuring instruments they are using are properly calibrated. They may find it useful to follow the manufacturer's instructions to determine when and how extensively to calibrate.

Relative intensity of common noise

Activity	*Noise Level (dBA)*
Normal conversation	50–60
Motorcycle	110
Firecrackers	150
Hunting weapon	160
Printing machine	90
Truck transportation	90
Textile mill	100
Underground mining	110
Metal-tool operations	110
Heavy equipment	110
Gunfire by field weapon	190
Gunfire by hand-held weapon	160

Evaluation of Hearing

1. **Test of spoken words:** The simplest form of hearing evaluation may be performed in a quiet room without any sophisticated equipment. The patient is asked to repeat spoken words of increasing intensity while competing noise (the crumpling of paper or the sounds from a Barany noise box) are presented to the opposite ear. Test results may be expressed as the ability to hear a soft whisper, loud whisper, soft-spoken voice, loud-spoken voice or shout.

Fig. 8.7: Noise level meter

2. **Tuning fork tests:** Tuning fork test should be performed with a 512 Hz tuning fork because frequencies below this level will elicit a tactile response. Tuning fork tests are not used in rating hearing impairment because they are qualitative rather than quantitative measurements. From a diagnostic standpoint, however, these tests do provide the physician with valuable preliminary information concerning whether the hearing loss is conductive or sensorineural in nature and whether one or both ears is/are involved. Generally, the otologist will make these tests to confirm audiometric test results. Three tests are routinely employed: (a) The Rinne test compares the subject's ability to hear the tuning fork by airborne sound with his or her ability to hear it by bone conduction, (b) the Weber test determines which ear, if either, hears the tuning fork better by bone conduction, and (c) the Schwabach test compares the subject's ability to hear the fork by bone conduction with the ability to a listener with normal hearing to hear it in the same position. Usually, the otologist makes the comparison to his or her own hearing.
3. **Pure-tone audiometry:** Pure-tone audiometry is the measurement of hearing. The results of such testing are recorded on an

audiogram, a graph showing hearing (threshold) level as a function of frequency. Hearing (threshold) level is the number of decibels that the subject's threshold of hearing lays above the zero reference for the frequency. Thus, a pure-tone audiogram compares the hearing of the person being tested with a standard hearing level considered to be 'normal' (i.e. the audiometric zero reference level).

Normal hearing is the median hearing level of a large group of young adults of ages between 18 and 25 years having no known history of ear disease and no appreciable exposure to high-level noise. A set of sound pressure levels which represent the normal hearing threshold has been established as the zero reference level for audiometry.

A manual, pure-tone audiometer is an instrument in which the tester has control of the frequency and sound pressure level of the pure tones presented to the subject. The employee whose hearing is being tested responds to the test tone by raising a finger or hand or pushing a signal button. The technician selects a particular frequency, presents short tone pulses with a tone-interrupter switch, manipulates the hearing-level dial to determine the subject's hearing level, and records that level.

Audiometric testing not only monitors the sharpness or acuity of an employee's hearing over time, but also provides an opportunity for employers to educate employees about their hearing and the need to protect it.

Audiometer: The manual audiometer presents a variable sound level in an earphone. The audiometric technician measures at each frequency the minimum sound pressure level that the person can hear. The decibel difference between audiometric zero and the threshold is reported as the subject's hearing level, or hearing loss, at each frequency. A pure-tone audiometer is essentially an electroacoustic generator that produces pure tone at predetermined frequencies and calibrated sound intensities. The frequency range is typically from 125 to 8000 Hz and divided into discrete steps. The intensity is variable in discrete 5 dB steps. Threshold measurement is done in 5 dB intervals on standard manual audiometers.

Hearing testing is a most critical part of the examination. Proper hearing tests imply use of adequate sound-treated hearing test room (audiometry booth), carefully calibrated audiometric equipment, and trained technicians. Bone conduction pure-tone tests and air conduction pure-tone tests may all be required.

Fig. 8.8: Audiometry booth

Air-conduction (AC) threshold: The pure-tone air conduction test measures the sound conducted to the eardrum through air. A pure-tone threshold for each tested frequency is determined by presenting a pure tone at the test frequency at intensities varies according to an established pattern. The threshold represents the lowest intensity at which a person is able to hear approximately 50% of the tone presentations. The subject's hearing thresholds are established at various octave intervals, usually from 250 through 8000 Hz, with interoctave interval of 3000 Hz usually measured as well.

Bone-conduction (BC) threshold: Although the air-conduction test establishes the subject's hearing thresholds, it does not determine definitely whether the hearing loss, if any, is of conductive or sensorineural

(perceptive) type. To make this determination, the bone-conduction threshold test is made. In this test, sound vibrations generated by a bone vibrator held against the skull (mastoid bone) are conducted directly to the inner ear through the skull (mastoid bone), bypassing the outer and middle ear. If a hearing loss (Fig. 8.9) is indicated by the air conduction test, but normal hearing is indicated by the bone-conduction test, the hearing loss is conductive rather than sensorineural. Such a result indicates the loss is not due to the prolonged exposure to noise. Bone-conduction can be estimated up to 4000 Hz in the pure tone audiometric test.

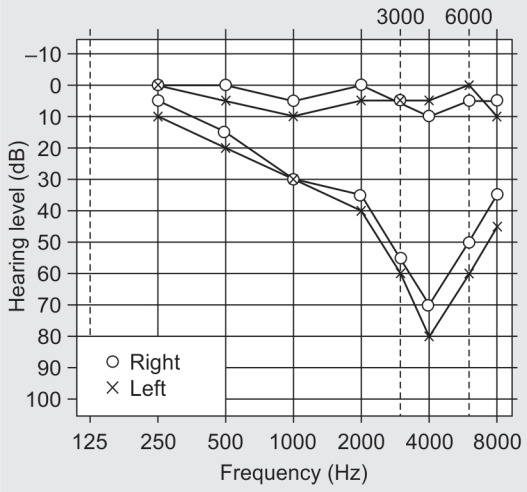


Fig. 8.9: An example of a typical audiogram from a normal individual (dashed lines) and an individual with a bilateral sensorineural hearing loss resulting from excessive noise exposure. Note the maximum loss at 4,000 Hz and the spread of loss to the lower frequencies

Masking: Masking is the stimulation of one ear of the subject by controlled noise to prevent the subject from hearing, with that ear, the tone or signal given to the other ear. Since there is a chance that the test signal will cross over through the bones in the skull to the nontest ear, the nontest ear must be masked to eliminate it from the measurement process.

Unmasked bone-conduction thresholds indicate only that the better inner ear is functioning at the levels measured. No statement can be made about the other ear without masked thresholds, unless hearing in each ear is normal or the air-conduction threshold for each ear is equal and the unmasked bone-conduction threshold equals the air-conduction threshold. The examiner's skill is critical in obtaining bone-conduction thresholds. It is not uncommon for too much masking to be presented to the nontest ear. This masking then crosses back over the skull and interferes with the test ear results.

Audiogram: An audiogram basically consists of a graph with frequency indicated along the top or horizontal axis and intensity along the side or vertical axis.

The frequency scale across the top of the audiogram is expressed in either cycles per second (cps) or Hertz (Hz). The numbers on the left side of the audiogram indicate the intensity or the loudness of the sound, which is measured in decibels. The smaller the number, the fainter the sound.

When the subject's hearing is measured, the threshold of hearing (Fig. 8.10) at each test frequency is established at the intensity level at which the sound can just barely be heard. The further a subject's threshold is below the zero line of audiogram, the greater the loss of hearing. Generally, the pure-tone thresholds determined by air conduction for the right ear

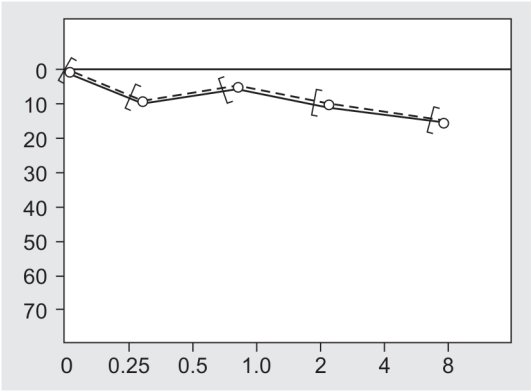


Fig. 8.10: Normal hearing

are recorded on the audiogram as circles (O) and those for the left ear are recorded as Cross (X).

Bekesy audiometry: Pure-tone threshold can also be measured by Bekesy audiometry, in which the patient uses self-directed techniques that involve pressing and releasing a signal button. This procedure is used in some occupational screening programs, but it is generally not as reliable as procedures that are administered by an audiologist.

Speech audiometry: Ability to hear speech is the preferred standard for rating hearing loss for compensation purposes. It has, however, been impossible standardized speech test materials because of language difficulties and the differences in intelligence and educational levels of the persons whose hearing is being tested. Because of these variables, pure-tone threshold audiometry is recognized as the most reliable method available for establishing a quantitative measure of hearing acuity. Speech audiomtry, which provides essential diagnostic information concerning the type and cause of hearing loss, is used to supplement pure-tone audiometry. Two commonly used speech tests are the *Speech Reception Test* and the *Speech Discrimination Test*.

Impedance (immittance) audiometry: The mechanical aspects of the middle ear sound transformer system can be assessed by *Tympanometry* and *Acoustic Reflex Testing.*

Evoke-response audiometry (brain stem audiometry): In patients who demonstrate unilateral or asymmetric sensorineural hearing loss, retrocochlear lesions (lesions of the eighth cranial nerve, brain stem, or cortex) must be ruled out. Evoked potentials, which typically are elicited in response to clicking noises and recorded via scalp electrodes, provide information about the location of sensorineural lesions. For individuals with normal hearing, as well as most patients with cochlear hearing losses, a series of five electroencephalographic waves may be detected, representing the central auditory system from the eighth cranial nerve (wave 1) to the inferior colliculus (wave 5). The discovery of any significant delay or even a complete absence of response may indicate a cerebellopontine angle tumour (e.g. acoustic neuroma) or a lesion of the brain stem. More definitive diagnosis of retrocochlear lesions requires computed tomographic (CT) scanning or magnetic resonance imaging (MRI).

Stenger test: The test is useful for detecting feigned unilateral hearing loss. The Stenger principle states that when two tones of the same frequency but of different loudness are presented to both ears simultaneously, only the loud tone will be heard. When the louder tone is presented to the ear with a feigned hearing loss, the patient stops responding because the patient perceives that all the sound is coming from that side. Patients with true unilateral loss indicate that they continue to hear the sound in the opposite ear.

Otoacoustic emissions: Otoacoustic emissions (OAEs) are a recent addition to objective hearing testing. OAEs are produced when the cochlea receives an external sound stimulous, and the mechanical properties of the outer hair cells act in a manner in which a measurable sound is produced and emanated laterally through the middle ear to be recorded in the external auditory meatus. There are two types of evoked OAEs used clinically today. They are transient-evoked otoacoustic emissions (TEOAEs) and distortion-product otoacoustic emissions (DPOAEs). OAEs are useful in occupational hearing loss because OAEs are rapid (30 seconds to 3 minutes), reproducible, frequency-specific (1000–10,000 Hz) and asses the injury to the outer hair cells caused by noise. However, OAEs do not test inner hair cells, the auditory nerve, and higher auditory structures, their current role in occupational hearing loss will be in screening and supporting pure-tone audiometry.

Functional hearing test: The hearing in noise test (HINT) is a direct measure of functional speech perception in noise. This test has been validated for use in screening

applications for hearing-critical jobs. The HINT measures speech intelligibility in quiet and spectrally matched noise at supra-threshold levels using sentence materials.

Identification of Deafness in Audiogram

Conductive deafness: Hearing loss may occur when there is a difficulty in conducting sound waves to the inner ear such as blockage in the outer ear, rupture of the tympanic membrane, ossicle fixation, breakage, etc. The inner ear perception response remains intact. Although, work-related conductive hearing loss is not common, it can occur when an accident results in a head injury or penetration of the eardrum by a sharp object, or by any event that ruptures the eardrum or breaks the ossicular chain formed by the small bones of the middle ear (e.g. impulsive noise caused by explosives or firearms). The audiometric pattern of a typical conductive deafness is that the air conduction is below the normal range (25 dB) and bone conduction is within normal range (25 dB). Conductive hearing loss may be reversible through medical or surgical treatment. It is characterized by relatively uniformly reduced hearing across all frequencies in tests of the ear, with no reduction during hearing tests that transmit sound through bone conduction.

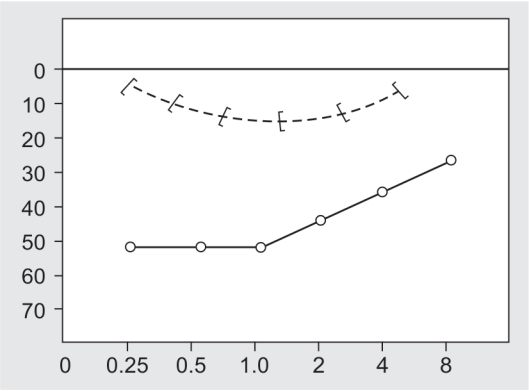


Fig. 8.11: Conductive deafness

Sensorineural or perceptive deafness: Sensory neural hearing loss is a permanent condition that usually cannot be treated medically or surgically and is associated with irreversible damage to the inner ear. It is a permanent damage. The normal aging process (presbycusis) and excessive noise exposure in industry (NIHL) or environment (sociocusis) are notable causes of sensory neural hearing loss. Studies show that exposure to noise damages the sensory hair cells that line the cochlea. Even moderate noise can cause twisting and swelling of hair cells and biochemical changes that reduce the hair cell sensitivity to mechanical motion, resulting auditory fatigue. As the severity of noise exposure increases, hair cells and supporting cells disintegrate and the associated nerve fibers eventually disappear. Occupational noise exposure is a significant cause of sensory neural hearing loss, which appears on sequential audiograms as declining sensitivity to sound, typically first at high frequencies (above 2000 Hz), and then lower frequencies as damage continues. Often the audiogram of a person with sensory neural hearing loss will show a 'NOTCH' at 4,000 Hz. This is a dip in the person's hearing level at 4,000 Hz and an early indicator of sensory neural hearing loss. Though many theories are offered to explain notch at 4,000 Hz in noise induced hearing loss (NIHL), the acceptable one is '*Mechanical Theory of Tanndorf*'. According to this theory, the region in the cochlea for perception of 4,000 Hz is set into vibration when responding to any acoustic stimuli and hence this region is subjective to more torsion. As the condition becomes worse the neighbouring areas are also destroyed and thus hearing loss widens. Sensory neural hearing loss may also appear without the notch at 4,000 Hz. The typical audiometric pattern is that both air-conduction and bone-conduction responses are below the normal range and both the responses are equal. Sensory neural hearing loss can also result from other causes, such as viruses (e.g. mumps), congenital defects, and some medications (like streptomycin).

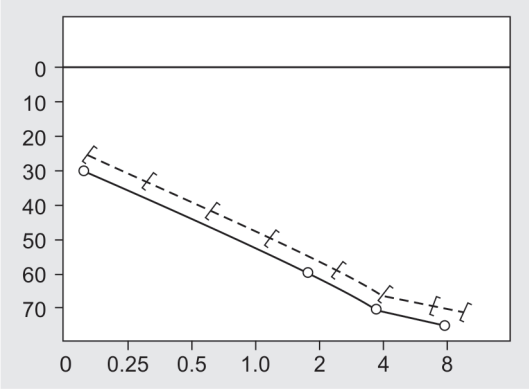


Fig. 8.12: SN deafness

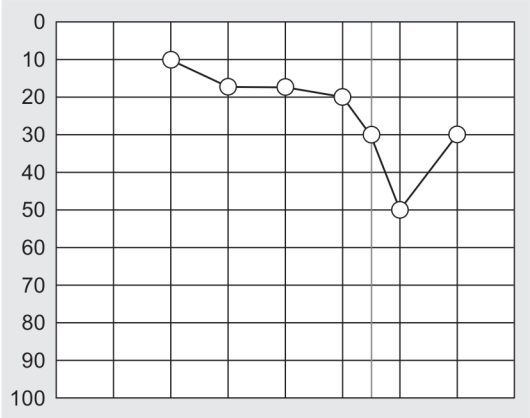


Fig. 8.13: NIHL with notch at 4000 Hz

Mixed type deafness: Both conductive and perceptive deafness may be present in the same ear. The audiometric pattern is that both air-conduction response and bone-conduction response are below the normal range and there is a gap between these two responses. Example of *injury causing mixed conductive and sensorineural hearing loss:* Temporal bone injuries sometimes involve both the middle and inner ear, resulting in mixed, conductive and sensorineural hearing loss.

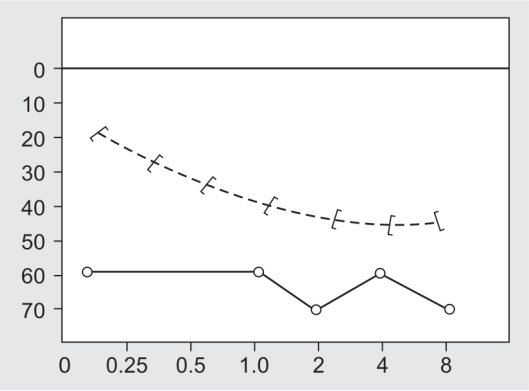


Fig. 8.14: Mixed type deafness

Health Effects of Noise

Auditory effects: One of the earliest descriptions of the adverse effects of noise on hearing was by Francis Lord Bacon in 1627. He relates his own experience of what was a temporary threshold shift. Nearly a century later, a report by Ramazzini recognized the relationship between copper hammering and hearing impairment and in 1782, Admiral Lord Rodney was deafened for two weeks following the firing of 80 broadsides from his ship HMS Formidable. In 1831, Fosbroke accurately described noise-induced hearing loss in blacksmiths and coined the phrase 'blacksmith's deafness'. It was the arrival of industrial age, however, that led to a more widespread recognition of the deleterious effects of intense or prolonged noise on hearing: Roosa and Holt in the United States of America, Bezold in Germany and Barr in Great Britain. Following the introduction of audiometry, Fowler in 1929 observed the characteristic 4-kHz dips and the first systematic audiometric studies were reported in 1939 by Bunch and by Larsen. The technological advancements seen after the Second World War paralleled by ever-increasing noise levels in the workplace. While loss of earning due to hearing loss caused by acute trauma at work was compensable from the early part of the last century, it is only about in the last few years that the legislative bodies of developed countries have recognized occupational noise-induced hearing loss, put into place mechanisms to confer responsibility on the manufacturing industries and compensate those individuals deemed to suffer this disorder.

Occupational noise-induced hearing loss can be defined as a partial or complete hearing

loss in one or both ears arising in, or during the course of, and as the result of one's employment. This includes acoustic traumatic injury as well as noise-induced hearing loss. Noise-induced hearing loss is generally used to denote the cumulative, permanent loss of hearing that develops gradually after months or years of exposure to high levels of noise.

Occupational noise-induced hearing loss arising out of traumatic injury, such as explosions or a blow to the head is compensable under the Workman's Compensation Act. Noise levels much lower than those producing acoustic trauma may produce hearing loss if the exposure is sufficiently intense and prolonged. This type of hearing loss is termed sensory neural loss. Sensory neural hearing impairment cannot be assessed in terms of the loss of sensory cells and neurons; instead, some change in hearing function must be measured. The changes in function that are commonly measured are (1) the hearing threshold sensitivity for pure tones and (2) some index of the ability to hear and understand speech. The determination of hearing threshold sensitivity for pure tones in a quiet environment is a standard procedure. Methods for assessing the ability to understand speech have not been well standardized, because the understanding of speech is affected by such variables as vocabulary, education, intelligence, and the nature of speech test material, in addition to hearing ability.

Noise exposure can produce a permanent hearing loss in exposed individuals, and whether it does is dependent upon a number of factors, including (1) the sound intensity, (2) the length of time an employee is exposed to the noise, and (3) individual susceptibility to noise-induced hearing loss. The more intense the given noise, the more it will produce hearing loss – also, the longer an employee is exposed to a particular noise, the greater the probability of injury to the auditory system from that noise. Individual vary a great deal in term of how much hearing loss they will develop from exposure to a particular noise level for a particular length of time. If a group of workers were exposed to the same noise level over their working lifetime, some would have their hearing affected more rapidly and to a much greater extent than others.

Occupational noise-induced hearing loss is considered one of the most common occupational disorders in industrial countries. Apparently, between 1 and 4 per cent of the population are exposed to harmful or potentially harmful noise levels. The rigorous epidemiological study in 1980 (UK National Study of Hearing) found that 12% in the UK had a sensorineural hearing defect. Around one-third of these were accounted for by age and 5% by noise. This would imply that around 1 in 200 of the adult population has noise-related sensorineural hearing impairment. Variations in individual susceptibility are now considered to be multi factorial.

Auditory effects of sound stimulation and hazardous noise

Adaptation (perstimulatory fatigue): This is an immediate physiological phenomenon that occurs when a sound is presented to the ear. For sound pressure levels up to 80 dB, the greatest elevation in threshold occurs at the same frequency of the stimulating tone. There follows an exponential recovery which occurs within 1 second. For higher stimulating sound intensities, true temporary threshold shift sets in though the intensity of stimulation required varies from individual to individual and depends on the frequency of the stimulating tone.

Noise-induced temporary threshold shift (NITTS)/post-stimulatory fatigue: The magnitude of a temporary threshold shift is proportional to the intensity and the duration of the stimulus and the recovery, unlike adaptation is slow. Recovery usually occurs within 16 hours, but take several days with higher intensities. The risk of developing permanent threshold shift has been studied by Mills and

is complex. Interactions between the level, nature and number of noise exposures, their duration and frequency and the susceptibility of the individual are all factors. This is often accompanied by tinnitus and after a short rest period, the rise in threshold recovers. With repeated exposure, there is a tendency to acquire resistance to the auditory effects in that the degree of temporary threshold shift lessens though at some arbitrary point, continued exposure leads to a permanent threshold shift.

Noise-induced hearing loss (NIHL) or noise induced permanent threshold shift (NIPTS): This is characterized by irreversible audiometric effects and pathological changes in the cochlea. The 4 kHz notch tends to deepen but also insidiously widens, encompassing adjacent high frequencies. Classically, there is a high frequency rise in threshold with a characteristic notch at 4000 Hz, though the notch may centre at 3000 or 6000 Hz. Once the audiometric changes encroach upon the speech frequencies (2 and 3 kHz in particular), the effected individual becomes aware of the diminished acuity in his or her hearing. Speech discrimination with background noise becomes difficult and the associated tinnitus (which is highly variable in character) may become intrusive. The rate of progression again depends on the noise parameters cited previously and on individual susceptibility. Generally, progression at 4 kHz is initially rapid, but slows down after 10–12 years. Progression to involve the lower frequencies is associated with a flattening of the audiogram in the highest frequencies such that the characteristic notched audiogram is not a prerequisite for a diagnosis of occupational noise-induced hearing loss. At this stage, the impaired speech discrimination in noise is accompanied by the complaint of sound generally being too quiet. With the progression of time, the increasing effects of ageing on auditory function come into play and this aspect assumes importance in the evaluation process of those being assessed with possible occupational noise-induced hearing loss.

The Department of Health and Social Security publication 'Occupational Deafness' states that apart from the characteristic audiometric changes, there are no signs and symptoms specific to noise-induced deafness. This implies that the diagnosis of occupational noise induced hearing loss in an audiometric one. However, the characteristic audiogram with its 4 kHz notch is not a pathognomic feature of noise induced deafness. The notch may lie between 3 and 6 kHz and with progression over time the notch may widen and be lost as the thresholds at the higher frequencies rise. Additionally, noise exposure is not the only cause of a 4 kHz notch and the hearing loss may not necessarily be symmetrical.

Characteristics of Noise-induced Hearing Loss

1. It is gradual.
2. It is painless.
3. It is usually bilateral.
4. It may be either symmetrical or asymmetrical.
5. It is preceded by NITTS.
6. It is irreversible.
7. It is a sensorineural (perceptive) deafness.
8. In the early stage of the disease, speech range is not affected.
9. It depends on individual susceptibility.
10. Usually there is a notch at 4 kHz.
11. NIHL can be measured.
12. It expands in both the upper and lower frequency ranges when exposure is increased.
13. It is usually not amenable to treatment.
14. In audiometric test both air-conduction and bone-conduction responses are below the normal range and both are equal.

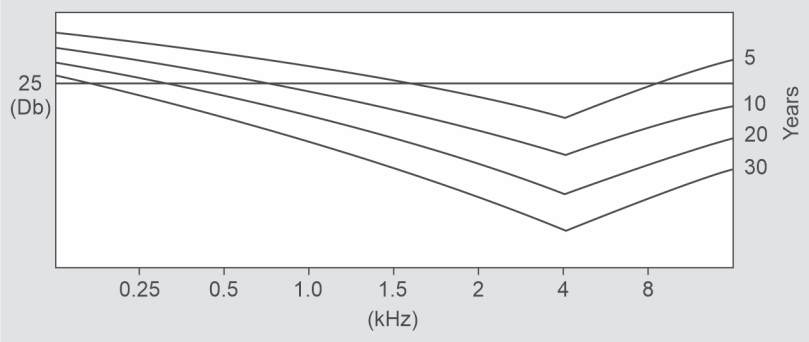


Fig. 8.15: Duration of exposure and NIHL

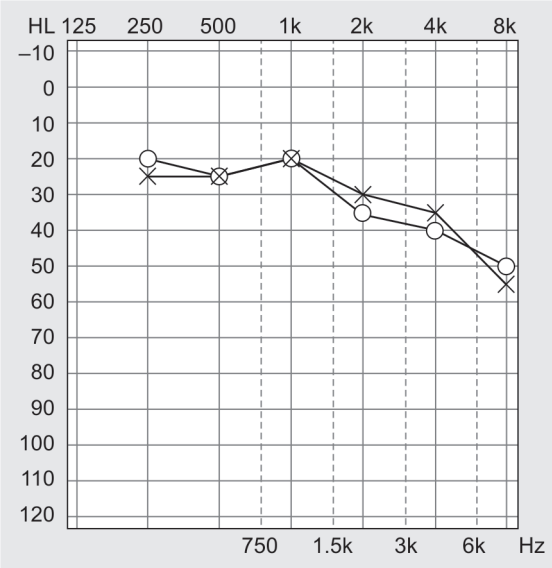


Fig. 8.16: NIHL of both ears (symetrical)

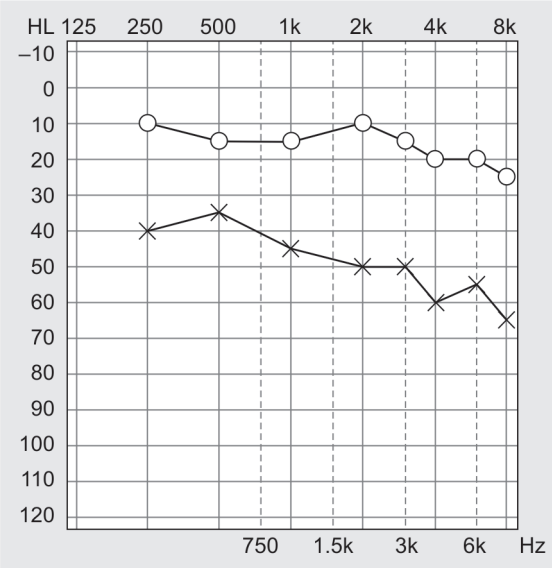


Fig. 8.17: NIHL at left ear only (asymetrical)

Pathology of Noise-Induced Hearing Loss

The correlation of occupational noise-induced hearing loss with cochlear pathology was first described by Habermann in 1890. He reported that it was the organ of Corti, particularly the sensory hair cells and occasionally the spiral ganglion cells of the auditory nerve fibres that were affected. Studies by Igarashi and colleagues and Bredberg showed that the site of prediction in the organ of Corti was 11 mm and 10.5–14 mm, respectively, from the beginning of the basal turn of the cochlea. This corresponds to an area that is responsive to sound frequencies around 4 kHz.

Progressive degree of stereocilial damage and hair cell death correlate with temporary and permanent threshold shift. The sensitivity of afferent inner hair cells and it is postulated that dysfunction of the latter has an adverse effect on the function of the former. The time-honoured belief that the pathological site correlate with the threshold shift has also been challenged as there is a variation in the distribution of the hair cell loss and loss of cochear sensitivity, related perhaps to the response of the middle ear muscles to different types of hazardous sound stimulation. In addition to these mechanical factors, meta-

bolic and vascular factors have been postulated.

It is becoming increasingly apparent that the damage to the cochlea caused by excessive noise is the result of cellular injury from reactive oxygen species (ROS). ROS are ions or small molecules that include oxygen ions, free radicals (hydroxyl radical, OH, peroxynitrite radical, ONOO) and peroxides (e.g. hydrogen peroxide, H_2O_2) and are produced in the mitochondria. They are a natural product of oxygen metabolism and have an important role in cell signaling. They may, however, be produced in excess in response to stress and are then capable of causing serious damage to cell structures, a condition known as 'oxidative stress'. They have a role in programmed cell death (aptoptosis), and also have some beneficial effects in the induction of host defense genes. The damaging effects of ROS include damage to DNA, oxidation of fatty acids and amino acids and inactivation of enzyme systems. Normally, there are enzyme systems that protect the cell from damage from ROS, such as superoxide dismutase, catalases, glutathione peroxidases. In addition, antioxidants, such as ascorbic acid (vitamin C) and tocopherol (vitamin E), have a major role in scavenging free radicals.

The cochlea is metabolically highly active and produces ROS which are in normal circumstances neutralized by endogenous antioxidant mechanisms. There is evidence that oxidative stress can be responsible for cochlear impairment as a result of an accumulation of harmful free radicals. Noise exposure is the most common cause of this stress, but similar mechanisms are at play in cochlear injury from ototoxic medication. One factor which seems to play a significant part in causing these metabolic changes is the role of reduced cochlear blood flow following stimulation with loud noise.

Noise and solvent interactions: Animal experiments have indicated that combined exposure to noise and solvents induces synergistic adverse effects on hearing. Experimental studies have explored specific substances, including toluene, styrene, ethylbenzene, and trichloroethylene. A number of epidemiological studies have investigated the noise–solvent relationship in humans. Overall, the evidence strongly suggests that combined exposure to noise and organic solvents can have interactive effects (either additive or synergistic), in which solvents exacerbate noise-induced impairments even though the noise intensity is below the permissible limit value. A review found the incidence of sensoryneural hearing loss to be higher than expected in noise-exposed workers who were also exposed to solvent containing toluene. In addition to the synergistic effects with solvents, noise may also have additive, potentiating, or synergistic ototoxicity with asphyxiants (such as carbon monoxide) and metals (such as lead).

Ototoxic substances came gradually to the attention of occupational health and safety professionals in the 1970, when the ototoxicity of several industrial chemicals, including solvents, was recognized. The possibility of noise/solvent interaction was raised when Bergstrom and Nystrom (1986) published the results of a 20-year epidemiological follow-up study in Sweden, started in 1958 and involving regular hearing tests in workers. Interestingly, a large proportion of workers employed in the chemical divisions of companies suffered from hearing impairment, although noise levels were significantly lower than those in sawmills and paper pulp production. It is suspected that industrial solvents were an additional causative factor in hearing loss.

Workers are commonly exposed to multiple agents. Physiological interactions with some mixed exposures can lead to an increase in the severity of harmful effects. This applies not only to the combination of interfering chemical substances, but also in certain cases to the co-action of chemical and physical factors. In this case, effects of ototoxic substances on ear function can be aggravated

by noise, which remains a well-established cause of hearing impairment.

Examples of relevant literature on interactions between noise and specific substances include:

1. Toluene (Brandt-Lassen et al., 2000; Johnson et al., 1988; Lataye and Campo, 1997; Lund and Kristiansen, 2008).
2. Styrene (Lataye et al., 2000; Lataye et al., 2005; Makitie at al., 2003).
3. Ethylbenzene (Cappaert et al., 2001).
4. Trichloroethylene (Muijser et al., 2000).
5. Carbon monoxide (Lacerd et al., 2005).
6. Lead (CDC-HHE, 2011).

Clinical Findings of Noise-Induced Hearing Loss

Patients with NIHL frequently complain of gradual deterioration in hearing. The most common complaint is difficulty in comprehending speech, especially in the presence of competing background noise. Because patients with noise-induced hearing loss have a higher frequency bias to their hearing loss, they hear vowel sounds better than consonant sounds. This leads to a distortion of speech sounds when they are listening to people with higher-pitched voices. Background noise, which is usually low frequency in bias, masks the better-preserved portion of the hearing spectrum and further exacerbates the problems with speech comprehension.

Noise-induced hearing loss frequently is accompanied by tinnitus. Most of patients describe a high-frequency tonal sound (ringing), but the sound is sometimes lower in tone (buzzing, blowing, or hissing) or even non-tonal (popping or clicking). This sensation may be intermittent or continuous and usually is exacerbated by further exposure to noise. Tinnitus is usually the most bothersome to patients when there is little ambient noise present. Therefore, some patients may complaint of inability to fall asleep or to concentrate when in a quiet room.

The 4000 Hz notch, which frequently develops relatively early in the worker's exposure to hazardous noise, generally will move laterally as further exposure continues; thus, lower and higher frequencies become affected somewhat latter if the exposure continues. Because the most important thresholds for comprehension of human speech are between 500 to 3000 Hz, a significant decrease in speech discrimination threshold does not begin until frequencies of 2000 Hz and below are affected. The speech discrimination score is normal in the early stages of noise induced hearing loss but may deteriorate as the loss becomes more severe.

Most frequently, the hearing loss in NIHL is bilateral, although asymmetry can exist, particularly when the source of noise is lateralized (e.g. rifle or shotgun firing). Tinnitus may or may not be present. Tinnitus is a subjective complaint, and measurements of tinnitus are based on the patient's ability to match the ringing in loudness and frequency.

Individuals who have acute noise-induced hearing loss can present with a variety of audiometric patterns, including temporary or permanent high-frequency sensorineural hearing loss, cupped sensorineural hearing loss (mid frequency), and flat sensorineural hearing loss. These patients frequently experience tinnitus, and a few will have symptoms of hyperacusis (it is a debilitating hearing disorder characterized by an increased sensitivity to certain frequencies and volume ranges of sound and, occasionally, vertigo).

Diagnosis of Occupational Noise-induced Hearing Loss

The process of establishing a diagnosis of occupational noise-induced hearing loss should be no different from the process of reaching a diagnosis when faced with any other clinical problem. The fundamental principles of taking a comprehensive and pertinent history, conducting a thorough examination and undertaking appropriate investigations are just as applicable. However, certain factors cloud this process and as a

result the diagnosis is usually circumstantial. When faced with an individual with hearing loss and a history of occupational noise exposure, the clinician has to make a decision as to whether the former is a result of the latter. This gives rise to the first problem. The Department of Health and Social Security publication 'Occupational Deafness' states that apart from the characteristic audiometric changes, there are no signs or symptoms specific to noise-induced deafness. This implies that the diagnosis of occupational noise-induced hearing loss is an audiometric one. However, the characteristic audiogram with its 4 kHz notch is not a pathognomic feature of noise-induced deafness. The notch may lie between 3 to 6 kHz (a notch centered at 6 kHz is considered to be an artifact), and with progression over time the notch may widen and be lost as the thresholds at the higher frequencies rise. Additionally, noise exposure is not the only cause of a 4 kHz notch and the hearing loss may not necessarily be symmetrical. The history must include not only audiovestibular symptoms, but also details of previous and intercurrent focal and systemic disorders that may affect the ear. This includes the administration of potentially ototoxic drugs. The history of noise exposure, whether occupational, military or otherwise, can be difficult to construct in temporal terms as individuals may have difficulty in recalling past accurately. Increasing recognition of non-syndromic familial sensorineural deafness and the familial nature of otosclerosis and some cases of Meniere's disease underline the importance of obtaining a good family history. Autoimmune deafness may be associated with systemic symptoms and these should be enquired about. The examination of the ear, nose and throat must be complete. Much information about the health of the external and middle ear can be collected from pneumatic otoscopy and if the clinician is in any doubt, microscopic examination of the ear should be readily available, if nothing else, to clear the external meatus of wax that precludes a view of the drumhead. Aside from the audiometric evaluation, additional investigations are dictated by the clinical assessment and include haematological, serological and relevant immunological laboratory tests, in addition to radiology if indicated. Evaluation of the historical, clinical and investigative evidence usually allows, if present, the causal relationship between hearing loss and noise exposure to be established, but as there is a currently no single clinical or investigative feature unique to noise-induced hearing loss, the diagnosis remains on the clinical probability and audiometric compatibility.

Degree of Hearing Loss (as Per the Ability to Understand the Speech)

As said earlier, the speech frequency range is usually from 500 Hz to 3000 Hz. As noise induced permanent threshold shift is not seen in this range in the early stages, the affected individual will have no difficulty in communicating with speech and hence the loss remains unnoticed. Only the audiometry testing can detect such loses. If affected individual is further exposed to the noise without protection, the loss will extend to the neighbouring frequencies thus affecting the speech frequency range. At this stage, the individual will experience difficulty in speech communication.

Though the type of hearing loss is important, of equal importance is the degree of impairment. Because the critical range of hearing speech is usually 500 Hz to 3000 Hz, most professionals focus on this range of hearing when describing the extent of a hearing loss. All individual's hearing loss for speech can be estimated by taking an arithmatic average of thresholds for pure tones, as seen in the audiograms for four test frequencies of 500, 1000, 2000 and 3000 Hz. The following table shows the classification of degree of hearing loss.

Degree of ability to understand speech		
Degree	*Average hearing level for 500, 1000, 2000 and 3000 Hz*	*Ability to understand speech*
None	Below 25 dB	No difficulty
Slight	26–40 dB	Difficulty with faint speech
Mild	41–55 dB	Frequent difficulty with faint speech
Moderate	56–70 dB	Frequent difficulty with loud speech
Severe	71–90 dB	Can hear only shouted speech
Profound	91 dB and above	Very limited usable hearing

It is known that noise-induced hearing loss is not seen at speech frequency range during early stages. The average hearing level is below 25 dB and hence the affected individual will have no difficulty in the ability to understand speech in the early stages.

How the Hearing may be Impaired

Partial or total deafness may affect either one or both ears. Hearing loss may result from infection, obstruction, trauma, prolonged noise exposure, toxic agents, allergies, and many diseases. Determining the cause of hearing loss involves a careful evaluation of the medical history, the occupational history, noise exposures, the medical examination, and the audiogram. The audiogram measures the loss of hearing but does not evaluate the cause. Because loss of hearing is only a symptom of damage in the hearing mechanism, multiple causes may exist. The differentiation of that part of the hearing loss due to noise exposure from that due to other causes sometimes is difficult and requires otologic consultation. The following is the outline of the major causes of hearing loss and is not intended to be exhaustive or complete. (1) *Infection of the ear*: Infection of the external ear (otitis externa) may cause swelling in the external auditory canal and result in obstruction and hearing loss. Chronic infection of the middle ear (otitis media) may cause mild to severe conduction hearing loss. Acute infections may heal completely. The cause of the infection generally is secondary to infection elsewhere, usually in the respiratory tract, (2) *Obstruction or injury by physical agents*: Impacted cerumen (wax) in the external canal is a frequent cause of hearing loss due to plugging of the canal. Foreign bodies in the ear canal or, rarely, in the middle ear, if not remove, may cause infection, with damage to the entire ear. Trauma of the ear may cause serious hearing losses by rupture and/or dislocation of the drumhead and ossicles. The inner ear may be damage by dislocation of the stapes or fracture of the temporal bone containing the cochlea. A sudden, intense pressure wave, such as produced by a blast, may cause hearing loss that may be partial or total, temporary or permanent. Prolonged noise exposure may cause hearing loss. The intensity, vibration, frequency, duration of exposure to noise, as well as the age of the subject must be considered before causal relationship to hearing loss can be estimated. Barotrauma (aerotitis) is middle ear damage due rapid changes in altitude. This occurs most frequently in persons taking an airplane trip while they have a cold or sinusitis. The greatest difficulty arises during descent, when air cannot enter the Eustachian tube from the throat to equalize the middle ear pressure. Excessive lymphoid tissue (adenoids) in the nasopharynx must be considered an important cause of hearing loss in children. (3) *Toxic agents and allergies*: Quinine and its derivatives, tobacco (nicotin) and aspirin (salicylates), are possible cause of hearing loss in hypersusceptible patients. Discontinuation of the drug with the onset of ringing in the ear (tinnitus) or auditory nerve irritation reverses the process; continued use of the drug may cause permanent and irreversible hearing loss. Widely used antibiotics may cause inner-ear hearing loss (nerve deafness) or labyrinthine disturbance (difficulty with balance). Some of these antimicrobial drugs are streptomycin, dihydrostreptomycin, neomycin, kanamycin,

vancomycin and viomycin. Other drugs or materials may cause labyrinthitis or nerve deafness in individuals sensitive to these substances, or impaired hearing may result from the effects of allergy on the upper respiratory passages. (4) *Diseases*: Maningitis can cause hearing loss through involving any part of the hearing mechanism within the skull. Tumors, haemorrhage, vascular disease or spasm, and certain neurologic diseases may involve the central auditory area, the auditory nuclei or the eighth nerve, with the production of hearing loss. Many infectious diseases (mumps, measles, scarlet fever, diphtheria, respiratory infections) have been responsible for hearing loss mainly in children. The effects may be due to direct infection in the middle and/or inner ear or as a secondary infection in the middle ear, (5) *Acoustic trauma and blast trauma:* The term "acoustic trauma" describes permanent hearing loss following brief exposure to a single very loud noise. This encompasses, for example, the effects of gunfire, but also includes the effect of industrial impulse noise associated with drop forging and riveting. Gunfire noise in particular has the potential to be extremely hazardous. Peak sound pressure levels of 160 dB for hand-held weapons to 190 dB for field weapons have been recorded and, therefore, permanent injury to the inner ear may arise from the first exposure. It is worth bearing in mind that with rifle fire, the forward ear (left ear in a right-handed individual) is closer to the muzzle, thereby bearing the greater brunt of the acoustic trauma than the opposite ear and this is often discernible audiometrically. Second, occupational noise-induced hearing loss and acoustic trauma may coexist in same individual. Blast trauma (otic blast injury) is due to the effects of an explosion such as bomb blast, but may also be a component of the acoustic trauma from large caliber weapons. This is characterized by a greater severity and may be associated with damage to the tympanic membrane and the middle ear structures. The shock wave from an explosion is longer than that of hazardous sound and consists of a short (5 ms) positive pressure followed by a longer (30 ms) negative pressure. Clinically, there may be a history of bleeding from the ear following the injury and examination shows a spectrum of tympanic membrane changes ranging from hyperaemia to frank perforation which may be clear or ragged. It is the initial positive pressure that perforates the drumhead and the subsequent negative pressure that leads to the characteristics everted edges of the perforation. The resultant deafness and tinnitus are immediate and severe but recovery, though incomplete for the higher pitches, is not uncommon and over three-quarters of drumhead perforations heal spontaneously.

Factors in Hearing Loss Claim Evaluation

Many complex acoustic, medical, and legal factors must be considered when determining the causal relationship between noise exposure and the resulting hearing loss. Those who investigate and adjust occupational hearing loss claims must understand the medical, acoustic, loss prevention, and legal aspects of the problem. Although it is difficult to predict the outcome of the hearing loss claim based upon a given set of circumstances, any conclusion must be drawn on the basis of knowledge of applicable laws, regulations, and legal precedents of the jurisdiction involved. The decision of the person responsible for determining the causal relationship of occupational hearing loss must be based on an evaluation of the available information. When appropriate evidence is presented in a logical and orderly sequence, when major issues are identified, and when the basis for any presumption of cause is defined, then the decision-making process is facilitated, and as equitable a decision as possible is likely to result. Unfortunately, information on a worker's past noise exposures is often unavailable, inadequate, or incomplete. All individuals are not alike and do not react in the same way to similar exposures to noise.

Off-the-job exposures may contribute to or be a primary cause of hearing loss. These are some of the factors that must be considered in the decision-making process.

Evidence of Hearing Loss

The first consideration of determining the probability of a cause–effect relationship between an occupational hearing loss and excessive noise at the workplace is to establish: (1) That a hearing loss does, in fact, exist and (2) that the particular manifestations of hearing loss appear to be the result of exposure to excessive noise at the workplace.

Occupational hearing loss can be defined as the loss of hearing in one or both ears as the result of one's employment. This last phrase is very important; the loss of hearing must be "as the result of one's employment". The hearing loss may be the result of hazardous noise exposure or due to acoustic trauma.

Early Signs of Hearing Loss

Early sign of hearing loss:

1. Difficulty in understanding spoken words in a noisy environment.
2. Need to be near or look at the person speaking to help understand words.
3. Familiar sounds are muffled.
4. Complaints that people do not speak clearly.
5. Ringing noises in the ears.

Audiometric Examination Procedure

Audiometry is the measurement of hearing. The results of such testing are recorded on an audiogram, a graph showing hearing (threshold) level as a function of frequency. Hearing (threshold) level is the number of decibels that the subject's threshold of hearing lies above the zero reference of that frequency. Thus, a pure tone audiogram compares the hearing of the person being tested with a standard hearing level considered to be 'normal' (i.e. the audiometric zero reference level).

A manual, pure tone audiometer is an instrument in which the tester has control of the frequency and sound pressure level of the pure tones presented to the subject. The employee whose hearing is being tested responds to the test tone by raising a finger or hand or pushing a signal button. The technician selects a particular frequency, presents short tone pulses with a tone-interrupter switch, manipulate the hearing-level dial to determine the subject's hearing level, and records that level.

The degree of hearing loss can be determined by an audiometric examination, which tests the person's ability to hear pure tones at defined frequencies. Audiometry is, in part, a subjective test—individuals vary in response during different tests made at different times. The resulting audiograms, as a result, may not accurately reflect the true degree of permanent hearing loss. Several sources of variance may confound this estimate: Testing environment, the skill of the person administering the test, the degree of temporary hearing loss resulting from a recent exposure to either occupational or nonoccupational noise, and (since financial awards are involved) attempts by the claimant to exaggerate the true loss. The audiometry examination should be administered only by skill technicians who have received training in audiometry. In addition, a physician or audiologist should provide supervision to the audiometric testing program and set policies and procedures for its administration. For example, the program supervisor should spell out the criteria for referral, and specify how and when individuals should be informed of audiometric results and recommendations should be made concerning protection and/or placement of applicants or employees. Professional supervision of the audiometric testing program will support the acceptance of audiograms as evidence in legal proceedings.

It is important that the test be made in a room where the background noise is below certain specified limits. Excessive noise in the

test room tends to mask the sound delivered by the audiometer to the earphones, thus producing a test record of some degree of hearing loss which does not actually exist. Ordinarily, room background noises are low in frequency; their masking effect is pronounced at frequencies below 1000 Hz.

As assurance of reliable testing and consistency between tests, it is important that the audiometer be of a design that meets the specifications for audiometers approved and published by American National Standards Institute (ANSI). It is also important that the calibration of the audiometer be checked frequently, because errors do occur through usage or abuse.

Hearing tests in compensation cases should be made after full recovery from any temporary threshold shift. Opinions differ among experts concerning the maximum period of recovery or stabilization of hearing after noise exposure in any given individual. Statements have been made that the average recovery time is about 16 hours—this is only an average figure and would not necessarily apply to any given individual. Also, this statement applies to relatively minor hearing loses; the higher the hearing loss, the longer the recovery time. Some authorities think that 48 hours' separation from noise is adequate for recovery; others estimate that the necessary recovery time to be spent away from the noise ranges from 1 to 6 months. Accurate evaluation of permanent hearing loss may be difficult to establish if the claimant is working in a noisy environment at the time the audiometric tests are to be made. The physician must make sure that the worker has been away from the noise for a sufficient period of time to eliminate any temporary threshold shift.

The baseline and periodic audiometric examinations made by the employer previously should be obtained and compared with present audiometric test results. The sensorineural hearing loss that results from noise exposure is similar to hearing loss caused by other less apparent factors. Without a baseline or pre-employment audiogram, it is difficult to state what part of the employee's hearing loss was caused by the conditions at his or her present place of employment and what part of the hearing ability was already lost before he or she was hired.

A similar problem presents itself when the employee has worked for several companies and each one might have contributed to the total hearing loss—which employer should be held responsible for the hearing loss? Several states of United States have enacted statutes that hold that the last employer is responsible for the entire hearing loss, unless there is competent audiometric evidence documenting the employee's baseline hearing level at the beginning of his or her employment.

If the hearing tests disclose a sensorineural loss, a carefully developed history of the loss is an important tool in objectively evaluating the cause. Once this type of loss has been diagnosed, there is no specific test that can either confirm or eliminate noise as the sole cause. In many cases, however, much can be learned by a careful examination of the subject's history. In some cases, it may be found that the hearing loss existed prior to the worker's employment but was not realized until disclosed by audiometric tests. In other cases, it may be disclosed that the worker's hearing was impaired by nonoccupational causes, such as otologic and infectious diseases, other noise exposure, or toxic drugs. A history of military service with exposure to gunfire or jet engine noise, for example, indicates that some hearing loss is to be expected regardless of industrial noise exposure. Having to pay compensation award has alerted industry to the need for preplacement/pre-employment tests to established baseline audiograms.

Differential Diagnosis of NIHL

Noise-induced hearing loss is used to describe the cumulative loss of hearing, always of the sensorineural type, that develops over months

or years of hazardous noise exposure. Employees who have sudden hearing loss may present problems to the consulting physician. A sudden hearing loss may occur as a result of acoustic trauma. However, sudden hearing loss does not result from exposure to typical occupational noise levels. Sudden hearing loss frequently occurs in the general population as the result of viral infection or vascular occlusion and from ruptured labirynthine windows, leading to the escape of perilymph.

There are a number of indicators that will suggest the need for careful consideration of causes other than noise. Some of these are listed below and are based upon commonly expressed viewpoints by experts in this field:

1. A conductive loss is not caused by continuous exposure to noise. It may be occupational in origin, but in such case, it would be the result of a traumatic injury to the external ear, the eardrum, or the middle ear.
2. A mixed conductive and sensorineural loss indicates that exposure to noise is not the only cause.
3. Inconsistent responses during different tests suggest the possibility of malingering or functional loss.
4. If the speech perception threshold is 15 dB or more than the average of the pure-tone levels at 500, 1000 and 2000 Hz, a psychogenic loss or malingering should be suspected.
5. A pronounced loss in one ear, with the hearing in the other essentially normal, indicates ordinarily that the hearing loss is not due to noise exposure.
6. A record of exposure to gunfire or airplane engine noise in military service will indicate that significant losses, particularly at about 4000 Hz, are to be expected regardless of industrial exposure.
7. Some hearing loss is to be expected among persons of advanced age, due to presbycusis and the common noise exposures affecting the entire population.

Presbycusis (Age-associated Hearing Loss)

Aging is associated with a progressive loss of auditory function, a condition which has been described in the past as 'Presbycusis'. It is recognized that most of the impairment arises as a result of progressive cochlear dysfunction with loss of hair cells from the organ of Corti, affecting the higher frequencies first, but advancing through the cochlea to affect eventually the whole frequency range to some extent. Other structure may also undergo degenerative change, for example the auditory nerve and the central auditory pathways. There may also be a central loss of cognitive function. It is clearly important to be able to make some allowance for the effects of 'natural' ageing on the hearing of those middle-aged and elderly individuals who have also been exposed to the harmful effects of noise during their working lives. The role of presbycusis (hearing loss due to the aging process) in hearing loss claims has received considerable attention. Research indicates that hearing loss increases due to aging, even for persons not exposed to occupational noise. Such studies show a gradual decline in the hearing sensitivity of the population. There is some disagreement as to how much of this loss is due to the aging process alone (presbycusis) and how much is due to environmental noise exposure (sociocusis). It is also not known how the aging process interacts with the growth of occupational hearing impairment. Usually, in workers' compensation, the entire impairment is compensable as long as occupational factor in any way increases the impairment caused by the physical condition.

Presbycusis usually affects the high frequencies more than the low frequencies. The audiogram shows the sound have to be made louder before they are heard in the higher frequencies (in the right side of the audiogram), leading to a slop to the audiogram. The audiogram shows normal hearing up to 1 kHz (mid-frequency) and a mild hearing loss in the mid to high frequencies. Depending on the degree on the hearing loss, the sound may

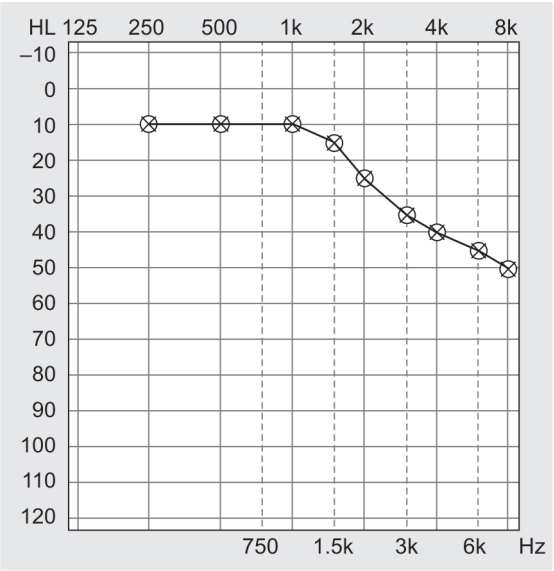


Fig. 8.18: Preshycusis

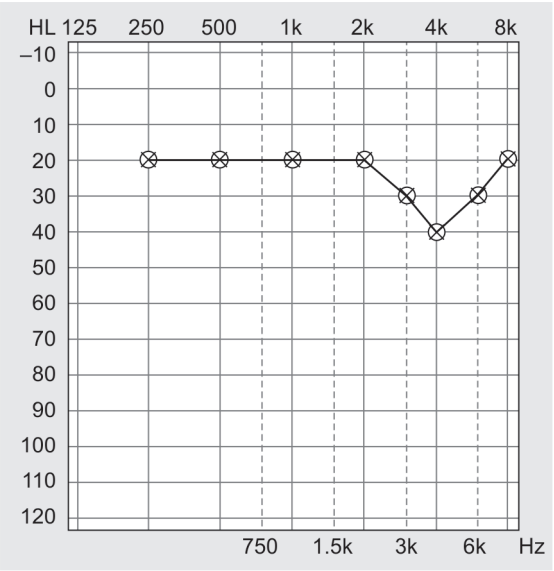


Fig. 8.19: NIHL

have to be made louder before they were heard but the general pattern is likely to be similar for all presbycusis hearing loses. A right-hand slopping hearing loss with the left and right ears usually deteriorating at equal rates. Noise-induced hearing loss (NIHL) most commonly occurs at 4 kHz. Therefore, there is a notch at 4 kHz.

Data on this subject are available from many sources. International Standard ISO 7029 (1984) gives values of age-associated hearing loss as deviation relative to the median thresholds of young otologically normal subjects. The data are available for sex, for the age range 20–70 years, for frequencies ranging from 125 Hz to 8 kHz, and for percentiles of the population from the 5th to the 95th. They are available in table form for ease of reference so that allowance can be made in the individual case for the effects of aging. Several countries' compensation boards require a deduction for the hearing loss that accompanies age, for example, a deduction from calculated hearing impairment of one-half decibel for each year of age over 40 years. The rationale for his is the desire to substrate the average amount of hearing loss from non-occupational causes found in the population at any given age. The International Standard ISO 1999 (1990) "Determination of occupational noise exposure and estimation of noise-induced hearing impairment" assume a direct additive effect between noise and age effects, although that is not true at profound levels of deafness. A word of caution is necessary. Burns and Robinson have drawn attention to the complicated interrelationship of occupational noise-induced hearing loss and ageing. The former tends to produce an elevation in threshold which is initially rapid, but slows down with subsequent exposure. Age-related hearing loss takes a progressively accelerating course with time. Thus, the contribution of occupational noise-induced hearing loss to the total sensorineural hearing loss decreases with age, and by the age of 80 it would make virtually no difference what the noise had been.

Assessment of Hearing Impairment: Impairment, Disability and Handicap

These consequences of disease are differentiated by the World Health Organization (WHO):

1. *Impairment*: Impairment relates to a loss or impairment of the structure or function

of an organ or system. Within the auditory system, there are a number of impairments of function which could be measured. The one which has most relevance for the measurement of hearing disability is the alteration in the pure tone threshold.

2. *Disability*: Disability is an index of the loss of an individual's functional performance as a consequence of impairment of the diseased organ or system. It thus represents a disturbance at the level of the individual. Hearing disability has been described as the restriction or lack of ability to perceive everyday sounds in the manner that is considered normal for healthy young people.
3. *Handicap*: Handicap encompasses the disadvantages experienced by an individual as a consequence of impairments and disabilities. It thus reflects interactions between the individual and his social and working environment. Hearing handicap is the disadvantage to an individual resulting from a hearing impairment or disability that limits or prevents the fulfillment of a role that is normal for the individual. Disability and handicap are thus seen to be multifaceted. A given degree of hearing impairment could represent a much greater handicap for a professional musician than for a sculptor for example. The complex picture of disability and handicap that may result from occupational noise-induced hearing loss has been pointed by Hetu et al.

In 1981, a working party of members of the British Association of Otolaryngologists (BAOL) and the British Society of Audiologists (BSA) was set up to address the problem of assessment of hearing disability for the purpose of compensation. Up to then, there was a plethora of conflicting recommendations, from a number of different sources. The results of the working party deliberations were published in the so-called 'Blue Book' of 1983. Because of some misgivings about its conclusions, a new group called the Inter-society Working Group was established in 1986 with a wider membership including representatives of the British Association of Audiological Physicians (BAAP), and the British Association of Audiological Scientists (BAAS).

The results of this group's efforts are published in 'Guidelines for medicolegal practice', although the BAAP did not sign up the final document because of the problems on the definition of disability and on agreeing the scale of degrees of disablement. After that, several studies have been carried out to identify the audiometric descriptors which best related with hearing disability. It was concluded that the three-frequency average of 1, 2, and 3 kHz was the best predictor. Errors were introduced by the inclusion of 0.5 kHz and 6 kHz. It may seem strange that 4 kHz is excluded from this formula, when one considers that this is the frequency most likely to be affected by occupational noise; however, the notch at 4 kHz may be narrow and quite deep, so that there is a danger of obtaining an inflated average hearing threshold measurement which does not truly reflect the degree of disability. Furthermore, it is convenient not to include 4 kHz in the estimate because of the difficulty in obtaining accurate bone conduction thresholds above 3 kHz. Finally, the three-frequency average coincides with the UK statutory hearing loss criteria, even though the fence values of the latter may be subject to some criticism.

Fence Values

The low fence is that notional point on the continuum of elevation of the hearing threshold level at which disability is deemed to commence. It has never been satisfactorily identified, and indeed cannot be seen as anything other than an artifice erected for administrative convenience, with little relevance to the individual case. Robinson et al.

identified 30 dB hearing threshold level averaged over 1, 2, and 3 kHz as the threshold of disability and this coincides with British Standard BS 5330 (1976). A low fence as high as 50 dB HL (1, 2, 3 kHz average) is employed in the UK statutory compensation scheme. Even the much lower value of 26 dB (0.5, 1, 2 kHz) that prevailed under the American Academy of Otolaryngology and Ophthalmology (AAOO) scheme underestimated the disability of those individuals who began life with hearing that was 'normal'. The high fence is that point in the continuum of elevation of hearing threshold level at which disability is deemed to be total. High fence values have been judged to be any level from 70 dB upwards, and are just as arbitrary as low fence values.

Hearing Loss Detection Formula

The formula for hearing loss compensation awards needs to be based on a reasonable cost of such a programme to society. Damage risk criteria involve psychophysical, political, and socioeconomic considerations. Generally, these decisions are compromises between providing total protection, at exorbitant cost, and providing a reasonable amount of protection to a given segment of the population at costs that can be absorbed by the government, industry, and ultimately by the taxpaying consumer.

Hearing impairment can be described by three parameters:

1. The first parameter is the selection of the hearing test frequencies that will be used to determine the severity of hearing impairment.
2. The second parameter is the 'low fence', which can be defined as the minimum hearing threshold shift which has to be exceeded for legal hearing impairment to exist.
3. The third parameter is a scale for measuring the severity of hearing impairment based on a 'multiplier' of decibels of hearing threshold shift above the 'low fence'.

Procedure: The following procedure is used to convert hearing threshold levels into percentages of hearing impairment, according to the 1979 AAOO (American Academy of Ophthalmology and Otolaryngology) formula:

1. The average of the hearing threshold level at 500, 1000, 2000 and 3000 Hz is calculated for each ear.
2. The percent impairment for each ear is calculated by 1.5% the amount by which the aforementioned average hearing threshold level exceeds 25 dB (low fence). A maximum of 100 % is reached at 92% dB (high fence).
3. The binaural impairment assessment should then be calculated by multiplying the smaller percentage (better ear) by 5, adding this figure to the larger percentage (poorer) and dividing the total by 6.

Sample of Hearing Loss Calculation

Following are examples of the calculation of hearing loss:

A. Mild to marked bilateral hearing loss:

Ear	*500 Hz*	*1000 Hz*	*2000 Hz*	*3000 Hz*
Right	70	80	80	90
Left	75	80	90	95

1. Calculation of average hearing threshold level:
   Right ear = (70 + 80 + 80 + 90)/4
   = 80 dB = 82.5% loss
   Left ear = (75 + 80 + 90 + 95)/4
   = 85 dB = 90% loss
2. Calculation of the hearing handicapped:
   Smaller number (better ear):
   82.5 × 5 = 412.5%
   Larger number (poorer ear): 90 × 1 = 90%
   Total = (412.5 + 90)/6
   = 84% (rounded off)

B. Severe to extreme bilateral hearing loss:

Ear	*500 Hz*	*1000 Hz*	*2000 Hz*	*3000 Hz*
Right	70	80	80	90
Left	75	80	90	95

1. Calculation of average hearing threshold level:
   Right ear = (70 + 80 + 80 + 90)/4
   = 80 dB = 82.5% loss
   Left ear = (75 + 80 + 90 + 95)/4
   = 85 dB = 90% loss
2. Calculation of the hearing handicapped:
   Smaller number (better ear):
   82.5 × 5 = 412.5%
   Larger number (poorer ear):
   90 × 1 = 90%
   Total = (412.5 + 90)/6
   = 84% (rounded off)

Management of Noise-Induced Hearing Loss

With the exception of the transient phenomenon of temporary threshold shift, the hearing loss caused by noise exposure is permanent and is due to the loss of or damage to the organ of Corti and neuronal structure in the inner ear. There is, as yet, no medical and surgical treatment that can reverse the damage to these structures. Management of occupational noise-induced hearing loss is therefore based on prevention of further damage, suitable amplification with hearing aids, and the provision were indicated of other assistive devices. It is generally felt that the process of injury to the inner ear cease when the subject is removed from, or protected from, his noisy working environment, but subjects should be warned of further possible damage from other sources, such as recreational noise exposure.

The effects on the inner ear are most marked in the high frequencies commencing at 4 kHz, but spreading downwards across the frequency range. The early effects, therefore, are most marked for the high frequency components of speech, the consonants, the sibilants and fricatives. These are those elements which convey most of the meaning in speech. Neuronal damage causes a further loss of speech discrimination which may not be correctable by simple amplification. In addition, there are other psychoacoustic deficits which may come into play and for which hearing aids cannot compensate, such as impaired frequency discrimination, frequency selectivity or temporal acuity. Furthermore, the phenomenon of recruitment, a feature of cochlear deafness, may limit the usefulness of amplification. This is characterized by a disproportionately great increase in the perceived loudness for a small increase in the actual sound pressure level. This is a major factor in causing distortion with hearing aids, and may limit their use. The ability to discriminate speech in the presence of background noise is lost early in the evolution of sensorineural deafness and involves factors other than a simple elevation of the hearing threshold. It is not restored by fitting a hearing aid and it remains one of the main grievances of hearing aid users who find it impossible to converse with one individual in a noisy environment. Despite these shortcomings, it is, however, clear those individuals with noise induced hearing impairment, can derive appreciable benefit from hearing aids provided that careful attention is given to the type of aid and the design and engineering of the ear mould. Different hearing aids have different frequency responses. Some preferentially amplify the higher frequencies and this would intuitively seen a sensible type of device for a subject with a high frequency hearing loss. The prescription of an aid with a customized frequency response for an individual hearing loss is not proper and has commanded much attention in recent years. It is, nevertheless, a much more challenging problem than the prescription of spectacles for a simple refractive error. Different hearing aids have different gain or amplification (i.e. some are stronger than others), and again it is clearly important to have the appropriate aid for the severity of deafness. Hearing aids, like

car engines, have a point or a range at which they function best. If one attempts to get more performance from an underpowered aid, there is a marked fall of in the efficiency of the device. It is better to get a stronger aid and have it working at its most efficient level. Compression aids incorporating automatic gain control, attempt to overcome the problem of the loud sound which exceeds the discomfort level of the listener. Systems which address the problems of understanding speech in the presence of background noise have been developed and are available.

In addition to hearing aids, there are other assistive devices or environmental aids that can be used with or independent of conventional hearing aids including amplifying devices, alerting and alarm system, telephone modification and speech-to-text transcription. They are not necessarily expensive and may provide significant personal benefit. They include induction loop systems, television listening aids, tactile and visual devices activated by sound stimuli (door bells, fire alarms, alarm clocks, telephone). Inductive coupling between coils in the handpiece and the hearing aid greatly helps telephone conversation. Text telephones using a computer link or a relay service via an operator may help the very deaf. Speech-to-text transcription, such as Palantype, is a major help at public meetings, but is expensive.

Cochlear implantation: Cochlear implant is an electrical device inserted into the inner ear of certain profoundly deaf individuals to take the place of a severely damaged organ of Corti and convey to the brain a processed sequence of electric stimuli which the brain perceives as sound. Cochlear implants have now reached such a level of sophistication that the best implantees can converse almost effortlessly even over the telephone. To 'qualify' to an implant, a subject must have a degree of deafness that cannot be aided with any conventional hearing aid. Pure occupational-induced hearing loss very rarely if ever produces deafness to that degree of severity, and with the present selection criteria for implantation, cochlear implantation is not a treatment option for such individuals. There are indications, however, that selection criteria may become less strict in the future.

Antioxidant treatment (a possibility for the future): Glutathione has been shown to limit noise-induced hearing loss in the guinea pig both on auditory evoked brainstem response audiometry and on histological studies of cochlear hair cell loss. Acetyl-L-carmitine is an endogenous mitochondrial membrane compound that helps maintain mitochondrial function in the face of oxidative stress. Carbamathione is a glutamate antagonist. Glutathione levels are reduced in the noise-damaged cochlea. Kopke and coworkers administered acetyl-L-carmine, carbamathione and a glutathione repletion drug D-methionine to three groups of chinchillas and demonstrated protection from damage by showing significant reduction in hearing threshold elevation and significant reduction in cochlear hair cell loss at postmortem examination. Human trials have not yet commenced, but the animal results would suggest that they cannot be far off. Early clinical trial with the antioxidant N-acetylcysteine (NAC) is anticipated.

Noise monitoring: If there is reason to believe that worker noise exposure will equal or exceed a time-weighted average (TWA) of 85 dBA, then noise monitoring is required. A sampling strategy must be designed to identify all workers who need to be included in the hearing conservation programme. The noise present must be characterized in terms of frequency (predominantly high, predominantly low or mixed), intensity (how loud it is), and type (continuous, intermittent, or impulse) using appropriate noise-monitoring instrument. Anytime there is any change in production, process, equipment, or controls, all noise monitoring tests must be repeated.

Engineering controls: The information collected during noise monitoring (particularly octave-band analysis, which indicates

the sound level at selected frequencies) may be used to design engineering noise controls. Designers conceptualize possible engineering solutions in terms of the source (what is generating the noise), the path [the rout(s) the generated noise may travel], and the receivers (workers exposed to the noise). The noise control may involve the use of enclosures (to isolate sources or receivers), barriers (to reduce acoustic energy along the path), or distance (to increase the path and ultimately reduce the acoustic energy at the receiver) to reduce worker noise exposure. In general, engineering controls are preferred but are not always feasible because of their costs and limits in technology.

Administrative controls: Administrative controls include (1) reducing the amount of time a given worker might be exposed to a noise source in order to prevent the TWA noise exposure from reaching 85 dBA and (2) establishing purchasing guidelines to prevent introduction of equipment that would increase worker noise dose. While simple in principle, the implementation of administrative controls requires management's commitment and constant supervision, particularly in the absence engineering or personal-protection controls. In general, administrative controls are used as an adjunct to existing hearing conservation programme noise control strategies rather than as the exclusive approach for controlling noise exposure.

Occupational Noise-induced Tinnitus

Many individuals have experience of the tinnitus that occurs after exposure to loud music, such as that of a rock concert. This temporary tinnitus may be short-lived or last for days. Depending on the duration and intensity of the exposure and may be associated with measurable temporary threshold shift. Similarly, tinnitus is reported often to accompany industrial noise exposure and while initially it is temporary, over a period of years it may become permanent in as many as 60%, particularly in those exposed to impact noise. There is some debate, however, regarding the importance of tinnitus in the assessment of occupational noise-induced hearing loss. The debate is not so much as to whether tinnitus and occupational noise-induced hearing loss go hand in hand, but the weight that should be attributed to the tinnitus component during the evaluation of a claimant.

There is no cure for tinnitus resulting from noise induced hearing loss, although numerous amelioration measures are available. In the absence of further inner ear injury, tinnitus will diminish gradually, usually over a course of weeks or months. A subtle degree of tinnitus often persists and is especially obvious when the patient is in a quiet room. For the few patients who find this to be extremely troublesome, masking the tinnitus with music or some other form of pleasant sound is often helpful. In patients with significant hearing loss, the most successful treatment may be appropriate hearing amplification. Modified hearing aids (tinnitus maskers) designed to produce masking noises generally have been of limited success. Psychiatric referral to manage associated depression sometimes is necessary.

Occupational Noise-induced Vertigo

Temporary vestibular disturbance in response to loud noise is referred to as the Tullio phenomenon. In a state of health, high sound intensities are required to evoke this response. In various ontological disorders, however, lesser sound levels may have a similar effect in certain individuals. Firm evidence that occupational noise-induced vertigo is a real entity is lacking. However, there is some evidence to substantiate a link between acoustic trauma and delayed endolymphatic hydrops in the symptomatic group (a reduced caloric response due to electrophysiological changes suggestive of endolymphatic hydrops) but further investigation or research

into this aspect of hazardous noise exposure is required.

Effects of Infrasound and Ultrasound

Evidence that excessive sound above or below the normal human auditory range may have an adverse effect on inner ear function remain scant. Infrasound (conventionally sound below 20 Hz) is commonly felt as vibration rather than heard and is a component of natural phenomena such as earthquakes and thunder. Heavy industrial machinery, high speed car travel with open windows and ship's engine rooms are potential source of infrasound. More specifically, Pyykko and colleagues examines 203 lumberjacks over a six-year period, compared the hearing in those with and without vibration induced white finger syndrome and found a statistically significant difference in thresholds at 4 kHz in those with the syndrome than those without. They postulated that the higher thresholds in the lumberjacks with vibration induced white finger were due to reflex sympathetic vasoconstriction in cochlear blood vessels in response to vasospasm of the hands rather than a direct mechanical effect on the cochlea.

The auditory effects of ultrasound (sound above 20 kHz) have been reviewed by acton. These include a full feeling in the ear, tinnitus and headache. Utilizing low frequency ultrasound (10–28 kHz can produce auditory electrophysiological effects, including alteration in the cochlear microphonic and increased thresholds—noted by Ishida and colleagues in guinea pigs. Currently, the adverse auditory effects of hazardous levels of both infrasound and ultrasound in human remain to be defined.

Systemic Effects of Noise Pollution

Recently, the World Health Organization Noise Environment Burden of Disease (BoD) Working Group has identified environmental noise (as distinct from occupational noise) as a stressor in the evolution of cardiovascular disease. In addition, there is increasing concern about the burden of sleep disturbance, of annoyance, of hearing impairment, of tinnitus and of cognitive impairment from environmental noise. It has been estimated that the severe annoyance due to noise from traffic, trains and aircraft, may account for approximately 3% of coronary heart disease deaths in Europe each year. Chronic noise exposure has been incriminated in elevation of systolic blood pressure in workers in metal manufacturing industry and in shipyard workers. Risk tends to be associated with night-time noise exposure and the noise threshold for cardiovascular disease has been stated to be a night-time exposure of 50 dBA. The mechanism is thought to be the high levels of stress hormones, such as cortisol, adrenaline and noradrenaline, released into the circulation at times of stress at a subcortical level in the amygdale. This mechanism may be active during sleep and there are several reports in the literature that indicate that exposure to traffic noise and aircraft noise during sleep may cause automatic changes, leading to hypertension and myocardial infarction. It has been suggested that the neuroendocrine changes associated with noise stress may modify immune function. Willich et al. reported that the risk of myocardial infarction from chronic noise burden appears more closely associated with sound levels than with subjective annoyance.

Sudden impact noise can produce startle reaction consisting of contraction of blood vessels, increased blood pressure, dilatation of pupil and both voluntary and involuntary muscles become tense (contraction). Vertigo, nausea, peptic ulcer, fatigue, increased pulse and respiratory rates, increased physical and psychological stress, irritability and socially undesirable behavior, reduced work efficiency and productivity, and increased accident and error—all are the effects of continuous exposure of noise in industry.

As a means of further assessing the noise-related disease burden, the WHO working group have devised disability adjusted life year (DALY). These allow one to qualify the amount to which life expectancy is reduced by premature death or disease-related disability. It was estimated that in 2002, Europeans lost 880000 DALYs to coronary heart disease related to road traffic noise. The group also recognized possible interaction between traffic noise and air pollution in the increases BoD. Outdoor air pollution represents approximately 2% of cardiopulmonary disease mortality, and it is not clear whether the impact of noise on ischaemic heart disease independent, additive or synergistic to the impact of outdoor air pollution.

It is also suggested that sleep disturbance caused by environmental noise (noise-induced insomnia) can be a contributory factor to loss of performance and predispose to accidents at home, at work and when driving. The deleterious effects on noise on cognitive function are also under scrutiny. There are four components of cognitive impairment (reading, recall, recognition and attention) which appear to show a consistent relationship with noise exposure. It must be emphasized, however, that there are immense difficulties in acquiring robust data to establish a casual relationship. There are uncertainties in quantifying noise exposure, in separating the effects of noise from other variables, and agreeing and in qualifying outcome measures.

In 2004, the WHO published a guide for occupational health professionals aimed at the provision of a tool for carrying out detailed disease burden estimates of the hearing loss from occupational noise. In Europe, environmental noise is becoming a major health concern for policy-makers. The European Directive related to the assessment and management of environmental noise (Directive 2002/49/EC) addresses the action plans to reduce harmful effects of noise exposure. The summary of policy implications of the initiatives:

1. Estimates of the incidence of occupation-related NIHL in a given country or study population will provide quantitative information on the importance of the problem and help motivate intervention to reduce the risks and impact of health.
2. While incurable and irreversible, NIHL is nevertheless preventable and it is essential that preventive programmes are implemented.
3. Hearing conservation programmes should be integrated into the overall hazard prevention and control programmes for the workplace.

On this basis, the WHO publication stipulates that such programmes require political will and decision-making, high-level management and workforce commitment, adequate human and financial resources, technical knowledge and experience, communication and monitoring mechanisms and continuous programme improvement. The WHO also recognizes the importance of local socio-cultural aspects and finance proportion of population works in the informal sector—a group that represents a major challenge in terms of occupational hazard prevention.

Hearing Conservation Programme

According to American National Standard Institute (ANSI), any unwanted sound is known as noise. Hearing, a precious sense which is responsible for effective speech communication can be damaged due to continuous exposure of high noise levels. In recent years, noise has become one of the major environmental pollutants mainly in industries.

Noise is not only a health problem but also a legal one as noise induced hearing loss (NIHL) in industrial workers due to industrial noise is compensable as per the Workman's Compensation Act. The compensation claims may amount to huge sums affecting the

economy of the industries. Hence, to safeguard workers from noise as well as to reduce compensation claims, 'Hearing Conservation Programme' plays a vital role.

The industries recognized the need for evaluating hearing and for preventing occupational hearing loss. For those industries having industrial medical programme, the measurement of hearing threshold has been carried out by medical department personnel. The evaluation of the noise problems has involved not only medical and industrial hygiene personnel, but also management, safety, engineering, production and maintenance personnel. The type and magnitude of the noise evaluation and control programme may vary considerably, depending upon the nature of industry and the probability of hazardous noise exposure. The successful programme must include the coordination and integration of four phases:

1. Physical evaluation of the noise exposure (noise measurement) and noise reduction.
2. Control of noise exposure (provision of personal ear protection).
3. Medical evaluation of hearing of exposed personnel (audiometric test).
4. Information and education.

Most manufacturing industries create some potentially harmful noise but the hazard will vary markedly with the type (continuous, impulse or interrupted), intensity and the duration of noise exposure. For that reason, each industry and plant must evaluate its own problem and organized its own hearing conservation programme.

Physical evaluation of the noise exposure (noise measurement)—the relationship of hearing loss to noise exposure: With the development of accurate methods of measuring both the sound stimulus and the hearing level, more

Noise level and duration of exposure are combined to give 'noise exposure points'

Sound pressure level L_{Aeq} (dB)	*Duration of exposure in hours*							
	1/4	*1/2*	*1*	*2*	*4*	*8*	*10*	*12*
105	320	626	1250					
100	100	200	400	800				
97	50	100	200	400	800			
95	32	65	125	250	500	1000		
94	25	50	100	200	400	800		
93	20	40	80	160	320	630		
92	16	32	65	125	250	500	625	
91	12	25	50	100	200	400	500	600
90	10	20	40	80	160	320	400	470
89	8	16	32	65	130	250	310	380
88	6	12	25	50	100	200	250	300
87	5	10	20	40	80	160	200	240
86	4	8	16	32	65	130	160	190
85		6	12	25	50	100	125	150
84		5	10	20	40	80	100	120
83		4	8	16	32	65	80	95
82			6	12	25	50	65	75
81			5	10	20	40	50	60
80			4	8	16	32	40	48
79				6	13	25	32	38
78				5	10	20	25	30
75					5	10	13	15

Total daily exposure points converted to a daily personal exposure. Note the first and second action levels at 80 and 85 dB

Total exposure points	*Noise exposure L_{EPd} dB*
3200	100
1600	97
1000	95
800	94
630	93
500	92
400	91
320	90
250	88
200	88
160	87
130	86
100	85
80	84
65	83
50	82
40	81
32	80
25	79
20	78
16	77

precise determination of relationship between hearing loss and noise exposure has become possible. Burns and Robinson reduced the number of significant parameters to two: The noise level and the duration of exposure. Daily personal noise exposure (L_{EPd}) can be determined by wearing a personal sound dosimeter over a given period. A more accurate picture may, however, be established from the measured values of A-weighted sound pressure levels and the duration of exposure at work using a Noise Exposure Ready Reckoner as recommended in the 2005 Health and Safety Executive document 'Controlling Noise at Work'. This employs a noise exposure points system relating average sound pressure levels (L_{Aeq} dB) with duration of noise exposure in hours. It is a simple way of working out the daily personal noise exposure, especially in situations where the noise is available throughout the day, for example, if an employee spends part of a day in a very noisy environment and part of the day in a less noisy activity. In the noise exposure points scheme, the upper exposure action value is 100 points (equivalent to an L_{EPd} of 85 dB) and the lower exposure action is 32 points (equivalent to an L_{EPd} of 80 dB). The 'first action level' of 80 dB is the level at which ear protectors should be available on demand. The 'second action level' of 85 dB is the level at which the use of ear protections is mandatory. A similar scoring system can be used to work out weekly exposure and is of particular value if there is a significant variation in noise exposure from day to day during the working week.

There are also occasions when one wants to establish the L_{EPd} caused by repeated 'single event' noise produced for example by impact or cartridge-operated tools. Peak pressure meters provide rapid information and are easy to use. If the reading exceeds 125 dBA, more accurate measurements should be made. An integrating sound level meter (BS 6693: IEC 804) is the most convenient instrument for general use. It calculates the equivalent continuous sound pressure level according to the equal energy principle. A peak sound pressure level of 140 dB is defined as the 'peak action level'. At this level, the use of ear protectors is mandatory. It is clearly the responsibility of employers to ensure that all measurement equipment is accurately and regularly calibrated.

Noise reduction: Noise reduction can be helped by the enclosure of noisy machines, the use of mufflers, damping, silencers, anti-vibration mountings, the treatment of reflective surfaces with absorbing materials, the limitation of the use of noisy equipment to times when it is actually required, and the distancing the workers from the areas of maximum noise.

Control of noise exposure (provision of personal ear protection): Ear protection provides a system of attenuation of the incoming sound and thus minimizes the sound arriving at the tympanic membrane. Hearing protective

Fig. 8.20: Noise dosimeter

devices is considered the last option for controlling noise exposure. These are usually used during the time it takes to implement engineering or administrative controls, or when such controls are not feasible. Unless great care is taken in establishing a hearing conservation programme, workers will often receive very little benefit from hearing protective devices. The best hearing protector, when fitted correctly, is one that is accepted by the worker and worn properly. If the worker exposure is above 85 dBA (8-hour TWA), hearing protection must be made available, along with the other requirements in the hearing conservation programme. Various types of protectors are available.

Plants having hearing conservation programme generally assign their medical departments the task of fitting employees with ear plugs, ear muffs and hearing protective helmets. These personal protective equipments are frequently issued in the field along with general instruction on the need of good adjustment of the cushion to prevent leaks. Whenever personal protection is warranted, both the exposed employees and their supervisors should be convinced that ear protection is not only beneficial but also necessary to conserve hearing. The medical department has an important role in a hearing conservation programme both in evaluation and protection.

Ear muff: Ear muffs fit over the ears and are sealed to the side of the head by a soft cushion seals filled with soft plastic foam or a viscous fluid. They are usually held in position by means of a headband, but may alternatively be attached to a soft helmet. Attenuation may be 'frequency selective', protecting some frequencies more than others, particularly the higher frequencies rather than the speech frequencies. 'Amplitude selective' devices are designed to provide attenuation that increases with sound level. Such a device usually has a small hole which acts as a mechanical filter, allowing low sound pressures to pass but offering more resistance at high pressures. 'Active devices' incorporate electronic circuitry which limits the transmission of sound at high intensity, and are of value when workers are exposed to short burst of high intensity sound or impulsive sounds. 'Antinoise' devices incorporate circuitry which cancels out incident noise, especially at low frequency. These devices are still largely experimental. Many of the newer muffs incorporate an inbuilt communication system.

While the ear muffs usually provide an effective level of attenuation, their value may be limited by any effect which decreases the efficiency of the seal, for example, spectacle frames, goggles, beards and long hair, or scarves worm under the muffs. Furthermore, wear and tear may decrease the efficiency of the seal. It should also be emphasized that if protection is removed in noisy areas, even for a short period, the amount of protection will be severely limited. For example, if a protector with an 'assumed protection' of 20 dB(A) is

Fig. 8.21: Ear muff

removed for 30 minutes per day the actual reduction in noise dose received by the wearer will be only 12 dB(A).

Ear plug: Ear plugs are generally thought to be less efficient attenuators of sound than muffs, but have the advantage that they are easy to use. They may be disposable or permanent. Disposable plugs are made of a compressible or conformable material. They are readily available commercially, but have a finite 'life expectancy' after which their effectiveness decreases. Permanent and custom moulded plugs have a longer survival, and are usually comfortable if made by an experienced technician. One of the main disadvantages of plugs is the increased propensity to otitis externa, although this too can occur with muffs. Dual protection with the use of both muffs and ear plugs may be indicated when the noise levels are extremely high (e.g. when L_{EPd} exceeds about 115 dBA).

Hearing protective helmets (noise-excluding helmets): An extremely high levels of sound (e.g. in tunnelling), the protection offered by muffs and plugs, either alone or in combination, may be insufficient. Sound may still reach the ears through nose, mouth, eye sockets and the skull itself. The attenuation provided by the protector should comply with British Standard BS 510866 (ISO 4869), and the basic design features should comply with BS 6344. The 'assumed protection' of a device will vary with frequency and most will attenuate higher frequency sound better than low frequency sound. The 'assumed protection level' of noise exposure is obtained by subtracting the assumed protection of the device at each octave band frequency from the sound pressure level at each frequency.

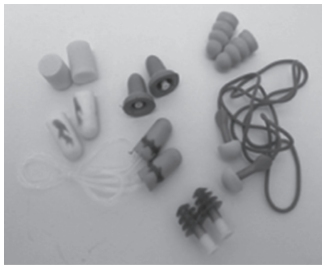


Fig. 8.22: Ear plugs

Noise reduction ratings: When OSHA promulgated its Hearing Conservation Amendment in 1983, it incorporated the EPA labelling requirements for hearing protectors which required manufacturers to identify the noise reduction capability of all hearing protectors on the hearing protector package. This measure is referred to as the noise reduction rating (NRR). It is a laboratory-derived numerical estimate of the attenuation achieved by the protector. It became evident that the amount of protection users was receiving in the workplace with the prescribed hearing protectors did not correlate with the attenuation indicated by the NRR. OSHA acknowledged that in most cases, this number overstated the protection afforded to workers and required the application for certain circumstances of a safety factor of 50# to the NRR, above and beyond the 7 dB subtraction called for when using A-weighted measurements. For example, consider a worker who is exposed to 98 dBA for 8 hours and whose hearing protectors have an NRR of 25 dB. We can estimate the worker's resultant exposure using the 50# safety factor. The worker's resultant exposure is 89 dBA in this case.

The 50# safety factor adjusts labelled NRR values for workplace conditions and is used when considering whether engineering controls are to be implemented.

Estimated dBA exposure

$$= 98 \text{ dBA} - [(25 - 7) \times 50\#] = 89 \text{ dBA}$$

However, when assessing the adequacy of the hearing protection for hearing conservation (HC) purposes, CSHOs should only subtract 7dB from the NRR.

Exposure for PPE/ HC enforcement

$$= 98 \text{ dBA} - (25 - 7) = 80 \text{ dBA}$$

Single/double hearing protection: Dual hearing protection involves wearing two forms of hearing protection simultaneously (e.g. earplugs and ear muffs). The noise exposure for workers wearing dual protection

may be estimated by the following method: Determine the hearing protector with the higher rated NRR (NRRh) and subtract 7 dB if using A-weighted sound level data. Add 5 dB to this field-adjusted NRR to account for the use of the second hearing protector. Subtract the remainder from the TWA. It is important to note that using such double protection will add only 5 dB of attenuation.

Medical evaluation of hearing of exposed personnel (audiometric test): Industrial environments are seldom suitable for accurate tests of normal hearing, even though the medical facilities may be located in a relatively quit area. To determine the suitability of an area for audiometric testing, it is necessary to carry out environmental noise measurements during the noisiest anticipated conditions. From these studies, the degree of attenuation required to permit accurate threshold audiometry can be determined. In most instance, it will be cheaper and more satisfactory to purchase a prefabricated testing room (audiometry room/booth) having sufficient attenuation to permit accurate threshold measurements under the worst conditions. To assure that audiogram taken in the plant prove acceptable technically, the programme should be under the supervision of the medical director/in-charge. In addition, the technician taking the measurement should be adequately trained. An industrial audiometric technician should be competent in obtaining medical and occupational exposure histories and measuring pure tone audiometric thresholds, when he observes abnormalities, he should refer the employee to a physician for examination. If the physician feels that special diagnostic tests are required, he may recommend that the employees receive diagnostic evaluation from an outside otologist or a hearing and speech centre. Employee identification is important because it permits verification of the record and avoid duplication. This identification usually consists of the employee's name, age, sex, badge and staff number, etc. It is important that no two persons be identified by the same number and that individual numbers remain the same for each person during the period of study. A brief history of ear disease, previous noise exposure and family history of deafness should be included along with present occupational and para-occupational history should be included and a simple examination of the external canal and drum should be performed and recorded. If environmental noise measurement has been made in the area where the individual works, that location should be described as accurately and completely as possible. It is also described. It is also desirable to record audiometric test conditions such as location and type of the test booth, manufacturer and type of audiometer, name of the examiner and method of procedure. Such factors as the day of work week, the hour of work day, and the interval between noise exposure and testing help to interpret the data. A hearing conservation programme includes preplacement, periodic (follow-up) and terminal audiogram for the exposed personnel. The specific interval between follow-up examinations will depend upon many factors including statutory requirement, the environmental noise exposure and the availability of the medical facilities. Where the sound pressure level in the working environment exceeds 85 dBA, follow-up threshold examinations should be instituted. The more the sound pressure level exceeds 85 dBA, the more urgent is the need for hearing conservation measure.

Measurement of hearing using electro-acoustic devices is generally referred to as audiometry. Pure tone audiometry records the threshold at octave intervals covering the frequencies most commonly encountered in everyday life and in particular in everyday speech. The frequencies usually tested in the clinical setting are 125 Hz, 250 Hz, 500 Hz, 1 kHz, 2 kHz, 4 kHz and 8 kHz. In the medico-legal and compensation fields, 3 kHz are always tested and 6 kHz may be. Air and bone conduction thresholds are measured to establish the presence or absence of the

conductive component of the overall hearing loss. Bone conduction thresholds are inaccurate above 3 kHz. Audiometry may be carried out by an audiometrician (manual audiometry) or using a self-recording audiometer. Hinchcliffe has pointed out the possible shortcomings of manual audiometry. Two different audiometricians might obtain significantly different thresholds at 3 kHz and 4 kHz on the same patient on the same day. Test-retest reliability on the same patient by the same audiometrician may yield differences of up to 25 dB. Self-recorded audiometry eliminates the variability due to audiometrician. By utilizing a combination of continuous and pulse test tones, there is a greater likelihood of picking up a spurious (nonorganic) hearing loss. A permanent record is obtained without the need for transcription with the risk of errors. Sweep frequencies are used because this technique may pick up notches at intervals not tested on fixed frequency audiometry. Thus, the preferred technique is sweep frequency self-recorded audiometry using both continuous and pulse tones. An audiometry programme ideally should consist of a pre-employment test followed by test at regular intervals. Important side issues include the calibration of the equipment, the acoustic requirements of the test area, instructions to the subject and the format and storing records. Electric response audiometry is an objective method of assessing hearing thresholds which has no place in the routine evaluation of hearing loss, but has a role in the medicolegal arena.

The interpretation of individual audiograms is usually straight forward, particularly if a pre-placement audiogram is routinely performed and presented to the physician, who interprets changes in threshold levels which may have occurred between periodic audiometric tests. When differences of 10 dB or more are observed at more than one frequency at or below 2000 Hz or when differences are found to be of 20 dB or more at 4000 Hz or above, an explanation should be sought. If there is no medical explanation for such a change and if the situation suggests a noise factor, a study of all employees working in the noisy area may be desirable. When the number of employees is small, and when no engineering control of the noise hazard is readily available, the most practical control measure may be personal protection. Where statistically significant group difference has been demonstrated and correlated with noise exposure, it is easier to convinced management that the expensive engineering or personal control measures may be a good investment. However, to be effective, hearing conservation requires close supervision and medical follow-up as well as an educational programme.

In evaluating the audiograms of employees from noisy environments, the possibility of temporary threshold shift should be recognized. Temporary threshold shift can be reduced or eliminated only by examining employees after they have been away from hazardous noise for 16 hours or more.

When permanent threshold shift is recognized in a large number of employees from a certain noisy department, management must be informed of the problem and its consequences. One or more control programs should be presented. When the permanent threshold shifts are limited to a small percent of exposed personnel who show progressive and significant changes in hearing threshold, a decision must be made as to whether the employees should be removed from the noisy exposure. Such a decision is very difficult and must be based on complete evaluation of the individual, the environment and the economic implications to both the individual and management. In the final analysis, the decision to remove an employee from a given noise exposure is a management decision but the evaluation and recommendation of the plant medical officer will weigh heavily upon it.

Audiological criteria: There are two types of audiological criteria, to be applied to baseline and periodic audiograms, respectively. A

baseline audiogram is the first audiogram performed; in some cases, this will be a pre-employment audiogram. Periodic audiograms, performed yearly, are to be compared to the baseline. The criteria for referral for threshold shifts seen on periodic audiograms should be clearly distinguished from criteria for threshold shift used to trigger 'in-house' action by the hearing conservation programme, such as refitting of hearing protectors or counseling. The Occupational Safety and Health Administration (OSHA) defines a 'standard threshold shift' as a shift of 10 dB or more, in either ear, for the pure tone average of 2, 3, 4 kHz; this is intended to detect small changes in hearing so that preventive action can be taken. The criteria for threshold shift listed below, on the other hand, are intended to detect larger changes which are more likely to be significant, both medically and in terms of communicative difficulties. Programme directors may choose to have repeat audiograms done to verify apparent threshold shifts prior to taking action. If a periodic audiogram or retest demonstrates a 'standard threshold shift' as defined by OSH, this audiogram becomes the new baseline, to which subsequent periodic audiograms will be compared. However, for purpose of otologic referral, the original baseline should continue to be used. Otherwise, a slowly progressive hearing loss could become severe over a period of years: a series of small threshold shifts would occur, each triggering 'in-house' action and resulting in a new baseline, but none large enough to result in otologic referral.

In addition to the quantitative criteria listed below, workers showing variable or inconsistent responses or unusual hearing loss curves should be referred.

A. Baseline audiogram:
   1. Average hearing level at 0.5, 1, 2, and 3 kHz greater than 25 dB, in either ear.
   2. Difference in average hearing level between the better and poorer ears of: (a) more than 10 dB at 0.5, 1, and 2 kHz, or (b) more than 20 dB at 3, 4, and 6 kHz.

B. Periodic audiogram: Change for the worse in average hearing level, in either ear, compared to the baseline audiogram of:
   a. more than 10 dB at 0.5, 1, and 2 kHz, or
   b. more than 20 dB at 3, 4, and 6 kHz.

Medical Criteria

1. History of ear pain, drainage, dizziness, severe persistent tinnitus, sudden, fluctuating, or rapidly progressive hearing loss, or a feeling of fullness or discomfort in one or both ears within the preceding 12 months.
2. Visible evidence of cerumen (wax) accumulation or a foreign body in the ear canal.

If a person has received otologic evaluation previously on the basis of failing the foregoing criteria, he should be re-evaluated if he develops ear pain, drainage, dizziness, disequilibrium, imbalance, or severe persistent tinnitus or shows significant change in hearing levels as defined in Section B above. In patients with puzzling ear symptoms, such as diplacusis, fullness, and inconsistent audiometric finding, it is better to refer than possibly overlook a significant problem.

Information and education: Workers at risk of occupational noise-induced hearing loss need to be educated about the harmful effects of noise and the importance of hearing conservation measures. They need to understand the important of wearing properly fitting and appropriate ear protections. The attitude not to wear ear protectors must be changed. Workers should recognize and report the first symptoms of deafness, such as temporary threshold shift and tinnitus. Oral explanation, individual counseling and lectures as well as leaflets, posters, films and videos may all be employed. Education must

be extended to management so that their responsibilities are clearly defined, and managers exposed to potentially damaging sound levels must themselves be conscientious in their observation in safety measures.

Audiogram as an educational tool: By showing the worker his personal record of hearing sensitivity, the nurse or physician has a most effective device for the promotion of hearing protection. If the audiogram is used to explain the status of worker's hearing at the time the hearing protector is being fitted for the first time, success almost invariably is ensured. The wearer now knows that his hearing is so good that it must be preserved at all cost. If the audiogram indicates that his acuity is deteriorating in the higher frequencies, the need to do all humanly possible to save the amount remaining is imperative. Utilization of graph type of audiogram is reported to be the physician's and nurse's most useful tool in obtaining 100% successful protection in the plant or workplace. Too often the 'why' and 'how' of hearing protection are completely ignored. It is only assumed that the worker should be delighted to wear protective devices. Hearing protection is the matter of counseling the worker and this require time. For instance, in the course of an audiometric examination or a fitting for a protective device, employees should be alerted to and advised about 'off-the-job' noise exposures as well as noise hazards at the workplace.

Workers and management must understand the potentially harmful effects of noise to ensure the hearing conservation programme is successful in preventing noise-induced hearing loss. A good worker's education programme describes: (1) Programme objectives, (2) existing noise hazards, (3) how hearing loss occurs, (4) purpose of audiometric testing, and (5) what workers can do to protect themselves. In addition, roles and responsibility of employees and the workers should be stated clearly. Training is required to be provided annually to all workers included in the hearing conservation programme. Opportunities for maintaining awareness occur during periodic safety meetings, as well as during audiometric testing appointments when testing results are explained.

Nonorganic (or Exaggerated, or Functional) Hearing Loss

In the medicolegal context, with the attendant lure of financial gain from compensation, the doctor assessing an industrial deafness case has to be constantly aware of the possibility of exaggeration of a hearing loss. Experience clinicians and audiometricians may develop a finely honed sixth sense, based on the inappropriate or pantomimic behaviour of an individual subject. There may be a clear discrepancy between the perceived hearing of the subject in interview, and the volunteered thresholds on pure tone audiometry. Further discrepancy may exist between the pure tone threshold and the score on speech audiometry. There may be test-retest unreliability on audiometry, and there may be a lack of agreement between pure tone thresholds obtained with ascending and descending stimulus intensity. Sweep frequency audiometry may reveal a pattern suggestive of non-organic hearing loss. Not frequently, however, one may have to turn to electric response audiometry for verification. Electric response audiometry (ERA) or evoked response audiometry provides a method of objectively estimating the auditory thresholds, by the recording of the electrical events which occur in the auditory pathways following the exposure of the ear to an acoustic stimulus. The stimuli used are either broadband clicks containing a wide spectrum of frequency, or more frequency-specific stimuli, such as filtered clicks or tone bursts. A single such stimulus will give rise to electrical potential changes in a series of neural structures from the cochlea to the auditory cortex. These events have a characteristic form and latency depending upon which part of the auditory pathway is being studied.

The best estimate of hearing is a standard pure tone audiogram carried out on a willing and cooperative subject. Electric response audiometry (ERA) is used when that ability or willingness are absent. The preferred technique in the evaluation of a subject suspected of exaggerated hearing loss is corrected electric response audiometry (CERA), but two other techniques, auditory brainstem response (ABR) and electrocochleography (ECochG) are occasionally employed.

INDUSTRIAL VIBRATION

Introduction

Occupational vibration reaches the worker through different paths or transmission routes. In 'whole-body vibration' it is conducted through a contacting or supporting structure that is itself vibrating like a ship's deck, the seat of a vehicle traversing rough terrain, a vibrating platform. There are vibrating work processes affecting the whole body in coal, iron ore and cement machinery that prevent blow holes in the final castings. In other industrial processes, the route of entry is through paths to the hands, wrists and arms of the subjects, so-called 'segmental vibration' or 'hand-arm vibration'. The examples of hand-arm vibration are generated from pneumatic tools, electric-driven grinding, burrowing tools, rock drilling, and cross cutting timber. The prolonged use of vibrating tools and equipment can lead to a number of pathological effects. Physicians supervising a vibration-exposed work force should know the general principles involved in the recognition, evaluation, and control of vibration as a physical agent.

Occupational health professional needs to have some understanding of vibration measurement and its implications when assessing individual cases. Vibration is an oscillatory motion that can be represented by a simple harmonic sine wave with the properties of displacement, a velocity and acceleration. In practice, the wave is a complex, of differing frequencies and acceleration. Vibration can move in three orthogonal directions (x-, y- and z-axes). They differ from when the subject vibrates from front to back (x-axis) or from side to side (y-axis) or in the vertical mode (z axis). To assess the medical effects, the accelerations and frequencies must be measured along the three coordinates of the biodynamic system proposed by the International Standard Organization (ISO), the British Standards Institution (BSI), the American National Standards Institution (ANSI) and the American Conference of Governmental Industrial Hygienists (ACGIH). It is the vector sum that is normally calculated (ISO 5349). The measurement of interest, in terms of biological effect, is the magnitude of vibration or 'A(8)' which incorporates intensity, duration and direction. The daily exposure to vibration of a person is ascertained using the formula:

$$A(8) = a_{hw}\sqrt{\frac{T}{T_o}}$$

where a_{hw} is the vibration magnitude in metres per second squared (m/s^2); T is the duration of exposure to the vibration magnitude a_{hw} and T_o is the reference duration of eight hours (28800 seconds). A frequency weighting up to 16 Hz is factored into calculations to take account of the supposedly more damaging lower frequencies suggested by laboratory studies.

Segmental Vibration: The Hand–Arm Vibration Syndrome (HAVS)

The medical effects of using hand-held vibrating tools are more serious and more clearly defined than those caused by whole-body vibration. With the increasing technical developments since 1900, the use of vibrating tools has greatly expanded. Outbreaks of 'white finger' arising from both air-driven and electric-power tools were reported by Seyring, Hunt, Telford and Agate. Throughout the early literature this vibration hazard is

described variously as traumatic vesospastic disease, dead fingers, spastic anaemia, Raynaud's phenomenon of occupational origin, vibration induced white finger, and finally, since 1985, the hand-arm vibration syndrome.

Classification: The vascular and sensorineural components of HAVS are graded separately by Stockholm workshop scale (SWS). The scale is a clinical grading and not a disability scale. It has, however, been used to make recommendations on employability. Limitation in definitions used in the scale has led to suggested modifications.

Pathophysiology: There is uncertainty as to the exact pathogenesis of hand-arm vibration syndrome. The vascular and neurological components of the hand-arm vibration syndrome may, however, share a common pathogenesis. Vasospasm may be initiated by nerve fibre dysfunction in the vessels wall, or conversely numbness and tingling may be due to damage to the intraneural vessels. In addition, nerve injury, carpal tunnel syndrome and Dupuytren's contracture can all be associated with vascular dysfunction.

Sensorineural hand-arm vibration syndrome: In the natural history of the condition, neurological (sensorineural) symptoms tend to occur first. Most surveys record them as twice as common as vascular symptoms and generally causing greater disability. The latent period for sensorineural symptoms varies from few months to many years. Use of vibratory tools causes transient paraesthesia in the fingers of most users. This usually passes off within 20 minutes. Often, the first abnormal symptoms are an extension of this time. Tingling or numbness usually starts at the fingertips, but can affect all, or the whole, of the fingers. With continuing exposure, these symptoms may become persistent lasting for hours or even all day. Although aggravated by coldness, the tingling and numbness can occur in a warm environment, causing considerable distress. The sensory loss causes workers to describe their fingers as feeling thick or 'like bananas' in many instances. There is a loss of feeling, of temperature appreciation and of pain sensation. In severe cases, workers can experience clumsiness and loss of manual dexterity, with poor finger coordination causing inability to do fine work. Assessing a case of suspected neurological HAVS requires details of vibration exposure, past medical history and a clinical history of date of onset of symptoms, site of paraesthesia, time of occurrence and duration of symptoms. Nocturnal symptoms may occur,

The Stockholm workshop scale for the classification of HAVS

Stage	*Grade*	*Description*
Vascular component		
0		No attacks
1V	Mild	Occasional attacks affecting only the tips of one or more fingers
2V	Moderate	Occasional attacks affecting distal and middle (rarely also proximal) phalanges of one or more fingers
3V	Severe	Frequent attacks affecting all phalanges of most fingers
4V	Very severe	As in stage 3, with trophic changes in the fingertips
Sensorineural component		
0SN		Vibration-exposed but no symptoms
1SN		Intermittent numbness with or without tingling
2SN		Intermittent or persistent numbness, reduced sensory perception
3SN		Intermittent or persistent numbness, reduced tactile discrimination and/or manipulative dexterity

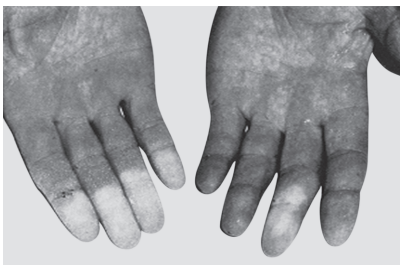


Fig. 8.23: White finger

but are less frequent than in case of carpal tunnel syndrome. There is no gold standard to diagnose sensorineural HAVS and no single test with sufficient specificity or sensitivity; researchers have recommended the use of vibrotactile threshold test (VTT) and thermal aesthesiometry test (TA).

Modification of Stockholm workshop scale for classification of SN component of HAVS

Stage	*Criteria*
0SN	Vibration exposure, but no symptoms
1SN	Intermittent numbness and/or tingling with tingling with a sensory neural score of ≥ 3 to < 6
2SN (early)	Intermittent and persistent numbness and/or tingling, reduced sensory perception with a score of ≥ 6 to <9
2SN (late)	As 2SN (early), but with a score of ≥ 9 to <16
3SN	Intermittent and persistent numbness and/or tingling, reduced manipulative dexterity and a SN score of ≥ 19

Vascular clinical features: The vascular component is one of arterial vasospasm, and the clinical features are indistinguishable from Raynaud's phenomenon. Vibration induced Raynaud's phenomenon is graded on the Stockholm scale according to the severity of the symptoms and the extent of the blanching. The onset of the HAVS is dependent on two factors. The first is the susceptibility of the individual using vibrating tools to the harmful effects of vibration, and the second is the vibration dose received, higher dose leads to an earlier onset. The first symptom or sign of digital arterial vasospasm is the onset of blanching affecting the tip of one or more digits and occurring in response to cold. Gradually, the extent of the blenching increases until the full length of each finger may be affected. The thumbs tend to spared, but they can be affected particularly in more severe cases of HAVS, or when the thumb is particularly exposed to vibration. The blanching of the fingers may just extend on to the most distal aspect of the palms. The blanching is circumferential and sometimes obliquely so, affecting the territory of one digital artery, but not both. A typical attack lasts for between 20 and 30 minutes, although it can last longer up to an hour or so. Recovery can be hastened by warming of the hand, although this may be painful. True digital artery vasospasm is accompanied by numbness in the fingers due to transient ischaemia of the digital nerves. The area of blanching is normally sharply demarcated from the surrounding normal skin. In the recovery phase, the circulation is restored to the digit from the base towards the tip, and there is usually a reactive hyperaemia, reflecting the acidosis or ischaemia that will have developed in the digit during the period of ischaemia. The finger often appears a prominent red colour as recovery proceeds, with ultimately a normal pink colour being resumed.

Hand–Arm Vibration and Muscles Weakness

Patients suffering from HAVS are frequently noted to suffer from weakness of grip. The workers exposed to the use of vibrating tools frequently complain of weakness of grip and research studies have shown the vibration can cause damage to the small muscles of the hand and the nerves that supply them. The onset of weakness requires further investigation to identify any possible dose response. This will allow development of guidelines for working practice. All studies concerning grip strength need to be tightly controlled for age. In addition to muscle weakness, vibration may affect bones also and bone cyst or osteoarthritis to wrist or elbow may develop.

Diagnosis of HAVS

Examination models for the diagnostic work up of subjects with possible injury from work with vibrating tools and equipment vary with the purpose of the assessment. These models range from questions about symptoms, signs,

and the possible association between exposure and injury; to questions on fitness for work, disability. The clinical examination is done to confirm a possible association between vibration exposure at work and injury, the assessment may include the following aspects:

1. Evaluation and judgment of symptoms and signs, and grading of severity;
2. Exposure assessment that includes both vibration and ergonomic aspect; and
3. Excluding other potential differential diagnosis.

If, after history and examination, there is no or low suspicion on an underlying disease, then there is no need for further specialized testing. If there is a suspicion that the patient has secondary Raynaud's phenomenon, then further evaluation is needed. The musculoskeletal examination should follow standard clinical orthopaedic examination, specifically looking for signs of carpal tunnel syndrome, Dupuytren's contracture and osteoarthritis.

A physical examination is followed by a series of objective tests if needed, for which following tests are proposed:

Vascular

1. Adson's test (neck rotation) Doppler (finger blood flow).
2. Lewis Prusik (nail-bed compression).
3. Allen's test (ulnar, radial compression at wrist).
4. Cold provocation test (3 minutes at 10°C).

Neurologic

1. Light touch (cotton wool)
2. Pain (pin prick)
3. Temperature (probe)
4. Vibrotactile threshold test (VTT)
5. Thermal aesthesiometry test (TA)
6. Phalen (wrist flexion)
7. Tinel (tunnel percussion)

Musculoskeletal

1. Grip strength (dynamometer).
2. Pinch test (thumb and forefinger).
3. Moberg pick-up object Recognition.

In addition to above screening tests, the following laboratory data are required: blood count, ESR, blood glucose determination, thyroid studies, vitamin B_{12} level, renal and liver functions, rheumatoid factor, cryoglobulins, serum protein electrophoresis, cold agglutinins, complement (C3 and C4) and ANA (if positive, tests for antitopoisomerase I and anticentromere antibodies are indicated); and X-ray of cervical spine, hand and chest (for cervical rib and bone changes). If the screening tests for carpal tunnel (Phalen and/or Tinel) are positive, nerve conduction velocity tests will be required for the median and ulnar nerves. The nerve conduction data will be necessary for the medicolegal cases. In view of the possible synergistic effect with noise, an audiogram is advised. The results of the objective tests and the subjective history of HAVS attacks, together with an estimate of vibration dosage (intensity and duration), enable the physician to formulate a dose-response relationship and thus establish first a diagnosis of HAVS and second a stage assessment (vascular and sensorineural) for each hand with reasonable certainty. *Finger systolic blood pressure:* Raynaud's phenomenon can often be diagnosed on the basis of a medical interview, a questionnaire and by identification of white fingers on a colour chart. In some countries, medicolegal demands are to conduct finger systolic blood pressure measurement (comparing distal systolic blood pressure before and after cooling). Quantitative sensory testing (QST), Electrodiagnostic testing (for muscles weakness), biopsy of skin and nerve and MRI are sometime recommended.

Workplace Health Surveillance

Workplace health surveillance is primarily for the detection of adverse health effects at an early stage. It can also be used as a means of identifying and protecting susceptible individuals at increased risk from the effects of hand-arm vibration. In addition, it can provide feedback on the effectiveness of control

measures that have been introduced, and also to prevent the progression of HAVS to the more severe and disabling stages.

Preventive Measures

1. The vibration level or intensity and its spectral content: The known hazardous frequency range is assumed to lie in the 30 Hz to 300 Hz. Recent research suggests that high frequencies partly neglected by a 'weighting curve' should not be discarded at the present cutoff 1500 Hz frequency but are to be extended to 5000 Hz, when estimating HAVS dosage.
2. Vibration exposure time: There is a clear correlation between increasing impairment and deterioration with increasing vibration exposure time.
3. Continuous vibration, as opposed to interrupted work schedules, should be avoided; use 10-minute breaks or rest periods every continuous exposure hour.
4. An increasing grip force on tool handles (coupling at the hand-tool interface) increases the vibration energy to the hand with greater damage to arteries, nerves, tendons, joints, and muscles.
5. The lighter the tool, the more energy is transferred to the hand since there is a less damping. Reduction in the weight in a hand-held vibratory tool reduces the workload on joints (elbow, shoulder) but increases the vibration.
6. Gloves are inefficient as protection from HAVS. Conventional glove designs provide little attenuation below 500 Hz. Gloves with built-in absorbent (damping) material is usually bulky, thus reducing manipulative efficiency. The absorbent material may in some designs actually increase vibration through material resonances.
7. As with all occupational hazards, there is an individual susceptibility. The so-called latent interval, the period between first exposure to vibration and the appearance of the first white finger (s) tip, is a valuable factor in assessing HAVS damage. The shorter this interval, the more hazardous the vibration. A subject with a very short latent interval compared to the mean of this vibration-exposed population is 'susceptible' to vibration effects.

Treatment of HAVS

There is no active treatment for neurological symptoms. The treatment of vascular phenomena is the same as the treatment of Raynaud's disease. The general measured are aimed at the prevention of the signs and symptoms. The workers should specifically keep the hands warm, as well as the body. Simply wearing gloves is not enough if the body is cold. Working in a warm environment is the ideal, but often not feasible. Drug treatment is often of little benefit and the side effects of medication usually preclude its use. Alpha-adrenergic blockers will cause vasodilatation, but are associated with tachycardia, hypotension and a stuffy nose. Calcium-channel blockers, such as nifedipine, may be used with some success but the beneficial effects are not sustained in the long term with side effects. Other agents, such as endothelin receptor antagonists, have been tried in the management of severe Raynaud's phenomenon.

Whole Body Vibration

Whole body vibration (WBV) occurs when the human body is supported on a surface that is vibrating. Vibration is an oscillatory motion characterized in terms of the frequency of the oscillatory cycle, its magnitude and its direction. The magnitude of vibration can be quantified by its displacement (m), its velocity (ms^{-1}) or its acceleration (ms^{-2}). However, it is usually expressed in terms of an average measure of the motion's acceleration [to give the root mean square (rms) value in ms^{-2}] so that a complex time-varying pattern is not unduly influenced by a few unrepresentative peaks. The rms magnitude can be related to

the energy imparted and hence to the injury potential.

The frequency of vibration is expressed in cycles per second (Hz). The adverse health effects of WBV seem to be related mainly to exposures in the low frequency range, and frequencies below 3 Hz can cause motion sickness. Thus, to account for differences of response, current standard for exposure evaluation recommended weighting the frequencies of measured vibration according to the assumed deleterious effects of each frequency. Frequency weightings are applied to measurements taken in three orthogonal directions (x-, y-, and z-axes) at the interfaces between the body and the vibration.

Health Effects of WBV

The best-recognized health effect of WBV relates to the outcome of low-back pain (LBP). LBP is a symptom rather than a disease and consequently case definitions of outcome in research studies have varied in relation to anatomical distribution symptoms and in relation to their frequency, intensity and impact in terms of disability, medical consultation and sickness absence. Other health effects include radiating leg pain and sciatica and prolapsed intervertibral disk. Other suggested effects of WBV are neck–shoulder pain or cervical disc degeneration, autonomic disturbance, disorder of balance and digestion, and effects on menstruation and perhaps labour.

There may be motion sickness include drowsiness, nausea, vomiting (often with little warning), stomach fullness, loss of appetite, facial flushing, dizziness, headache, inability to concentrate and sweating. It depends on the frequency (e.g. cycles per second) of vibration in Hz (usually below 3 Hz).

Pathophysiological Mechanisms

The role of WBV in the pathogenesis of LBP remains uncertain. A popular working hypothesis is that vibration causes mechanical overloading which leads to premature degeneration of the lumber spine. Biodynamic and physiological experiments suggest the possibility of several interacting mechanisms, e.g. muscle fatigue, reduced disc height and bending of the spine jointly leading to high strain on the vertebral column. Direct mechanical injury may be one of the causes.

Factors Affecting Exposure and control Measures

In addition to magnitude, duration and pattern of WBV, several other factors may relate to the injury potential, including individual susceptibility to injury, the dynamic response of the human body (mechanical impedance, vibration transmissibility, absorbed energy), body posture and health status. Dose may be related to the design of the vehicle, the cabin seating, the suspension, the road condition, the road speed and ultimately the behavior and training of the driver. It is required that the employer to assess and if necessary, measure the level of WBV to which workers are exposed.

Actions as Preventive Measures

1. Measures the vibration and evaluate vibration exposure time.
2. Adjust the subject's work schedule vis-a-vis vibration level.
3. Examine the ergonomic design (seat, instruments, control) with appropriate antivibration absorbent materials—advances have been made in "air-ride" vibration isolators.
4. Institute pre-employment and periodical medical examinations with special reference to thoracic and lumber vertebra. If the exposure is severe, X-ray examination is recommended every 4 years.
5. Undertake routine engineering maintenance.
6. Measure vibration of new plant machines, usually installed as replacements to increase production.

Health Surveillance

Health surveillance, the result of which are taken into account in the application of preventive measures at a specific workplace, shall be intended to prevent and diagnose rapidly any disorder linked with exposure to mechanical vibration. Such surveillance shall be appropriate where:

1. The exposure of workers to vibration is such that a link can be established between that exposure and an identifiable illness or harmful effects on health.
2. It is probably that the illness or the effects occur in a worker's particular working conditions.
3. There are tested techniques for the detection of the illness or the harmful effects on health.

The main virtues of health monitoring may lie in identifying extremes of unacceptable exposure, in ensuring systematic coverage, and in gaining a group level impression of the success in controlling health problems. Where surveillance is undertaken, it should be managed by certified occupational health personnel.

IONIZING RADIATION

Introduction

Ionizing radiation is any radiation consisting of moving particles or electromagnetic waves that carries sufficient energy to produce ions in matter. Ionization occurs when enough radiation energy is transferred to atoms in the material through which it is passing to displace an orbital electron, thus, it leaves these atoms as electrically charged ions. In tissue, the ionization of atoms within cells produces immediate biochemical changes that may result in immediate or late biological effects.

All matter is made-up of atoms. Each atom contains a nucleus around which electrons orbit. In the nucleus, there are protons and neutrons. All atoms of the same chemical element have an identical number of positively charged protons in the nucleus and negatively charged electrons on the orbits. So, an undisturbed atom is electrically neutral. The number of protons defines the atomic number of the element. The mass of the atom is determined by the number of protons and neutrons and the total number is called the mass number. The same element can have different number of neutrons and subsequently different mass numbers. These variants of the elements are known as isotopes. Some of these isotopes are unstable and eventually transform into atoms of another element with the simultaneous emission of alpha (α)- or beta (β)-particles and accompanied usually by gamma (γ)-rays. This property of the unstable atom is called radioactivity; the change itself is called radioactive decay and the unstable is said to be a radionuclide. The time necessary to reach a stable form depends on the particular isotope and may take a few fractions of a second to several thousand years. The time for the activity to decay by one half is termed the half-life ($t_{1/2}$). For example, sodium-23 (^{23}Na) is the stable form of sodium and ^{24}Na is a radioactive isotope with a $t_{1/2}$ of 15 hours. The later decays emitting a α-particle to become ^{24}Mg, a stable isotope of magnesium. This activity is measured by the numbers of disintegrations per unit time. The unit by which radioactivity is measured is the Becquerel (Bq) and 1 Bq equals one atomic disintegration per second.

Ionizing radiation may be higher energy electromagnetic radiation (X-rays and α-rays) or energetic subatomic particles such as beta (β)-particles and neutrons. According to their energy, X-rays and α-rays interact with matter and tissue and although the mechanisms may be different, they all produce positively and/or negatively charged ions, which then interact with the absorbing matter to produce physiochemical changes by adding or subtracting electrons. The energy of these electromagnetic radiations will also determine

their penetration, higher energy photons penetrating further than low energy one. When they do interact with tissues and cells, energy is deposited within the tissue.

The different radiations penetrate matter in different ways, the properties being determined by the size, charged and energy of each type; α-particles are stopped by a thin piece of paper or the dead layer of the skin, while β-particles can penetrate the hand but will be stopped by a thin sheet of aluminium; X-rays and γ-rays penetrate the body and an aluminium sheet, but are stopped by lead. Neutrons penetrate most materials, but may be stopped by thick polythene or concrete (hydrogenous materials); high energy neutrons have a high penetrance in tissue but low energy neutrons can be absorbed in the body because of the body's high water content. These overall properties of radiation affect the degree of cellular damage following exposure and the methods needed for protection.

In general, α-particles do not constitute a significant hazard as an external source, but are hazardous when taken into the body. When incorporated, they can irradiate adjacent cells in, for example, the liver. Neutrons, because of their absence of electrical charge, produce ionization indirectly and tend to be more penetrating. Ionizing radiations have sufficient energy to break chemical bonds and ionize atoms and molecules, producing an ion pair. These ions are charged and capable of causing further ionization and energy deposition leading to physicochemical changes in cellular constituents.

Units of Radiation

The activity of a radioactive material is the number of nuclear disintegrations per unit of time. The unit of activity is a Becquerel (Bq). One Bq is equal to one disintegration per second. Formerly, the unit of activity was Curie (Ci) and one Bq corresponds approx. to 17 pico-curie.

The potency of radiation is measured in three ways: (a) *Roentgen*: It is the unit of exposure. It is the amount of radiation absorbed in air at a given point, i.e. number of ions produced in 1 ml of air. Now, Roentgen is being replaced by the new SI unit (International System Unit) to Coulomb per kilogram (C/kg). One Roentgen is equal to 2.58×10^{-4} C kg^{-1}. (b) *Rad*: It is the unit of absorbed dose. It is the amount of radioactive energy absorbed per gram of tissue of any material. As per SI Unit, Rad has been replaced by Gray (Gy). It is the unit of absorbed dose, defined as the dose of ionizing radiation that imparts 1 joule of energy to 1 kg of absorbed material. One Rad is equal to 0.01 Gy. (c) *Rem*: It is the product of the absorbed dose and the modifying factors. It indicates the degree of potential danger of health. Sievert (S_V) is now replacing Rem. It is the SI Unit of dose equivalent. The dose equivalent of 1 sievert is equal to 100 Rems.

Dose Quantities

Some of these physicochemical changes in cellular constituents may be of no biological consequence and others may be repaired, but there is a finite probability that the damage

Properties of ionizing radiation

Type	*Range in air*	*Range in tissue*	*Hazard*	*Examples*
Alpha (α)	Few cm	10 s of μ	Internal	Plutonium
Beta (β)	Up to several mtrs	Few mm	External and internal	Caesium
Gamma (γ)	Many mtrs	Many cm	External and internal	Cobalt source
X-Ray	Many mtrs	Many cm	External and internal	Hospitals
Neutron	Many mtrs	Many cm	External and internal	Reactors

Source: National Radiological Protection Board, Chilton, UK (now the Radiation Protection Division of the Health Protection Agency)

may cause cell death or irreparable damage to vital cell constituents. The absorbed dose is a measure of the mean energy absorbed by unit mass of tissue, and the absorbed dose in grays (Gy) is equal to the deposition of one joule (J) of energy in 1 kilogram (kg) of tissue. Overall, the greater the dose, the greater the likelihood of a biological effect being seen. Energy is deposited along the path of ionizing radiation as it traverses tissues in the form of ionizations. The average deposition of energy per unit length is called the linear energy transfer (LET).

Charged particles tend to have higher LET values than X-rays or α-rays. The International Commission on Radiological Protection (ICRP) has introduced a weighting factor related to LET to take into account these differences. These radiation weighting factors (w_R) may range from 1 to 20 for different radiations. Thus photons, such as X-rays or α-rays, are assigned as w_R of 1 (low LET) and α-particles 20 (high LET). Tissues are also assigned weighting factors (w_T) to differentiate the wide variation in tissue sensitivity to radiation, the lymphopoietic stem cells and gonadal germ cells being the most sensitive and bone being relatively insensitive. These radiation and tissue weighting are used to convert the absorbed dose in grays to an effective dose in sieverts (SV). This system allows external and internal exposure to be combined into one dose—on the basis of equality of risk. One a radionuclide is incorporated; it will continue to expose surrounding tissues until final decay or it is excreted. It is usual to calculate this committed effective dose following the ingestion or inhalation so that extra care can be taken to reduce future external exposure.

Submultiples of the sievert are commonly used, such as the millisievert (mSv), which is one-thousandth of a sievert; for example, the world average individual dose received due to exposure to natural background radiation is about 2.4 mSV per year compared with the occupational dose limit of 20 mSv average per year over defined periods of five years. It is sometimes useful to have a measure of a total dose to groups of people or a population. The quantity used to express this total is the collective effective dose: It is obtained by summing the product of all the doses in a group and the number of people in the group and is usually expressed in person-sieverts.

Equivalent Dose

Equivalent dose is the absorbed dose averaged over a tissue or organ (rather than a point) and weighted for the radiation quality involved in the exposure. The gray specifies an absorbed dose of radiation energy, but it turns out that observed biological effects do not always relate directly to the absorbed dose. Thus, 1 Gy of X-ray dose does not have the same effect as 1 Gy of neutrons. A radiation quality factor (QF) is used in calculating an equivalent dose to adjust for such differences. The QF is a LET-dependent factor used to weight absorbed doses and calculate (for radiation-protection purposes) equivalent doses that express the health effects risk for all types of ionizing radiation on the common scale. The QF factor is 1 for X-rays and gamma rays, 1 for electrons, 5 for protons, 10 for neutrons, and 20 for alpha particles. In the latest recommendations of the International Commission on Radiological Protection (ICRP), the QF has been replaced by the radiation weighting factor (W_R), which serves the same purpose as the QF. The recommended numbers for the W_R of various radiation types are similar to those given for the QF. The only difference is the neutron values, which are now 5, 10 or 20 depending on the neutron energies involved. These factors are arbitrary values which are generally based on the relative biological effectiveness of that radiation type in inducing stochastic effects, such as cancer, at low doses. Reactive biological effectiveness (RBE) is a factor used to compare a specific type of biological effect caused by absorbed radiation doses in Gy (rad) of a particular radiation

under study with that effect for the same doses of 250 kVp X-rays. The SI unit of equivalent dose, name the Sievert (Sv), is the dose of 1 J/kg weighted by the appropriate QF or W_R. The Sv is equal to the absorbed dose of one Gy times the radiation weighted factor or QF.

Equivalent dose (H) = Dose (D) × QF

Detection of Ionizing Radiation

Physical: Ionizing radiation cannot be directly detected by the human senses, but they can be detected and measured by a variety of means, such as photographic films, Geiger tubes and scintillation counters. There are also relatively new techniques using thermoluminescent materials and silicon diodes. Some of these techniques are used to measured individual doses on dosimeters (generally called film badges). Measurements made with such dosimeters can be interpreted to represent the energy deposited in the body or in a particular part of the body by the radiation concerned.

Biological: When a radionuclide is deposited in an internal organ, for instance via ingestion or inhalation, dose is generally calculated, because the activities are too small to be measured. The dose (or activity) can be assessed or measured by taking urine or blood samples and applying biokinetic models or be measured directly by special detection systems, e.g. whole body monitors. These systems are only available at special sites in the United Kingdom, such as British Nuclear Fuels, the Atomic Weapons Establishment, the Health Protection Agency, Sellafield Ltd or Harwell.

In an accident situation, estimating the whole-body dose by changes in circulating absolute lymphocyte counts in the first 48 hours is possible and by cytogenetic assays for chromosome aberrations in cultured lymphocytes. These techniques use either dicentric counting or more recently fluorescence *in situ* hybridization (FISH) painting of chromosomes and measuring the translocation of genetic material. The threshold for whole body dose-validated chromosomal changes is around 1–200 mSv. More recently, electron paramagnetic spin resonance techniques have developed using teeth of, in the case of cadavers, bone, which permits a retrospective assessment of the dose. The accurate range for this technique is from around 100 mGy to lethal doses. Another technique using post-subcapsular lens opacities also offers a possible way to assess historic doses; however, its accuracy is not yet proven.

Health Effects of Ionizing Radiation

Any organ or tissue may be affected, the degree varying with the dose and the radiosensitivity of the given organ or tissue. Distinguishing two types of effects is possible, somatic and hereditary. The somatic effects relate to the individual who is exposed, and may be early or late and in the embryo or foetus may be teratogenic. The hereditary effects would occur in the offspring, through genetic damage to germ cells of the exposed individual.

Somatic effects: Somatic effects are classified as *deterministic* and *stochastic*. Deterministic effects are those for which the severity increases with the dose and for which a threshold exists. Stochastic effects are those whose probability of occurrence increases with the dose and whose severity is independent of the dose and without a threshold. With deterministic effects, cause and the effect can be seen. However, due to the random nature of the interaction of radiation with matter, for stochastic effects the inference of cause only be based on an increase in the probability of that effect. Where the dose is low(as in occupational exposures), only stochastic effects might be seen. The severity of stochastic effects is not dose dependent as it is with deterministic effects. So, an increase in a dose produces an increase in the probability of a stochastic effect.

Deterministic effects: General dose–response relationships are sigmoid in shape and exhibit a threshold. For each deterministic

Approximate threshold levels of dose (ED_0) for deterministic effects (adult exposure)

Organ	*Effect*	*ED_0 (threshold dose) (Gy)*
Whole body	Early death	1.5
	Prodromal syndrome (e.g. vomiting)	0.5
Bone marrow	Early death	1.5
	Depression of haematopoiesis	0.5
Lung	Early death	6
	Pneumonitis	3–5
Skin	Prompt erythema, dry desquamation	^>8
	Moist desquamation and necrosis	>15
Thyroid	Hypothyroidism	3
Lens of the eye	Detectable opacity	0.5–2
	Cataract with loss of vision	5
Testes	Temporary sterility	0.15
	Permanent sterility	3.5–6
Ovaries	Permanent sterility	2.5–6
Embryo or fetus	Teratogenesis	0.10

effect, the two main parameters to consider are the threshold dose (ED_0), at which the given effect may appear, and its relative severity. These effects are characterized by the median dose, ED_{50}, at which 50% of exposed individuals will exhibit the effect, and by the slop of the curve at the median. The dose-response relationships are generally quoted for acute exposure; protraction of exposure increases the median dose for the effect. Doses below which selected deterministic effects should not occur in a normally distributed population (ED_0) are given below. These levels take into account the individual variation and sensitivity rather than the average value. They are not complete and are for guidance only. The values should not be used in conditions of known radiosensitivity, e.g. ataxic telangiectasia.

Haematopoietic system: The bone marrow is the main concern since exposure to penetrating radiation at high dose rate can lead to death within a few weeks. This early mortality results from stem cell depletion in the marrow. The lymphocytes are the most sensitive indicators of injury to the bone marrow. Acute doses of 1–2 Gy reduce their concentration in the blood 50% of their normal level within about 48 hours. Neutrophils and platelets also show a dose-related decrease in concentrations and the levels can be used to predict the likelihood of survival and the necessity for treatment. People with bone marrow aplasia show an increased susceptibility to infection and frequently spontaneous bleeding (from thrombocytopenia) as a direct result of damage to the immune and haematopoietic systems. In severe cases of radiation injury, marrow aplasis is the likely cause of death. The median lethal dose for human is not precisely known. Several estimates have been published ranging from 2.4 to 5.1 Gy bone marrow dose. The higher values of the estimates of the LD_{50} involved cases where significant supportive and therapeutic medical treatment was provided. With minimal medical treatment involving no more than basic first aid, a value for LD_{50} of 3 Gy has been adopted by United Nation Scientific Committee on the Effects of Atomic Radiations (UNSCEAR), the National Radiological Protection Board (now part of the Health Protection Agency) and the Nuclear Regulatory Commission, USA (NUREG). For supportive medical treatment which includes procedures such as reverse isolation procedures, the use of antibiotics, white cell and platelet transfusions and intravenous feeding, NUREG 11 recommend a value for LD_{50} of 4.5 Gy. Recent advances in rescuing depleted marrow with stem cell stimulating growth factors would now be included in treatment. These offer hope that the LD_{50} will be raised, possibly to the point where death no longer depends on haematopoietic damage. The lymphocyte count decreases within a few hours of irradiation and the platelet and granulocyte counts within a few days or weeks, while the erythrocyte count begins to decrease rather slowly only after a number of

weeks. The exposed individual may die from infection or from haemorrhage. The consensus value for the median lethal dose within 60 days ($LD_{50/60}$) is estimated to range from 2.5 to 5 Gy, after homogenous exposure.

Gastrointestinal tract: There is considerable variation in response as the different parts of the gastrointestinal tract have markedly different radiosensitivity. The oesophagus and rectum are relatively radioregistant, while the stomach and small intestine are much more sensitive. The small intestine is the most sensitive because of the rapidly proliferating mucosal cells of the mucosal epithelium in the crypts of Lieberkuhn. Single acute doses to the abdomen of around 6–16 Gy produces early onset of nausea, vomiting and diarrhea, the symptoms occurring earlier and being prolonged broadly in proportion to the dose. These symptoms are thought to be caused by the release of 5-hydroxytryptamine (5-HT) into the bloodstream which stimulates the nausea/vomiting centres in the brain and other 5-HT receptors. There is a concomitant increase in bowel motility which may be caused by bile salt and other substances acting on the damage mucosa. For gastrointestinal symptoms, the following figures are given as guidelines for adults: Anorexia may be seen in 5% of those at 0.4 Gy and 95% at 3 Gy, nausea in 5% at 0.5 Gy and 95% at 4.5 Gy, vomiting in 5% at 0.6 Gy and 100% at 7 Gy, and diarrhea in 5% at 1 Gy and over 20% at 8 Gy. If the time from exposure to onset of any of the above symptoms is less than one hour, the whole body dose is likely to be >3 Gy, if more than three hours <1 Gy and if they last for more than 24 hours, the dose is likely to be >6 Gy. These signs and symptoms are sometimes referred to as the acute radiation syndrome.

Lung: The lung can be exposed to external radiation from a beam or internal radiation after inhalation of radioactive materials. Radiation pneumonitis appears some weeks or months after exposure. It is a complex phenomenon, including oedema, cell death, cell desquamation, fibrin exudates in the alveoli, fibrous thickening of alveolar septa and proliferative changes in the blood vessels. The main effect is interstitial pneumonitis, followed by pulmonary fibrosis, resulting principally from the damage and response of the fine vasculature and the connective tissues. The development of the lesions is highly influenced by the volume of the organ irradiated and the dose. An acute exposure of both lungs shows a threshold at about 6–7 Gy, with an ED_{50} at about 9 Gy.

Thyroid: The adult thyroid is not especially sensitive to radiation; however, it should always be considered if iodine isotopes are inhaled or ingested by some employees as it accumulates rapidly in the gland. The radiation-induced diseases include acute radiation thyroiditis and hypothyroidism. Total ablation of the required high dose, about 1000 Gy within a short period (two weeks). Hypothyroidism is produced by much lower doses, with 50% incidence at about 60 Gy for acute external exposure and 300 Gy for prolonged internal exposure. The childhood thyroid is more sensitive to radiation.

Skin: The skin is relatively radioresistant organ, but is likely to be exposed in any type of accident. Skin responses depend upon various factors, such as size of the radiated area, depth distribution of dose, duration of exposure and dose rate. Radiation damage to the skin may be observed as erythema, moist desquamation and necrosis, with threshold of 2, 12-20 and >18 Gy, respectively. Acute desquamation often results in chronic changes, with hyperkeratosis and talengiectasis of the capillaries and of superficial and deep blood vessels. The chronic phase may lead to ulceration, atrophy and necrosis. Protraction of exposures of 1–14 days will increase the threshold and ED_{50} values by a factor of about two compared with acute exposure. As radiation burns involving large areas can precipitate the same systemic effects as thermal injury, overall management may be

complicated, especially where other body system has been affected, e.g. bone marrow.

Eye: The radiation doses received by the lens may result in lenticular opacities or cataracts. There is currently a debate as to whether these changes are deterministic or stochastic. The eye may be exposed either after local irradiation – acute or protracted – or after whole body irradiation. The threshold level for detectable opacity is estimated to be less than 0.5 Gy. Protraction does not increase the threshold so much as for some other organs. The cataract does not appear early after exposure; the latent period varies from 6 months to 35 years, with an average of three years.

Gonads: The germ cells of the reproductive system are highly radioactive. The threshold dose for transient sterility lasting for several weeks average 0.15 Gy for men and about 5 to 10 times higher for women. Recover time in men is dose dependent and may take many years. Doses of 2.5-6 Gy or more are required for permanent sterility in both men and women.

Embryo and fetus: The embryo and fetus should also be considered, as women in the workforce may become pregnant. The developmental effects of radiation in the embryo and fetus are strongly related to the gestational age at which the exposure occurs, i.e. whether it occurs during organogenesis. The most serious health consequences of prenatal exposure are embryonic death, gross congenital malformation, growth retardation or severe mental retardation. For exposure at high dose rates, severe mental retardation has been shown for exposure occurring during the 8th–15th week and to a lesser degree during the 16th–25th week after conception. The threshold dose is estimated to be around 0.4 Gy between 8th and 15th week and 0.1–0.2 Gy between the 16th and 25th, respectively.

Central nervous system: The acute central nervous system effects are generally reached only when the whole-body radiation dose exceeds about 50 Gy. The survival time is usually less than 48 hours. Death is believed to be a functional of several causes, including vascular damage, meningitis, myelitis and encephalitis. Fluid also infiltrates the brain causing marked oedema. However, any person who exhibits even mild symptoms of central nervous system syndrome would inevitably die from gastrointestinal or haematopoietic damage.

Effects of acute whole body radiation exposure: The effects which are likely to be seen following a homogenous whole-body irradiation over a short period are shown in the table. Experience from several accidents has shown that most casualties have only part of their bodies exposed to the radiation. The nonuniform and heterogeneous nature of the exposure leads to variable signs and symptoms and the outcomes are different. Some high localized doses do not present with the classical prodromal signs of nausea and vomiting. This complicates the initial triage and subsequent therapy. For example, in patient with severe but partial bone marrow irradiation residual stem cells in unaffected bone marrow can reject bone marrow grafts causing host-versus-graft disease.

Effects of chronic whole body radiation exposure: In a chronic exposure (measured in days, weeks, months), the symptoms are usually more subtle. The usual feature is of general malaise, with influenza-type symptoms, fever and or diarrhoea and vomiting. It has been reported that chronic radiation exposures have been implicated in life-shortening, accelerated ageing and premature atherosclerosis.

Effects of partial body radiation exposure: In almost all partial body exposures, whether acute or chronic, the skin will be affected. Depending on the type of exposure, deeper tissue may also be affected. Incidents where small sources have been carried in shirt pockets for short periods have given rise to exceptional doses (measures in hundreds of Gy) leading to serious necrotic lesions of the chest wall.

Acute whole-body radiation		
Dose	*Signs/Symptoms*	*Diagnosis*
0–100 mSv	No signs or symptoms	Dose clinically undetectable
100–500 mSv	No signs or symptoms	Expected laboratory findings: Small decrease in lymphocyte count in blood (1-4 days), increase in accuracy of chromosome dosimetry from limit of sensitivity
500 mSV-2 Sv	Nausea and vomiting probable, onset usually >2 h	Laboratory findings: Diagnostic changes in blood within days, accurate chromosome dosimetry
2–6 Sv	Nausea, vomiting and headache, onset within 30 min to 2 h	Laboratory findings: Early diagnostic changes in blood, accurate chromosome dosimetry (lethal dose 50% without treatment ~3 S_V)
> 6 Sv	Rapid onset of nausea, vomiting, headache and pyrexia (within 30 minutes)	Early diagnostic changes in blood
> 8 Sv	Chromosome dosimetry becoming saturated, GI syndrome–diarrhoea	
10 s of Sv	Rapid onset of apathy, prostration, convulsion and death	

Stochastic Effects

Carcinogenesis: Following irradiation, a viable but modified somatic cell may retain its mitotic capacity and may result, after a prolonged and viable period, in the development of a malignancy. The cancer induced by radiation, with or without the contributions of other agents, is indistinguishable from those occurring 'spontaneously' or from other causes. Since the probability of cancer resulting from the radiation is related to dose, this type of effect of radiation can only be detected by statistical means in epidemiological studies carried out on exposed population groups. If the number of people in an irradiated group and the doses that they have received is known, and if the number of cancers eventually observed in the group exceeds the number that could be expected in an otherwise similar but nonirradiated group, the excess number of cancers may be attributed to radiation, and the risk of cancer per unit dose may be calculated, i.e. the risk factor. The probability of causation can be ascribed based on the radioepidemiological table.

Effects of radiation on the skin with increasing dose	
Dose	*Effects*
0–100 mGy	Nil
100–400 mGy	Nil expected
400 mGy–4 Gy	Transient erythema expected within 3 days of exposure
4–8 Gy	As above followed by fixed erythema after variable latent period, temporary hair loss
8–15 Gy	Prompt erythema followed bt vesiculation, permanent hair loss
>15 Gy	Severe vesiculation, tissue slough, very slow to heal, possible site of malignant changes

On the basis of available information, United Nation Scientific Committee on the Effects of Atomic Radiations (UNSCEAR) and ICRP have assessed that the risk factors obtained directly from observations at high doses and high-dose rates should be reduced by at least a factor of two to give more realistic risk factors for low doses and dose rates. The risk factor or lifetime fatality probability coefficient from ICRP for a reference population of both sexes and of working age is 4.1×10^{-2}/Sv for the sum of all fatal malignancies, i.e. a dose of 1 Sv in a working lifetime results in a 4% chance of a fatal cancer occurring. More recent epidemiological studies involving lower dose exposures from the UK National Registry for Radiation Workers have shown that the leukaemia incidence trend with dose is consistent with the ICRP risk estimates.

Probability of causation: The concept of the probability of causation (PC) has been adopted to answer the question: If a person has been exposed to ionizing radiation and subsequently develops a cancer, what is the probability that the cancer was due to the earlier exposure? According to the US National Institutes of Health and International Atomic Energy Agency (IAEA), the probability a radiation dose to an organ of a person's body leads to the subsequent development of cancer can generally be calculated as:

$$PC = \frac{ERR}{(1 + ERR)}$$

where ERR is the excess relative risk associated with the exposure, i.e. ERR is the relative increase in risk, with the value 1 subtracted. For example, an ERR of 1 represents a doubling of the risk, relative to that in the absence of exposure. This gives a probability of causation of

$$PC = 1/(1+1) = 0.5$$

i.e. 50% chance that the cancer was induced by radiation. The ERR can depend on number of factors i.e. the size of the radiation dose, the sex of the person exposed, the age of the exposure, the time between exposure and the onset of the disease, the degree of dose protraction.

In the United Kingdom, the nuclear industry has set up a no-fault compensation scheme based on PC calculations, to avoid unnecessary litigation and allow the current scientific risk factors to be used in a generous manner for those claiming a radiation induced cancer.

Hereditary Effects

The other main late effect of radiation is the concept of hereditary damage and arises through irradiation of the germ cells. Ionizing radiation induces mutations which are frequently harmful in the germ cells or their precursors. The hereditary disease that mutations may cause range from afflictions, such as colour blindness or minor disorders of metabolism (e.g. disorders of amino acid metabolism) to serious defects which may cause early death or severe mental retardation (e.g. Down's syndrome).

Biological Basis for Radiation Effects

It is generally accepted that for carcinogenesis, the cellular DNA of the genome is the critical molecule. Damage of this molecule leading to cancer can be mediated through direct ionization by the radiation or by indirect action in the formation of free radicals in the fluid in close proximity to the genome. This indirect effect accounts for about two-thirds of the biological effect in the case of low LET radiation, but the direct effect predominates with high LET radiation. Evidence is building that there are fragile sites unusually prone to breakage and rearrangement in DNA, which could explain particular sensitivities.

In some medical conditions, such as telangiectasia and Li-Fraumeni syndrome, there exist genetic defects in cell repair mechanisms which produce an increase in individual sensitivity to ionizing radiation. At high doses, cell death will predominate leading to organ malfunction, whose severity is dependent on the dose.

Factors Influencing Radioactivity

Susceptibility of carcinogenic effect of radiation can be affected by a number of factors, such as genetic constitution, sex, age, physiological state, smoking habits, drugs and various other physical and chemical agents. The genetic basis of some diseases including increased cancer susceptibility is becoming clearer. For example, it has been shown that the full expression of ataxic telangiectasia, including enhanced cancer susceptibility, results from homozygous mutated genes on chromosome. In addition, the heterozygous carriers, who do not express the full-blown disease, are known to be subject to a higher cancer incidence, especially for breast cancer. Cancer rates are highly age dependent and, in general, increases exponentially in older age

groups. The expression of radiogenic cancers varies with age in similar way. Smoking and prolonged exposure to inhaled α-particle emitters interact synergistically and this is then said to be a multiplicative effect. In general, baseline rates for radiological cancer in male exceed those in female, possibly because of increased exposure to carcinogen and promoters in occupational activities and lifestyle factors, such as increased smoking and consumption of alcohol.

Occupational Doses

Legal controls require that the dose received by the more exposed employees is routinely assessed and records kept of their doses. These employees are often referred to as 'classified' persons. A significant proportion of the non-classified employees are also monitored, but the records for this group, who generally receive low doses anyway, are less comprehensive. Approximately, every four years, the Radiation Protection Division of the Health Protection Agency, UK conducts a major review of the radiation exposure of the UK population.

UK annual dose limits (mS_V)

Part of the body	*Classified person*	*Nonclassified person*	*General public*
Whole body	20	6	1
Any single organ or tissue	500	150	50
Hands, forearms, feet and ankles	500	150	50
Lens of the eye	150	50	15

Radiation Protection

The main principles of radiation protection are set out and promulgated by the ICRP in publication 103. They can be summarized as follows:

1. No practice involving exposure to radiation should be adopted unless it produces at least sufficient benefit to the exposed individuals or to society to offset the radiation detriment it causes (termed 'justification of a practice').
2. In relation to any particular source of radiation within a practice, all reasonable steps should be taken to adjust the protection so as to maximize the net benefit, economic and social factors being taken into account (termed 'optimization of protection').
3. Finally, a limit should be applied to the dose (other than from medical exposures) received by any individual as the result of all the practices to which he is exposed (termed 'application of individual dose limits').

In simple terms, this framework is derived from three principles that apply to many human activities, and especially to medicine:

1. The justification of a practice implies doing more good than harm.
2. The optimization of protection implies maximizing the margin of good over harm.
3. The use of dose limits implies an adequate standard of protection even for the most highly exposed individuals.

These three principles overall avoid the possibility of deterministic effects and minimize the risk of stochastic effects in normal operation.

Protection against radiation: For protection against external radiation, the general principles deployed in the workplace to keep external doses as low as reasonably practicable are to keep exposure as short as possible, keep as far away from the source as possible and whenever possible place shielding between the source and the employee (e.g. lead aprons and special rooms for exposure). This simple concept of time, distance and shielding should be practiced routinely. For protection against external contamination, the use of protective clothing prevent contamination of the skin; however, loose contamination on such clothing will still irradiate the skin and so exposure time need to be strictly limited. Containment of contamination is essential wherever possible to prevent activities producing an

inhalation hazard. For protection against internal irradiation, the use of containment, such as with fume cupboards or glove boxes, is effective and is used at a level appropriate to the level of hazard. Work with α-emitted radionuclides presents a major hazard and these facilities require special detection system for leaks to reduce the possibility of either inhalation or contamination of wounds.

Medical Surveillance

Workers should be medically examined prior to employment (pre-employment medical examination), and thereafter their medical fitness should be reviewed at periodic intervals through periodical medical examination (normally annually). The primary purpose of this medical surveillance is to assess the initial and continuing fitness of the employees for their intended tasks. The nature of the periodic reviews will depend on the type of work that is undertaken. Three situations may arise where special surveillance may require:

1. Fitness of wearing respiratory protection devices.
2. Fitness of handling unsealed sources in the case of employees with skin disease or skin damage.
3. Fitness of employees with psychiatric or psychological disorders.

Employees who wear respiratory protective equipment in the course of their radiation work, for example, inside contaminated confined spaces, need to be checked periodically to verify their lung function. Employees with skin diseases, such as psoriasis, may need to be excluded from work with unsealed radioactive materials, unless the levels of activity are low and appropriate precautions are taken, such as covering the affected parts of the body. However, there may be a need for periodic medical checks to ensure that unprotected areas have not become affected by the skin disease. In employees with psychiatric or psychological disorders, the primary concern is whether the employees could pose a danger to themselves or their colleagues, particularly in high radiation dose-rate areas and the handling of unsealed and portable sources.

There is no particular reason why employees who have previously been treated for malignant disease should be excluded from work with radiation if they are otherwise fit for the job. However, it may be necessary to restrict their employment in emergency teams where high doses in accident situations might be permissible, and essential when saving life.

Two types of employees may need special reassurance by the physician, these are:

1. Women who are, or may become pregnant.
2. Individual employees who have been or may have been exposed substantially in excess of the dose limits.

Once the physician or the management has been informed that the woman believes she is pregnant, arrangement may need to be made to change her conditions of work. Physician should also be able to inform the pregnant woman of the risks to her conceptus associated with her work. Separate dose limits apply to pregnant women.

In case of accidental exposure or over-exposure, the physician needs to liaise with management and other safety specialists to ensure that all suitable arrangements for evaluating the scale of the exposure are undertaken.

Medical Management of Accidentally Exposed Employees

As soon as the unexpected exposure is suspected, management should undertake an investigation to determine the dose to the employees. If the dose is established, an injury is sustained, or contamination occurs, then the appointed doctor or occupational health service should be informed and measures to be taken as follows:

a. *External exposure (direct radiation exposure)*: It is convenient to divide exposures into three categories of increasing doses. *Doses close to the dose limits:* Normally,

such doses do not require any special clinical investigations or therapy. The role of the occupational health personnel is to counsel the overexposed employee that the exposure is unlikely to produce adverse health effects.

- *Doses well above the dose limits*: Where the exposure is significantly higher, but below the threshold for deterministic effects, the prime role of the occupational physician is to advise the employee of the risks. Then they need to determine whether biological dose indicators, such as lymphocyte counts and chromosome aberration assays, are needed to confirm the dose estimates. Normally, no further action is required other than counselling.
- *Doses at or above the threshold for deterministic effects*: If the assessed external doses are around the threshold for deterministic effects, therapeutic action may need to be undertaken. In order to make this decision, the overexposed employee needs to be examined clinically and any abnormal findings or symptoms recorded. Haematological examination will need to be undertaken in order to monitor the clinical course of the overexposure. If the exposure is severe enough to lead to the acute radiation syndrome, early transfer to a designated hospital is essential. The occupational physician should institute the initial investigations and treatment of the early symptoms. Immediate life-threatening injuries, such as fractures and burns, must be treated as a priority before transfer to a hospital.

  The clinical management of such highly exposed individuals is dealt with as it is in case of internal exposure.

b. *Internal exposure (internal deposition of radioactive materials)*: The employee should be removed temporarily from the workplace to prevent any further exposure, even when the dose is expected to be close to, but below, the dose limit. This action will allow more accurate dose estimates from sequential counting either of the body, an organ or body fluids. High exposure may warrant interventional therapy to accelerate the excretion of radionuclides. Such therapeutic measures might include the administration of chelating agents to enhance the excretion of transuranic radionuclides, for example, in plutonium internal contamination intravenous administration of diethylene triamine pentoacetic acid (DTPA) will enhance excretion by chelation or give stable iodine for radioiodine uptake. Many of these therapeutic procedures would be undertaken only at a major hospital, for example lung lavage for a large inhalation of plutonium.

c. *External contamination*: Where an employee has been externally contaminated, decontamination, by simple washing, should be undertaken as quickly as possible. Significant skin contamination with β-emitting radionuclides can result in radiation burns if not treated quickly. It should be remembered that thermal burns could complicate skin decontamination and both may need to be treated simultaneously. Some radionuclides may be absorbed through the skin depending on their chemical form, and can lead to internal contamination. This is particularly true where skin contamination occurs with **tritiated** water and some compounds of iodine and caesium.

Examples of treatment for internal contamination

Isotope	*Treatment*
Iodine	Administration of stable iodine
Tritium	Forced diuresis
Plutonium	Chelating agents (DTPA)–by IV or aerosol inhalation
Caesium	Prussian blue given orally inhibits intestinal absorption

Return to Radiation Work

Exposures which do not approach deterministic levels need not affect an employee's

fitness for further radiation work. An employee should be advised by the physician on the level of the increased risk for stochastic effects. Where their own actions contributed to an overexposure, consideration should be given by management to retraining before return to work. Return to work after internal contamination may be delayed until an adequate dose assessment has been made. Where there is partial body overexposure which produces deterministic effects, the employee should be advised on the future risks involved in radiation work.

NONIONIZING RADIATION

Introduction

The electromagnetic radiation spectrum has been divided into a number of frequency regions. The most useful divisions are between ionizing (X-rays, gamma rays, cosmic rays) and nonionizing radiation (ultraviolet radiation, visible rays, infrared radiation, radiofrequency waves). Nonionizing radiation is that part of the electromagnetic spectrum which does not have sufficient energy to ionize matter, but can excite atoms by raising their outer electrons to higher orbitals, a process which may store energy, produce heat or cause chemical reaction (photochemistry). The nonionizing radiation of wavelength below 300 nm is absorbed in cornea, 300 to 400 nm is absorbed in lens and more than 400 nm can reached to the retina. The wavelengths of nonionizing radiation of major importance to the eye are grouped under the following headings:

Nonionizing radiation	*Wavelength*
Ultraviolet	UV(C) – 100 to 280 nm UV (B) – 280 to 315 nm UV (A) – 315 to 400 nm
Visible	400 to 760 nm
Infrared	IR (A) – 760 nm to 1.4 µm IR (B) – 1.4 µm to 3 µm IR (C) – 3 µm to 1 mm
Microwave	1 mm to 1 m

The major and natural source, of these wavelengths is sunlight which has a broad emission ranging from far UV (200–280 nm) to far infrared (3000–10000 nm). However, it should be noted that the eye is not normally exposed to the far UV component of the spectrum since this solar component is blocked by the atmosphere. In addition to sunlight, there is an ever-increasing amount of man-made candescent and incandescent sources which cover the full non-ionizing radiation spectrum.

Ultraviolet Radiation

Ultraviolet radiation exposure can occur from natural or man-made sources. Sunlight creates an important exposure for outdoor workers. Industrial process such as welding, plasma torch operations, and some hot-metal work produces UV radiation. UV radiation is also used for its germicidal properties in settings such as hospitals. Other applications include chemical synthesis and analysis, photoengraving, illumination, sterilization of foodstuffs, lasers, vitamin production, medical diagnosis and treatment and identification technologies. The UV radiation is subdivided into three regions: the UV-A from 315 to 400 nm, termed the blacklight region, UV-B, from 280 to 315 nm, the erythemal region, and UV-C, the region below 280 nm, the germicidal region. Wavelengths below 295 nm are absorbed in the epidermis, while longer wavelengths penetrate into the dermis. Wavelengths greater than 300 nm are transmitted through the cornea and are absorbed by the lens.

Absorption of UV can produce photochemical reactions and result in effects including protein-DNA cross-linking. Some of the effects of UV exposure include photokeratitis and conjunctivitis, especially after exposure to wavelengths of 270–280 nm. Symptoms include pain, tearing, intolerance of light, visual haze, and a sensation of sand in the eyes. Cataracts have been produced by excessive exposure to UV in the 295 to 310 nm region.

Skin burn is also associated with exposure in the 300 nm band, with 297 nm wavelength considered to have the maximum biological effectiveness. Exposure produces the erythema or reddening of the skin we term sunburn. Tanning is also initiated by such exposure, with the migration of melanin pigment and resultant darkening of the skin. Long-term effects include premature skin aging with damage to connective tissue and the loss of elasticity that results in wrinkling (elastosis). More important is increased risk of skin cancers. The most serious of these, melanoma, often appears on body areas that may receive relatively less UV exposure such as the trunk or legs; it has, however, been associated with blistering sunburns received when young.

UV exposure can also create a photosensitization reaction if the individual has also been exposed to phototoxic agents such as psoralens, coal tar, and some cosmetic preparations. Photoallergic responses mediated by the immune system can occur after exposure to pharmaceuticals such as salicylamides, hexachlorophene, and certain antibiotics, as well as cosmetics.

Outdoor workers can use sunblocking creams as well as appropriate clothing (long-sleeved shirts, trousers, broad-brimmed hat, and/or neck shield) to prevent skin burn and tanning. Workers' eyes can be protected against low-sensitive sources of UV (low pressure mercury vapor lamps, sunlamps, and black light lamp) by use of glass or plastic spectacles, goggles, or shields. Light-weight clothing or UV absorbing creams will protect the skin. Exposure to low-intensity sources can also be controlled by limiting exposure

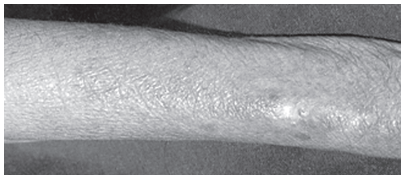


Fig. 8.24: Elastosis

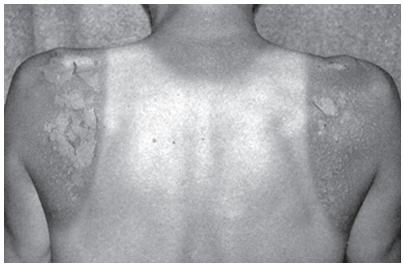


Fig. 8.25: Sunburn

time so that total irradiance on the eye or skin is controlled.

Sources of UV radiation such as welding arcs must be shielded from workers near the operation with use of materials opaque to UV to prevent 'arc eye' or 'flash eye'. For the welder who is directly exposed to the UV from the arc, use of personal protective devices including tinted goggles and face shields and densely woven or leather clothing will prevent excessive exposure. UV exposure for welders' helpers and other workers near welding operation can be mitigated by using shielding booths or curtains and by coating walls, ceilings, and other surfaces with pigment-based paints that have low reflectance in the UV range. Various types of glass and flexiglass shielding can protect against different UV wavelengths. Where these are not used, adjacent workers will require eye and skin protection.

The UV radiation produced by an arc welding can also initiate the chemical reaction that creates ozone and nitrogen oxides from oxygen and can decompose vapor of chlorinated solvents to produce highly toxic phosgene gas. The UV radiation control programme must deal with these potential hazards as well.

Visible Radiation

Intense exposure from very bright short pulsed sources such as pulsed lasers and high intensity electronic flash lamps can induce thermal injury in the retina. Wavelengths in the blue range (400 to 500 nm) can also cause

a photochemical injury in the retina. Shielding and exposure avoidance are the main controls.

Photic retinopathy may occur, due to photochemical damage by the blue light component of the arc welding, to individuals welding without eye protective goggles. In general, patients present with a decrease in vision which occurs immediately or soon after the injury; fundus appearance and visual recovery resemble those of solar retinopathy.

Solar retinopathy: Solar retinopathy is a well-recognized form of acute light damage caused by the direct or indirect viewing of the sun. Socrates advised that "a solar eclipse should be only observed by looking at its reflection in water". Galileo injured his eyes while viewing the sun through his telescope. Retinal damage is considered to be photochemical since sunlight exposure is insufficient to raise the retinal temperature by 10°C provided macular choroidal blood flow is maintained. The patient complains of blurred central vision immediately or soon after (actual within minutes) and the damage is usually, but not always, bilateral. The fundus presents initially with a yellow spot at the fovea which after several days changes to a reddish area with surrounding pigmentary change. There is a marked drop in visual acuity with a central scotoma. By two weeks, a red, well-circumscribed lamellar hole (100–200 µm diameter) often with a larger area of retinal pigment epithelium mottling is apparent. Visual acuity usually improves to near normal by six months, although a slight-to-moderate loss in visual acuity may persist in some individuals. A very small central red spot may remain throughout life. Oral corticosteroids have been advocated as a treatment for acute lesions with severe visual loss, but their efficacy remains unproven.

The quantity and quality of light available in the workplace must also be evaluated. Glare is a frequent problem where intensities are too great and where workers must view reflecting surfaces such as video display terminal (VDT) screens. Lighting must be sufficient for performance of the task but not so high as to produce glare or visual fatigue. Lighting criteria for different occupations have been developed for most work environments.

Infrared Radiation

Any hot body emits infrared radiation (IR). Industrial sources of significant infrared radiation include furnaces, hot metals, dehydration equipment and equipment for drying and baking of paints and surface coatings and spot heating. IR is produced by certain lasers and medical physiotherapy equipment. IR does not penetrate below the superficial layers of skin; its effect is to heat the skin and tissue beneath it. Radiation in the near infrared region (0.75 to 1.5 µm) can cause acute skin burn and may result in increased skin pigmentation. The shorter wavelengths are also absorbed in the iris and cornea of the eye, from which heat is transferred to the lens. Excessive exposure of the eyes to IR from furnaces and other hot bodies has traditionally been thought to produce 'glass blower's cataract' or 'heat cataract'. Overexposure to near infrared may also damage the retina by protein denaturation. Chronic exposure to excessive IR can also cause a 'dry eye' irritation of cornea.

Reflective aluminum shielding and aluminized clothing are often used to reduce infrared exposure and thermal loading for workers at furnaces and other hot metal working operation. Protective eyewear and face-shields should be components of the protective outfit. National Institute of Occupational Safety and Health (NIOSH) noted that glassworkers, arc welders and other may be exposed to both UV and IR, necessitating use of special glasses that absorb in both regions of the spectrum. This specific hazard was studied by Okuno who developed a theoretical model of the human eye and its exposure to infrared radiation. Temperature distributions within it were calculated, suggesting that IR cataracts in the workplace result from the generation of heat by absorp-

tion of IR in the cornea and heat conduction to the lens. The threshold IR irradiances for cataract formation were determined from the relationship between the incident irradiance and the lens temperature in the range 163 to 178 mW/cm^2 for long-term exposures (greater than about 5 minutes) under normal condition. However, these values may be reduced by 50% for workers who perform heavy work at high ambient temperature. It may be possible to set IR exposure limits in the workplace based on these threshold data.

Safe infrared exposure levels: The available data indicate that acute ocular damage from the incandescent hot bodies found in industry can occur with energy densities between 4 and 8 Wsec/cm^2 (1 to 2 cal/cm^2) incident on the cornea. The ocular tissue involved would depend on the wavelengths that are absorbed. As these relate to threshold phenomena, it would appear that a maximum permissible dose of 0.1 to 0.8 Wsec/cm^2 (0.1 to 0.2 cal/cm^2) could limit the occurrence of these acute effects. A further reduction by a factor of 10 should prevent the more chronic effects of intraocular tissues.

Infrared and UV radiation filtration is readily achieved in glass filter with additives such as iron-oxide, but the visible attenuation determines the shade number, which is a logarithmic expression of attenuation. Normally, a shade 3–4 is used for gas welding (goggles), 10-14 for arc welding and plasma arc operations (helmet protection required). The rule of thumb is that if the welder finds the arc to view, adequate attenuation is provided against ocular hazards. Supervisors, welders' helpers and other people in the work area may require filters with a relatively low shade number (e.g. 3–4) to protect against welder's photokeratitis (arc-eye). In recent years, a new type of welding filter, the autodarkening filter has appeared on the scene. Regardless of the type of filter, it should meet American National Standard Institute (ANSI) Z87.1 and Z49.1 standards for fixed welding filters specified for the dark shade.

Microwave Radiation

Microwave radiation is found in radar and communications equipment, diathermy applications, and industrial and consumer ovens. Microwave intensities can be sufficient to cause significant local heating of tissues. The effect is related to wavelength, power intensity, and time of exposure as well as body size, its electrical properties, and whether reflecting surfaces are present. Generally, the longer wavelengths will produce a greater temperature rise in deeper tissues than will the shorter wavelengths. However, the given power-intensity, there is less subjective awareness to the heat from longer wavelengths than from shorter wavelengths because of their absorption beneath the body's surface. An intolerable rise in body temperature, as well as localized damage, can result from an exposure of sufficient intensity and time. The eye, which has a limited blood supply to dissipate localized heat, can suffer lens opacities, cataracts, corneal damage and retinal lesions. Testicular lesions and decreased sperm counts have also been reported.

Acute, accidental exposure of the eyes to microwave radiation may lead to skin burns, conjunctival infection and loss of corneal epithelium, as well as stromal oedema and opacification. Epithelial loss can be managed with topical antibiotics (e.g. chloramphenicol or fuscidic acid) and cycloplegia (e.g. cyclopentolate), while topical steroids (e.g. prednisolon acetate) may also be considered if stromal damage and inflammation occur (indicated by corneal oedema and opacification).

Radiofrequency Radiation

Radiofrequency fields (RF) are a segment of the nonionizing part of the electromagnetic spectrum. RF range from a few kHz to several hundreds of GHz. The public may be exposed to RF produced by AM, FM radio and television broadcast antennae, navigations systems, cordless telephones, wireless local area networks and increasingly by mobile

phone base stations and mobile phone handsets. Occupational exposures to RF may be experienced while operating and maintaining radio and television broadcasting and telecommunication systems. Additional significant sources of occupational RF exposures are dielectric heaters used for heating, sealing and melting vinyl and other materials and from military and civilian use of radar systems. In the medical field, exposures can occur from the use of magnetic resonance imaging (MRI), diathermy or electrosurgery. Workers in the electric utility industry may also be exposed to RF from antennae located on or near electric utility facilities. RF exposure in the far fields (at least several wavelengths away from the antennae) is typically measured as power density in W/m^2 or mW/cm^2. In near fields (close to the source of exposure relative to wavelength), exposure to RF is measured as electric and magnetic fields, in V/m and A/m, respectively. Since the actual amount of exposure, a person receives from an RF field is also dependent on many factors, such as body size, grounding state of the person and frequency, uniformity and polarization of the field, in addition to field intensity, the internal exposure is typically expressed as specific absorption rate (SAR) in W/kg.

Effects of RF: Exposure to RF radiation induces heating in body tissues and imposes a heat load on the whole body, thus thermal mechanisms are accepted and prevention of excessive heating (above 1°C) serves as a basis for most international guidelines. High RF may interfere with pacemakers and other implanted medical devices. Direct contact with or very close proximity to an RF source, through an electric arc, may result in severe burns which tend to heal poorly. The lens of the human eye, due to lack of circulation and insufficient mechanisms to eliminate thermal energy, is known to be sensitive to heat. Therefore, heating due to high RF exposure could result in cataract. Various studies did not suggest that long-term, low-level RF exposure is associated with cataract development. Many different effects at low RF energy, thought to be nonthermal in nature, have been reported in recent studies. Long-term exposure may produce some adverse effects include nervous, neuroendocrine, reproductive, immune and sensory system effects, including cataracts, decrease birth-weight, behavioral changes and change in blood cell concentrations.

Occupational exposure to RF radiation may exceed exceptionable limits for maintenance crews on broadcast towers and radar devices, operators of dielectric heaters, shortwave diathermy devices and microwave oven. Exposure should be reduced by engineering controls, such as total or partial enclosure to limit leakage, interlocks, and additional shielding where necessary. Other controls including limiting access to RF field areas and reducing power during maintenance operations. Present guidance on occupational exposure is based on restricting the RF-induced whole body SAR to less than 0.4 W/kg (a safety factor of 10); a heat load sufficiently small that its contribution to other possible heat loads, generated from hard physical work and/or imposed by high ambient temperatures, can be neglected. Basic restrictions on localized SARs, average over any 10 g of contiguous tissue, are 10 W/kg in the head and trunk and 20 W/kg in the limb. These are intended to restrict local tissue temperature rises to acceptable levels.

LASER

Laser is an acronym for 'light amplification by the stimulated emission of radiation'. Lasers are sources of nonionizing radiation that can operate in the UV, visible and infrared region of the electromagnetic spectrum. The light emitted has a unique combination of spatial coherence (i.e. all the waves are in step), monochromaticity (one colour or narrow wavelength range) and usually high collimation. Furthermore, the emission may be continuous wave or pulsed for either long or short (Q-switched) duration.

The known effects of acute light damage to ocular tissues are exploited in preventive ophthalmology. Optical breakdown damage from Q-switched or mode-locked lasers are important procedures in iridotomy and posterior capsulotomy. Photoreactive keratectomy is now a standard procedure in Europe for the correction of refractive error less than 4 diopters, as well as becoming fashionable for the treatment of high myopia and is routinely undertaken using an excimer laser emitting in the UV-C region of the spectrum. Providing irradiance, exposure time, pulse rate and beam alignment are optimal, the operation is successful, however, poor quality control can result in corneal fibrosis and opacification. Argon, krypton and diode lasers are routinely used in retinal scatter photocoagulation to induce the regression of subretinal and preretinal vessels in conditions such as proliferative diabetic retinopathy.

Lasers are commonly used both in industry (e.g. drilling, cutting, welding, and communication) and military (e.g. rangefinders, tactical target designators, night vision) settings and there have been numerous reports of accidental exposure resulting in immediate loss of central vision. Depending on the type of laser, damage occurs by either a thermal or mechanical mechanism. In the case of retinal damage, a gliotic scar develops and a paracentral or central scotoma may persist.

In addition to potential optic hazards, lasers may create airborne contaminants generated by the process or the laser fuels used, including noise, fire, electrical, and cryogenic hazards. A laser impacting on a hazardous metal part, for example, could generate a fume and other small particles.

Understanding the laser hazard requires a knowledge of wavelength or wavelength range, the average power output (or total energy pulse for pulsed lasers), the length of exposure, and laser source radiance and viewing angle. Lasers are divided into four major classes depending on the potential safety hazard. These classes are defined by the American National Standards Institute (ANSI) Z-136.1: 2007 and British Standard BS/EN/IEC 6082:2007:

1. *Class 1*: Nonhazardous lasers, (i) the output is so low the laser is inherently safe or (ii) the laser is part of a totally enclosed system. A class 1M laser product is safe to view without optical aids, but otherwise is potentially hazardous.
2. *Class 2*: Low power visible continuous wave and pulsed lasers which, whether repetitively pulsed or continuous wave lasers, are not hazardous within the eye's aversion response (i.e. $\leq$ 0.25 second). Normally, only procedural controls such as not pointing the laser at the eye are required. Class 2M laser products pose the same risk if viewed without optical aids, but otherwise are potentially hazardous to view with telescopes.
3. *Class 3*: Divided into class 3R and class 3B.
   - Class 3R: Low to medium power lasers where the risk is minimal largely because of the extremely low probability of the pupil being large, of all the beam energy entering the eye and the eye accommodated to focus the beam to a minimal spot. Nevertheless, the eye may be exposed technically to levels up to five times the maximum possible exposure (MPE). Hazard can be controlled by relatively simple procedures (e.g. use of beam stops and ensuring that beam paths are not at eye level). The only documented injuries from this type of laser have occurred from intentional direct-beam exposure.
   - Class 3B: medium power lasers where the viewing beam either directly or by specular reflection is hazardous, but diffuse reflections are almost always safe. Hazard can be controlled by the use of beam enclosure, beam stops (ensuring that beam paths are not at eye level) and, if needed laser eye protection.

4. *Class 4*: High power lasers which are not only a hazard from direct viewing and from specular reflections, but also from diffuse reflections. The direct beam may also be a skin and fire hazard. Their use requires extreme caution.

Depending on the hazard posed by a particular laser, a complete laser safety programme will include a combination of controls. Engineering controls such as protective housing and enclosures, interlocks, filtered viewing optics, warning lights, and remote controls combined with the use of appropriate personal protective equipment under the control of a laser safety committee and/or laser safety officer, will attenuate the hazard. Use of warning signs, level and standard operating procedures, training of workers, limitation of entry to authorized individuals, and effective maintenance are other elements of a successful laser safety programme.

Exposure limits of lasers: Occupational exposure limits have been promulgated by a number of organizations. In the United States, both the American Conference of Governmental Industrial Hygienists (ACGIH) and the American National Standards Institute (ANSI) have produced a comprehensive set of exposure which apply for pulse duration of 100 fs to 30 ks (eight hours) for wavelengths from 180 nm in the ultraviolet (UV) to 1 mm in the extreme infrared (IR). These exposure limits are virtually identical, although exposure limits are termed 'threshold limit values' by ACGIH and 'maximum permissible exposure limits' in the ANSI standard Z-136.1. On the international scene, the International Commission on Nonionizing Radiation Protection (ICNIRP) published guidelines for limits of human exposure to laser radiation in 1996. The basic resource used for the development of the laser exposure limits was the Environmental Health Criteria Document of the World Health Organization (1982). The International Electrotechnical Commission (IEC) and European guidelines (EU Optical Radiation Directive) are all based upon the ICNIRP, ACGIH and ANSI exposure limits.

EXTREMELY LOW FREQUENCY ELECTRIC AND MAGNETIC FIELD (EMF)

Electric Fields

A charge is surrounded by an electric field in the same way that the earth is surrounded by a geomagnetic field. One charge acts on another charge with a given force. 'Electric field strength' at a given point is the force F_E acting on a unit of positive charge, q, per unit of charge. By definition, $F_E = E \times q$. The unit of electric field strength is N/As, i.e. equivalent to V/m. Electric field strength is therefore proportional to voltage (V). When a conductor is connected to an alternating current, the field around the conductor changes direction as the voltage changes direction. An alternate current field induces a charge in unearthed, conducting objects in its vicinity. It also induces alternating currents in the body's tissues and fluids.

Magnetic Fields

Certain materials are capable of attracting iron, nickel, or cobalt and are called magnets. The earth is itself a natural magnet surrounded by a magnetic field. This magnetic field exerts a force which acts on any electric charge moving in the field. A positive charge, q, moving with a velocity, v, perpendicular to a magnetic field, is affected by a force $F_B = q \times v \times B$, where B is magnetic flux density, that is, a measure of the field's strength. This strength is measured in N/Am, that is, the same as Vs/m^2 (= 1 T). The unit A/m is another measure of a magnetic field strength. The factor for converting from flux density to the latter measure is $1.36 \times 10^{-6} \times A^2/N$. Thus, magnetic field strength is proportional to current (A). If an alternating current passes through the conductor, the direction of the magnetic field around the conductor alternates as current alternates. The flux of such a field induces an electromotive force in a

conductive circuit. This force induces, in turn, a current, depending on the circuit's conductivity. The current induced in such instances is expressed in current density with the unit A^2/m. Sometimes, the time derivative of the magnetic field (T/s), which varies with time, is employed as an indirect measure of the induced current. (A = current, q = positive charge, N = force, E = electric field, s = time in second, A= q/s, q= As, N = q × E, E = N/q = N/As = V/m, V= Voltage, m = distance in metre, B= magnetic flux density = velocity, mT = micro tesla = μT).

Electromagnetic Fields

In the strictest sense of the word, there are no 'electromagnetic fields', but there is an electromagnetic force effect which is the sum of the electric field and the magnetic field: $F = F_E + F_B = q (E + v \times B)$.

Extremely-low-frequency radiation produced by power frequency has been considerably studied in the recent past. Electricity is generated and usually transmitted as alternating current (ac) in the United States at 60 cycle per second, or 60 hertz (Hz), and in Europe at 50 Hz. The magnitude of the electric fields, measured in kV/m, is directly proportional to line voltage, while magnetic fields, measured in tesla (T) or microtesla (μT), are determined by the magnitude of the electric current. These fields are found around every electrical conductor, motor and appliance. In office buildings, computers and copy machines are common sources of magnetic fields. Power distribution facilities and large motors used to drive building air conditioning systems can also contribute significantly to the magnetic field environment. In factories, high magnetic fields are encountered near large electric machine, electric heating equipment and other high current-carrying devices. TV sets, refrigerators, mobile phones, mobile phone towers, computers, electric cookers (stoves), microwave oven, electric razors, hair dryers, electric wiring, electric heaters are the domestic source of EMF.

Occupational exposure: Occupational EMF exposures are considerably higher than non-occupational ones, which are usually in the 0.01–0.3 μT range. Occupational exposures have been studies most extensively in the electric utility industry. Average exposures have been found to be higher in electrical occupations than in other occupations, such as office work, ranging from 0.4–0.6 μT for electricians and electrical engineers to approximately 1.0 μT for power line workers. Welders have the highest average exposure at 3.7 μT. The employees of other concerned occupations are: Railway conductors, shunters, and track workers, blast furnace workers, Miners, telephone operators, telephone repairmen.

Measurement of EMF

Measurement of 50–60 Hz electromagnetic fields (EMF) is easily carried out with hand-held field meters. Sensitive, reliable meters (voltmeters and amperemeters) are commercially available for measuring electric and magnetic fields. Small, portable dosimeters have also been developed are on the market, for example, the positron (from Canada) and Emdex (from USA) instruments.

Health Effects of EMF

A number of evaluations have been made, for example, by World Health Organization, WHO (1984, 1987), the International Radiation Protection Association, IRPA (1989), the Swedish Academy of Engineering Science (1983, 1987), and the Institute of Occupational Health, IOH (1990). These evaluations have described several well-documented effects.

Hair vibration and spark discharges are secondary effects of E-field exposure. Otherwise, the various evaluations are unanimous in the view that the underlying causal mechanisms are most likely to be found in the current the fields induce in tissue. Both E- and B-fields induce current in the body. We now know the magnitude of these currents in

relation to the various degrees of and types of effects.

Acute effects: Electric fields can be detected as a slight tingling of the hair at field strengths of 5–10 kV/m. Electrical workers may also be affected by microshocks which can be experienced by a person touching a conducting object in an elevated electric field greater than about 2 kV/m. Microshocks have been suggested as a source of certain effects in cells and tissues, such as chromosomal anomalies. It should be noted, however, that there is no substantial evidence implicating microshocks as harmful beyond their nuisance effects.

Human cannot sense time-varying magnetic fields of 50–60 Hz, except at very high field strengths (on the order of 3–5 mT). At this field strength, human perceive visual flashes of light, i.e. magnetophosphenes, which are transient and not thought to produce any permanent retinal lesions. Extremely high fields may result in nerve stimulation.

Some experimental studies suggest that electric and magnetic fields can, under certain exposure conditions, produce physiologic effects in human volunteers. Changes in heart rate, heart rate variability, cognitive function and evoked cerebral potentials were observed to be slightly affected by exposure to EMF.

Long-term effects: The first publication linking to human cancer appeared in 1979. The paper reported an association between childhood cancer and presumed residential exposure to EMF. The health effects that have received most attention are cancer, reproductive effects, neurobehavioural and neurodegenerative conditions and cardiovascular diseases.

Cancer: The studies of the hypothesized association of EMF exposure with human cancer have mostly followed these two lines of investigation: Cancer among children whose exposure to EMF is residential in origin, and cancer among workers. Based on the epidemiological studies childhood leukaemia risk appears to double at average residential exposure levels above 0.3–0.4 μT. More recent and better quality studies have assessed magnetic field exposures through job-exposure matrices (JEMs) populated with personal measurements of time-weighted average (TWA) magnetic fields. The major limitation of magnetic field JEMs is that occupation is not the main determinant of exposure. A person's occupational exposure depends on the performed job tasks, field sources encountered during work, the average strength of sources and the proportion of time spent at different locations relative to sources. Consequently, measured exposure varies widely among individuals within the same occupations. Furthermore, JEMs based on contemporaneous measurements may not properly represent historical exposures. In numerous studies investigating occupational EMF exposures and several cancers, leukaemia and brain cancer and more recently, breast cancer have received the most attention, although many of the breast cancer studies focused on residential exposures. Sporadic reports of elevated risks for other cancers, such as malignant melanoma, non-Hodgkin's lymphoma, testicular cancer and prostate cancer, have been reported.

Lack of authentic biophysical mechanism and lack of evidence showing carcinogenic effects of magnetic fields in laboratory studies also weakens the argument for the causal effect. This uncertainty of the nature of the observed relationship is reflected in the classification of power-frequency magnetic field exposure as a possible human carcinogen (group 2B) by the International Agency for Research on Cancer (IARC) and World Health Organization (WHO).

Reproductive effects: The potential influence of occupational EMF exposure on reproduction has been examined among women working with video display unit (VDU). Initial concern was based on reports of several clusters of spontaneous abortions and congenital malformations among VDU operators. Subsequently, several epidemio-

logical studies provided very little or no evidence of an association between VDU use and spontaneous abortion or other adverse pregnancy outcome. Early studies were concerned with childhood leukaemia as a possible consequence of parental exposure. But no association was found in recent studies. More recent analysis of childhood brain tumour studies found no association with parental exposure of EMF.

Neurobehavioural and neurodegenerative effects: A number of studies have focused on two categories of possible neurobehavioural effects in occupationally exposed populations: Generalized neurasthenic effects and suicide. A series of studies investigated the potential relationship between occupational magnetic field exposure and neurodegenerative diseases, most notably Alzheimer's disease and amyotrophic lateral sclerosis (ALS). These investigations also suggested biological pathways that might be responsible for the association.

Cardiovascular effects: The magnetic field exposure on heart rates are variable, and by several prospective cohort studies suggested that reductions in some patterns of heart rate variability are associated with increased risk for heart disease, overall mortality rate in survivors of myocardial infarction and sudden cardiovascular death. The potential for interference with implanted medical devices, such as pacemakers, need to be considered in the case of very high electric and magnetic fields. All patients with pacemaker and defibrillators should be informed of the potential problems that could be associated with exposure to EMF. Interference to electric and magnetic fields is usually temporary and moving away from the source will alleviate the response. Advice to patients with these devices is to consult their cardiologist and the device manufacturers if concerned about potential EMF interference.

Exposure guidelines: The exposure standard are based on studies that provide information on the health effect of EMF, as well as the physical characteristics and the sources in use, the resulting levels of exposure and the people at risk. Exposure standards generally refer to maximum levels to which whole or partial body exposure is permitted from any number of sources. This type of standard normally incorporates safety factors and provides the basic guide for limiting personal exposure. There are two main international standards or guidelines: The International Commission on Non-ionizing Radiation Protection (ICNIRP) and the Institute of Electrical and Electronic Engineers (IEEE).

Acute effects on the nervous systems form the basis of international guidelines. None of the guidelines consider potential long-term effects, such as cancer, to be sufficiently established to serve as a basis for standards. In particular, exposure limits are based on the acute effects on electrically excitable tissues, particularly those in the central nervous system (CNS). The current ICNIRP limits for workers are 10 kV/m and 500 μT for 50 Hz and 8.3 kV/m and 420 μT for 60 Hz. The IEEE exposure levels are 20kV/m and 2710 μT at 60 HzμT. The differences in the guidelines derived independently by the IEEE and the ICNIRP, result from the use of different adverse reaction thresholds, different safety factors and different transition frequencies.

The International Radiation Protection Association (IRPA) has published Guidelines on Limits of Exposure to 50/60 Hz Electric and Magnetic Fields, and the Institute of Occupational Health in Sweden has, in collaboration with the National Institute of Radiation Protection, the National Swedish Board of Occupational Safety and Health and labour marker parties, drawn up a criteria document (1990) for low-frequency magnetic fields. The guidelines are based on the effects of the current induced in the body by the fields, that is, of about the same magnitude as the current normally occurring in the body (up to about 10 mA/m^2). Occupational exposure to 50 to 60 Hz magnetic fields is limited to 0.5 μT for a

complete workday and 5 μT for short-term (2 hours) exposure.

BAROMETRIC PRESSURE

Introduction

On the surface of the Earth at sea level, we live in an air environment where the air pressure results from the mass of the air in the atmosphere above and the effect of the Earth's gravitational field. On ascent to altitude on mountains or in aircraft, the mass of the air above is less and hence the pressure is also lower. On descent underwater, the environmental pressure results from both the mass of air above and the mass of the water. Since the water is many times denser than air, there is a very rapid increase in pressure with progressive descent underwater and pressure changes occur when compressed air is used in engineering works. This change in pressure is the most critical environmental factor in both diving and compressed air work.

Pressure can be measured in many alternative units which are in everyday use. The following units are used to measure environmental pressure with their normal value at sea level:

Equivalent Units of pressure

Unit	*Abbreviation*	*Value*
Standard atmosphere absolute	ata	1
Bar (bar)	bar	1
Torr	torr	760
Millimitres of mercury	mmHg	760
Kilopascal	kPa	100
Pounds per square inch	psi	14.7
Kilogram force per sq. centimetre	$kgf.cm^{-2}$	1.033
Feet of seawater	fsw	33
Metres of seawater	msw	10

Gases are compressible, so become denser as pressure increases. As a result on ascent to altitude, air density reduces with ascent and so the pressure deduction is not linear with height. Water is not compressible and hence underwater the rises linearly with depth of 1 atmosphere for every 10 m additional depth. The soluble pressure underwater in ata can simply be calculated from the water depth in meters divided by 10 and adding 1 for surface air pressure. Hence, pressure at 50 m depth is 50/10 + 1 = 6 ata. In compressed air work, 1 bar (gauge) is equivalent to 2 bar absolute pressure (i.e. 1 bar atmospheric pressure + 1 bar gauge pressure).

Both divers and compressed air workers operate in an environment where the ambient pressure is raised. Historically, compressed air work has rarely involved pressures above 3–4 ata, but in recent years the development of tunnel boring machines used in modern excavation has led to much higher pressures being required. Diving is regularly undertaken at pressure of up to 35 ata and the world record exceeds 70 ata.

In construction, work is done in pressurized environments in caissons used in building bridge piers and in pressurized tunnels. The caisson is simply an inverted box, opens at the bottom, which is pressurized to keep water from entering. Compressed air is used in tunnels to dry out the working face and hold out water when digging through wet, porous ground or under rivers and lakes.

For every foot a diver descends below a surface, or the tunnel worker descends below the local water table, there is a linear pressure increase of 0.433 pounds per square inch (psi) for fresh water and 0.445 psi for salt water. Thus, a diver working in 100 feet of sea water would be subject to a pressure of 44.5 psi gauge (psig). Since the diver was already exposed to 14.7 psi absolute (atmospheric pressure) at sea level, the absolute pressure experienced at 100 feet is 59.2 psi. In the practical work situation, gauge pressures that show only the pressure differential from atmospheric are used. However, in calculating decompression schedules, absolute pressure is required.

Units of measure for pressure also need definition. One atmosphere equals 14.7 psi.

This is equivalent to the weight of a column of sea water 33 feet high of 1 square inch cross section. Thus, for every 33 feet a driver descends the pressure on his body increases by 1 atmosphere. At the depth of 99 feet, the diver is at 3 atmospheres gauge or 4 atmospheres absolute (ATA). It is usually customary when referring to pressure in atmospheres to mean atmospheres absolute, unless otherwise noted.

The metric system vastly simplifies diving calculations, since 1 kg/cm^2 very closely approximates one atmosphere. 10 m of seawater pressure is equal to 1 kg/cm^2. Thus, changes in atmospheres or meters in depth can be treated decimally. Nevertheless, the metric system is not used for diving orcompressed-air work in the United States, although it is now common in England, Canada, and the rest of the world.

High Barometric Pressure

Physics of the Pressure Environment

The physical properties of the environment are defined by the gas laws. *Boyle's law* states that for a fixed mass of gas at constant temperature the volume is inversely proportional to the absolute pressure. Hence, if pressure is double, the volume is half. *Charles' law* states that for a fixed mass of gas at constant pressure, the volume is proportional to the absolute temperature. *Dalton's law* states that 'in a mixture of gases, the pressure exerted by each of the constituent gases is the same as it would exert if it alone occupied the same volume'. The pressure exerted by each constituent is called a partial pressure. The total pressure in a gas mixture is the sum of the partial pressures of the constituents, and these partial pressures can be calculated from the total pressure and the fractional concentrations of the constituents. Hence, in air at sea level where oxygen forms 20.9% of the gas mixture, the partial pressure of oxygen is 20.9% of 1 ata or 760 mmHg i.e. 0.209 ata, 159 mmHg. *Henry's law* states that 'at constant temperature, the amount of gas which dissolves in a liquid with which it is in contact is proportional to the partial pressure of the gas. Hence, while the amount or mass of the gas which dissolves in a liquid is also dependent on that gas's solubility, an increase in partial pressure of the gas will increase the amount that goes into solution.

The constituent gases of the diver's breathing mixture have important pathophysiological effects. Oxygen is critical to metabolic processes and a reduced partial pressure of oxygen will result in hypoxaemia. However, the increase in oxygen partial pressure may also be toxic. Oxygen toxicity is more likely with increasing pO_2 and with duration of exposure. The lung may be affected with prolonged exposure to a pO_2 above 0.6 ata and, in the central nervous system (CNS), prolonged exposure often manifests as convulsions with relatively short duration exposure to pO_2 above 1.6 ata. CNS toxicity enhanced by exertion, so a diver at rest in a chamber can tolerate a pO_2 of 2.8 ata which would result in convulsions if the diver was active in the water. Oxygen tolerance can be extended if exposure is intermittent. Nitrogen which is normally an inert component of the air we breathe has narcotic and anaesthetic effects at high partial pressures, which limits the depth at which air can be used as a breathing mixture and represents a distinct safety hazard. The narcotic effects begin to be noticeable at a depth of about 30 msw (pN_2 approximately 3 ata) and increase as the pressure goes up. Nitrogen narcosis and gas density issues become an important contributor to accidents or errors. Breathing air is considered to be a medical contraindication for routine commercial diving applications at depth greater than 50 m. In practice, this is the reason the Royal Navy prohibit air diving for military purposes at depth greater than 50 m and in the health and safety executive (HSE)—approved compressed air tables breathing air is limited to a 3.45 bar (gauge)

maximum pressure in the United Kingdom. At higher pressure, the nitrogen requires to be replaced either partially or completely by an alternative 'inert' gas usually helium. Helium has the advantages of having no narcotic effect and being very light, so also overcomes some of the respiratory problems caused by gas density. However, it diffuses very easily, has high thermal conductivity, and so induces significant thermal control problems and produces speech distortion which affects vocal communications. Other gas may also be encountered. Argon is used as a shielding gas in welding operations. It is breathable but more narcotic than nitrogen and hence a hazard. Hydrogen has been used in some experimental deep dives for its minimal density. It has high thermal conductivity and introduces the potential for explosive gas mixture.

Health Hazards of High Barometric Pressure

Acute Effects

Barotrauma:

1. *Middle ear barotrauma:* The most immediately recognized effect of change in environmental pressure is the result on the middle ear cavity. The Eustachian tube is normally closed and hence the middle ear cavity is a closed gas space with one flexible side, the tympanic membrane. The body tissues behave like a fluid and transmit pressure so any change in environmental pressure is transmitted to gas-containing spaces in the body. An increase in pressure results in a reduction in middle ear gas volume and causes movement of the tympanic membrane. This is really sensed and can cause pain if the membrane is stretched enough. If the diver opens the Eustachian tube, air at ambient pressure passes up into the middle ear and the tympanic membrane moves back to the normal position. As the diver descends in the water or a tunneller is compressed in a gas environment, this action of 'equalizing' ear pressures is required recurrently until the diver reaches stable depth. If equalization is not performed, the differential pressure across the Eustachian tube may effectively prevent the diver from opening the tube and result in pain and trauma to the tympanic membrane or inner organs. The tympanic membrane may be bruised and haemorrhagic or may rupture, an event, which although painful, equalizes pressure. In most cases, middle ear barotraumas resolves within two weeks with complete healing of the ear drum. It is important to ensure Eustachian function has returned to normal before returning to diving or other pressure exposure. However, the transmitted pressure effects may also damage the organ of Corti and produce a permanent hearing loss. Vigorous attempts to equalize pressure by holding the nose and doing a valsalva manoeuvre increase intracerebral pressure and hence the pressure difference across the round window. Rupture of this membrane results in leak of endolymph into the middle ear and a gradual onset of vertigo, nausea and hearing loss. This is an emergency and requires urgent otological assessment.

The degree of barotraumas to the tympanic membrane can be judged by its appearance. Wallace Teed, a US Navy diving medical officer, devised a four-point scale for classifying middle ear injury. This scale is used by US Navy:

Teed 1: Congestion in Shrapnell's membrane and along the handle of the malleus.

Teed 2: Redness of the entire tympanic membrane with retraction.

Teed 3: Haemorrhage into the tympanic membrane but not into the middle ear.

Teed 4: Entire middle ear filled with blood, drum bluish in appearance and possibly ruptured.

2. *Sinus barotrauma:* Other gas containing spaces in the body may also cause problems. Sinus cavities should normally communicate with the nasal airways and hence not behaves close spaces. However, minor inflammatory change in the nasal mucosa may readily close sinus ostea and so prevent free gas movement. The openings of the sinuses must be patent, or the application of increased pressure will force the capillaries lining them to distend and burst, filling the sinuses with blood. The most painful type of sinus squeeze involves the frontal sinuses. The maxillary, ethmoid, and sphenoid sinuses are less painful and also less commonly affected. If the worker's frontal sinus is blocked, the exquisite pain produced makes it impossible for him to compress deeper than a few feet.

   If the sphenoid sinus is blocked, the pain is generally referred to the occiput or vertex of the skull. Decongestants and nasal spray are the only readily available treatment for sinus blockage. In case of severe sinus obstruction or Eustachian stenosis, an otolaryngologist may be able to artificially widen the opening to the sinus or place a tiny polyethylene tube through the eardrum in the caisson worker. Divers not using dry suits cannot be helped by piercing the eardrum with a tube, since water would then enter the middle ear. Some compressed-air workers have used pressure-equalization tubes for months during the extent of the contract.
3. *Pulmonary barotrauma:* The lungs are the largest gas-containing space in the body. In normal breathing, the lungs communicate freely with the upper airways and mouth so changes in volumes of gas in the lungs can be easily compensated for during normal breathing. Should the diver stop breathing, hold their breath or change depth rapidly, gas volume may change enough to injure the lung. Compression of the lung (squeeze) is extremely rare in normal diving or compressed air work, but a common issue in extreme breath-hold diving. Overexpansion of the lung (burst lung) is a much more important problem in diving with potentially fatal results. If during a rapid ascent, the diver does not exhale adequately, the lung will expand beyond normal volume and may rupture allowing gas to escape through the interstitial tissue of the lung. The gas may trace to the pleural cavity causing pneumothorax often with tension, or into the mediastinum, or it may rupture into the pulmonary venous system and pass directly into the left heart and result in arterial gas embolism. Rarely, gas may track through the retroperitoneal space and cause pneumoperitoneum. Both tension pneumothorax and gas emboli may be rapidly fatal. Pneumothorax presents in a similar manner to spontaneous pneumothorax in other situations with chest pain and breathlessness and is treated in the same way. Pneumomediastinum often presents slowly after the dive, often several hours later with crepitation over the anterior chest and neck, discomfort in the neck and hoarse voice.

   There is no specific treatment available, but breathing 100% oxygen accelerates the removal of air from the tissues. Arterial gas embolism usually presents with an acute cerebral event, convulsion, loss of consciousness, sudden blindness or hemiplegia and is usually almost immediately following surfacing from the dive. Treatment is required rapidly and includes administration of 100% oxygen and therapeutic recompression using an appropriate treatment procedure. In severe cases, like pneumomediasinum, pneumothorax or pneumoperitoneum, again recompression is usually followed by hyperbaric oxygen therapy, which has

three aims: (a) It reduces the inert gas load within the vascular space and other tissues, (b) it may provide oxygenation to tissues that remain hypoxic as a result of the injury and (c) it may help to prevent reperfusion injury in those tissues which had lost their blood supply.

The pathogenesis of these disorders is incompletely understood. Arterial gas embolism occurs when the lung ruptures as a result of pulmonary barotrauma and gas is released into the pulmonary venous system. When gas escapes into the pulmonary venous system, its main target organ is the brain. Gas enters the cerebral circulation and, although some will pass through, it triggers circulatory change with local hyperaemia and an increase in cerebral blood volume, systemic hypertension, a loss of cerebral autoregulation and rapid development of cerebral oedema. The later is almost certainly related to the injury to vascular endothelium. In those who survive the acute injury, the progress of the illness may be significantly affected by the presence of inert gas already dissolved in brain tissue. The dissolved gas may enhance the volume of gas involved exacerbating the pathological impact. Hence, an episode of arterial gas embolism occurring in the presence of a significant dissolved gas load may have a worse prognosis than one occurring without.

The risk of sudden change in pressure is much less in compressed air work where the working chamber pressure and compression and decompression rates are controlled by a pressure lock operator. However, there remains a risk of explosive decompression resulting from engineering or equipment failure.

3. *Barotrauma of other organs*: The bowel usually contains gas, but is distensible and ultimately open ended. Hence while gas movement may be recognized by the diver or tunneller, it is rarely pathological. Gas spaces around the body are also affected. The gas space between a diver's breathing mask and face is affected by pressure change. A diver's mask includes the nose, allowing the diver to exhale through the nose to equalize pressure. Without this action, the diver's periorbital soft tissues are affectively sucked into the mask space producing oedema and haemorrhage. This results in a dramatic appearance, but is not a serious injury. Divers wearing a dry suit have an enclosed gas space around them which will reduce in volume during descent. Eventually, the suit will be compressed around the diver and folds in the suit material may trap subcutaneous tissues of the diver and result in linear bruising (suit squeeze) which may be painful. Many modern suits now have a gas injection system allowing the diver to introduce additional gas and release the pressure. This provides more comfort, but introduces an additional hazard due to buoyancy (floatability) effects.

Decompression sickness: Decompression sickness is a spectrum of clinical syndromes which result from the effects of the formation of excessive amount of gas microbubbles in the tissues and bloodstream, during and after a decompression, from gas which had previously been dissolved in body tissues according to Henry's law.

When a diver or compressed air worker is exposed to increased environmental pressure and breath a gas mixture containing an inert gas (e.g. nitrogen or helium), body tissues are exposed to an increase in partial pressure of that gas. As a result, additional gas dissolves. Some gas may diffuse through the skin directly into subcutaneous tissues, but the majority of the gas will dissolve initially in the pulmonary blood flow and be distributed gradually to all body tissues. This process of solution, distribution and diffusion is gradual and will take six to eight hours at a constant

pressure until complete equilibrium in all body tissues is established. At this point, the diver is said to be saturated with the inert gas at that particular pressure. On return to the surface, the partial pressure of the gas reduces and so the tissues become effectively supersaturated with gas which is then released from the solution. On slow ascent, this excess gas will mostly be transferred through the circulation to the lungs and exhaled. However, the difference in partial pressure between the diver's tissues and the environment may be sufficient to cause gas to come out of the solution and form bubbles. Bubbles are not inert and the bubble surface is capable of triggering a reaction in tissues and in blood involving the coagulation system and an inflammatory process. Recognized risk factors for clinical episodes of decompression sickness fall into two major categories: (i) Pressure exposure-related factors, such as depth (pressure) and duration of exposure, previous exposures over the preceding hours and days; and (ii) the ascent/decompression rate, decompression methodology and individual diver's predisposing clinical factors, such as intracardiac shunt or personal susceptibility. Symptoms of decompression sickness are very variable, but fall into following several main categories:

1. *Neurological:* Neurological symptoms include paraesthesiae of any area, sensory loss, focal weakness and disturbance of balance. More pronounced spinal cord involvement may cause marked weakness and paraplegia or even tetraplegia and be associated with spincteric disturbance. Low back pain or pain in a girdle distribution is often a precursor of spinal cord symptoms. Vestibular symptoms (staggers) of nausea, vertigo and loss of balance may occur either with or without other neurological symptoms. Disturbance of cerebral function is less common, but well documented, and may include behavioural change or even loss of consciousness. Although the ultimate prognosis for neurological disease is usually for considerable recovery, this is the main cause of permanent disability resulting from decompression illness.
2. *Limb pain:* Limb pain (bends) symptoms most commonly affect larger joints, typically the shoulder, elbow, wrist and knee, but can affect any joint. Joint pain varies from being a mild ache (niggle) to severe and incapacitating pain, tend to be constant and unaffected by movement of joint. Limb pain not localized to a joint sometimes occurs and may be due to oedema and swelling of the muscle. This is historically the most common presentation in compressed air workers often affecting the knees and typically presenting 4–12 hours after exposure.
3. *Cutaneous:* Cutaneous symptoms, include itching which is probably due to gas diffusing through the skin, urticaria and a more persistent rash, called 'cutis marmorata' which comprises a patchy rash of erythematous and petechial lesions interspersed with normal skin spread over variable areas but usually over the trunk or proximal limbs. It is usually painless and not itchy and hence not observed until the diver removes their suit. This rash is often associated with more serious neurological manifestations of the illness which may be concurrent or follow later. Localized areas of oedema of the skin may occur which are probably due to local lymphatic obstruction (peau d'orange).
4. *Constitutional:* Many patients also report constitutional symptoms of tiredness, nausea, feeling cold or weak. These symptoms are most likely a result of hypovolaemia resulting from loss of fluid through damaged capillary endothelium into interstitial tissues and often improve rapidly with rehydration and therapeutic recompression.
5. *Pulmonary:* Pulmonary involvement (chokes) results from massive bubble load

in the venous circulation and obstruction of the pulmonary circulation. This results in cough, breathlessness and haemoptysis. The resulting high pulmonary arterial pressure results in intrapulmonary shunting and passage of bubbles to the arterial circulation and severe neurological symptom usually follow. Individuals with significant pulmonary involvement are critically ill and there is a high mortality. Symptoms may develop at any time after the commencement of the ascent phase of a dive, but are most common in divers in the first one to two hours following surfacing.

Immediate first aid treatment includes administration of 100% oxygen and rehydration by the most effective means available. Patient presenting with decompression illness should receive specialist assessment and transfer to a recompression unit for further treatment with intravenous fluids and recompression.

Long-term effect of high barometric pressure: Divers are at risk of acute diving-related illness, such as decompression sickness and barotraumas, as well as other work-related injury. Incomplete recovery from these events may leave the diver with a significant residual deficit or disability which will never recover.

1. *Bone necrosis:* Both divers and compressed air workers are at risk of developing 'dysbaric osteonecrosis'. This is a form of aseptic bone necrosis which results as a long-term health effect. It may occur after a single pressure exposure, but is more likely after deeper and longer pressure exposure and is more likely with history of acute decompression illness to occur in individuals.
2. *Neurological:* Divers are at risk of long-term neurological injury as a result of diving in the absence of a history of acute decompression illness resulting in a permanent disability like memory difficulties and concentration problems.
3. *Pulmonary:* Divers have been demonstrated to have larger lungs than the normal population and this has been associated with abnormalities in the expiratory flow pattern consistent with small airway obstruction.

Low Barometric Pressure

High Altitude illness

The discomfort experienced by mountaineers on ascent to high altitude is compounded by the additional hardship of exertion, fatigue, exposure, low temperature, gastrointestinal upsets, alteration in diet and dehydration. In 1913, TH Ravenhill, medical officer to a mining district in the Chilean Andes, described the features of altitude sickness affecting miners transported by rail from Antofagasta, a seaport on the Pacific Ocean, to mines situated above 4600 m. Based on the clinical observations, Ravenhill identified three types of high altitude sickness: Normal puna (acute mountain sickness), cardiac puna (high altitude pulmonary oedema) and nervous puna (high altitude cerebral oedema). It is tribute to Ravenhill's clinical and observational skills that his classification of the 'benign' (acute mountain sickness) and 'malignant' (high altitude pulmonary oedema, high altitude cerebral oedema) forms of altitude sickness remains in use in the modern era.

Acute Effects

Acute mountain sickness: Acute mountain sickness is a self-limiting condition characterized by headache, sleep disturbance, anorexia, nausea, vomiting and cerebral symptoms, such as profound fatigue, dizziness, irritability, lack of concentration and confusion. Physical signs include periorbital and peripheral oedema and manifestations of normal physiological response to high altitude exposure, such as shortness of breath on exertion and tachycardia. Acute mountain sickness (AMS) affects unacclimatized visitors at elevations above 2500 m. Symptoms

become manifest after a period of approximately six hours at high altitude and reach their maximum severity at 24–48 hours. Symptoms subside gradually after two or three days' acclimatization. If the condition is ignored and ascent to greater altitude is attempted, AMS may progress to potentially fatal high altitude cerebral oedema (HACO) or high altitude pulmonary oedema (HAPO). The incidence of AMS in sea-level residents ascending to altitudes greater than 3000 m has been estimated from large population studies of trekkers and soldiers on single ascents.

Lake Louise Acute Mountain Sickness (AMS) score: A diagnosis of AMS is based on a recent rise in altitude, the presence of a headache and at least one other symptom and a total score of at least 3. AMS = Altitude Gain and Headache and at least one other symptom and a total score of 3 or more. Add up the responses to each of the questions of the self-report score (questions 1–5):

Self-report questionnaire:

1. *Headache*	0. *No headache*
	1. Mild headache
	2. Moderate headache
	3. Severe headache, incapacitating
2. GI symptoms	0. No GI symptoms
	1. Poor appetite or nausea
	2. Moderate nausea or vomiting
	3. Severe nausea and vomiting, incapacitating
3. Fatigue and/or weakness	0. Not tired or weak
	1. Mild fatigue/weakness
	2. Moderate fatigue/weakness
	3. Moderate fatigue/weakness, incapacitating
4. Dizziness/lightheadedness	0. Not dizzy
	1. Mild dizziness
	2. Moderate dizziness
	3. Severe dizziness, incapacitating
5. Difficulty sleeping	0. Slept as well as usual
	1. Did not sleep as well as usual
	2. Woke many times, poor nights sleep
	3. Could not sleep at all

Additional lake louise scoring: The self-report score stands alone and is recommended for nonmedical mountain travelers. Additional observations are sometimes used by researchers. The clinical assessment score (questions 6–8) can be added to the self-report score, in which case, in the context of a recent rise in altitude, a score of 5 or more would be taken as AMS. In this case, AMS = altitude rise and headache and at least one other symptom (from questions 1–5) and a total score of 5 or more (questions 1–8).

Clinical assessment:

6. Change in mental status	0. No change in mental status
	1. Lethargy/lassitude
	2. Disoriented/confused
	3. Stupor/semi-conscious
7. Ataxia (heal-toe-walking)	0. No ataxia
	1. Manoeuvre to maintain balance
	2. Steps off line
	3. Falls down
	4. Can't stand
8. Peripheral oedema	0. No peripheral oedema
	1. Peripheral oedema in one location
	2. Peripheral oedema in two or more locations

Functional score: Overall, if you had any symptoms, how did they affect your activity?

0. No reduction in activity
1. Mild reduction in activity
2. Moderate reduction in activity
3. Severe reduction in activity (e.g. bedrest).

High altitude pulmonary oedema (HAPO): High altitude pulmonary oedema is a noncardiogenic form of pulmonary oedema. Pulmonary hypertension is the principal pathological process causing leakage of oedema fluid from intravascular space into the alveolar bed in association with an exaggerated pulmonary vasoconstrictive response to hypoxia and exercise, which is distributed unevenly throughout the lung vasculature. In addition, abnormal endothelial synthesis of vasoactive substances disturbs the balance between vasodilators (e.g. nitric oxide) and vasoconstrictors (e.g. endothelin-1). The signs of HAPO are marked breathlessness at rest, a rapid respiratory rate (>30 breaths/min) and a dry cough, frothy or blood-streaked sputum. The victim appears deeply cyanosed and may complain of chest discomfort. Extensive bilateral crepitations (rales) are heard on auscultation. In extreme cases, gurgling sounds from the lungs may be audible to the victim and his companions without the use of a stethoscope. Signs of cardiac failure are not evident, although tachycardia (pulse rate >120 beats/min) and a low systemic blood pressure are frequently present. Cardiac murmurs are not a feature of HAPO. However, the second heart sound is accentuated and sometimes palpable, reflecting elevation of pulmonary artery pressure. An erroneous diagnosis of bronchopneumonia may be suggested by the presence of low-grade pyrexia and moderate leukocytosis, but respiratory infection may coexist with HAPO.

Arterial blood gas measurements confirm severe hypoxaemia with low arterial partial pressure of oxygen (paO_2) from hyperventilation resulting in respiratory alkalosis. Prominence of the pulmonary arteries and patchy infiltrates seen in chest radiographs in early disease progress to homogenous bilateral infiltration in severe cases. Right ventricular overload pattern (right axis deviation, right bundle block and right ventricular strain) is present on electrocardiograph tracing. Echocardiography may demonstrate incompetence of tricuspid valve. Cardiac catheterization studies performed on HAPO victims before treatment or descent show a high pulmonary arterial pressure. Descent to low altitude and administration of oxygen lowers pulmonary artery pressure, clears radiographic pulmonary infiltrates and relieves symptoms within one or two days. Failure to descent or to treat HAPO effectively may result in a fatal outcome within a few hours of onset: The mortality rate is quoted in the range of 4–11 per cent. The development of HAPO is related to the speed of ascent, the altitude achieved, the exertion involved and the susceptibility of the individual.

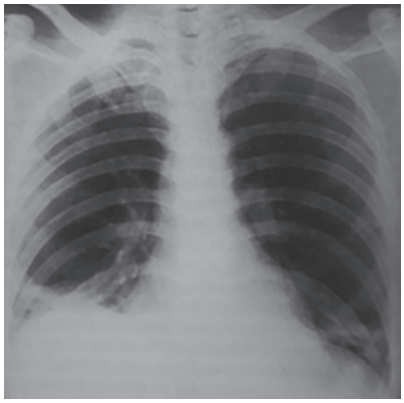


Fig. 8.26: Lung collapse at high altitude

High altitude cerebral oedema (HACO): High altitude cerebral oedema presents typically with intense headache, profound lethargy, ataxia, impaired coordination, confusion, disorientation and hallucinations. Convulsions and focal features, such as cranial nerve palsies and visual field loss, may occur. Cerebral oedema occurs in people exhibiting 'benign' AMS: The progression to HACO is indicated by deterioration in existing AMS, despite treatment and no further ascent, or the appearance of neurological signs, such as ataxia on walking (poor heel to toe walking) or sitting (truncal ataxia), cranial nerve palsies and visual field loss. Convulsions may occur. Papilloedema develops in 50% of HACO victims. Rapid deterioration in conscious level

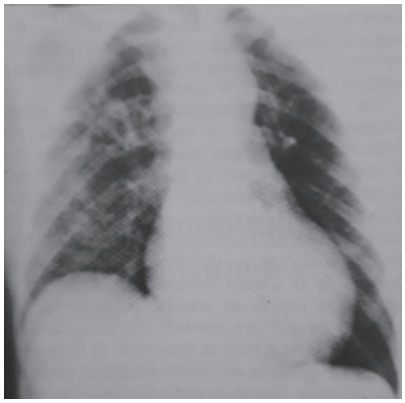


Fig. 8.27: High altitude pulmonary oedema

leading to death has been widely reported. Investigation off the mountain show raised cerebrospinal fluid pressure on lumber puncture and increased signal in white matter reflecting oedema of the corpus callosum on T-2 and diffusion-weighted magnetic resonance imaging (MRI). Postmortem examination reveals extensive cerebral oedema, intracerebral haemorrhage and thrombosis in cerebral veins and dural sinuses.

Current thinking is that HACO is due to vasogenic oedema related to disruption of the blood–brain barrier with extravasation of fluid into the interstitial space rather than cytotoxic oedema related to cell death due to increased intracellular osmolarity. Damage to the blood-brain barrier may be 'mechanical' induced by impairment of cerebrovascular autoregulation or 'chemical' related to release of mediators of barrier permeability, such as bradykinin and hydroxyl-free radicals. Cerebral hypoxia is exacerbated by exercise which reduces oxygenation further.

Cerebral oedema occurs usually in unacclimatized people above 3000 m elevation, although cases of HACO in well-acclimatized climbers at extreme altitudes (>7000 m) have been demonstrated.

Treatment

Acute mountain sickness of moderate severity – without respiratory distress or neurological dysfunction – may be managed at intermediate altitudes between 3000 and 4000 m, particularly if the victim is able to sleep at a lower altitude. Exercise induces sodium and water retention, decrease oxygen saturation and increases pulmonary artery pressure, thus aggravating the pathological processes leading to altitude sickness: Rest is an important component of treatment for all grades of severity of AMS. Abstinence from alcohol, adequate fluid intake and frequent small meals of carbohydrate content are recommended. Nonsteroid anti-inflammatory drugs (e.g. ibuprofen) have been proven effective in relieving high altitude headache. To ease nausea and vomiting, anti-emetics (e.g. prochloroperazine 10 mg oral or intramuscular injection three times a day) may be administered. To aid sleep short-acting sedatives (e.g. temazepam) should be used at the lowest effective dose for the shortest time possible. A low-dose acetazolamide (250 mg) may relieve symptoms of AMS, arterial oxygenation improves and pulmonary gas exchange is established.

Treatment of severe AMS, HAPO and HACO is immediate descent to the lowest altitude feasible and administration of oxygen. All other therapeutic measures are of secondary importance. In addition to descent and oxygen, dexamethasone is administered in severe AMS (4 mg every six hours for 24 hours) and HACO at a dose of 8 mg initially followed by 4 mg every six hours by mouth, IM or IV injection. Nifedipine, a calcium channel blocker, reverses hypoxic pulmonary hypertension and lowers pulmonary arterial pressure in HAPO victims. Oxygenetion improves and the clinical manifestations of HAPO are relieved. Nifedipine is prescribed at a dose of 10 mg sublingually and 20 mg slow release capsule stat followed by 20 mg slow release capsule every six hours until decent for established HAPO. HAPO is not caused by cardiac failure and the use of frusemide may worsen the condition of a hypotensive, shocked HAPO victim.

High altitude retinal haemorrhage (HARH) may occur at altitude above 14,000 ft but are seldom symptomatic and, unless sought by funduscopic examination, may be overlooked. Noticeable defect in peripheral vision rarely occur. When discovered, treatment is oxygen, with early descent to a lower altitude. The progression is good for healing (by resorption) in several weeks, without impairment of visual acuity. Rarely, persistent visual field defects may occur.

Chronic Mountain Sickness (Monge's Disease)

This is a disorder that develops in some native highlanders after many years of residence at altitude above 4000 m (Health and Williams. 1989). The patient, who is usually a young or middle-aged man, exhibits excessive fatigue, decreased exercise tolerance and marked central cyanosis. The cyanosis combined with excessive polycythaemia gives the lips and mucous membranes an almost black colour. There is moderate pulmonary hypertension with hepertrophy of the right ventricle, but right ventricular failure is seldom seen. The haemoglobin concentration exceeds 23 g/dl and the arterial oxygen tension and oxygen saturation are severely depressed (Penaloza, Sime and Ruiz, 1971). The underlying dysfunction is one of chronic alveolar hypoventilation due to the loss of long-established acclimatization. The normal ventilator responses to hypoxia are lost. The increased arterial hypoxaemia gives rise to a marked increase in the concentration of haemoglobin and to a very high haematocrit (73 to 83%).

The treatment of Monge's disease is descent to a lower altitude. The symptoms and the blood and circulatory disturbances improve rapidly on descent to sea level, but usually recur if the patient returns to high altitude.

High-altitude Pulmonary Hypertension

It is a form of high-altitude related disease in long term residents. It is marked by pulmonary hypertension and cor pulmonale without polycythemia. The affected individuals present with dyspnoea, cough, cyanosis. Peripheral edema, and physical examination findings consistent with right heart failure. Right-axis deviation, right ventricular hypertrophy, and p-pulmonale may be present on the electrocardiogram, while chest imaging reveals right ventricular and right atrial enlargement and prominence of pulmonary arteries. The optimal treatment for the disorder is relocation to lower elevation, but as in chronic mountain sickness, descent may not be feasible and pharmacologic options may be necessary.

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