Chapter

1

Occupational Health

MEANING OF OCCUPATIONAL HEALTH AND OCCUPATIONAL HEALTH HAZARD

Occupational Health

Occupational health is the promotion and maintenance of the highest degree of physical, mental and social well-being of the workers in all occupations. It should aim at the promotion and maintenance of the highest degree of physical, mental and social well-being of workers in all occupations, the prevention among workers of departure from health caused by their working conditions; the protection of workers in their employment from risks resulting from factors adverse to health; the placing and maintenance of the worker in an environment adapted to his physiological and psychological capabilities and, to summarize, the adaptation of work to man and of each man to his work. It is a division of general medicine which is devoted to the prevention of occupational disease and injury and to the promotion of health of people at work.

Today's concept of occupational health has greatly been changed from yesterday's concept of 'industrial health'. Now, it is changing to 'environmental health' because of greater awareness of all concerned about the ecological problems arising from rapid industrialization, urbanization, use of complex process and hazardous chemicals in industries.

The modern industrial processes present many hazards to the health of the employees. It is an established fact that there is no occupation which does not have any hazard. It is also true that there is no effective way of cure available for many of the occupational diseases. But there is a rosy part also that most of the occupational diseases are preventable. Preventive medicine and occupational health have the same aim. One of the declared aims of occupational health is to provide a safe occupational environment in order to safeguard the health of the workers and to step up industrial production and development.

Hazard and Risk

Hazard is the potential for causing harm and risk is the likelihood (or, the probability) that such harm will result. The term risk is now extended to include consideration of the severity of effect. If handling sheets of paper at work is just as likely to cause cuts (paper cut) to the skin, as sharpening knives, the probability of injury from both processes is the same, but cut from knives are more severe than paper cuts. Therefore, of the two processes with equal likelihood of skin injury, sharpening knives is viewed as riskier because of the severity of the effect. The distinction between hazard and risk is important in regard to choose to suitable measures to reduce the likelihood of occupational injury or disease. Minimizing risk may be more cost-effective than eliminating hazards.

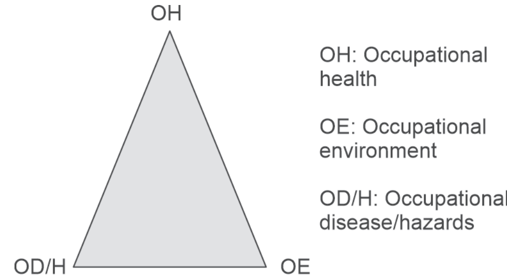

Fig. 1.1: Trangle of interrelation between OH, OE and OD/H

Occupational environment is the sum of external conditions and influences prevailed at the place of work and which have direct and/or indirect effects on the health of working populations. If occupational environment is polluted, it will affect the health of working population and may cause occupational disease or hazard. The disease which arises out of or in course of occupation is known as *occupational disease* (Fig. 1.1).

Improvement of environmental health is a major concern of many industries. The objective of health education in environmental health promotion through improvement of personal hygiene are to:

1. Educate the people in the principle of environmental health with a view to bring about desired changes in health practice.
2. Secure adoption, wide use and maintenance of environmental health practice.
3. Promote active participation of the people to improve the personal and environmental health.

An agent is defined as a substance living or non-living or a force, tangible or intangible the excessive presence or relative lack of which may initiate or perpetuate a disease process. Basically, there are three types of interactions in a working environment between environment and hazardous agents, they are:

1. Workers vs physical, chemical and biological agents.
2. Workers vs machine, tools, housekeeping.
3. Workers vs workers.

OCCUPATIONAL HEALTH HAZARDS

An industrial worker may be exposed to five types of hazards, depending upon his occupation:

1. *Physical hazard*: Physical hazard is due to the interaction between workers and physical agents. Examples of physical agents are heat, cold, noise, vibration, ionizing radiation, non-ionizing radiation, illumination, barometric pressure, electricity, electromagnetic fields (EMF).
2. *Chemical hazard*: Chemical hazard is due to the interaction between workers and chemical agents (Fig. 1.2). Chemical hazards are due to excessive airborne concentrations of vapors, gases, or aerosols in the form of dusts, fumes, or mists. These may present an inhalation hazard; some may act as skin irritants or be toxic by absorption through the skin and gastrointestinal tract.

   There is hardly any industry which does not make use of chemicals. The ill effects produced depend upon:

   a. Nature of exposure
   b. Severity of exposure

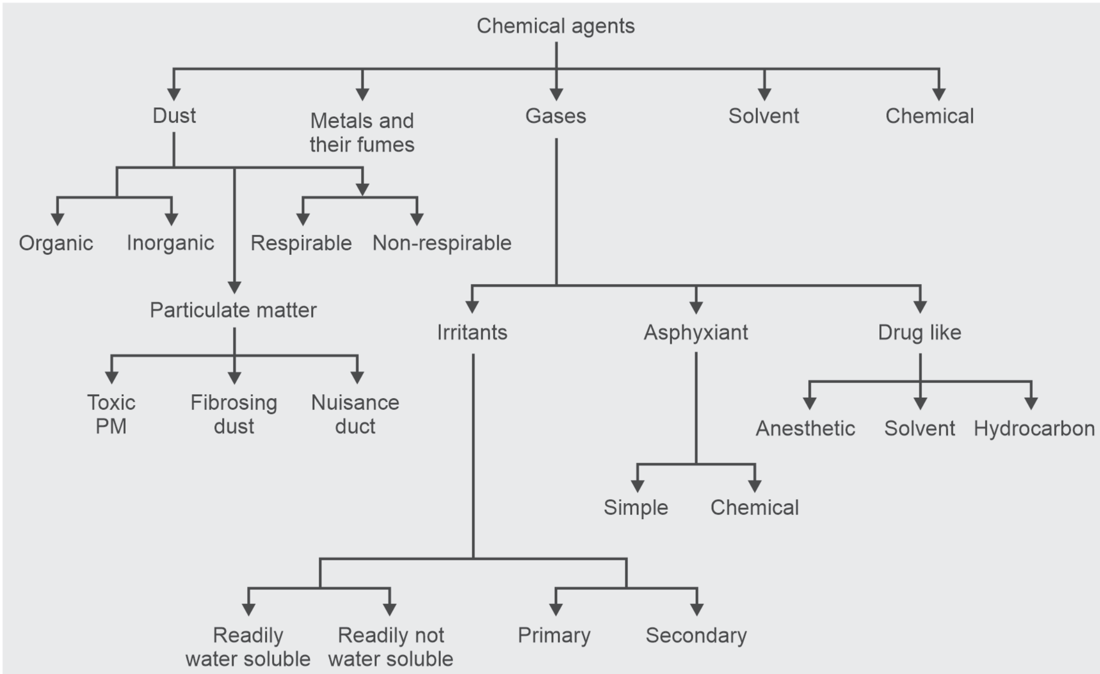

Fig. 1.2: Type of chemical agents

  c. Duration of exposure
  d. Quantum of exposure
  e. Individual susceptibility

3. *Biological hazard*: Biological hazard is due to the interaction between workers and biological agents. Examples are bacteria, virus, and fungus, insects, etc. These hazards may be produced by handling or processing biological specimens, plants, or animals and by improper removal of waste or sewage; inadequate food handling procedures; and deficiencies in personal hygiene, sanitation, and housekeeping procedures. Occupational biohazards may act as infectious, allergenic, toxic, or carcinogenic agents in humans.
4. *Mechanical hazard*: Mechanical hazard is due to the interaction between workers and machine, tools, housekeeping, etc. The unguarded machine, protruding and moving parts, poor installation of the plant, lack of safety measures are the causes of accidents which is a major problem in industries (Fig. 1.3). Working for long hours in un-physiological postures is the cause of fatigue, backache, diseases of joints and muscles and impairment of the workers' health and efficiency. Accident, injuries and cumulative trauma disorder are the examples of mechanical hazards. Factors responsible for mechanical hazard are two: environmental factors (heat, noise, vibration and machine without any

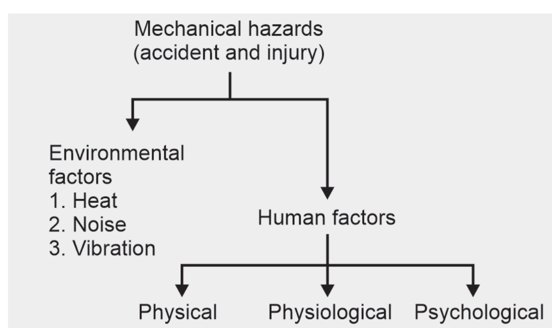

Fig. 1.3: Factors responsible for mechanical hazards

guard) and human factors. Human factors may be physical (color blindness, deafness), physiological (age, sex, experience and shift work) and psychological (accident proneness and over enthusiasm).

5. *Psychosocial hazard*: Psychosocial hazard is a hazard that affects the mental well-being or mental health of the workers by overwhelming individual coping mechanisms and impacting the workers ability to work in a healthy and safe manner. There are numerous psychological factors which operate at the place of work. These are the human relationships amongst workers themselves on the one hand and those in authority over them on the other hand. Examples of psychosocial factors include type and rhythm of work, work stability, service conditions, job satisfaction, leadership style, security, workers' participation, communication, etc. In modern occupational health, the emphasis is upon the people, the condition in which they live and work, their hopes and fears and their attitudes towards their job, their fellow-workers and employees. Psychosocial effects may be of two types: Psychological (behavioral changes like industrial neurosis) and psychosomatic (systemic changes like high blood pressure due to mental stress) (Fig. 1.4).

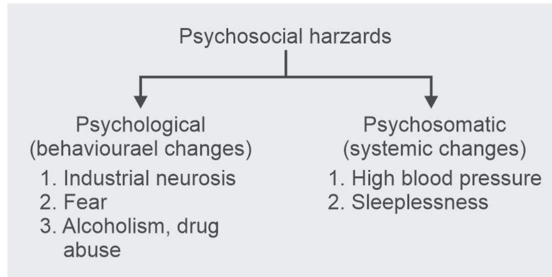

Fig. 1.4: Type of psychosocial hazards

OCCUPATIONAL HEALTH AWARENESS PROGRAMME

There is a growing awareness that improved working conditions and the working environment are a positive contribution to the national development and a necessity in the achievement of sustainable development. But despite the efforts of all concerned, the incidence of diseases is still too high and the workplace remains hazardous. Hazards related to the use of chemicals are widespread and according to ILO estimate, two out of every three workers worldwide are exposed to them. It shows how important it is to multiply our efforts to protect working people and the environment against these hazards. Prevention is the treatment of choice in case of occupational health hazards to save lives of workers and economic losses in industries as well as these are essential for the excellence in industrial development.

Health awareness is a process that bring out changes in workers' knowledge, attitude, skill and behaviour. A positive knowledge, attitude and participation of the employees should be there through the process of health awareness for the promotion and maintenance of the highest degree of physical, mental and social well-being of the workers in all occupations. Health awareness helps a worker:

a. To recognize the problem
b. Analyse the problem
c. To set a realistic goal

Health awareness comes mainly through: (a) Health education; (b) Training.

Health Education

The aims of health education are:

a. To encourage workers to be healthy
b. To know how to stay healthy through scientific information.

The steps of health education are:

a. To culture a health awareness
b. To culture a healthy lifestyle
c. To culture a group responsibility
d. To culture interest, attitude and value.

The practice of health education may be:

a. Individual health education
b. Group health education—through lecture, group discussion, panel discussion, symposium, workshop, role playing, demonstration, programme instruction and simulation exercise.

Training

Workers often experience work related health problems and do not realise that the problems are related to their work, particularly when an occupational disease, e.g. is in the early stage. Besides the other more obvious benefits of training are:

a. Skill development
b. Hazards recognition.

A comprehensive training programme in each workplace will help workers:

a. To recognize early stage/symptom of any potential occupational disease before they become permanent
b. To assess their work environment
c. To insist the management to make changes before hazardous condition can develop. Employee training should be designed to influence employees to comply with appropriate work practice. Such training should be conducted periodically for all employees.

Training should include the following areas:

a. Potential risk of overexposure
b. The importance of industrial hygiene, biological monitoring and notification of test results
c. Description of work practice control including personal protective equipment
d. Firs-aid and emergency training.

WORKPLACE HEALTH PROMOTION

Workplace health promotion is an approach towards health gain by encouragement of all aspects of positive health. It is a process of enabling people to increase their control over their own health and to improve it. It is a continuation towards well-being or optimal health. It also states that health promotion is not just the responsibility of the health sector, and goes beyond healthy lifestyles to well-being. Reduction of health care costs, disability claim and absenteeism and increase in productivity are the benefits of health promotion. Job satisfaction and work stability are also the results of health promotion. Workers' lifestyles may have a specific or a general impact on their health, safety and working capacity. Workers should be advised on avoiding lifestyles harmful to health as well as on adopting healthy and safe working practice.

For occupational health professionals the promotion of health in the workplace means conducting an occupational health practice which involves protecting and promoting workers' health through the prevention of occupational and other work-related injuries and diseases. For specialists in health promotion, it may mean introducing non-smoking policies, tackling alcohol and drug abuse, encouraging physical exercise and healthy diets, changing behavioral patterns, and undertaking activities to bring these things about.

Wellness may be defined as optimal health. Achieving it may involve improving physical and mental ability, developing reserve capacities and adaptability to changing circumstances, and reaching new heights in creative and other work. In a working environment, some of these factors can be evaluated quantitatively in relation to absenteeism, presenteeism, job satisfaction and work stability.

Absenteeism: Absenteeism or absence from work is defined as nonattendance when expected to work, for any reason at all, medical or other. *Presenteeism*: A loss of workplace productivity resulting from employee health problems and/or personal issues even though the employee is physically present at work.

COMMON WORK-RELATED (OCCUPATIONAL) DISEASES OR HAZARDS

Physical Hazards

1. *Heat*: The common physical hazard in most industries is heat. Heat stress is the amount of heat that is to be eliminated from human body to remain the body in thermal equilibrium and estimated as the metabolic heat load and heat loss or gain through the process of convection, conduction, radiation, and evaporation. The direct effects of heat exposure are burn, heat cramps, heat syncope, heat exhaustion, heat hyperpyrexia and heat stroke. The indirect effects of heat are decrease efficiency, increase fatigue, enhanced accident, decrease productivity, sleeplessness and fatigue (Fig. 1.5).

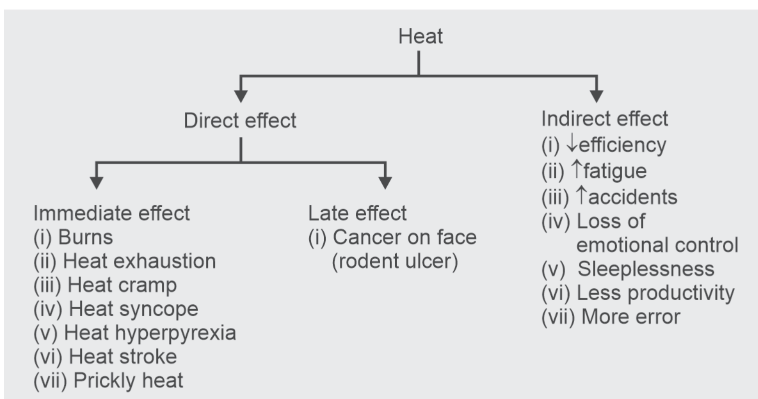

Fig. 1.5: Effect of heat

2. *Cold*: The effect of cold or subnormal atmospheric temperature may be of two types—generalized effect and local effect (Fig. 1.6). Generalized effects include coldness, hypothermia, shivering, weakness, desire to sleep, coma and death. Local effects depend upon the type of subnormal temperature like freezing and non-freezing temperature. Important hazards associated with freezing (ice forming) temperature are frostnip, frostbite (superficial, deep and freeze-thaw-refreeze injury). The example of non-freezing (non-ice forming) temperature is immersion foot or trench foot which is due to the

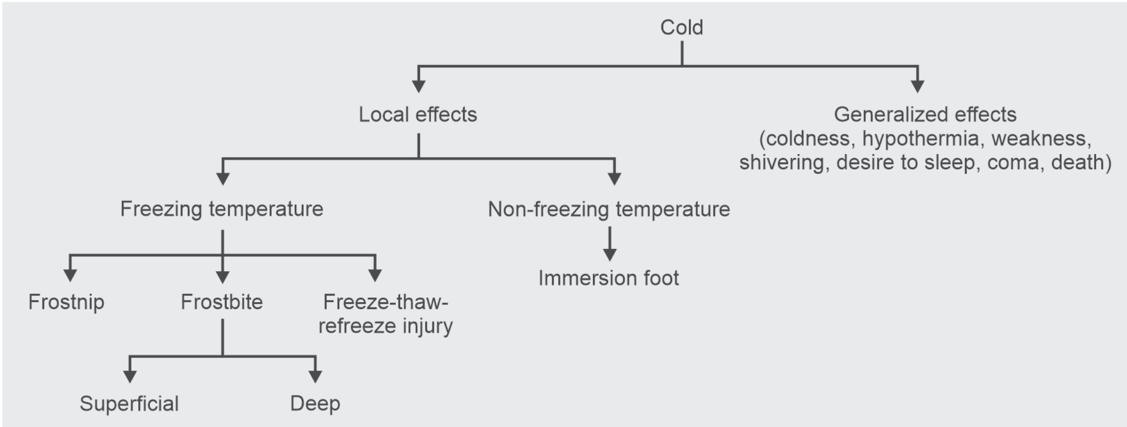

Fig. 1.6: Health effect of cold

immersion of feet in cold water during work usually in standing posture. There may be arterial thrombosis, and sudden cardiac death.

3. *Noise*: According to American National Standard Institute (ANSI), any unwanted sound is known as noise. The characteristics of noise are: Intensity or loudness measured by the unit 'dB' and frequency or sharpness measured by Hz or cycle per second. The effects of noise are two types:
   a. Auditory effect consists of noise induced temporary threshold shift (NITTS) and noise induce hearing loss (NIHL) or noise induced permanent threshold shift (NIPTS)
   b. Non auditory effects.

NITTS: The magnitude of a temporary threshold shift is proportional to the intensity and duration of the stimulus and the recovery. Recovery usually occurs within 16 hours but may take several days with higher intensities. This stage is reversible. The sign of developing permanent threshold shift is a complex process. Interaction between the level, nature and number of noise exposure, their duration and frequency and the individual susceptibility are all factors. Classically, there is high frequency rise in threshold with a characteristic notch at 4 kHz though the notch may center at 3–6 kHz. 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.

NIPTS/NIHL: When NITTS is repeated for years, it tends to lose the sensitivity to acoustic stimuli due to the destruction of structure in the inner ear (hair cells), thus leading to permanent hearing loss or NIHL. This is characterized by irreversible audiometric effects and pathological changes in the cochlea. The 4 kHz notch tends to deepen in audiogram and also insidiously widen, encompassing adjacent high frequencies. Once the audiometric changes encroach upon the speech frequencies (2–3 kHz in particular) the affected individual becomes aware of the diminished acuity in his or her hearing. Speech discrimination with background noise becomes difficult and the associated tinnitus (which are 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. The deafness due to noise in industry is called sensory neural (SN) or perceptive deafness. In the hearing conservation program, the noise-exposed people are audiometrically tested usually once in a year. The aims of such periodic examination are to:

a. Observe changes in hearing threshold.
b. Assess the ability to communicate.
c. Assess proper job placement.
d. Diagnose
e. Follow up.

The main characteristics of NIHL are:

a. It is permanent
b. It is subtle in earlier stage
c. It is not present in all the subjects who are exposed to noise. Hence, protection and periodic audiometric examination for all the noised exposed people are essential.

Non-auditory effect of noise:

a. It is a common observation that any sudden noise will produce 'startle' reaction in anyone and it cannot be voluntarily controlled. It can occur even during sleep. The characteristics of startle reaction are:
   i. The blood vessels contract
   ii. Blood pressure increases
   iii. Pupil dilates
   iv. Both voluntary and involuntary muscles become tense (contract). After a few minutes startle reaction becomes normal.
b. Noise stimulates adrenal glands to produce hormone catecholamines and this hormone is responsible for certain heart and circulatory diseases.
c. Nausea and vomiting.
d. Pulse and respiratory rates increase.
e. Noise increases physical and psychological stresses.
f. Sleeplessness.
g. Fatigue.
h. Irritability and socially undesirable behavior.
i. It reduces work efficiency and productivity.
j. Increases accident and error.
k. Tiredness and insomnia.
l. Peptic ulcer.
m. Sickness absenteeism and presenteeism.
n. Tendency to speak loudly.

4. *Vibration*: Vibration are two types: Hand-arm vibration (HAV) and whole-body vibration (WBV). The unit of vibration is Hz or cycle per second. Occupational vibration reaches the worker through different paths or transmission routes (Fig. 1.7). 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. 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 tools producing hand-arm vibration are 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. Hand-arm vibration may cause hand-arm vibration syndrome (HAVS) like white finger and whole-body vibration may cause motion sickness and low back pain.

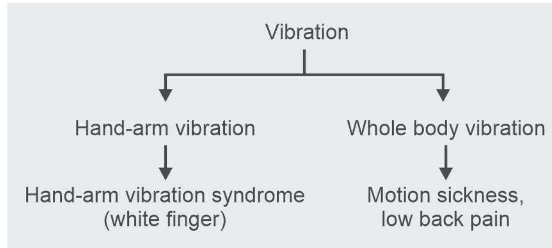

Fig. 1.7: Health hazard of vibration

5. *Non-ionizing radiation*: 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 non-ionizing radiation (ultraviolet radiation, visible rays, infrared radiation, radiofrequency waves) (Fig. 1.8). Non-ionizing 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).

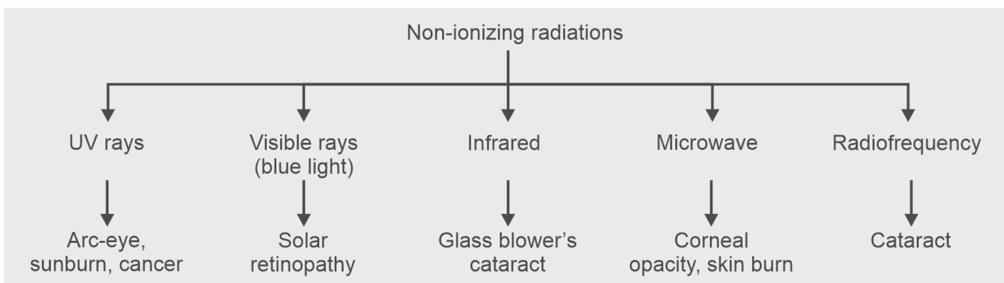

Fig. 1.8: Health hazard of non-ionizing radiations

Ultraviolet radiation (UV ray) may produce 'arc eye' or 'flash eye' and skin burn. 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'. Excessive exposure of the eyes to *infrared (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. 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 edema and opacification. Long-term exposure of radiofrequency may produce some adverse effects include nervous, neuroendocrine, reproductive, immune and sensory system effects, cataracts formation, decrease birth-weight, behavioral changes.

LASER: Laser is an acronym for light amplification by the stimulated emission of radiation. Lasers are sources of non-ionizing 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 (all the waves are in step), monochromaticity (one color 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. Some 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, 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. It may cause retinal damage. 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, e.g. could generate a fume and other small particles.

Classifications of laser are defined by the American National Standards Institute (ANSI) Z-136.1: 2007 and British Standard BS/EN/IEC 6082:2007:

Class 1: Non-hazardous lasers:

i. The output is so low the laser is inherently safe
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.

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.

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, it needed laser eye protection.

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.

6. *Ionizing radiation*: The radiation which can penetrate the human cells and deposits its energy within it is known as ionizing radiation (Fig. 1.9). The effects of ionizing radiation are:
   a. Somatic effects consist of:
      i. Immediate effects like ionizing burn, radiation sickness and acute radiation syndrome
      ii. Delayed effect like leukemia, carcinogenesis, fetal developmental abnormalities and shortening of life.

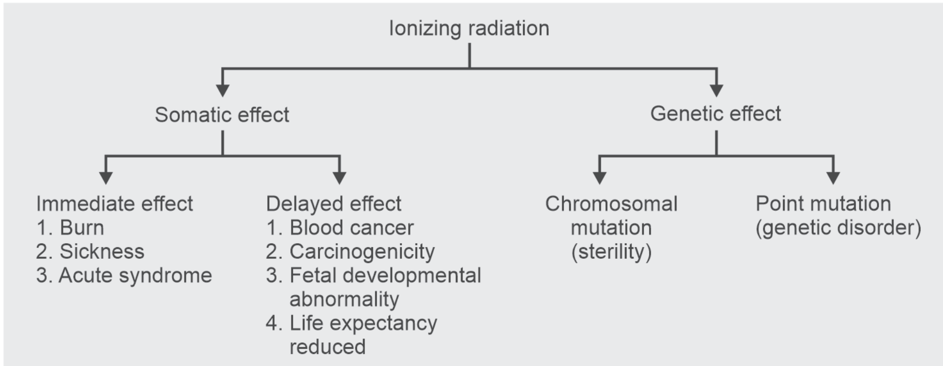

Fig. 1.9: Health effect of ionizing radiation

b. Genetic effects these are:
   i. Chromosomal mutation—causes sterility
   ii. Point mutation—affects gene.

7. *Illumination*: The workers may be exposed to the risk of poor illumination or excessive brightness. The acute effects of poor illumination are eye strain, headache, eye pain, lacrimation, congestion around the cornea, eye fatigue, etc. The chronic effect of poor illumination is 'miner's nystagmus'. Exposure to excessive brightness or 'glare' is associated with discomfort, annoyance and visual fatigue. Intense direct glare may also result in blurring of vision and lead to accident.
8. *Atmospheric pressure*: Atmospheric or environmental pressure may be high or low (Fig. 1.10).
   a. High barometric pressure: The increases of barometric pressure which have been experienced by the sea depth as great as 701 m (7000 kPa), some 70 times the normal barometric pressure present at the earth's surface (100 kPa). Acute effects may be two types:
      i. Compression barotrauma: Effects during work at high atmospheric pressure within the caisson or working at the bottom of sea or tunnelling work are middle and inner ear barotrauma, sinus pain, teethache, etc.
      ii. Decompression illness: Health effects occur when coming back to normal atmosphere from high barometric pressure. The effects are musculoskeletal pain,

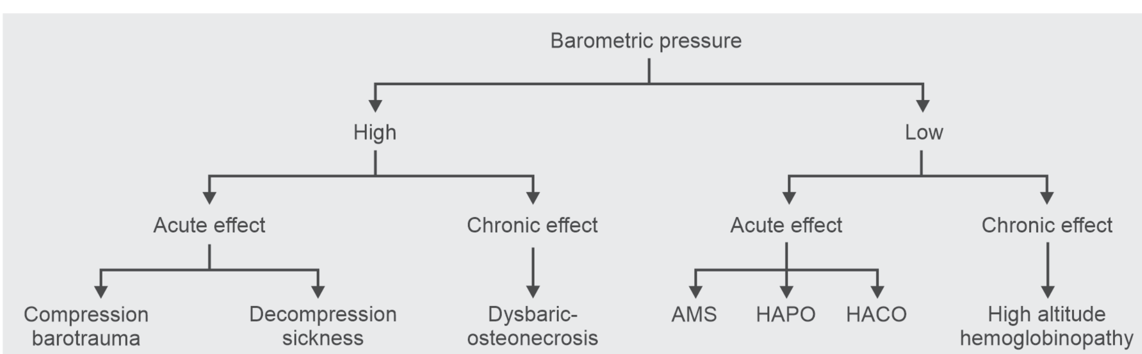

Fig. 1.10: Health hazard of barometric pressure

cardiopulmonary decompression, chest pain (pulmonary barotraumas), neurological decompression (psychosis), latency (loss of consciousness on coming to surface due to gas embolism) and Caison's disease. The chronic effects are hearing loss, neurological sequela and dysbaric osteonecrosis.

   b. Reduced barometric pressure: It is found in high altitude. The acute effects are acute mountain sickness (AMS), high altitude pulmonary edema (HAPO) and high-altitude cerebral edema (HACO). The example of chronic effect is high altitude hemoglobinopathy.

9. *Electromagnetic field (EMF)*: Electricity is generated and usually transmitted as alternate current (AC) at 50–60 cycles per second or 50–60 Hz. The magnetic field is generated around it is known as electromagnetic field. It is a very low intensity electromagnetic field. Electromagnetic field (EMF) is measured in tesla (T) or micro-tesla (mT), are determined by the ACGIH's ceiling value (at 60 Hz magnetic field) for guideline exposure limit is 1 mT. EMF is available around every electrical conductor, motor, electrical appliances, House wiring, current floor on the safety grounding system, utility line outside the home, computers, copy machine, power distribution facility and large motor, near large electric machine, electrical heating equipment, television, cellular phone and radar. Long-term health effects are:
   a. Cancer formation: Leukemia (blood cancer), brain cancer, breast cancer, lung cancer, malignant melanoma, cancer of prostate, testes and pituitary gland.
   b. Reproductive effects are:
      i. Parental exposure and brain tumor: If father is exposed then offspring may develop childhood brain tumor, neuroblastoma.
      ii. Pregnancy outcomes: Spontaneous abortion and congenital malformation.
   c. Neurobehavioral and neurogenerative effects: General neurasthenic effects—depressive symptoms, suicidal tendency, Alzheimer's disease. EMF has definite effect on pacemaker.
10. *Electricity*: The nature and severity of electrical injury depend on:
   a. The type and voltage of electrical current.
   b. The resistance of the body at the point of entry of electrical current.
   c. The pathway of the current flowing through the body.
   d. The duration of exposure.

   Alternate current (AC) produces greater damage than a direct current (DC) of the same amperage. The AC flow of 60 cycles (Hz) that will produce a tingling sensation, the threshold of perception, is from 1 to 2 milliamperes (mA) and for DC it is 5 mA.

   The effects of electricity:
   i. Immediate or life-threatening, causing cardiac or respiratory arrest which requires CPR.
   ii. Electric burn. It cannot be measured as fire burn is measured.
   iii. AC of 60 Hz at 110–220 volts traveling across the chest for <1 second is capable of inducing ventricular fibrillation at current as low as 60–80 mA.
   iv. Electrical injuries may visibly burn the tissue at the point of contact with the electrical source and the point of exit (or grounding point) as well as there is burn in-between. There is usually more tissue damage than is readily apparent.
   v. Current flow flows the path of blood vessels and muscles and may cause thrombosis and necrosis of tissue at sites remote from the apparent injury.

vi. There may be associated musculoskeletal injuries not only from the titanic contraction of muscles but also from the not uncommon fall of the electrically injured victim.
vii. Oliguria due to renal damage may be a sequela of electrical injury due to destruction of muscles and the production of myoglobin pigment in the urine.
viii. Severe pain.
ix. Dehydration.
x. Shock and death.

Chemical Hazards

Hazards due to gases:

1. *Carbon monoxide (CO) poisoning*: CO poisoning is the most common of all poisoning in industry today. It is known as 'silent killer' because it is colorless, odorless, tasteless and non-irritant, therefore, gives no warming of its presence under any circumstances. It is a chemical asphyxiant. The mechanisms of toxicity are:
   a. It has 200–300 times more affinity towards hemoglobin than oxygen and produces carboxyhemoglobin. Therefore, cells will not get oxygen for cellular respiration.
   b. It inhibits cytochrome A_3 oxidase (cytochrome-monoxide binding) at the cellular level.

   The acute effects are dizziness, nausea, weakness and collapse. Acute heavy exposure may result in loss of consciousness without warning, followed by coma, convulsion and death. The classical cherry pink color of the skin is a rare sign but venous blood frequently looks arterial. Patient recovering from CO poisoning may suffer neurological sequelae including tremor, personality changes, memory impairment, loss of visual acuity, inability to concentrate and parkinsonian features. Moist oxygen inhalation or in some cases hyperbaric oxygen therapy is the treatment of choice.
2. *Hydrogen cyanide (HCN) poisoning*: Hydrogen cyanide is a chemical asphyxiant. Hydrogen cyanide and its sodium, potassium and calcium salts manifest their toxicity by way of a common mechanism, namely, inhibition of the respiratory enzyme cytochrome oxidase. The inhibition causes cellular anoxia and, if a large enough dose is received, results in death. Victims of acute poisoning present with headache, dizziness and vertigo. There may be agitation and confusion, with nausea and vomiting. Acute cyanide poisoning induces sustained seizures, endogenous catecholamine release and cardiovascular shock. Skin contact with solution of cyanide salt can cause itching, discoloration or corrosion. Cyanide salt aerosols can cause upper respiratory irritation. Enlargement of the thyroid gland has also been reported. An increased occurrence of the objective symptoms of headache, changes in taste and smell, irritation of the throat, vomiting, breathlessness on effort, lacrimation, abdominal colic, precordial pain and nervous instability has been noted among workers having long-term occupational exposure to low concentration of HCN.
3. *Hydrogen sulfide*: Hydrogen sulfide is acute acting toxic substance and a leading cause of sudden death in the workplace. It is a chemical asphyxiant. Brief exposure at high concentrations cause unconsciousness, respiratory paralysis and death. Eye irritation, cough, sore throat, vague gastrointestinal symptoms and photophobia may occur with low-level exposure, with overwhelming exposure, collapse and respiratory arrest can develop within minutes.
4. *Ammonia*: Ammonia is a colorless gas and is one of the most widely used industrial chemicals ranking forth in volume of production after sulfuric acid, lime and oxygen.

Ammonia vapor is a severe irritant to the eyes, respiratory tract and skin. It may cause corneal irritation, breathlessness, bronchospasm, chest pain and pulmonary edema that may be fatal. Production of pink frothy sputum often occurs. Complication can include bronchitis or pneumonia. Liquid anhydrous ammonia in contact with the eyes may cause serious eye injury or blindness, on the skin, it causes first and second-degree burns that often are severe and if extensive, may be fatal.

5. *Chlorine*: Chlorine gas is a severe irritant of the eyes, mucous membrane, skin and respiratory system. A major accidental exposure to unmeasured but high concentration for a brief period caused burning of the eyes with lacrimation, burning of nose and mouth with rhinorrhea, cough, choking sensation and substernal pain. These symptoms frequently were accompanied by nausea, vomiting, headache, dizziness and sometime syncope.
6. *Sulfur dioxide*: It is a severe irritant for the eyes, mucous membrane and skin. Exposure to concentration 10 to 50 ppm for 5 to 15 minutes causes irritation of eyes, nose and throat, rhinorrhoea, choking, cough, reflex bronchoconstriction. The liquid form may cause corneal and skin burns.
7. *Benzene:* Inhalation of high concentration of benzene (3000–7500 ppm) may cause a state of excitation and euphoria (benzol jag) followed by drowsiness, fatigue, vertigo, nausea and vomiting, convulsion followed by paralysis, loss of consciousness and death from respiratory failure. Inhalation of small amount of benzene over a long period has caused blood dyscrasias, including aplastic anemia, leukemia (blood cancer), thrombocytopenia. Additional signs and symptoms of chronic toxicity may include headache, dizziness, fatigue, loss of appetite, irritability, nervousness, nose bleeding and other hemorrhagic manifestation.
8. *Phosgene*: In most fatal cases, pulmonary edema reaches a maximum in 12 hours followed by death in 24–48 hours. With moderate exposure the presenting symptoms often are a dryness or a burning sensation in the throat, vomiting, pain in the chest, and dyspnea. Delayed onset of pulmonary edema is characterized by cough, abundant 'foamy' sputum, progressive dyspnea, and severe cyanosis. Pulmonary edema may progress to pneumonia, and cardiac failure may intervene. Exposure to high concentrations may also cause severe conjunctivitis and subsequent turbidity of the cornea of eye. Chronic bronchitis and emphysema have been reported as a consequence of acute exposure. Because of the often-delayed onset of symptom following potentially lethal exposures of phosgene, education of employees and medical observation following suspected overexposure are extremely important. Medical examinations, including pulmonary function tests, are necessary to evaluate recovery from overexposure and to detect chronic lung changes.
9. *Methyl isocyanate*: It is a widely used chemical, but it gained worldwide notoriety after its disastrous release from the Union Carbide pesticide plant in Bhopal, India in 1984. It was being used in the chemical synthesis of carbaryl pesticides, and had been stored in liquid form in two steel tanks. The cause of the incident is still not known, but at 00.30 on December 3, 1984 an exothermic reaction took place in one of the storage tanks, resulting in the escape over a few hours of 40 tons of methyl isocyanate. A dense cloud of methyl isocyanate flowed over an area of the city about 40 km^2 at a time when there was a temperature inversion and a light wind. It is considered that >10000 people were killed and hundreds of thousands injured in the disaster. Methyl isocyanate is an intense

lacrimator and irritates eyes, mucous membrane and skin. The liquid in contact with the eye may cause permanent damage. It may cause pulmonary irritation and sensitization. Exposure of man to high concentration can cause cough, dyspnea, increase secretions and chest pain.

10. *Carbon dioxide*: It is a colorless and odorless gas but when breathed from an anesthetic machine it has a faintly acid smell. The effects of breathing carbon dioxide are very characteristic. 2–3% CO_2 in air will pass unnoticed at rest, but tidal volume is increased by 30% and the minute volume by 5%; on exertion there may be marked shortness of breath. At 3% CO_2 in air, breathing becomes noticeably deeper and more frequent at rest, with the effect becoming more marked until at 5% CO_2 in air there is severe dyspnea, with limits exposure for most people. Subjects complain of headache and are sweaty and have a bounding pulse. At 10% CO_2 in air, respiratory distress develops rapidly with loss of consciousness in 10–15 minutes, even though the oxygen concentration is reduced to 19% only. Exposure to carbon dioxide levels above 15% are intolerable and rapid loss of consciousness ensues after a few breaths of a mixture of 30% carbon dioxide in air. Even at this level, the oxygen concentration is 15%, not enough to cause loss of consciousness from hypoxia, but death will occur if the carbon dioxide level is maintained. Thus, it is important to note that monitoring oxygen in air is an inadequate guide to the carbon dioxide hazard; carbon dioxide should be directly measured. A working or living area should be immediately evacuated when concentration exceed 1.5% by volume (the occupational short-term exposure limit value). Carbon dioxide absorbs the solar energy and prevents the energy from radiating back into the space. As a result, the atmosphere gets heated up. This is commonly known as the greenhouse effect that causes global warming. Normally, the heat from the sun is radiated to the earth and this heat is again radiated back into the space.

Hazards due to Dusts

The term 'pneumoconiosis' is defined by the Fourth International Conference of experts in 1971 as 'the accumulation of dust in the lungs and the tissue reaction to its presence'.

Diseases	*Causes*	*Source of dust*
1. Baritosis	Barium sulfate	Mines of barium sulfate
2. Siderosis	Iron oxide	Welding
3. Stannosis	Tin oxide	Smelting
4. Kaolinosis	Hydrated aluminum silicate	China clay
5. Aluminosis	Stamped aluminum	Paints
6. Anthracosis/CWP	Coal dust	Coal mining
7. Silicosis	Free silica dust	Mining, quarrying, etc.
8. Talcosis	Hydrated magnesium silicate	Rubber industry
9. Berylliosis	Beryllium compounds	Atomic reactor, aero engineering
10. Asbestosis	Asbestos fiber	Mining, brake lining, asbestos-cement product

Damage to the lungs caused by dust or fumes or noxious substances inhaled by workers in certain specific occupations is known as *occupational lung disease (OLD)*. All pneumoconiosis(s) are OLD but all OLD are not pneumoconiosis. Pneumoconiosis is defined as "the accumulation

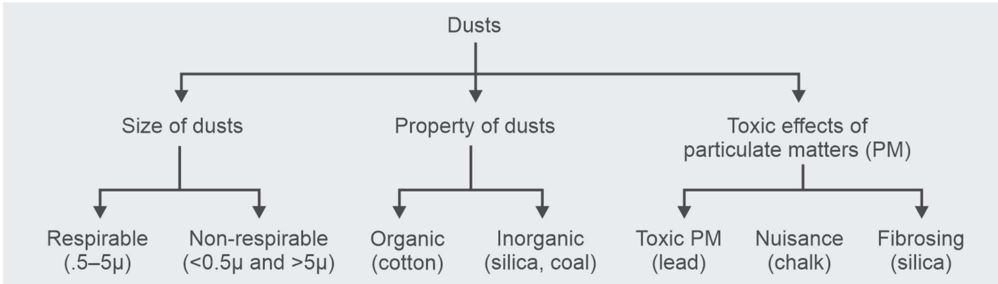

Fig. 1.11: Types of dusts

of dust in the lungs and the tissue reactions to its presence". The clinical and pathological consequences that result from the inhalation of dust are variable mainly according to (Fig. 1.11):

1. The properties of the dust
2. The intensity and the duration of the exposure
3. Host susceptibility.

Dust constitutes one of the most common respiratory hazards. Dusts may be *respirable* (0.5–5 microns in diameter) or *non-respirable* (below 0.5 micron and above 5 micron), or dusts may be *organic* like cotton, tobacco or sugar cain fibre, or *inorganic/mineral dusts* like silica, coal dust, asbestos, or, dusts may be:

1. *Toxic particulate matter:* When these dusts are inhaled, they will not be restricted/retained in the lungs but they will be absorbed in the system for toxic systemic effects, e.g. lead, cadmium, chromium dusts.
2. *Fibrosing dusts*: When these dusts are inhaled, they will be retained in the lungs and produce fibrosis of the lung tissue, e.g. free silica, coal dusts and asbestos fiber.
3. *Nuisance dusts*: In contrast to fibrogenic dusts which cause scar tissue in lungs when inhaled in excess amounts, so-called 'nuisance' dust has a long history little adverse effect on lungs and do not produce significant organic disease or toxic effect when exposures are kept under reasonable control. The nuisance dusts have also been called (biologically) 'inert' dusts, but the later term is inappropriate to the extent that there is no dust which does not evoke some cellular response in the lung when inhaled in sufficient amount. Example of nuisance dust is chalk dust.

Some Common Pneumoconiosis

1. *Silicosis*: Silicosis is a fibrotic disease of the lung caused by the inhalation of, retention of and pulmonary reaction to crystalline silica. The three important crystalline form of silica are quartz, tridymite, and cristobalite. These forms are also called free silica to distinguish them from the silicates. There is mounting evidence that freshly fractured silica may be more toxic than aged silica, perhaps related to reactive radical groups on the cleavage planes of freshly fractured silica. This may offer a pathogenic explanation for the observation of cases of advanced disease in both sandblasters and rock drillers where exposure to recently fractured silica is particularly intense.

   Types of silicosis: Acute silicosis, chronic silicosis and accelerated silicosis.

   Complications of Silicosis:

   a. Tuberculosis and other infection: When active tuberculosis is common in the community, many patients with silicosis develop combined diseases with an extremely poor prognosis and called 'silicotuberculosis'.

b. Progressive massive fibrosis (PMF).
c. Pulmonary heart disease (cor pulmonale).
d. Rheumatoid syndrome: With high titers of circulating rheumatoid factor without overt arthropathy.
e. Nephropathy: Kidney damage with proteinuria and systemic hypertension.
f. Carcinogenicity: It can produce cancer.

Prevention: There is no specific therapy for silicosis. Prevention remains the cornerstone of eliminating this occupational lung disease. Prevention of silicosis depends on preventing exposure to respiratory silica dust which presupposes that the risk has been recognized; regrettably this is not always the case. Most countries now recognize a silica dust standard aimed at preventing exposure of the workforce to more than permissible level of quartz. Ideally, the use of quart should be avoided if a safer substitute material is available. There is some evidence that inhalation of quartz mixed with other dusts leads to a less severe and progressive disease. The education of workers and employers regarding the hazards of silica dust exposure and measures to control exposure is important. Improved ventilation and local exhaust, process enclosure, wet technique, personal protection including the proper selection of respirators and where possible, industrial substitution of agents less hazardous than silica reduces exposure. Silicosis is a reportable disease. Workers exposed to a risk of silicosis should be offered chest radiography at regular intervals, probably at least once in every 3 years, in order that the earliest signs of disease are detected and the workers protected from further exposure. If silicosis is recognized in a worker, limited future significant exposure is advisable. Unfortunately, the disease may progress even without further silica exposure.

2. *Asbestosis and asbestos fiber*: The mineral known as asbestos, a name derived from the Greek word for unquenchable, belongs to one of two mineralogical groups, the serpentines and amphiboles. Asbestos is a collective term for some of the metamorphic, fibrous, mineral silicate. Chrysotile or white asbestos is the only asbestos that is a serpentine; it is a magnesium silicate with a curly configuration and is able to shear into many smaller fibrils. It is a soft and silky fiber, has high tensile strength, high flexibility, good spin ability and high resistance to alkali. It undergoes thermal decomposition at about 675°C. All the other forms of asbestos are amphiboles. This group of minerals consists of a large number of structurally similar but chemically different chemicals. It is straight and needle like fiber. It is generally more brittle and appears to be dustier. It has greater resistance to acid and heat. Asbestos has a long history. It was first used in Finland about 2500 BC to strengthen clay pot. In Greek it was used as 'magic cloth' which did not burn in fire. Asbestos is derived from the Greek word meaning 'inextinguishable' or 'indestructible'. They have different physical and chemical properties, but share a fibrous form or habit. Mineralogists have generally taken a particle with a length-to-breath ratio (aspect ratio) of 10 : 1 or more to be a fiber (as per the Indian Factories Act, 1948, TLV of asbestos fiber is for fiber >5 μm in length and <5 μm in breath with length to breath ratio equal to or >3 : 1). Prevention and screening of asbestos related hazards are: Screening of asbestos-exposed populations can be carried out for practical and scientific purposes. There are four goals of screening: to identify high-risk groups, to target preventive actions, to discover occupational diseases, to develop improved tools for treatment, rehabilitation and prevention. Of the three routes of exposure (dermal, ingestion and inhalation) to asbestos

fibers, the inhalation route is responsible for most if not all of the serious health effects (Fig. 1.12). Effects of asbestos fibers are:

a. *Asbestosis*: Asbestosis is defined as diffuse interstitial fibrosis of the lung as a consequence of exposure to asbestos fiber. Symptoms of asbestosis include dyspnea and cough. Common findings are inspiratory basilar crackles and less commonly, clubbing of the fingers. Functional disturbances can include gas exchange abnormalities, a restrictive pattern and obstructive features due to small airways disease. Asbestosis is generally associated with relatively high exposure levels with radiological signs of parenchymal fibrosis. Diagnosis of asbestosis is done by sputum examination for asbestos body, X-ray chest, pulmonary function test, HRCT, lung biopsy, etc.
b. *Malignant mesothelioma*: Malignant mesothelioma effecting any serosal membrane (pleura and peritoneum) may be induced by asbestos inhalation. Therefor these can produce pleural or peritoneal malignant mesothelioma. Inhaled asbestos fibers mainly blue asbestos (crocidolite) can penetrate the structure in between lung and abdomen and reach peritoneum to develop peritoneal mesothelioma. Mesothelioma is frequently present with pleural effusion, dyspnea, and chest pain.
c. *Lung cancer*: Clinical signs and symptoms of asbestos-related cancer do not differ from those of lung cancer of other causes. The presence of asbestosis is an indicator of high exposure. Asbestosis may also contribute some additional risk of lung cancer beyond that conferred by asbestos exposure alone. The interaction between smoking, asbestos exposure and lung cancer is the best-studied example of the influence of smoking on occupational disease. Studies of asbestos workers indicate a substantially increased risk of lung cancer, and lung cancers occur at greater than additive rates (usually synergistic or multiplication effect) in person exposed to both asbestos and cigarette smoke.
d. *Skin corn or asbestos wart*: Workers with a great deal of unprotected skin contact with asbestos (from handling this material with their bare hands) would sometimes develop nodular skin lesions termed asbestos corns or warts. When asbestos fibers get into the skin as a result of abrasion, they provoke a low-grade inflammatory reaction with hyperkeratosis which eventually swells out, taking the fibers with it and leaving no scar. These benign lesions are a foreign body reaction from fibers embedded in the skin.

3. *Coal workers' pneumoconiosis (CWP)*: Coal is not a pure mineral. It is formed by the accumulation of vegetable matter covered by sedimentary rock to seal it from air and

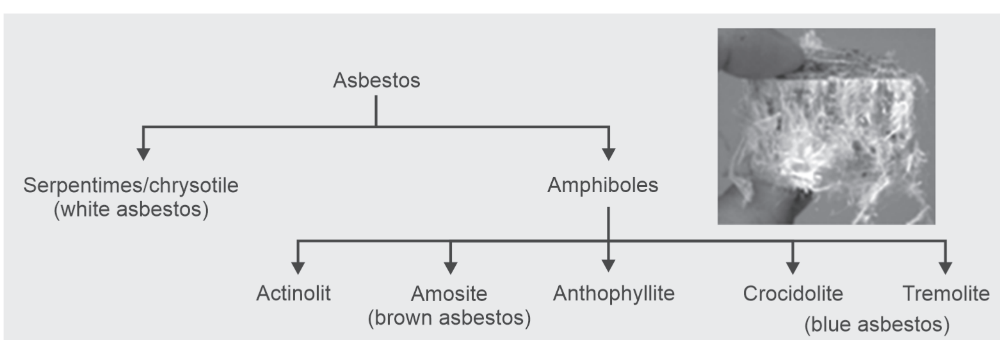

Fig. 1.12: Types of asbestos

subjected to pressure and temperature. It causes the physical and chemical property to change. Coal is comprised of moisture, pure coal (carbon) and mineral matter (mainly sulfur). Coal workers' pneumoconiosis is virtually confined to underground coal-miners, though it may occur in any place where a worker is exposed to high levels of coal dust in poorly ventilated conditions. Thus, it has been described in coal trimmers, loading coal in the holds of ships, and in men and women sorting coal on surface screens. Some other sources of coal/carbon are carbon black and coke.

Effect of Coal Dust Inhalation

a. *Coal-workers' pneumoconiosis (CWP)*: CWP results from the inhalation and deposition of coal mine dust and the lung's reaction to its presence. Typically, CWP is described as simple pneumoconiosis and progressive massive fibrosis (PMF). Simple coal-workers' pneumoconiosis is the early stage of CWP and it is reversible. The miners with simple CWP have no symptoms or sign attributable to the disease. As the disease progresses to PMF, the combination of fibrous masses and associated emphysema leads to increasing breathlessness which is initially on exertion. These lesions may also cavitate and the worker may expectorate an ink-like fluid known as melanoptysis. This stage may progress further till death even though the workers are withdrawn from the exposure. The X-ray findings at the last stage of the disease looks like a typical angel's wings or bat's wings.
b. *Caplan's syndrome*: Caplan's syndrome is a rare condition occurring in dust-exposed workers with rheumatoid diathesis (with or without clinical arthritis).

HEALTH HAZARDS OF METALS AND THEIR COMPOUNDS

'Metal' is a general term referring to elements with a typical lustrous appearance, which the good conductors of heat and electricity and which take part in chemical reactions as positive ions (cations). The 'metal' refers to those elements in the periodic table that have specific characteristics. They are good conductors of heat and electricity; they are bright, shiny and malleable; and they produce a ringing sound when struck. Metals lose electrons to form cations, and the resulting electropositivity gives it an affinity to bind with sulfur, chlorine and other non-metals to form compounds. Alloys are formed from a homogenous mix of metals with other elements. Examples are brass (copper and zinc), bronze (copper and tin), and steel (iron and carbon). These often have physical properties that are different from their components. The term 'superalloys' which has been used for alloys with excellent mechanical strength, and the ability to resist deformation at high temperature. Many superalloys are based on nickel or cobalt. Metaloids are elements, such as antimony, arsenic, polonium and tellurium. They have properties in between those of metals and non-metals.

1. *Lead and its compounds*: Lead is a bluish-grey metal with the symbol Pb, atomic number 82 and atomic weight 207.2. Due to high specific gravity (sg 11.34), ease to casting and fabrication, low melting point (327.4°C), resistance to corrosion and opacity to X-rays, it has had a wide range of uses, such as soldering, printing type, ammunition, brass, piping and shielding against X-rays. Inorganic lead has been used in paint pigments, glass and ceramics. Among organic lead compounds, tetraethyl lead was used extensively as an anti-knocking agent in gasoline. Lead oxide is the most toxic and lead chromate is the least toxic inorganic lead. Tetraethyl lead is the most toxic and tetramethyl lead is the least toxic organic lead. Inhalation is the main route of entry for inorganic lead. Ingestion is often the

principal route of entry, particularly where good standards of personal hygiene are not ingested upon. Lead workers who are allowed to eat or drink in their workplace without first careful washing their hands are unnecessarily increasing the risk they run as are those who smoke at work. Inorganic lead compounds are not significantly absorbed through the skin but organic ones may be. Lead is mainly excreted through the kidneys (75%). Other routes include bile, sweat and the keratinized tissue.

a. *Hazards of inorganic lead*: Classically, the patient with lead poisoning presents with a history of abdominal pain, colic and constipation. Lead is nephrotoxic, cardiotoxic, and central nervous system symptoms including irritability, restlessness, insomnia and other sleep disturbances, fatigue, vertigo, headache, poor memory, tremor, depression, symptoms can progress to drowsiness, stupor, hallucinations, delirium, convulsion and coma. A typical poisoning in adults with lead, the major symptoms and signs are abdominal pain, anemia and muscle weakness (peripheral neuropathy). There may be 'wrist drop' and 'footdrop'. It has reproductive toxicity to both sexes. Another well-known manifestation of lead exposure is a bluish line on the gums. This is so-called Burtonian lead line.
b. *Organic lead*: Mild intoxication is characterized by nervous instability. Common symptoms include insomnia, troubled dreams, emotional instability, and heightened and erratic physical activity. In severe cases, the prodromal stage develops into violent symptoms, sometimes gradually but most often suddenly. These consist of delusions, hallucinations, and hyperactivity, with constant moving, loud shouting, and laughing. The hyperactivity may go on continually for several days before it ends up in coma and death or, alternatively, in recovery. Organic lead can reduce IQ. The biochemical tests include blood lead, urine lead, blood ZPP estimations, etc.

2. *Mercury poisoning*: Mercury (Hg) is a metal that is liquid at room temperature under normal pressure, with a high specific gravity (sg 13.59 at 12°C), high expansion coefficient and large surface tension. Many metals can be mixed with mercury to form amalgams. The various use of mercury includes thermometers, electrical appliances (lamps, switches, and batteries), extracting gold, and the restoration of teeth (dental amalgam). Mercury is present as inorganic and organic compounds. Mercury and its compounds are absorbed in all three routes. Mercury is excreted in the urine and feces and may also be found in sweat, saliva, and breast milk. Acute poisoning usually results from the accidental or deliberate ingestion of mercury compounds and the toxicity include vomiting and abdominal pain may develop and watery diarrhea, hematemesis and then shock cause death. The classic symptoms of chronic mercury poisoning are tremors, gingivitis and erethism (Greek *erethismos* means irritate). The earliest findings are usually gingivitis, hypersalivation (mercurial ptyalism) and an unpleasant, bitter, metallic taste in the mouth. Gingivitis is most marked in those with poor oral hygiene and may be severe enough to cause loosening or loss of teeth. There may be glossitis, pharyngitis and gastritis, tremors and change of handwriting. There may be mercuria lentis and Minamata disease. Minamata disease was first noted at the end of 1953 when an unusual neurological disorder began to affect the villagers living on Minamata Bay on the Southern Coast of the most southernly of the main island of Japan. It was commonly referred to as "kibyo", that is, the mystery illness. It was an outbreak of chronic methyl mercury poisoning due to the ingestion of contaminated fish from the Minamata Bay in Japan.
3. *Arsenic poisoning*: Today, arsenic is employed in metallurgy, agriculture (as a cotton desiccant), animal husbandry (for sheep dip and growth promotion of swine and poultry)

and forestry (as a silvicide). These industrial applications account for the majority of the uses of inorganic and organic arsenic compounds. Arsine (AsH_3) is a colorless gas which is odorless at low concentration but smells of garlic at high concentration. It is formed whenever arsenic comes in contact with nascent pure hydrogen: $As + 3H = AsH_3$. Toxicity may be acute when there is ingestion of inorganic arsenic. It causes acute diarrhea, shock and death. The chronic toxicity usually due to organic arsenic are like hyperpigmentation, eczematous dermatitis, ulceration of skin, skin cancer, loss of vibration sense, difficulty in walking, pain and burning sensation of calf muscles, hepatic cirrhosis, anemia, painless perforation of nasal septa, bronchitis, pneumonitis, broad wide strip type of line under the nailbed called 'mess line'. Arsine gas is the gaseous form of arsenic which is very toxic. It is the commonest cause of occupational jaundice.

4. *Chromium poisoning*: Chromium and its compounds protect against rust, provide color, conserve energy as components of catalysts, prevent decay, resist soiling, and are used in hundreds of other ways, such as in the manufacturing of foam rubber. Chromium is a very complex and versatile metal whose harmful effects are heavily dependent on valence. In addition to metallic chromium (valence 0), other valences found in industry are the +2, +3, +4, +5, and +6 combining states. Trivalent and hexavalent chromium are the only compounds known to be significantly associated with human disease. Hexavalent chromium is most toxic because it can be absorbed through the skin but trivalent cannot. It may cause contact dermatitis, chrome ulcer, perforation of nasal septa, asthma, pneumonitis, conjunctivitis and lungs cancer.
5. *Nickel poisoning*: Nickel is used for the production of stainless steel, nickel alloys, nickel cast iron, alkaline batteries (nickel cadmium batteries), pigments and it is used for electroplating and electroforming. It may cause itching called nickel itch, lungs cancer, cancer of nasal septa, etc. Nickel carbonyl is one of the most toxic within the all toxic metal fumes in industries. It causes lungs alveolar damage and enzyme system damage very quickly.
6. *Cobalt poisoning*: Cobalt (Co) is a silvery-gray, shiny, hard metal. The main route of absorption during occupational exposure is through inhalation. Refined cobalt is available to the industrial market primarily as broken or cut cathodes and to a small extent as electrolytic coarse powder. The toxicities of cobalt are—interstitial fibrosis called hard-metal pneumoconiosis, occupational asthma, cardiomyopathy, allergic dermatitis, affect thyroid gland (increase T4). The international Agency for Research on Cancer (IARC) concluded that cobalt and its compounds are possibly carcinogenic to human.
7. *Aluminum toxicity*: Aluminum is produced from ores (primary production) and scrap (secondary production). Aluminum is a light metal, which is used more widely throughout industry than any other non-ferrous metal. Its major applications are in transportation, building and construction, packaging and electrical equipment. A wide spectrum of respiratory symptoms and diseases, including interstitial fibrosis, pneumoconiosis, occupational asthma, granulomatous disease, chronic obstructive pulmonary disease, emphysema, desquamative interstitial pneumonia, and pulmonary alveolar proteinosis has been described to occupational activities involving exposure to aluminum dust or fumes. In the handling of minerals which contain aluminum (bauxite and corundum), exposure to aluminum accompanied by exposure to silica. This combined exposure may lead to the development of fibrosis of the lung (Shaver's disease). China-clay workers exposing to hydrated aluminum silicate may suffer from the disease known as Kaolinosis. It may cause Alzheimer's disease consists of deterioration of mental function

involving memory, judgement, abstract thinking as well as changes in personality and behavior.

8. *Beryllium poisoning*: Beryllium has chemical and physical properties that make it desirable for high technology applications. The application of beryllium and its alloys are widespread, including use in aerospace, the automotive industries, dental alloys, electronics, computers, nuclear weapons and telecommunications. Exposure to beryllium dust and fumes can cause an immune hypersensitivity reaction, dermatitis and lung diseases, including acute berylliosis, chronic beryllium disease and lung cancer, most hazardous exposures to beryllium come from dust or fume generated by disturbing the surface of a beryllium product through machining and polishing processes.

Occupational Carcinoma

1. *Skin carcinoma*: The causes may be: Coal tars and pitches, X-ray, dyes, certain oils, sunrays, arsenic and arsenic compounds, benzo(a)anthracene, benzo(a)pyrene.
2. *Lungs carcinoma*: The causes may be: Asbestos fiber, free silica dust, nickel, chromium, coal tar, radioactive substances, cigarette smoking, petroleum products, beryllium, cadmium, formaldehyde.
3. *Bladder carcinoma*: The causes may be: Beta-naphthylamine, benzene, para-aminodiphenyl, arsenic.
4. *Blood cancer*: The causes may be: Benzene, ionizing radiation, phenoxy acid herbicides and dioxins, some chlorinated organic compounds.

Occupational Dermatitis

- *Physical agents*, e.g.
  a. Heat
  b. Cold
  c. Moisture
  d. Friction
  e. Pressure
  f. X-ray.
- *Chemical agents*, e.g.
  a. Acid
  b. Alkali
  c. Dyes
  d. Solvent
  e. Grease, tar and pitch
  f. Chlorinated phenols.
- *Biological agents*, e.g.
  a. Living agents:
    i. Viruses
    ii. Bacteria
    iii. Fungi
    iv. Parasites.
  b. Plant products
    i. Leaves
    ii. Vegetables

iii. Fruits
iv. Flowers
v. Vegetable dust.

EXPOSURE OF TOXIC AGENTS IN FIRE HAZARD

The main threats to life in fires are toxic gases, heat and oxygen deficiency. The temperature in a room in a house fire can easily reach 500–1000°C, and as many as 400 toxic compounds can be demonstrated in the smoke. The principal toxic constituents of smoke are soot, carbon monoxide, carbon dioxide, nitrogen oxides, hydrogen cyanide, hydrogen chloride, sulfur dioxide, hydrogen fluoride, hydrogen sulfide, isocyanates, acrolein, benzene, phenol, formaldehyde and a range of chlorinated hydrocarbons. Carbon monoxide is an important factor in 50–80% of all fire fatalities. The role of hydrogen cyanide is less clear, although it is formed in many fires especially those involving wool, silk, nylon and polyurethane products. The chemistry involved in combustion reactions is extremely complex, even in the simplest example, such as the burning of jet of natural gas or methane in oxygen or air, when numerous reactive species, including free radicals (such as OH, O and CH_3) are produced which eventually form mainly carbon dioxide, water and large particles of carbon (soot).

$$CH_4 + 2O_2 = CO_2 + 2H_2O$$

Smoke comprises a mixture of gases, liquid and solid particles which arises as combustion and pyrolysis products. Predicting the products of combustion (oxidative degeneration) and pyrolysis (thermal decomposition) of burning materials and hence determining in retrospect the effects of exposure of fireman or other victims to the constituents of a smoke plume is not straightforward. Some general principles can be considered. Materials consumed by fires contain carbon, hydrogen and oxygen as their main elements, and hence the bulk of all combustion products will consist of compounds formed from these, e.g. carbon monoxide and carbon dioxide. The next important elements are halogens (mostly chlorine) and nitrogen, with smaller amounts of elements such as sulfur and phosphorus. Almost all the inorganic anions are released as the irritant acid gases, hydrogen chloride and hydrogen fluoride, if fluorine is present. For the nitrogen present in well-ventilated fires oxides of nitrogen are released, but in large fires in buildings, where the ventilation is poorer, a larger proportion of the nitrogen is released as hydrogen cyanide. Organic compounds may form a number of partially decomposed products, e.g. formaldehyde, acrolein, and crotonaldehyde and possibly free radicals that add to the irritancy of the smoke. Other compounds, such as decomposition products from isocyanates, styrene and phenol, may also be important. Fires in warehouses storing chemicals will potentially release a cocktail of irritants and toxic substances which may greatly add to the hazard of the fire plume.

Polyvinyl chloride (PVC) is the most widely used plastic and fires involving large amounts of this material at storage sites and recycling installations are not uncommon and attract notoriety because they are difficult for the fire services to put out while they generate toxic plumes. At over 300ºC, PVC decomposes to form hydrogen chloride and carbon monoxide together with small amounts of about 50 various hydrocarbons. About 50% of the polymer’s weight comes off as hydrogen chloride, and the irritancy of the plume is added to by the presence of acrolein, ammonia, sulfur dioxide, nitrogen dioxide, aldehydes and particles. Phosgene is produced only in rare circumstances. There is no doubt that dioxins are formed whenever chlorinated plastics are burned: in a large-scale plastics fire typically involving 500–1000 tons of PVC as much as 1–2 kg of dioxins could be produced.

When there is fire in a naturally pressurized oil well, the plume contained carbon dioxide, carbon monoxide, sulfur dioxide and particles, including soot. The major health concern revolved around the particle fallout that contained elemental carbon, rock/soil particles, metal oxides, silicates, vanadium, nickel and polycyclic aromatic hydrocarbons besides smoke rained oil drops.

The products released the combustion of wood are more difficult to measure mainly because the tendency for some to incinerate more rapidly at high temperature. The occupational health of forest firefighters and the workers who manage fire are at great risks. The number of possible products of vegetation combustion is almost limitless and every fuel and condition of vegetation burning products a unique pattern. It is possible and practical to select a limit number of by-products that are most likely to be involved in the acute toxicity of smoke. Two of the most important are formaldehyde and acrolein. Both appear to occur in all smoke. The toxicology of both is well studied, they are powerful respiratory irritants. There seems to be a reasonable prospect that free radical precursors with half-lives in the tens of the minutes are produced when cellulosic materials burn. If so, they will reach the respiratory pulmonary cells. Ozone is not produced in the fire, but the various hydrocarbons of smoke are substrates for reactions that eventually produce ozone, and that production may continue for miles down-plume. The effects of best-known component are particulate matters.

Air emissions from open tyre fires have been shown to be more toxic (e.g. mutagenic) than those of a combustor, regardless of the fuel. Open tire fire emissions include 'criteria' pollutants, such as particulates, carbon monoxide (CO), sulfur dioxides (SO_2), oxides of nitrogen (NOx), and volatile organic compounds (VOCs). They also include "non-criteria" hazardous air pollutants (HAPs), such as polynuclear aromatic hydrocarbons (PAHs), dioxins, furans, hydrogen chloride, benzene, polychlorinated biphenyls (PCBs); and metals such as arsenic, cadmium, nickel, zinc, mercury, chromium, and vanadium. Both criteria and HAP emissions from an open tyre fire can represent significant acute (short-term) and chronic (long-term) health hazards to firefighters and nearby residents. Depending on the length and degree of exposure, these health effects could include irritation of the skin, eyes, and mucous membranes, respiratory effects, central nervous system depression, and cancer. Firefighters and others working near a large tyre fire should be equipped with respirators and dermal protection. Unprotected exposure to the visible smoke plume should be avoided.

Toxicity resulting from the massive use of plastics in our modern society is not restricted to exposures sustained by workers during synthesis, formulation, or manufacture. Rapid and nearly complete oxidation of plastic products occurs with the application of extreme heat during a conflagration, resulting in carbon dioxide, water and oxide of nitrogen. Even so, partially degraded molecules result, and these may recombine to form new molecular species. Thermal degradation often begins at relatively low temperature (200–400ºC), producing hydrogen chloride, carbon dioxide, carbon monoxide, and some hydrocarbons. Neoprenes release sulfur dioxide and hydrogen sulfide. Rigid urathanes and isocyanates also produce CO_2, CO and small amount of $CFCl_3$, HCN, HCl and hydrocarbons. Although the expected toxicity results from those compounds released in greater amounts, those emitted in smaller amounts cannot be disregarded for two major reasons:

a. They are very toxic (e.g. benzene and hydrogen cyanide)
b. Certain workers are hypersusceptible for some reasons, e.g. allergy to isocyanates and pharmacobiochemical sensitivity as encountered occasionally with amines and certain halogenated hydrocarbons.

Not only should the major parent compounds be considered but also additives, such as accelerators, catalysts, copolymers, dyes, fillers, mold lubricants, pigments, plasticizers, solvents, stabilizers, and ultraviolet absorbers. There are a number of syndromes being reported that appear to result from thermal degradation products of plastics.

Teflon fume fever has been reported, especially in smokers exposed to teflon fume generated by temperatures far in excess of those achieved during the cooking with teflon coated utensils.

Meat wrapper's asthma is another alleged syndrome that appears to be associated with exposure to heated plastics, in this instance, hot wire cutting of polyvinyl chloride film. There are instances of adverse reactions when urethane foams, polystyrene, and other plastics are thermally degraded.

Fire in a pressurized tunnel burns more intensely for a given flame size, spreads more vigorously and is harder to extinguish than atmospheric pressure and all of these effects are magnified by oxygen enrichment. So, the fire risk depends on both pressure and percentage of oxygen in the environment and remains a major safety consideration in compressed air work.

Classification of Fire

1. Class 'A' fire (ordinary fire): Fire in ordinary combustible materials such as wood, paper, etc.
2. Class 'B' fire (oil substances fire): Fire in flammable liquid like petrol, diesel, etc.
3. Class 'C' fire (gas fire): Fire involving gaseous substances under pressure like liquid petroleum gas, etc.
4. Class 'D' fire (metal fire): Fire involving metals.
5. Class 'E' fire: Fire involving electrical equipment.

EFFECTS OF OXYGEN DEFICIENCY

Oxygen-deficient atmosphere can be encountered in mines, and other underground or confined spaces. The main danger of suddenly entering an atmosphere devoid of oxygen is that it will lead to an almost immediate loss of consciousness without warning; even very quiet breathing will produce sudden loss of consciousness within 50 seconds when all the remaining oxygen in the lungs has gone.

It is the partial pressure of oxygen, not its percentage, which is of physiological importance, but gas measuring instruments and alarms record volume concentrations. A drop of 3–4% of oxygen by volume is a little physiological significance in humans, but it will extinguish a candle flame. The level of oxygen has to fall to 13% volume before symptoms become very noticeable.

The effects of low oxygen concentration are as follows:

1. 16–13%: Dizziness and shortness of breath on exertion; pulse rate accelerated and volume of breathing increased. Ability to maintain attention is diminished but it can be restored with conscious mental effort.
2. 13–10%: Judgment faulty. Rapid fatigue and fainting on exertion. Severe injuries cause no pain. Emotional lability.
3. 10–6%: Nausea and vomiting. Loss of ability to perform any vigorous muscular movements or even to move at all.
4. $<$ 6%: Loss of consciousness with fainting or coma. Rapidly fatal.

Confined Spaces

Working in enclosed spaces with inadequate natural ventilation is a well-known cause of death from asphyxia due to oxygen deficiency or from a build-up of toxic gases and vapors, such as carbon monoxide, carbon dioxide, methane, ammonia, hydrogen sulfide, petroleum vapor and liquid petroleum gas. The examples of confined spaces are: trenches, pits, sewers, tunnels, submarines and spacecraft. Toxic substances are accumulated where workers are welding or flame-cutting. In addition, the presence of flammable gases such as butane, propane and petrol, all of which are normally heavier than air, can be responsible for explosions in tanks. The risk of fire and explosion may be increased by enrichment of the air by oxygen in the event of a leak from an oxygen cylinder forming part of welding equipment. Oxygen deficiency in pits can arise from an ingress of methane or absorption of oxygen by certain constituents of soil. The rotting of vegetation and the rusting of metalwork inside tanks also consume oxygen. Manholes, tunnels and trenches in limestone soil can partly fill with carbon dioxide formed by the action of acid groundwater. Apparently safe atmospheres can become suddenly dangerous if the residues and sludges in tanks or in sewers are disturbed by the worker walking in them, or by water surges following sudden heavy rainfall. In construction industry, pipe-freezing work is carried out using liquid nitrogen to solidify soil to enable drilling to be performed in wet conditions and fatalities have occurred from the cold gas pushing out the available air.

In order to avoid gassing accidents, safe systems of work must be adhered to and these often require the use of electronic monitors or detector tubes to test for toxic, flammable and asphyxiating gases before initial entry to a confined space and while the work continues. The space should be well ventilated before entry; otherwise, appropriate breathing apparatus has to be worn.

HEALTH HAZARDS OF METAL ARC WELDING IN INDUSTRY

Metal welding is a genetic term for any of the processes, ubiquitous in industry, of joining metals at areas or point softened or liquefied by the application of heat, sometime with associated local pressure.

The toxic agents responsible for health hazards:

A. *Particulate emission*: Two main groups of particulates may be emitted from electric arc welding process:
   1. Fractioned mixed metal oxide particles are formed from metal which is vaporized in the high temperature of the arc and then condensed, often reacting with oxygen in the air, to form chain and aggregates reflecting the composition of the electrode but with the more volatile constituents predominately. The most of these particles are in the respirable range, e.g. iron, zinc, aluminum, cadmium, and chromium.
   2. The second group of particles is term ‘spatter’. These are coarser, discreet, unfractionated particles of electrode, usually larger than the respirable range.

B. *Gaseous emission*: The principal gases are:
   1. Ozone
   2. Oxide of nitrogen
   3. CO

C. Heat

D. Noise.

Health Effects

Respiratory System

1. *Acute effect*:
   a. *Asphyxia*: When working in an inadequately ventilated confined space where oxygen has been displaced by inert shielding gases or depleted by combustion or rusting of ferrous structures.
   b. Inhalation fever: The term inhalation fever collectively describes as a group of acute, non-allergic, usually benign and self-limiting flue-like illness caused by heavy exposure to certain inhalable environmental pollutant. The most common of these is metal fumes fever, with polymer fumes fever.
   Metal fumes fever: It is one of the few ancient occupational diseases still encountered in modern industrial practice, is quite common among welders, the percentage reporting an attack when asked specifically has been variously reported as 10% and 30%. It is an unpleasant but uncomplicated, self-limiting, non-allergic, influenza-like reaction following a single exposure to high concentration of metal fumes. The syndrome consisting of fever, chills, muscle pains and malaise, generally with relatively mild respiratory symptoms, and classically little or no radiographic or functional abnormalities, although this is not always the case. The symptoms usually begin a few hours after a heavy exposure to metal oxides and they subside spontaneously after 24 hours or night's sleep. Zinc fume fever commonly occurs, e.g. after the smelting or thermal spraying of zinc without adequate exhaust, or after welding or flame cutting galvanized steel in a confined space. No specific treatment is required, except preventive hygiene measures.
2. *Chronic health effect*:
   a. Chronic bronchitis:
      - More in smokers
      - There is dose-response relationship.
   b. Occupational asthma
   It may be caused by inhalation of irritants or sensitizing agents, e.g. nickel, chromium, iron, and zinc.

Central and Peripheral Nervous System

1. Neuropsychiatric symptoms in aluminum toxicity
2. Manganese toxicity.

Reproductive System

Welding may cause reduction of fertility due to:
1. Exposure to radiant heat
2. Exposure to metal like chromium, nickel, manganese, and cadmium.

Physical Injury

Musculoskeletal system injury like accident, injury, shoulder pain, and low back pain.

Skin

1. Ray burn
2. Permanent pigmentation in the unprotected areas of skin

3. Welding may affect the exposed skin in other ways like siderosis
4. Contact dermatitis
5. Photodermatitis.

Ear and Hearing

1. Burn inside the ear is uncommon but very painful
2. Perforation of tympanic membrane with deafness
3. Facial paralysis
4. High rate of residual and recurrent perforation
5. NIHL.

Eyes

1. Eye injury from foreign body.
2. Arc-eye due to exposure of arc's ultraviolet radiation. Arc eye is an acute high flux ultraviolet injury to the corneal epithelium causing a punctuate superficial keratitis in the welder whose eyes have not been adequately protected when he has struck the electric arc to begin welding. Some injuries are caused by reflected radiation. It is also known as flash eye. Typical presentation after 4–5 hours like:
   a. Pain due to spasm of iris and ciliary muscles
   b. Photophobia
   c. Excess lacrimation
   d. Feeling of grittiness or foreign body sensation.

   The eyes will be normal if take rest for 48 hours.

Cardiovascular System

1. The toxic effect of soluble barium in fumes from certain filler wires and consumable electrodes shown to be cardiotoxic.
2. Electromagnetic field can develop disturbances to the pacemaker.

Welding fumes cannot be classified simply. The composition and quantity of both are dependent on the alloy being welded and the process and electrodes used. Reliable analysis of fumes cannot be made without considering the nature of the welding process and system being examined; reactive metals and alloys such as aluminum and titanium are arc-welded in a protective, inert atmosphere such as argon. These arcs create relatively little fume, but an intense radiation which can produce ozone. Similar processes are used to arc-weld steels, also creating a relatively low level of fumes. Ferrous alloys also are arc-welded in oxidizing environments which generate considerable fume, and can produce carbon monoxide instead of ozone. Such fumes generally are composed of discrete particles of amorphous slags containing iron, manganese, silicon and other metallic constituents depending on the alloy system involved. Chromium and nickel compounds are found in fumes when stainless steels are arc-welded. Some coated and flux-cored electrodes are formulated with fluorides and the fumes associated with them can contain significantly more fluorides than oxides. Because of the above factors, arc-welding fumes frequently must be tested for individual constituents which are likely to be present to determine whether specific TLVs are exceeded. Conclusions based on total fume concentration are generally adequate if no toxic elements are present in welding rod, metal coating and conditions are not conducive to the formation of toxic gases.

The following preventive measures must be taken to achieve adequate control of the welding hazards:

1. Prevent emission by selection/substitution of process; improve working practices by modification of process, process parameters or consumables to lower fume and gas emission rates or toxicity of the constituents.
2. Prevent exposure by natural ventilation and/or engineering controls, including isolation or segregation, control of pollutants at the source by the local exhaust ventilation.
3. Prevent exposure by wearing personal protective equipment.

OCCUPATIONS INVOLVING RISK OF CONTAINING THESE OCCUPATIONAL DISEASES

A. *Effects of work on health*: Work can affect health in several ways, like:
   1. Occupational exposure may be a direct cause of ill health, e.g. exposure to hydrogen cyanide gas encountered by firefighters during their work can cause cyanide poisoning.
   2. Occupational exposure, while not necessarily being the sole factor causing disease and be one of a number of contributory causes. Cigarette smoking multiplies the risk for lung cancer if there is exposure to asbestos fibers or radon gas. Each of these can cause lung cancer independently, but a worker who smokes, lives in a high environment radon area or is exposed to asbestos from work activities has a much-increased risk of lung cancer. Another example is that of exposure to carbon monoxide which can result from smoking, following exposure to methylene chloride (used as a paint stripper), and working in an enclosed space like garage which is heated by a gas-fire heater, and has a car engine running. All these factors act independently to increase blood carboxyhemoglobin, but when several of these sources of carbon monoxide are present there, there is an elevated risk of carbon monoxide poisoning.
   3. The nature of job may worsen pre-existing non-occupational disease. Hairdressing producer or working in kitchen can result in a worsening of endogenous eczema.
   4. The occupational environment may give easy access to potentially harmful materials, increasing the risk of their abuse. Adverse health effects resulting from availability to toxic agents are effects of anesthetic gases in anesthetists or other health care staffs or suicide in farm workers using pesticides.

B. *Effects of health on work*: Following a period of sickness absence, it is in a patient’s and society’s interest to encourage a return to work. Many people with some impairment of function either seek work or seek to remain at work. This may affect his work by the following ways:
   1. The patient’s condition may limit, reduce or prevent him performing the job effectively, e.g. the musculoskeletal conditions that reduce mobility or diminish manipulative ability.
   2. The patient’s condition might be made worse by the job (excessive physical exertion in some cardiorespiratory condition).
   3. The patient’s condition is likely to make it unsafe to do the job (liability to sudden loss of consciousness while working alone and at height).
   4. The patient’s condition is obviously/likely to make it unsafe for third parties, such as coworkers, visitors or members of the public, e.g. a bus or train driver who is prone to

episode of unconsciousness with no warning symptoms may cause an accident affecting other crew members, passengers and public.

5. The patient's condition might make it unsafe for the community (for consumer of a product, if a food-handler transmits infection).

Correlation Between Environmental Exposure and Health Status

The main factors which regulate the occupational health status are:

1. Nature of substance of exposure.
2. Intensity or severity of exposure.
3. Length of exposure.
4. Personal susceptibility.
5. Quantum of exposure.
6. *Bioavailability*: The bioavailability of a toxic substance indicates the extent of which the agent reaches the site of action. If it is not in bioavailable form, as in the case with many orally ingested toxic substances that cause vomiting or diarrhea, it will be removed promptly. In other cases, some of the agents will be inactivated before it reaches the site of action, e.g. when cyanide is taken orally, it is absorbed and passes to the liver, where the enzyme rhodanese may metabolize a portion of the ingested cyanide. On the other hand, if the cyanide in the form of gaseous hydrocyanic acid (HCN) is absorbed through the pulmonary circulation, it goes directly to the brain, where it may cause damage from hypoxia.

MODE OF CAUSATION OF OCCUPATIONAL DISEASES AND ITS EFFECTS

The main routes of entry of toxic materials are:

1. *Inhalation*: Inhalation by far the most important route of intake of airborne contaminants. In an industrial environment various substance, solid, liquid or gases are therein the air. In the process of breathing, these airborne contaminants come in the contact with the respiratory system. The respiratory system serves as the portal of entry into the body for a great variety of airborne substances, both gaseous and particulate matters. Many of these contaminants are capable of producing injury and diseases when they are deposited and accumulated in sufficient amount in the lungs or after transfer from the lung in sensitive sites deeper within the body.

   For all particles, a major determining characteristic of the behavior of the particle in the respiratory tract is the size. Size is measured not by geometric dimensions but by sedimentation. The 'aerodynamic diameter' of a particle is what the diameter would be of a perfect sphere sedimenting at the same velocity, correcting for density. Particles above 10 μm aerodynamic diameter tend to be deposited in the nose and larger airways due to inertial impaction. Particles with an aerodynamic diameter <10 μm but >5 μm attain sufficient inertia to become trapped in the smaller airways and at bronchial bifurcation. Particles (little particles) of <0.5 μm in diameter may either remain airborne and be exhaled or be pushed into the mucosa because of the phenomenon of brownian movement. They are kept aloft by molecular motion (heat) 'nudging' them from all sides. They also may be cleared by the process of phagocytosis. Thus, the particles in the midrange are most likely to penetrate to the alveoli and, once there, to sediment and to deposit on the airspace wall. This so-called 'respiratory range' between about 0.5 μm and 5 μm is a primary concern in the inhalation of particulates. They are known as respirable dust.

2. *Through skin*: Many gaseous and liquid materials are absorbed to a limited extent through the intact skin. When this occurs, the predominant action may be local, at the point of contact, or systemic. The amount of skin absorption generally is proportionate to the surface area of contact and to the lipid solubility of the toxic agents. Phenol, cresol, nitrobenzene, aniline, TEL, parathion, etc. may pose a great hazard through skin absorption than through inhalation. Absorption of solid materials through skin is not common. Absorption of toxic chemicals through skin is more through abraded than intact skin.
3. *Ingestion*: It may result from many sources like contaminated food and smoking. It is the least important rout of absorption in occupational scenario. The amount of absorption through GI tract is usually proportionate to the gastrointestinal surface area and its blood flow and depends on the physical state of the agent. Most toxic agents are absorbed in the small intestine.
4. *Ocular absorption*: Toxic chemical can also be absorbed through eyes. Eyes are also the site of absorption. When the chemicals enter the body through the conjunctiva, they bypass the hepatic circulation and are responsible for severe systemic toxicity. This may occur when the organophosphate pesticides are splashed into the eyes.

Metabolism of Toxic Chemicals

Absorption and Distribution of Chemicals

Foreign or exogenous chemical (xenobiotics) must be absorbed from the surrounding environment and transported to their target site in the body for a toxic effect to occur. The chemical has to cross the many cell membranes which form a lipoprotein barrier to the outside as well as maintain the integrity of the cell. The specific transport mechanisms that have evolved are to facilitate the absorption and distribution of nutrients rather than toxic chemicals. Consequently, most xenobiotics are transported by simple methods and not complex carrier-associated processes. A carrier for an endogenous substance unless the xenobiotic compounds have a very similar structure it will not usually be able to bind to the carrier. Lipid solubility is one of the major factors determining the extent and rate of simple diffusion through a lipoprotein membrane. Lipophilic molecules diffuse more readily than those that are hydrophilic, the rate of transport being dependent on the partition coefficient (the ratio of solubility in octane/water).

Toxicokinetic

The investigation and management of a patient exposed to toxic chemicals requires at least a basic knowledge of the concept of toxicokinetic alongside an understanding of the mechanism by which chemicals gain entry to the body and are biotransformed into nontoxic or occasionally, toxic metabolites. Toxicokinetic is the study of dynamic (kinetic) relationships between the concentration of a chemical (toxicon) in body fluids and tissues and its biological effects. Toxicokinetic analysis produces a mathematical description of the dynamics of absorption, distribution and elimination of chemical. Although involving the descriptive use of mathematical value in that the understanding can be uses to evaluate the significance of blood concentrations in biological monitoring and to determine the nature of exposure control (risk management).

Most toxic xenobiotics have no nutritional value and are metabolized primarily to reduce potential toxicity and facilitate their elimination from the body. The majority of compounds that gain entry most easily to the body are lipophilic and likely, therefore, to be retained and before they can be excreted must be converted into a form that is more easily eliminated. The liver is the major organ for the metabolism (biotransformation) of such compound. The principal function of biotransformation is to facilitate the elimination of a foreign agent by its conversion to a more polar (water soluble) metabolite and is therefore, a detoxification mechanism and then it is more readily excreted in the urine.

Toxic substances are excreted either unchanged or as metabolites. Excretory organs other than lungs eliminate polar (water-soluble) compounds more efficiently than no-polar (lipid-soluble) compounds. The kidney is the primary organ of elimination for most polar compounds and their metabolites. Many toxic substances metabolized by the liver are excreted first in the bile and later eliminated in the stool and reabsorbed into the blood. Some toxic substances may be excreted through saliva, breast milk, and there may be some minor removal in hair or skin. The volatile gases are readily excreted by the lung through passive diffusion from the blood, crossing the alveolar-capillary barrier in 'reverse' direction.

DIAGNOSTIC METHODS OF OCCUPATIONAL DISEASES

Occupational diseases are caused by a pathological adaptation of the patient to his working environment; therefore, in order to properly diagnose occupational diseases, one must evaluate both patient and environmental exposure. Very few occupational diseases present with specific pathognomic, clinical, or laboratory findings. Thus, the anemia of benzene intoxication, the peripheral neuritis of acrylamide poisoning, asthma of byssinosis, the fibrosis of asbestosis, the granuloma of berylliosis, the nodulation of silicosis cannot be adequately diagnosed as to etiological agent from clinical and laboratory findings alone. Only with knowledge of exposure, in addition to clinical factors, can an accurate diagnosis be made. Obtaining adequate environmental data and weighing their importance as causative factors can be an extremely difficult problem, especially for one not experience in this.

One should utilize the same process use in the diagnosis of condition, namely, history of symptomatology, past history, family history, and review of the symptoms.

The physical examination should include a general examination, with special emphasis on the organ system likely to be seen in the disease under investigation, e.g. the lead line (blue line) of gum in inorganic lead intoxication, the enlarged liver seen with overexposure to toluene, or the enlarge spleen in overexposure to benzene. Evaluation of exposure, which is necessary in diagnosing work-related conditions, adds a dimension to occupational medicine usually not found in other clinical practice.

Components of a Thorough Occupational History

1. Present job description and nature of work.
2. Hours of work and description of shifts system.
3. Types of workplace hazards:
   a. Physical
   b. Chemical
   c. Biological
   d. Mechanical
   e. Psychosocial.

4. Degree of exposure:
   a. Duration of exposure
   b. Exposure concentration
   c. Route of exposure
   d. Presence and efficacy of exposure controls
   e. Qualitative exposure data from inspections and monitoring.
5. Previous occupations:
   a. Lifetime history, with date of employment and job duties
   b. Hobbies and avocational exposures
   c. Sports.
6. Other jobs or 'Moonlighting'.
7. Time relation between job and symptoms:
   a. Symptoms occur or are exacerbated at work and improve away from work.
   b. Symptoms coincide with the introduction of new exposure at work.
   c. Symptoms coincide with other change in working conditions.
8. Previous significant medical history:
   a. Past illness
   b. Past injury
   c. Past surgery.
9. Presence of an occupational health program at work:
   a. Periodical health surveillance
   b. Workplace environmental assessment
   c. Regulation of workplace exposure
   d. Availability of trained medical and nursing care.
10. Workplace protection:
    a. Use of personal protective equipment on the job.
    b. Methods of material handling.
    c. Engineering protective measures.
11. First aid or acute care provided at the time of injury or illness.
12. Similar complaints among other workers.
13. Presence of any abnormal circumstances at work:
    a. Breakdown or shutdown machines or processes.
    b. Increase demand for production.
    c. Change in personnel, materials or processes.
14. Other environmental exposure:
    a. Home environment
    b. Indoor air quality
    c. Social lifestyle
    d. Smoking habits
    e. Alcohol use
    f. Drug use
    g. Addictive behaviors.

Need of Occupational History

The two main reasons why an occupational history is important:

1. Because of the effects of work on health: Occupational exposures may be a direct cause of ill health, e.g. exposure to hydrogen cyanide gas encountered by firefighters during their work can cause cyanide poisoning, or exposure to metallic mercury in gold miners, or in dentists causes mercury poisoning.
2. Because of the effects of health on the safe and efficient performance of work: The patient's condition may limit, reduce or prevent him performing the job effectively like musculoskeletal conditions that reduce mobility or diminish manipulative ability or patient's condition is likely to make it unsafe to do the job like liability to sudden loss of consciousness while working alone and at heights.

Laboratory tests done in connection with the occupational disease workup are in five categories:

1. *Tests for general assessment of health and to rule out other conditions*: Evaluation in cases of suspected intoxication should routinely include complete blood count, chest X-ray, electrocardiogram, urine analysis.
2. *Nonspecific tests of exposure*: These nonspecific tests of exposure include mean corpuscular volume, mean corpuscular hemoglobin concentration in case of exposure to hemotoxic agents, aspartate amino transferase (AST-SGOT), alanine amino transferase (ALT-SGPT) in exposure to liver toxins, a reduction in force expiratory volume in 1 second (FEV_1) of pulmonary function test (PFT) during the course of the working day when the patient is exposed to pulmonary irritants, delta aminolevulinic acid in red cells in inorganic lead intoxication.
3. *Tests for the agent or its metabolite that indicate exposure*: There are a number of these tests based on a knowledge of metabolism of the substance involved, e.g. it is known that gradual absorption of inorganic lead is characterized by increased lead in the blood. Exposure to toluene may be detected by analysis of hippuric acid in the urine and exposure to trichloroethylene by trichloroacetic acid in the urine and by breath analysis for the substance itself.
4. *Tests of genetic or acquired susceptibility*: There are an increasing number of tests that establish a susceptibility to a disease condition that may be stimulated, precipitated, or aggravated by occupational exposure. Some of these are as follows:
   a. Hereditary serum α_1 antitrypsin deficiency and chronic obstructive pulmonary diseases.
   b. Glucose-6-phosphate dehydrogenase deficiency and hypersusceptibility to hemolytic chemicals.
   c. Diaphorase deficiency and susceptibility to nitrite exposure.
   d. Immunologic screening tests (IgE, IgG) for hypersensitivity to organic compounds.
5. *Chromosomal alteration*: The rapidly expanding science identifies genetic injury from exposure to physical and chemical agents.

METHODS OF PREVENTION

The various measures for the prevention of occupational diseases may be grouped under four heads: Medical measures, engineering measures, statutory or legislation measures and administrative measures.

Medical Measures

1. *Pre-employment medical examination*: Pre-employment medical examination is done at the time of employment and includes the worker's family, occupational and social history, a thorough physical examination, biological and radiological examinations. The purpose of pre-employment medical examination is to place the right person in the right job and to preserve the data for further comparison.

Hazards	*Undesirable condition*
a. Lead	Anemia, hypertension, nephritis, peptic ulcer
b. Dyes	Skin, bladder and kidney diseases, asthma, precancerous lesion

2. *Periodic medical examination*: The slow development of some occupational diseases is very often, leads to their non-recognition in the early stages and this is harmful to the worker. Therefore, for early diagnosis of occupational diseases periodic medical examination is essential.
   The frequency and content of periodic medical examination will depend upon the type of occupational exposure. Ordinarily workers are examined once in a year. But in certain occupational exposure like lead, toxic dyes, and radium exposures monthly or 6-monthly examinations are indicated. Sometimes, even daily examination may be needed when irritant chemical like dichromate is handled.
3. *Pre-placement medical examination*: This medical examination is done when the worker is shifted from one hazardous area to another hazardous area within the same factory.
4. *Fitness-for-duty examination*: A fitness-for-duty examination is essentially the same evaluation and follows the same rules as a pre-placement examination. The only difference is that a fitness-for-duty examination determines whether a previously hired employee who was able to perform the essential functions of the job is still able to perform these essential functions safely. Ideally, a fitness-for-duty evaluation will determine whether an employee is able to perform his or her duties in a safe and effective manner.
5. *Pre-retirement medical examination*: Pre-retirement medical examination is done to the workers before the retirement to see the health condition at the time of retirement and these records are to be preserved in some occupations, like asbestos and silica prone industries.
6. *Special medical examination*: This medical examination is done in case of long-standing absent.
7. *Medical and health care services*: Occupational health service (OHS).
8. *First-aid center or ambulance room*: The ambulance room shall be separate from the rest of the factory and shall be used only for the purpose of first-aid treatment and rest. According to the Occupational Safety, Health and Working Condition Code, 2020 (Chapter VI), ambulance room in every factory, mine, building or other construction work wherein >500 workers are ordinarily employed.
9. *First-aid box*: There shall in every factory be provided and maintained so as to be readily accessible during all working hours first-aid boxes or cupboard equipped with the prescribed contents, and the number of such boxes or cupboards to be provided and maintained shall not be <1 for every 150 workers ordinarily employed at anyone time in the factory (Section 45 of Factories Act, 1948: First-aid appliances). According to the Occupational Safety, Health and Working Condition Code, 2020 (Chapter VI), adequate first-aid boxes or cupboards with contents readily accessible during all working hours.

10. *Industrial hospital*: As referral center.
11. *ESI scheme*: Employees' State Insurance (ESI) is a self-financing social security and health insurance scheme for Indian workers.
12. *Notification of notifiable occupational diseases*: National Laws and Regulations (According to the Occupational Safety, Health and Working Condition Code, 2020) require the notification of some cases and suspected cases of occupational diseases and these occupational diseases are called 'notifiable occupational diseases'. These diseases are recognized internationally for the purpose of workmen's compensation. The main purpose of notification in industry is to initiate measure for prevention and protection and ensuring their effective application and to investigate the working condition and other circumstances which have caused or suspected to have cause occupational diseases. There is a list of 29 diseases is included as notifiable occupational diseases as per Schedule III and Section 12 of this Code and recognized for the purpose of workmen's compensation as per the Workmen's Compensation Act, 1923. According to this OSH&WC, 2020, Section 12(1), where any worker in an establishment contracts any disease specified in the Third Schedule, the employer of the establishment shall send notice thereof to such authorities, and in such forms and within such time, as may be prescribe by the appropriate government.
13. *Supervision of work environment*: The information that may be obtained during a site visit includes a detailed description of the work process, prior results of industrial hygiene sampling and medical surveillance, list of toxic or hazardous materials used and, most important, a guided tour of the work site with a focus on the specific work areas where the patient has been working.
14. To supervise/acquainted with physical, chemical, biological, mechanical and psychosocial hazards.
15. *To study occupational physiology*: Fatigue and weight carried.
16. *To supervised night-work and shift work*.
17. *To advise on matter connected with health and welfare*.
18. *Maintenance and analysis of records*.
19. *Health education and counseling*.

Engineering Measures

1. *Design of building*: Measures for the prevention of occupational disease should commence in the blueprint stage. The type of floor, wall, height, ceiling, roof, doors, windows, cubic space, and color should receive attention.
2. *Good housekeeping*: It covers general cleanliness, ventilation, lighting, washing, food arrangements and general maintenance. It is a fundamental requirement for the control of occupational diseases.
3. *General ventilation*: There should be efficient exhaust ventilation in the room where dust, gas, and fumes are generated to decrease the airborne contaminants.
4. *Mechanization*: The plant should be mechanized to the fullest possible extent to reduce the hazard of contact with harmful substances.
5. *Substitution*: By substitution is meant the replacement of a harmful material by a harmless one or one of lesser toxicity, e.g. zinc or iron-based paints in place of lead-based paints and acetone or toluene can be used in place of benzene.

6. *Dust control*: Dust can be controlled at the point of origin by water spray (wet method or hydroblasting).
7. *Enclosure*: Enclosure the harmful materials and processes will prevent the escape of dust and fumes into the factory atmosphere.
8. *Isolation*: Sometime it may be necessary to isolate the offensive process in a separate building so that workers not directly connected with the operation are not exposed to the hazards.
9. *Local exhaust ventilation*: By providing local exhaust ventilation, dusts, fumes and other injurious substances can be trapped and extracted 'at source' before they escape into the factory atmosphere. The heart of local exhaust ventilation is the hood which is place as near as possible to the point of origin of the dust or fumes. In this way, the breathing zone of workers may be kept free of dangerous dust and poisonous fumes.
10. *Protection devices*: To provide respiratory and non-respiratory personal protective equipment (PPE).
11. *Environmental monitoring*: To maintain safe work environment.
12. *Statistical monitoring*: It comprises the review at regular intervals of collected data on the health and environmental exposure of occupational group.
13. *Research*: It can provide a better understanding of the industrial health problems. There are two kinds of research—pure research and research for improvement of or in connection with a manufacturing product.

Statutory/Legislation Measures

To comply (in India) some main code/act like:

1. Occupational Safety, Health and Working Condition (OSH and WC) Code, 2020.
2. The ESI Act, 1948.
3. The Workmen's Compensation Act, 1923.

Administrative Measures

Examples:

Acclimatization: It is done to the workers who are going to absorb in heat prone areas.

Change of Job: Shifting of the workers from noisy area to non-noisy area who are suffering from NITTS or initial stage of noise induced hearing loss (NIHL).

NOTIFIABLE OCCUPATIONAL DISEASES

Notifiable Occupational Diseases under Indian Factories Act, 1948

National Laws and Regulations require the notification of some cases and suspected cases of occupational disease and these occupational diseases are called notifiable occupational diseases. These diseases are recognized internationally for the purpose of workmen's compensation. The main purpose of notification in industry is to initiate measure for prevention and protection and ensuring their effective application and to investigate the working conditions and other circumstances, which have caused or suspected to have caused occupational diseases.

Section 89, Notice of Certain Diseases

1. Where any worker in a factory contracts any disease specified in the [third] schedule the manager of the factory shall send notice thereof to such authorities, and in such form and within such time, as may be prescribed.
2. If any medical practitioner attends on a person, who is or has been employed in a factory, and who is, or is believed by the medical practitioner to be suffering from any disease specified in the third schedule, the medical practitioner shall without delay send a report in writing to the office of the chief inspector stating:
   a. The name and full postal address of the patient.
   b. The disease from which he believes the patient to be suffering.
   c. The name and address of the factory in which the patient is, or was last, employed.
3. Where the report under sub-section (2) is confirmed to the satisfaction of the chief inspector, by the certificate of the certifying surgeon or otherwise, that the person is suffering from a disease specified in the third schedule, he shall pay to the medical practitioner such fee as may be prescribed, and the fee so paid shall be recoverable as an arrears of land revenue from the occupier of the factory in which the person contracted the diseases.
4. If any medical practitioner fails to comply with the provisions of sub-section (2), he shall be punishable with fine which may extend to [Rs. 1000].
5. The Central Government may, by notification in the official gazette, add to or alter the third schedule and any such addition or alteration shall have effect as if it had been made by this act.

Section 90, Power to Direct Inquiry into Cases of Accident or Diseases

1. The State Government may, if it considers it expedient so to do, appoint a competent person to inquire into the cases of any accident occurring in a factory or into any case where a disease specified in the third schedule has been, or is suspected to have been, contracted in a factory, and may also appoint one or more persons possessing legal or special knowledge to act as assessors in such inquiry.
2. The person appointed to hold an inquiry under this section shall have all the powers of a Civil Court under the Code of Civil Procedure, 1908 (V of 1908), for the purposes of enforcing the attendance of witnesses and compelling the production of documents and material objects and may also, so far may be necessary for the purposes of the inquiry, exercise any of the powers of an Inspector under this Act; and every person required by the person making the inquiry to furnish any information shall be deemed to be legally bound so to do within the meaning of section 176 of the Indian Penal Code (XLV of 1960).
3. The person holding an inquiry under this section shall make a report to the State Government stating the cause of the accident or as the case may be, disease, and any attendant circumstances, and adding any observations which he or any of the assessors may think fit to make.
4. The State Government may, if it thinks fit, cause to be published any report made under this section or any extracts there from.
5. The State Government may make rules for regulating the procedure at inquiries under this section.

List of Notifiable Occupational Diseases as per the Factories Act, 1948: It is also recommended in Scheduled III of "The Occupational Safety, Health and Working Condition Code, 2020":

1. Lead poisoning including poisoning by any preparation or compound of lead or their sequelae.
2. Lead tetraethyl poisoning.
3. Phosphorus poisoning or its sequelae.
4. Mercury poisoning or its sequelae.
5. Manganese poisoning or its sequelae.
6. Arsenic poisoning or its sequelae.
7. Poisoning by nitrous fumes.
8. Carbon bisulfide poisoning.
9. Benzene poisoning, including poisoning by any of its homologues, their nitro or amino derivatives or its sequelae.
10. Chrome ulceration or its sequelae.
11. Anthrax.
12. Silicosis.
13. Poisoning by halogens or halogen derivatives of the hydrocarbons, of the aliphatic series.
14. Pathological manifestation due to:
    a. Radium and other radioactive substances.
    b. X-ray.
15. Primary epitheliomatous cancer of the skin.
16. Toxic anemia.
17. Toxic jaundice due to poisonous substances.
18. Oil acne or dermatitis due to mineral oils and compounds containing mineral oil base.
19. Byssinosis
20. Asbestosis.
21. Occupational or contact dermatitis caused by direct contact with chemical and paints. These are of types, i.e. primary irritant and allergic sensitizers.
22. Noise induced hearing loss (exposure to high noise levels).
23. Beryllium poisoning.
24. Carbon monoxide.
25. Coal miners' pneumoconiosis.
26. Phosgene poisoning.
27. Occupational cancer.
28. Isocyanates poisoning.
29. Toxic nephritis.

COMPENSATION FOR OCCUPATIONAL DISEASES

The Workmen's Compensation Act (renamed as Employees Compensation Act, 1923) is the first piece of legislation towards social security. It deals with compensation for workers who are injured in the course of duty. The scheme of the Workmen's Compensation Act is not to compensate the worker in lieu of wages. The general principle is that a worker who suffers an injury in the course of his employment, which results in a disablement, should be entitled to compensation and in the case of a fatal injury his dependants should be compensated. Under the Workmen's Compensation Act it is the employer who is responsible to pay compensation (as opposed to the Employees State Insurance, establishments to which the Employees' State Insurance Act applies to the liability to pay compensation is on the ESI Corporation). The meaning of compensation in this Act is limited to compensation granted under the Act for employment injuries sustained during the course of work.

As per Section 3 of the Act, the employer is liable to pay compensation if the worker is injured by accident that:

1. Arises out of (i.e. while engaged in work)
2. In the course of his employment (i.e. during workhours)
3. Such an injury results in disablement of the worker.

If three conditions are met, the employer of an establishment covered by the Act is bound to pay compensation. While the second condition, i.e. during work hours is easy to prove, the first condition (i.e. the accident occurred while engaged in work) has been difficult to establish in certain cases.

Compensation is not payable by the employer in the following circumstances:

1. Where the disablement does not last for >3 days.
2. Where the disablement has arisen out of the following:
   a. Drugs or drink
   b. Disobedience
   c. Disregard for the safety measures prescribed.

An 'occupational disease' while in service, is a disease that inflicts workers in that particular occupation in which s/he was employed in and resulting from exposure to a hazardous working atmosphere, particular to that employment. If a worker contracts such a disease, then the employer is liable to pay compensation, provided that the worker was employed by him for a continuous period of 6 months. An occupational disease that is contracted in the course of employment will fall within the meaning of an 'accident' for the purposes of this Act. In the case of such a disease being contracted, the employer will be liable to pay compensation to the affected worker. The occupational diseases for which compensation is payable are specified in a list attached to the Act-specifically, Part A of Schedule III.

The compensation to be paid (according to the Chapter 3 of the Act) by the employer for injuries caused depends on extent of the disablement suffered by the worker; more severe disablements naturally receive higher compensation. This has been categorized as follows:

Compensation payable in case of:

1. Death
2. Disablement:
   a. Permanent total disablement.
   b. Permanent partial disablement.
   c. Temporary disablement.
      i. Temporary total disablement.
      ii. Temporary partial disablement.

In addition, the guiding principle is the higher the age of the injured worker, the lower the compensation.

List of occupational diseases as per Schedule III

S. no.	*Occupational Disease*	*Employment*
Part A		
1.	Infectious and parasitic diseases contracted in an occupation where there is a particular risk of contamination.	(a) All work involving exposure to health or laboratory work (b) All work involving exposure to veterinary work (c) Work relating to handling animals, animal carcasses, part of such carcasses, or merchandise which may have been contaminated by animals or animal carcasses (d) Other work carrying a particular risk of contamination
2.	Diseases caused by work in compressed air	All work involving exposure to the risk concerned
3.	Diseases caused by lead or its toxic compounds	All work involving exposure to the risk concerned
4.	Poisoning by nitrous fumes	All work involving exposure to the risk concerned
5.	Poisoning by organophosphorus compounds	All work involving exposure to compounds the risk concerned
Part B		
1.	Diseases caused by phosphorus or its toxic compounds	All work involving exposure to the risk concerned
2.	Diseases caused by mercury or its toxic compounds	All work involving exposure to the risk concerned
3.	Diseases caused by benzene or its toxic homologues	All work involving exposure to the risk concerned
4.	Diseases caused by nitro and amino toxic derivates of benzene or its homologues	All work involving exposure to the risk concerned
5.	Diseases caused by chromium or its toxic compounds	All work involving exposure to the risk concerned
6.	Diseases caused by arsenic or its toxic compounds	All work involving exposure to the risk concerned
7.	Diseases caused by radioactive substances and ionising radiations	All work involving exposure to the action of radioactive substances or ionising radiations
8.	Primary epitheliomatous cancer of the skin the skin caused by tar, pitch, bitumen, mineral oil, anthracene, or the compounds, products or residues of these substances	All work involving exposure to the risk concerned
9.	Diseases caused by the toxic halogen derivatives of hydrocarbons (of the aliphatic and aromatic series)	All work involving exposure to the risk concerned
10.	Diseases caused by carbon disulfide	All work involving exposure to the risk concerned
11.	Occupational cataract due to infrared radiations	All work involving exposure to the risk concerned
12.	Diseases caused by manganese or its toxic compounds	All work involving exposure to the risk concerned
13.	Skin diseases caused by physical chemical or biological agents not included in other items	All work involving exposure to the risk concerned
14.	Hearing impairment caused by noise	All work involving exposure to the risk concerned

(Contd.)

S. no.	*Occupational Disease*	*Employment*
15.	Poisoning by dinitrophenol or a homologue or by substituted dinitrophenol or by the salts of such substances	All work involving exposure to the risk concerned
16.	Disease caused by beryllium or its toxic compounds	All work involving exposure to the risk concerned
17.	Diseases caused by cadmium or its toxic compounds	All work involving exposure to the risk concerned
18.	Occupational asthma caused by recognised sensitising agents inherent in the work process	All work involving exposure to the risk concerned
19.	Diseases caused by fluorine or its toxic compounds	All work involving exposure to the risk concerned
20.	Diseases caused by nitorglycerine or other nitroacid esters	All work involving exposure to the risk concerned
21.	Diseases caused by alcohols and ketones	All work involving exposure to the risk concerned
22.	Diseases caused by asphyxiants: carbon monoxide, and its toxic derivates, hydrogen sulfide	All work involving exposure to the risk concerned
23.	Lung cancer and mesotheliomas caused by asbestos	All work involving exposure to the risk concerned
24.	Primary neoplasm of the epithelial lining of the urinary bladder or the kidney or the ureter	All work involving exposure to the risk concerned
25.	Snow blindness in snow bound areas	All work involving exposure to the risk concerned
26.	Disease due to effect of heat in extreme hot climate	All work involving exposure to the risk concerned
27.	Disease due to effect of cold in extreme cold climate	All work involving exposure to the risk concerned
Part C		
1.	Pneumoconioses caused by sclerogenic mineral dust (silicosis, anthracosilicosis, asbestosis) and silicotuberculosis provided that silicosis is an essential factor in causing the resultant incapacity or death	All work involving exposure to the risk concerned
2.	Bagassosis	All work involving exposure to the risk concerned
3.	Bronchopulmonary diseases caused by cotton, flax hemp and sisal dust (byssinosis)	All work involving exposure to the risk concerned
4.	Extrinsic allergic alveolitis caused by the inhalation of organic dusts	All work involving exposure to the risk concerned
5.	Bronchopulmonary diseases caused by hard metals	All work involving exposure to the risk concerned
6.	Acute pulmonary edema of high altitude	All work involving exposure to the risk concerned

EVALUATION OF INJURIES

Annexure 3

Schedule I

List of injuries deemed to result in permanent total disablement

S. no.	*Description of injury percentage of loss of earning capacity*	
Part I		
1.	Loss of both hands or amputation at higher sites	100
2.	Loss of hand and a foot	100
3.	Double amputation through leg or thigh, or amputation through leg or thigh on one side and loss of others foot	100
4.	Loss of sight to such an extent as to render the claimant unable to perform any work for which eyesight is essential	100
5.	Very severe facial disfigurement	100
6.	Absolute deafness	100
Part II		
1.	Amputation through shoulder joint	90
2.	Amputation below shoulder with stump <20.32 cm from tip of acromion	80
3.	Amputation from 20.32 cm from tip of acromion to <11.43 cm below tip of olecranon	70
4.	Loss of hand or of the thumb and four fingers of one hand or amputation from 11.43 cm below tip of olecranon	60
5.	Loss of thumb	30
6.	Loss of thumb and its metacarpal bone	40
7.	Loss of four fingers of one hand	50
8.	Loss of three fingers of one hand	30
9.	Loss of two fingers of one hand	20
10.	Loss of terminal phalanx of thumb	20
10A.	Guillotine amputation of tip of thumb without loss of bone	10
Amputation cases–lower limbs		
11.	Amputation of both feet resulting in end-bearing stumps	90
12.	Amputation through both feet proximal to the metatarsophalangeal joint	80
13.	Loss of all toes of both feet through metatarsophalangeal joint	40
14.	Loss of all toes of both feet proximal to the proximal interphalangeal joint	30
15.	Loss of all toes of both feet distal to the proximal interphalangeal joint	20
16.	Amputation at hip	90
17.	Amputation below hip with stump not exceeding 12.70 cm in length measured from tip of greater trochanter	80
18.	Amputation below hip with stump exceeding 12.70 cm in length measured from tip of great trochanter but not beyond middle thigh	70
19.	Amputation below middle thigh to 8.89 cm below knee	60
20.	Amputation below knee with stump exceeding 8.89 cm but not exceeding 12.70 cm	50
21.	Amputation below knee with stump exceeding 12.70 cm	50
22.	Amputation of one foot resulting in end-bearing	50
23.	Amputation through one foot proximal to the metatarsophalangeal joint	50
24.	Loss of all toe of one foot through the metatarsophalangeal joint	20

(Contd.)

S. no.	Description of injury percentage of loss of earning capacity	
Other injuries		
25.	Loss of one eye, without complications, the other being normal	40
26.	Loss of vision of one eye, without complications or disfigurement of eyeball, the other being normal	30
26A.	Loss of partial vision of one eye	10
Fingers of right or left hand index finger		
27.	Whole	14
28.	Two phalanges	11
29.	One phalanx	9
30.	Guillotine amputation of tip without loss of bone	5
Middle finger		
31.	Whole	12
32.	Two phalanges	9
33.	One phalanx	7
34.	Guillotine amputation of tip without loss of bone	4
Ring or little finger		
35.	Whole	7
36.	Two phalanges	6
37.	One phalanx	5
38.	Guillotine amputation of tip without loss of bone	2
Toes of right or left foot		
Great toe		
39.	Through metatarsophalangeal joint	14
40.	Part, with some loss of bone	3
Any other toe		
41.	Through metatarsophalangeal joint	3
42.	Part, with some loss of bone	1
Two toes of one foot, excluding great toe		
43.	Through metatarsophalangeal joint	5
44.	Part, with some loss of bone	2
Three toes of one foot, excluding great toe		
45.	Through metatarso-phalangeal joint	6
46.	Part, with some loss of bone	3
Four toes of one foot, excluding great toe		
47.	Through metatarsophalangeal joint	9
48.	Part, with some loss of bone	3

Note: Complete and permanent loss of the use of any limb or member referred to in this Schedule shall be deemed to be equivalent to be loss of that limb or member.

Examples

Sample of the evaluation of an injury: Loss of earning capacity assessment in case of head injury in an Oil Industry in India

Loss of earning capacity	*Criteria*
40%	Head injury without EEG abnormality
50%	Head injury with EEG abnormality but without epileptic fit
60%	(a) Head injury with EEG abnormality and epileptic fit (b) Head injury with brain scarring and EEG abnormality
80%	Head injury with mental and physical disability without complete recovery
100%	Head Injury followed by invalidity

Hearing impairment evaluation:

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 year.
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:

Mild to marked bilateral hearing loss				
Ear	*500 Hz*	*1000 Hz*	*2000 Hz*	*3000 Hz*
Right	15	25	45	55
Left	40	50	60	70

1. Calculation of average hearing threshold level:

   Right ear = (15 + 25 + 45 + 55)/4 = 35 dB = 15% loss

   Left ear = (40 + 50 + 60 + 70)/4 = 55 dB = 45% loss
2. Calculation of the hearing handicapped:

   Smaller number (better ear): 15% × 5 = 75%

   Larger number (poorer ear) = 45%

   Total = (75 + 45)/6 = 20%

Therefore, a person with hearing threshold levels shown in this diagram would have a 20% hearing handicapped.

MEDICAL SERVICE IN AN INDUSTRIAL ESTABLISHMENT AND ITS FUNCTIONS

Medical service in an industrial establishment means a service established in or near a place of employment for the purpose of:

1. Protecting the workers against any health hazard which may arise out of their work or the conditions in which it is carried.
2. Contribution towards the workers' physical and mental adjustment.
3. Contribution to the establishment and maintenance of the highest possible degree of physical, mental and social well-being of the workers.
4. Facilitate the complete treatment of the workers and their families.

Medical Service Organization in Industry may be of following types: It depends on the circumstances and the applicable standard, set up of Medical Service Organization. They are:

1. Possible:
    a. Should eighter be organized by the undertaking themselves or be attached to an outside body.
    b. Should be organized as a:
        i. Separate service within a single undertaking
        ii. Service common to a number of undertakings.
2. Immediately not possible: Where medical services cannot immediately be set up for the undertaking, such service should be established in first instance:
    i. For undertaking where the health risks appear greatest
    ii. For undertaking where the workers are exposed to special health hazards.
3. Not at all possible: The organizations where the medical service is not practicable, they should make arrangement with a physician or a local medical service for:
    i. Administering emergency treatment
    ii. Carrying out medical examination
    iii. Exercising surveillance over hygiene conditions.

Functions of Medical Services in Industry

1. Surveillance of all factors which may affect the health of the workers.
2. Job analysis in the light of hygienic, physiological and psychological considerations.
3. Supervision of personal protective equipment and its use.
4. Supervision of the welfare of the workers.
5. Medical examinations of the workers.
6. Surveillance of the adaptation of jobs to workers, mainly the handicapped workers.
7. Rehabilitation of disabled workers.
8. Placement of workers in a safe condition from hazardous condition.
9. Advice to individual worker at their request regarding any disorder in the course of work.
10. Immediate treatment of medical and surgical emergencies.
11. Training to the first-aid personnel.
12. Health education.
13. Compilation and periodic review of statistics.
14. To maintain relation with internal and external services and bodies dealing with health, safety, retraining, rehabilitation and welfare of workers.

15. Organized medical board.
16. Notification.
17. Maintain confidential personal medical file.
18. Referred the case to the referral center for appropriate treatment.
19. To conduct health exhibition.
20. Health care activities for workers and their families.
21. Disaster management activities.
22. Take part in the safety committee.
23. Work place health monitoring.
24. To provide health facility to the surrounding populations as a part of corporate-social responsibility.
25. To maintain first-aid boxes in the industry.
26. Screening for substances of abuse and combating abuses.
27. Development of health policy and programme.
28. To conduct biological monitoring and investigations.
29. To conduct work environment monitoring.
30. Research.

OCCUPATIONAL HEALTH SERVICES AND ITS ACTIVITIES

Occupational Health Services (OSH) are human-service organizations in modern society which have been explicitly designed to manage and promote the safety, health and welfare of citizens at work. The role of OHS should be essentially preventive. They will evaluate their preventive program, discover occupational hazards, and recommend the suitable placement of workers.

Principles of OHS

a. *Preventive*: Preventing occupational health hazards at work, like inoculations (vaccination), enclosure of hazardous process, machine guard, etc.
b. *Health promotion*: Promoting physical, mental and social well-being of workers like canteen facility, industrial training, etc.
c. *Protective*: Protecting workers' health against hazards at work, like use of PPE.
d. *Adaptation*: Adopting work and work environment to the capabilities of the workers, like acclimatization.
e. *Cure and rehabilitation*: Curing and rehabilitating occupational accidental injuries, occupational and work-related diseases, like early diagnosis and treatment, change of job, etc.
f. *Primary health care*: To provide primary health care to the workers and their families like to provide all facilities of primary health center.

Occupational Health Professionals

They include:
- Occupational health physicians and nurses
- Physiotherapists and ergonomists
- Occupational hygienists
- Safety engineers
- Occupational psychologists
- Managers of OHS units or organizations.

Activities of OHS

- Survey of factors which affect occupational health
- Job analysis
- Medical examinations of the workers
- Supervision of the welfare of the workers
- Supervision of personal protective equipment
- Adaptation of job for the workers
- Rehabilitation of disable workers
- Recommendation for the placement of workers in suitable job
- Notification of the notifiable occupational diseases
- Maintenance of confidential personal medical files
- Training on occupational health
- Health education
- To conduct medical board
- Referred the cases to referral center
- Maintained relation with external and internal services dealing with health, safety, hygiene, training, rehabilitation and welfare of workers
- Early diagnosis and treatment
- To provide ergonomic intervention technique
- Compilation and periodic review of statistics
- Research.

Action Plan of OHS

- Strengthening of international and national policies for health at work and developing the necessary policy tools
- Development of healthy work environment
- Development of healthy work practices and promotion of health at work
- Strengthening of occupational health care system
- Establishment of appropriate support services for occupational health
- Development of occupational health standards based on scientific risk assessment
- Development of human resources for occupational health
- Establishment of registration and data system, development of information services for experts, effective transmission of data and raising of public awareness through public information
- Strengthening of research
- Development of collaboration on occupational health with other organizations.

OCCUPATIONAL HEALTH HAZARDS IN HOSPITAL

Human health service sectors, like all complex employment settings or industries have the usual variety of routine employee hazards along with special risks unique for these environments. Because of the progression of the medical technology and the emergence of new and re-emergence of old diseases, human health service sectors like hospitals have not become safer places for work.

1. **Biological hazards:** Infectious disease continues to represent the most serious and life-threatening to the workers working in the health service sectors. Not only do hospital personnel contract disease through exposure to infected patients, they actually can transmit disease, since they are dealing with immunocompromised individuals. Hospital acquired infection is known as 'nosocomial infection' and usually resistant to normal treatment. Followings are some biological hazards (infective) of healthcare workers and risk of transmission to patients:
   a. *Hepatitis B*: Acute icteric hepatitis, sometimes fulminant, can occur 3–6 months after occupational exposure to the virus in non-immune workers. Acutely or chronically infected healthcare workers who undertake exposure-prone procedures can transmit hepatitis B infection to their patients.
   b. *Hepatitis C (HCV)*: It is now known that HCV is the cause of large proportion of cases of what was formerly called 'non-A, non-B hepatitis'. Acute or and even fulminant hepatitis can occur after occupational transmission of HCV.
   c. *Human immunodeficiency virus (HIV)*: A seroconversion illness typically occurs about 4–6 weeks after the exposure. The potential for HIV-infected healthcare workers to transmit to patients during exposure-prone procedures is a cause of great concern, given the very serious consequences of such an occurrence.
   d. *Viral hemorrhagic fevers*: It is caused by a range of viruses and they are severe and life-threatening diseases. The infections are transmitted to humans by mosquito bite (yellow fever, dengue), tick bite (hemorrhagic fever) or through contact with virus excreted by infected rodents or others animal reservoir (Lassa virus).
   e. *SARS coronavirus*: Very recently a pandemic outbreak started in the 1st week of December, 2019 and this coronavirus outbreak continued several months killing thousands of people worldwide. This pandemic infectious disease is spreading all over the world. World Health Organization (WHO) has given the official name of this Novel Coronavirus as COVID-19.
   f. *Varicella zoster*.
   g. *Cytomegalovirus*.
   h. *Measles, mumps and rubella*.
   i. *Tuberculosis*.
   j. *Scabies*.
   k. *Staphylococcal infection.*
2. **Chemical hazards:**
   a. *Waste anesthetic gases*: A variety of adverse health effects that have been attributed to exposure to waste anesthetic gases include cancer, somatic complaints like headache and nausea, stillbirth, low birth weight, spontaneous abortion, congenital anomalies, and premature death.
   b. *Chemotherapeutic agents*: The chemotherapeutic agents have well-known toxic effects like myelosuppression, immunosuppression, and specific organ toxicity. The obvious direct consequence is a concern for health care workers who utilize these agents on a regular basis.

c. *Other chemical agents*: A myriad of agents has been demonstrated to show toxic effects in health care workers, including latex surgical gloves (which can produce both cutaneous and systemic hypersensitivity reactions), glutaraldehyde (it is used as a tissue fixative and as a cold sterilizing agent like ethylene oxide for heat sensitive medical equipment, it is volatile, and irritant skin and mucous membrane symptoms are common, therefore protective gloves should be used), psyllium, and ribavirin aerosol. Biomaterials (methyl methacrylate monomer and chromium-cobalt-molybdenum particles) have been reported to produce occupational health hazards including pneumoconiosis in dentistry. Methyl methacrylate monomer is usually used by the orthopedic surgeons as bone cement for implantation of prosthetics or for fixation in the placement of stabilizing screws or devices. Formaldehyde is also produced chemical health hazard in hospital.

3. **Mechanical hazards (traumatic injury):** Hospital workers are susceptible to back injury, especially to the direct patient caregivers. One risk factor is the caregiver-patient size mismatch when a small nurse tries to reposition a large patient in bed. Needle pricks to the health workers mainly to the doctors and nurses are a major cause of spreading the infectious diseases like HIV and hepatitis-B.
4. **Physical hazards:** Ionizing radiation is one of the most well recognized hazards in the hospital environment. Each hospital must have safety officers who are responsible for the areas of diagnostic radiology, nuclear medicine with its multiple isotopes, and radiotherapy units. Personal monitoring (film badge) is used to check the adequacy of the radiation protection programme for the radiology employees. Pregnant lady is not allowed to be exposed to ionizing radiation, e.g. X-ray. X-ray technician should have to take adequate protection or personal protective equipment like lead coated apron or protected enclosure.
5. **Psychosocial hazards:** Work stress in most production-oriented organization is high but the stress for health care workers is compounded by the knowledge that mistake can cause significant harm to patients. Life and death decisions made almost instantly are not uncommon, and long hours with inadequate staffing compound the problem. House staff suffers extreme stress compounded by very long duty hours. Employee assistance programmes have proven to be useful in hospital setting, as they are in other environments.

Preventive and Control Measures

A. General measures:

   i. Personal hygiene and environmental sanitation: Keep it at high level in the hospital of any kind, is a mandatory requirement towards control of hospital infection.

   ii. Efficient housekeeping including clean supply of bed-liner, patients' dress, proper bed arrangement, frequent moping, periodic washing of ward floor.

   iii. Provision of auxiliary facilities like:

      a. Central Sterile Supply Department (CSSD)

      b. Mechanized laundry

      c. Hygienic kitchen and food supply system

      d. Prompt and coordinated system of waste management, and good housekeeping.

B. Special control:
   i. Air conditioning through fresh filtered air
   ii. Washing and disinfection of hospital ward in periodic interval
   iii. Periodical biological test of the swab
   iv. Plenty of water supply in the hospital
   v. Use of PPE like lead-coated apron for X-ray technician and use of protective gloves, masks, etc.

Principles of waste disposal in hospitals: Proper management of medical waste comprises collection, packaging, storage, transport, treatment, recycling, and disposal within the medical institution (station, ambulance, laboratory) up to the final disposal (recycling, thermal treatment, landfill).

ACTION PROGRAM FOR WORK RELATED DISEASES AT NATIONAL LEVELS

Hearing Conservation Program

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 Program' plays a vital role. The successful program 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.

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 precise determination of relationship between hearing loss and noise exposure has become possible.

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 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 program, 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 program. Various types of protectors are available. Plants having hearing conservation program generally assign their medical departments the task of fitting employees with ear plugs, earmuffs and hearing protective helmets.

Noise reduction ratings: When OSHA promulgated its Hearing Conservation Amendment in 1983, it incorporated the EPA labeling requirements for hearing protectors (40 CFR 211), 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, e.g. 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 labeled NRR values for workplace conditions and is used when considering whether engineering controls are to be implemented.

Estimated dBA exposure = 98 dBA – [(25 – 7) × 50#] = 89 dBA

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

Exposure for PPE/HC enforcement = 98 dBA – (25 – 7) = 80 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.

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.

Vision Conservation Program

A well-recognized Vision Conservation Program for an industry has five components:

1. Screening the vision of the individual worker.
2. Determining the vision requirements of different jobs among the workers.
3. Detailed assessment of the hazards of the jobs with special reference to the eye and the general working environment.

4. Encouraging the wearing of basic eye protective equipment at all times by every worker who enters the area potentially hazardous to the eye.
5. Providing properly fitting and approved type of safety eye-wears against special type of hazardous operations.

Individual workers should be examined and vision status recorded: Both at the time of initial recruitment (pre-employment medical examination) and periodical medical examinations. A 'vision tester' or an 'Orthorator' is a very useful and compact instrument to screen acuity of vision (both distant and near), field of vision, muscle balance (in both the planes), binocular vision and color vision. The visual standards of each worker should be carefully matched with the vision requirements of jobs performed by them, e.g. crane operators and drivers.

MAJOR ACCIDENT HAZARDS CONTROL—MEDICAL PROVISION

It is the statutory requirement of the occupier of any hazardous industry to have an emergency medical preparedness plan. Preparedness and preventive measures are not same.

Preparedness: It covers all actions taken with a view to organizing and facilitating timely and effective rescue, relief and rehabilitation.

Prevention: It means the formulation and implementation of long-term policies and measures to mitigate the impact. A well established and functioning enterprise level occupational health service (OHS) scheme forms the basis of Major Accident Hazard Control Scheme.

Medical Organization for Emergencies

The most important factor in the relationship between the prognosis of victims and the interval of time elapsing between sustaining the injuries are the availability of medical care. This later period is called the "delay-time or reaction time or response time". The emergency medical responses aimed at in reducing the 'delay-time'. As a general rule a casualty is ought to be offered the treatment at the earliest possible time.

The medical organization for getting the proper responses in emergency (industrial major accidents) has mainly three components.

1. Organization at Site (On-site Emergency)

Medical organization at site is needed for the purpose of:

a. Early identification.
b. Elimination of human error risk factors due to physiological and pathological causes.
c. Planning and executing the rescue operation.
d. Effective first-aid management.
e. Training for minimizing the after effects of major accident.
f. Constitution of 'Trauma team' for rescue and first-aid operations.

Effectiveness of on-site medical organization depends on:

a. Capability of the personnel.
b. Availability of trauma team.
c. Plan of action to receive the victims.
d. Their codification.

e. Their first-aid treatment.
f. Transportation.

Function of Trauma Team

Appropriate emergency first-aid treatment at the earliest:

1. To reduce delay time or reaction time: quick removal of the casualties from disaster site.
2. Prevention of respiratory failure and administration of oxygen.
3. Control of bleeding.
4. Replacement of suitable fluid for loss of blood.
5. Treatment of shock.
6. Replacement of suitable painkillers.
7. Supportive treatment for cardiac, respirator and other systems.
8. Splinting and immobilization in case of fractures.
9. To keep the body warm with blankets and to protect open wounds with suitable sterile dressing.
10. Moral support to the injured.
11. Maintenance of medical records.
12. Dead body, if any, is to be identified and dealt in the appropriate manner.
13. Distribution to the injured to referral center.

Action Plan

1. Employees should be trained first aid and be available at short notice to take part in the rescue operation.
2. Periodical mock drill should be conducted so that each one knows his exact role and responsibility.
3. Liaison with nearby reference hospital.
4. All employees should have had their blood grouped and routine immunization carried out.
5. A mutual-aid-scheme should be worked out in case of an industrial complex.
6. A system of 'Triage' is to be adopted to classified the victims into different groups depending upon the severity of the injury and urgency of transportation:
   a. Category I (Red tag): Highest priority.
   b. Category II (Blue tag): Highest priority.
   c. Category III (Yellow tag): Second priority.
   d. Category IV (Green tag): Lowest priority.
   e. Category X (Black tag): Least priority.

Triage System

The process of triage is used, to sort out patients into categories of priority for care and transport, based on the severity of injuries and medical emergencies. The sorting of patients begins as soon as trained personnel reach the site or sees the sick and injured persons.

Through careful triage, the patients can be placed into one of three categories: *Highest priority*, *second priority* and *lowest priority*.

Highest priority: Category I with red tag will get the highest priority from the medical point of view. The category I means: the victim is seriously injured, required immediate hospitalization in the referral center with life-supporting equipment during transportation. Category II with blue tag will get the same priority. The category II means the victim is seriously injured, required immediate hospitalization in the referral center without any life-supporting equipment during transportation. Examples of highest priority cases:

1. Respiratory arrest, airway obstruction, and severe breathing difficulties.
2. Cardiac arrest (remember that respiratory arrest is usually detected first).
3. Uncontrolled severe bleeding.
4. Severe head injuries.
5. Open chest wounds.
6. Open abdominal wounds.
7. Severe shock.
8. Burns involving the respiratory tract.
9. Severe medical problems (including heart attack cardiac stroke, heatstroke, poisoning and abnormal childbirth).
10. Unconsciousness.

Second priority: Category III with yellow tag will get the second priority. The category III means: victim is injured, required hospitalization in the referral center but not immediately.

1. Injuries to the spine (including cervical spine).
2. Moderate bleeding.
3. Conscious patients with head injuries.
4. Multiple fractures.

Lowest priority: Category IV with green tag will get lowest preference. The victim is injured required only first aid treatment and allows to go home. Category X with black tag will get least priority as the victim is dead or going to die.

1. Minor bleeding.
2. Minor fractures and minor soft tissue injuries.
3. Moderate and minor burns.
4. Obvious mortal wounds where survival is not expected.
5. Obvious death.

Transportation of Victims (Communication)

The transportation and allocation of victims to the hospitals need to be organized. The medical care rendered during transportation induces complications. There shall be two types of ambulances: one having life-supporting equipment and other without life-supporting equipment. Networking and round the clock readiness should be the essential feature of ambulance services. For this coordination between concerned agencies is necessary. Ambulance shall be manned by a full time driver-cum-mechanic and a helper trained in first aid, for the purposes of transportation of serious cases of accidents or sickness. The ambulance van shall not be used for any purpose other than the purpose stipulated herein and will normally be stationed at or near to the Occupational Health Center.

2. Organization in the Hospital (Off-site Emergency Plan)

Primary requirement for drawing plans for the hospitals to cope with emergencies arising out of major accidents is the identification of the victims, codification, distribution and early medical management should be there. The time period should be also so arranged that the initiation of each segment of the plan is made in the least possible time.

Crash team: A well-planed and optimum medical response helps the victims in a great way. The chances of survival and reducing the complication increase with this. The availability of Crash Team adds to the efficiency of such organization. The team consists of one Surgeon, one Anesthetist, one medical specialist, one or two theatre sisters, one casualty sister with other supporting paramedical staffs.

3. Medical Responses from Non-medical Personnel/Organizations

1. Police
2. Civil defence
3. Fire-brigade
4. Transport
5. NGO
6. Local administration.

OCCUPATIONAL HEALTH AUDIT AND SURVEY

Unlike hospital medicine, occupational health has always been closely associated with industry and therefore the efforts directed at improving the quality of occupational health service (OHS) has closely linked to the strategies already being used by industry to improve the quality of their own services and products.

Some occupational health departments have adopted the ISO-9000/BS-5750 approach and have incorporated elements of both medical and clinical audit into quality system. In India, the Bureau of Indian Standard (BIS) as per their clause 14489 has renamed the traditional safety audit as occupational safety and health audit and incorporated occupational health audit elements there.

Objectives of Occupational Health Audit

1. To determine the effectiveness and efficiency of an OHS organization as a whole.
2. To determine the need of change, e.g. in policy and in other planning and to evaluate any changes implemented.
3. To evaluate the contribution of the healthcare professionals and hence to help identify and plan education, training and other interventions.
4. To determine compliance with a standard, or with a service level agreement in terms of the structure of services, the quality of their process and/or the effectiveness of outcome.
5. To aid communication and find gaps in the evidence base for current practice, for setting the agenda for further research, evaluation and hence wider improvements in quality.

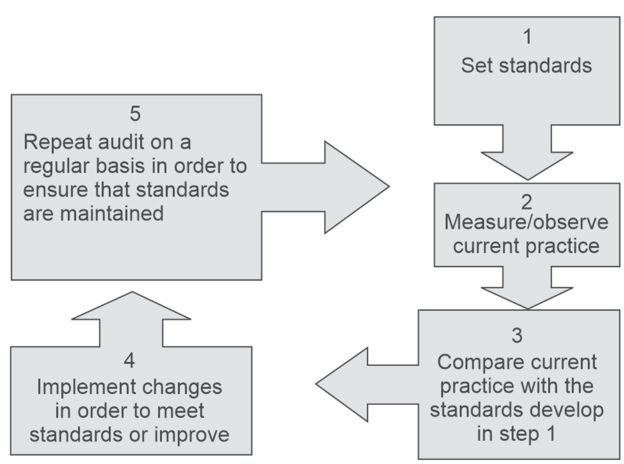

Fig. 1.13: The common model for OCC health audit

The most common approach to clinical audit on occupational health is based on Donabedian's structure, 'process and outcome' model (UK model) which uses techniques as the 'audit spiral' to help set standards, observe practice and compare current practice against the identified standard. In this model, changes can be introduced to ensure that the consistency of practice is maintained or to further improved the quality of practice. The standards set should be subjected to a rigorous process of continuous review, which takes account of new research and information generated from the audit process.

Elements of Occupational Health Audit

1. Occupational health administration.
2. Occupational health and hygiene program.
3. Occupational health service policy and procedures.
4. Health service resources facilities.
5. Health service program and activities.
6. Training program.
7. First-aid and medical program.
8. Records and forms.
9. Identification and assessment of occupational diseases.
10. Health surveillance and health monitoring.
11. Health and welfare measures.

Occupational Health Survey

Occupational health physicians are frequently asked to participate in work-site evaluation through occupational health survey. This evaluation may be performed to determine the work-relatedness of the single patient's symptoms, to investigate allegations of adverse health effects in a group of workers, to assist in the design of health and safety programs for a facility, or as a work-site 'follow-back' study of a sentinel health event. This workplace evaluation can range from brief walk-through surveys to more in-depth investigation including medical and environmental testing. The survey team usually consists of a medical officer and an industrial hygiene professional working together to perform a comprehensive evaluation of occupational hazards in a workplace.

Consultants conducting work-site evaluations should gather as much information as possible about the establishment before the site visit. Frequently this begins with a literature search centered around the materials and processes used. All work-site evaluation should begin with an opening conference led by the occupational health consultant. Attendees should include the plant manager, an individual responsible for plant health and safety, and at least one worker representative. The meeting should define the scope and nature of the investigation, address methodology of environmental and medical testing, and clarify concerns about trade secret and medical confidentiality issues. All attendees should be given the opportunity to ask questions or express concerns about the investigation process.

Once the opening conference is complete, the consultant team should devote some time to reviewing company health and safety records before actually beginning the walk-through. The reporting and recording systems can provide a good source of information about company injury and illness incidence rates. Logs from the company nurse, physician or first-aid center may give additional information about injury and illness that were not considered reportable. A material safety data sheet (MSDS) should be available for each toxic material used in the workplace; review of this information can identify many potential occupational hazards. Even a comprehensive set of MSDS may not reflect all chemical hazards in the workplace since reactive intermediates in the manufacturing process will not be reflected in this information. For example, the 1984 Bhopal disaster in India involved the release of methyl isocyanate, an intermediate in the production process of manufacturing the insecticide carbaryl.

Ideally, personal sampling data are available for a representative number of employees throughout the plant and have been correlated with appropriate medical or biological monitoring data. In reality, environmental monitoring data may be limited to area samples or to personal samples on a small number of workers. Individual work practices vary enough that two workers doing the same job in nearly identical settings may have quite different exposures. The use of personal protective equipment (PPE) may also modify individual exposures, although this is difficult to quantify. Workers may be using personal protective equipment without appropriate environmental data to demonstrate a need for it or to assist in appropriate selection. For example, many small firms require hearing protection in areas that are perceived as 'noisy', but have never conducted noise surveys. As a result, individuals who should be included in a hearing conservation program may not be, and individuals who work in noisy areas, especially those exposed to impact noise, may be inadequately protected. Similarly, the choice of appropriate respiratory protection also depends on adequate knowledge of the nature and level of air contaminants in the work environment.

When permitted, the occupational medical consultant should review any medical surveillance data collected through company programs. This may be difficult to access or look at systematically since a great percentage of occupational medical services are performed by outside clinics rather than in company facilities. Company may allow consulting physicians to review medical records that their employees have submitted in support of workers' compensation claims. If the consultation is being conducted in response to employee complaints, individual who have sought medical consultation relating to the occupational condition under investigation may voluntarily submit copies of their medical records for review.

In addition to the aforementioned medical and environmental information, the database of occupational health and safety consultations should be expanded to include information on company history, the formal statement of company goals, formal and informal organizational structure, labour-management relation, and regional economic trends.

The work-through evaluation itself is best accomplished by following the process from the entry of raw materials into the plant until the point where the finished product leaves the facility. This format should allow the consultant to see most job categories and departments and most routine job tasks. Maintenance activities and 'rework' of substandard product should be evaluated as well. Tasks that are performed infrequently or are not viewed as an integral part of the production process are often given little attention in company health and safety activities until they are associated with an injury or illness. The use of personal protective equipment in prescribed areas should be observed as well as employee hygiene practice such as eating, drinking, or smoking in potentially contaminated areas. The physicians should keep in mind that exposure may occur through inhalation, ingestion, dermal absorption, or a combination thereof, and should look for evidence of these as the walk-through is conducted.

During the walk-through, it is useful to ask employees about their work. Individual workers can often provide more insight into their specific job task than the plant representative present on the walk-through. Discussions regarding health problems should be brief, informal, and confidential. Initial question should be open-ended, rather than directed at specific symptoms or complaints. These can be followed with more specific questions about the nature, duration, timing and frequency of symptoms, as well as their association with work activities. It is also helpful to ask workers whether they know of any individuals who left employment with this company for health-related reasons.

If an industrial hygiene consultant is present, the walk-through survey may provide an opportunity for some limited environmental monitoring to help identify workplace contaminants and potential sources or exposure areas in the plant. Direct reading sampling equipment is available to assess airborne chemical contaminants, such as carbon monoxide, oxides of nitrogen, acid fumes and combusting hydrocarbons. Smoke tubes are a convenient means of observing airflow patterns relating to plant operations or ventilation systems; velocity meters can be used to obtain more exact information on plant ventilation.

The findings from the walk-through survey will suggest whether a potential occupational health hazard exists, and whether an in-depth medical or environmental survey, or both, is needed to characterize it. If environmental data demonstrates significant potential exposures, the industrial hygiene consultant may recommend either a thorough one-time survey or an ongoing monitoring program. Ongoing monitoring is particularly useful for exposures to agents whose health effects include long periods with asymptomatic changes and conditions that may benefit from early detection. Design of an in-depth medical survey should be based on findings from the walk-through and knowledge about the expected adverse health effects of substances used in the workplace.

When the work-site evaluation is complete, whether after the walk-through or following a more in-depth survey, a report should be issued that summarizes the findings of the investigation and makes appropriate recommendations for continuing health and safety activities within the establishment. For example, the industrial hygienist might recommend appropriate control technology or an ongoing environmental monitoring program. The occupational health physician may recommend, and help the company design, an ongoing medical surveillance program using appropriate screening tests to look for end-organ effects associated with the hazards encountered in the workplace. An understanding of the firm's organizational structure is critical to the consultants' ability to bring about change. The consultant must identify and have access to someone with enough authority to facilitate the acceptance and implementation of the final recommendations.

OCCUPATIONAL DISEASES RELATING TO CONSTRUCTION WORK, EMERGENCY MEDICAL TREATMENT OF INJURIES AND REHABILITATION AT CONSTRUCTION SITE

People might think that the active outdoor life in the construction industry would keep the workers fit and healthy. Quite the reverse is the true and the construction industry has a deservedly notorious reputation as being dirty, difficult and dangerous. Many people suffer fatal injuries on building sites every year. Many hundreds of thousands more people suffer serious injuries and ill health. Responsibilities for planning and coordination of health and safety are often unclear and compliance with health and safety law is generally poor. Informal contractual conditions in this sector make it difficult for workers to exercise their right and to push for more progressive and effective prevention initiatives based on workers participation, collective bargaining and training on skills, health and safety. The consequence of poor management standards in this sector is the deterioration of working and living conditions and an alarmingly high incidence of injuries.

Workers in construction industry are exposed to a wide range of hazardous substances and physical agents. Hazardous substances also have a serious impact on construction workers health. These may come in the form of liquids, gases, vapors, fumes or dusts. They are contained in a variety of commonly used products and materials in construction. The main exposure route is through inhalation, but substances such as solvents can also be absorbed through the skin. Very often, workers are not aware of what chemicals are contained in the products they use, and are not told about the health hazards and how to avoid them. The number one construction killer in any country is falling from height and this is principally due to the lack of proper edge protection in a variety of construction tasks. Height vertigo or height phobia is one of the important causes of fall from the height. Electrocution may also cause fatal and nonfatal injuries to the construction workers.

Most people are killed whilst carrying out perfectly routine work, where the hazards are well known. Deaths from these causes can and should be avoided by use of collective prevention measures. Lack of collective preventive measures is particularly dangerous when combined with work organization factors, i.e. management failures like poor housekeeping, chaotic working conditions, lack of planning and coordination, lack of training and supervision and intense productivity and time pressure.

Physical Hazards

Occupational deafness due to exposure of high noise level is very common in construction workers. Vibration syndrome mainly white fingers or Raynaud's phenomenon is common due to the use of vibrating pneumatic tools. Whole body vibration caused by operating heavy machinery and vehicles can cause damage to the spine. Extreme heat and cold, electrical injury, UV radiation from welding are some physical hazards in construction industry.

Chemical Hazards

Cement dust can cause serious respiratory problems over time, such as pneumoconiosis. Cement contains lots of chemicals, some of which cause skin problems like skin burn and contact dermatitis. Allergic dermatitis is caused by sensitivity to the chromium in cement and can be severe. Cement, fiberglass and wood preservatives can all cause irritant contact dermatitis, as well as mould oil in brick making. Cement burns are caused by wet cement trapped against the skin. Chromate and cobalt in cement, epoxy resin in special cements, softwoods and hardwoods, and rubber chemicals in boots and sealing strips are the main allergens.

Free silica dust can cause silicosis. This means irreversible scarring of the lungs, causing shortness of breath and premature death. Jobs such as stone masonry; concrete cutting or drilling; tunnelling and many demolition jobs can generate free silica.

Asbestos causes fatal diseases—asbestosis, mesothelioma and cancer of the lung. The use of asbestos in building and insulation materials has been widespread for many years. Millions of buildings all over the world contain asbestos, and workers carrying out maintenance, repairs, renovation or demolition work are often exposed without even being aware of it.

Wood dust causes respiratory system problems, irritation and allergy, asthma, rhinitis. Some types of wood dust and oils can cause nasal cancer, particularly certain hard woods. Fiber board and plywood contain glues and urea formaldehyde. Exposure to these chemicals may cause irritations.

Biological Hazards

Due to poor living and working condition of construction workers, many of them suffer from biological hazards like tuberculosis, cholera, parasitic diseases, dengue and malaria. Migration, including rural-urban migration, to seek work in large construction projects means being away from home and family for long periods. Therefore, the construction workers are at risk of HIV and AIDS (sexually transmitted diseases).

Mechanical Hazards

Back injuries may cause by manual handling of heavy loads, sometimes over long distances. For examples, bricks, cement blocks and cement bags with heavy load, confined spaces, awkward postures, heavy task and productivity demands, and long hours. Low back injuries, sciatica, hernias and slipped discs can put people out of the labor for ever. Other musculoskeletal disorders, injuries to muscles, nerves, tendons and joints are caused by physically demanding work. Risk factors include: uncomfortable postures, forceful and repetitive movements, awkward tools and sustained effort. Typical injuries include: Bursitis from kneeling, e.g. floor laying.

Psychosocial Hazards

Work stress of these workers may develop due to low socioeconomic condition, poor job security and high work stress.

Preventive Measures

Improvement can be made by substitution of hazardous materials for safer ones, by the introduction of safe working methods, by the use of appropriate PPE, through information, training and workers participation, access to occupational health services and health surveillance, providing social security and health schemes and strict implementation of law.

Caisson or Tunnelling Work

Caisson or tunnelling work is a part of civil engineering activity. Working at pressure becomes a requirement where excavation occurs beneath the level of the water table and where the ground is sufficiently porous to allow water to be forced into the tunnel or caisson as a result of the groundwater pressure. Hence, while it is possible to tunnel through solid impervious rock below water level without risk of flooding, tunnelling through softer water-permeable ground readily results in a water-filled tunnel with a risk of collapse of the tunnel apex or face. In this situation, to allow work to progress, the open end of the tunnel is closed off with a pressure tight structure and the tunnel is pressurized to a pressure adequate to prevent water ingress and stabilize the tunnel face. A pressure chamber or lock is built into the tunnel enclosure to allow workers to transfer from the surface to the pressurized environment without loss of pressure in the tunnel. This enables the tunnel construction to continue with work conducted in a dry environment.

Caissons are generally used for sinking bridge piers or structural foundation and furnish a means of removing mud and clay underwater so that bedrock may be reached upon which to place the foundation. A caisson is simply an inverted box that forms the bottom of the bridge pier. The edges of the box are bevelled so that they will cut into the clay or silt as weight is applied above. It is a large watertight chamber, open at the bottom, from which the water is kept out by air pressure and in which construction work may be carried out under water. Water is forced from the working compartment using compressed air, and the actual digging process can begin. Two shafts are provided which extend from the working chamber to the surface—one for removing mud and other excavated materials and one for the use of the working crew. A decompressed chamber is placed at the top where the working crew can decompress at the end of each shift.

Occasionally in caisson work, the physical layout makes it difficult to provide a large decompression chamber for the working crew in physical continuity with the shaft from the caisson below. In such cases, 'decanting' is used, wherein the workers decompressed very rapidly from the working pressure and then, within 5 minutes, move to a recompression chamber on an adjacent platform. However, decanting has the potential of producing a good deal of bubbling in the blood and tissues of the workers involved and, therefore, this process should be discouraged.

When the sand or muck becomes porous or the tunnel runs under a river or lake, there often is no practical way of keeping the water out except by putting considerable air pressure in the tunnel. To this purpose, an airtight bulkhead is erected across the tunnel so that pressure may be applied to the working face. Behind this bulkhead, a second bulkhead must be placed so that a decompression lock is created for the workers to enter and leave the work heading without having to drop the pressure on the heading itself.

Most tunnels are now constructed mechanically using a sophisticated tunnel boring machine (TBM) which lines the tunnel as it is constructed and hence the only human work requiring a pressurized environment is maintenance work conducted at the front face of the machine, such as blade or tool changing or technical or geological inspections. TBM requires short intense periods of compressed air interventions, sometimes at high pressures of 4–8 ATA. A shorter pressure exposure is now being adopted following recommendations from the medical advisers, especially where working pressure exceeded 2 ATA and this was eventually prescribed by HSE of UK in the 1996 regulations. Working pressure >3 ATA restrict shift duration. This has led to the need for the development and implementation of safer decompression techniques that give longer working time at higher pressure (>4 ATA) and with reduced risk of decompression sickness or dysbaric osteonecrosis. This means the application of diving technologies in the civil engineering context, which includes the use of mixed gas and saturated techniques, in addition to oxygen decompression. Tunnel workers may also require a gradual return to the surface pressure to prevent the onset of decompression illness. The decompression or ascent is conducted in a pressure lock so can be very accurately controlled.

Caisson and tunnel environments have other significant safety issues. They are enclosed (confine) spaces and require appropriate ventilation. Carbon dioxide retention may be a contributory factor in decompression illness. It is critical to ensure that the environment is not contaminated by toxic chemicals, particularly carbon monoxide (CO), and that all possible sources of ignition are controlled to avoid fire. Fire in a pressurized tunnel burns more intensely for a given flame size, spreads more vigorously and is harder to extinguish than atmospheric pressure and all of these effects are magnified by oxygen enrichment. So, the fire risk depends on both pressure and percentage of oxygen in the environment and remains a major safety consideration in compressed air work.

The followings are the conditions for compressed-air tunnel work:

1. Eat a well-balance diet.
2. Be temperate. Avoid excessive alcoholic beverages the night before or within 8 hours of going to shift.

3. Sleep at least 7 hours daily.
4. Take extra outer clothing into the tunnel when going on shift and during decompression to avoid chilling during that period.
5. Do not sit or rest in a cramped position during decompression.
6. Do not exercise during decompression. This does not mean you cannot move around to avoid sitting in one position.
7. Do not do hard exercise immediately after decompression.
8. Do not take a hot bath or shower within 6 hours of decompressing. A moderate warm bath or shower is permissible.
9. Do not go to sleep in a cramped position after decompression.
10. Do not allow yourself to become chilled within 6 hours after decompression.
11. Report at once to the physician incharge if you suspect you are suffering from "air pain" or decompression sickness. Do not give workers who are suffering from compressed-air illness any alcoholic drink.
12. If after decompressing, you develop 'niggles' or air pains that persists longer than a half hour, call the medical lock at once.
13. If you become ill away from the job site, communicate at once with the physician incharge.
14. Wear your identification bracelet so it will be known what to do with you in an emergency.
15. Stay within a 30-mile radius of the recompression facility for at least 1 hour after locking out.
16. Do not re-enter the man lock if suffering from 'air pains' or decompression sickness.
17. Do not fly in any aircraft for at least 12 hours after coming off shift.
18. Do not engage in any scuba diving (i.e. the simplest type of underwater breathing gear for the commercial diver, which is the self-contained rig consisting of bottles of air strapped to the driver's back and a demand regulator that supplies air to the diver via hose and mouthpiece at whatever pressure the person happens to be diving) at depths greater than 33 ft within 12 hours of coming off shift.
19. Do not engage in any scuba diving within 12 hours of going on shift.
20. Re-examination as per statutes.

The followings are the conditions that might disqualify a worker from compressed-air employment:

1. History of spontaneous pneumothorax.
2. History of ear surgery.
3. Active asthma.
4. Chronic embolism.
5. Addiction of narcotic or stimulant drugs.
6. Seizure disorders.
7. Pancreatitis.
8. Psychosis.
9. Gross obesity.
10. Advanced age.

Conditions that require close scrutiny:

1. Diabetes.
2. Hypertension.

3. History of thoracotomy.
4. History of fractures of the humerus or femur.
5. Migraine headaches.
6. History of taking systemic steroids.

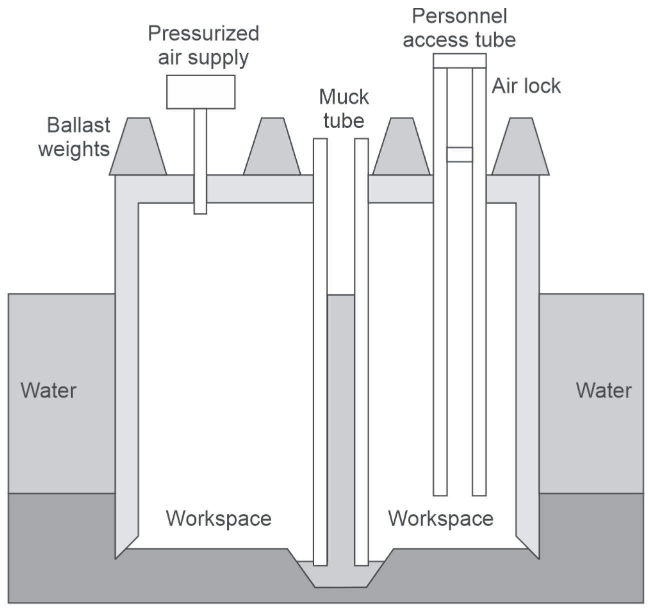

Fig. 1.14: Caisson engineering

Lifting of Loads in Industry

Lifting or carrying heavy loads, frequent bending or twisting, and heavy physical work may cause low back disorders. Lifting or carrying heavy loads has been linked to low back disorders in numerous publications. Epidemiological research has identified that the risk of injury increases when lifting loads from low heights, holding the load away from the body, or lifting while the torso is twisted or flexed. Biomechanical research supports these findings by demonstrating that loading of the spine increases in all of those conditions. Intervention studies have determined that lift tables, lifting hoists, lightening loads, and other engineering interventions can lower the risk of developing back injuries among workers.

The *revised lifting equation* results in two calculated values. The first is the *Recommended Weight Limit (RWL)* that corresponds to the AL in terms of acceptable weight of lift. The maximum possible RWL is 51 pounds. The second value is the *Lifting Index (LI)* that is defined as the actual weight lifted divided by the RWL. The LI gives a relative indication of the risk of injury associated with various lifting tasks. Available data does not allow prediction of the magnitude of risk for any individual or the exact percent of the work population who would be at an elevated risk for back injury as the LI increases above 1. The NIOSH perspective is that it is likely that tasks with a LI > 1 pose an increased risk of lifting related injury. Hence the goal should be to design all lifting jobs for LI of 1 or less.

As per Section 34 of the (Indian) Factories Act, 1948 (excessive weights):

1. No person shall be employed in any factory to lift, carry or move any load so heavy as to be likely to cause him an injury.
2. The State Government may make rules prescribing the maximum weights which may be lifted, carried or moved by adult men, adult women, adolescents and children employed in factories or in any class or description of factories or in carrying on in any specified process.

'Height Vertigo' or 'Height Phobia'

Vertigo can be described as abnormal perception of movement of the environment occurs as a result of a mismatch between the information about the person's position reaching the brain from the eyes. Vertigo is one category of dizziness (other categories are—faintness and miscellaneous head sensations). Vertigo is usually due to the disturbance in the vestibular system. Vertigo may represent either physiologic stimulation or pathologic dysfunction.

Physiological vertigo occurs in normal individuals when the brain is confronted with an intersensory mismatch among the three stabilizing sensory systems like car sickness, *height vertigo*, visual vertigo.

The most important diagnostic tool is a detailed history focused on the meaning of 'dizziness' to the patient. Is it faintness (presyncope)? Is there a sensation of spinning? Decrease in blood pressure, hyperventilation syndrome, hypoglycemia, somatic symptoms of a clinical depression, slight vestibular imbalance, etc. also can cause vertigo or other categories of dizziness. Appropriate investigations should be done to detect these disorders.

Emergency medical treatment of injuries and rehabilitation at construction site:

Trauma is the leading cause of death in most developed countries and is estimated to become the second or third most common cause of death across all age groups by 2020. Motor vehicle crashes are the most common etiology, accounting for the greatest number of injuries and deaths. Falls are the second most common cause of unintentional injury. It is clear that trauma is a large burden on the health care system.

Patients with blunt trauma frequently are encountered in the emergency department. The potential for early mortality and morbidity should be considered in all blunt trauma victims with a significant mechanism of injury. An organized assessment for life-threatening head, chest, abdominal, and pelvic injuries should be conducted with a recognition that definitive care may be provided after transfer to a certified trauma center. Proper stabilization measures including airway control, fluid resuscitation, and appropriate diagnostic studies should be performed prior to transfer. Airway control, assuring proper oxygenation and maintaining cerebral perfusion, are the hallmarks of early management of head injury. Attention to proper oxygenation and ventilation of patients with early tube thoracostomy is definitive in most patients with thoracic injury. Finally, pelvic stabilization and intravenous fluids are key management principles in abdominal and pelvic trauma.

Measures for Fracture

1. Do not move the injured unless the life is in danger from other cause.
2. Deal with the hemorrhage and breathing difficulties, if any.
3. Immobilize the fracture by the use of bandages using the injured body as support or by well-padded splint.
4. Immobilization should one point above and one point below. Remove the injured to the hospital.

Measures for Burn

1. Place the part under running cold water.
2. Cover the wound with several layer of sterilized cloth.
3. Artificial respiration if needed.
4. Immediate medical attention.

Measures for Wound

1. Stop the bleeding by anyone of the following methods.
   a. Direct pressure.
   b. Direct finger pressure into the wound in cases of larger bleeding wounds.
   c. Tourniquet (seldom needed)—use only as a last resort.

2. Avoid touching the wound with hands or unsterile material.
3. Clean the wound with running water and surrounding area with soap or spirit with clean gauze washing away from the wound. Apply ready-made adhesive gauze, bandage or sterile gauze and roller bandage as needed.
4. Keep the patient quiet; raising the extremity, if it is the bleeding part. Give no stimulants.

Cardiopulmonary Resuscitation for Unconscious Worker

Cardiopulmonary arrest is the abrupt cessation of spontaneous and effective cardiac output and ventilation. Cardiopulmonary resuscitation (CPR) is intended to provide artificial circulation and ventilation until other measures can be instituted so that spontaneous cardiopulmonary function is restored. Management of CPR is a team effort.

The scope of cardiopulmonary arrest and resuscitation has broadened enormously during the last two decades. Earlier it was confined to the management of unexpected catastrophes in hospital particularly in operation room. Subsequently it has embraced all the patients of 'sudden death' occurring both in and outside hospital with a systemic and uniform approach. CPR has now become a major public health endeavor involving a chain of personnel. The initial action starts by the lay persons around and is continued by paramedical personnel, nursing staffs, primary care physicians and finally by specialists and super specialists. Simple starting procedures 'mouth to mouth breathing' and 'cardiac massage', keep the patient alive and allow the further systemic approaches with equipment and drugs to act properly to give best resuscitation results.

CPR is further categorized as:

1. Basic life support (BLS).
2. Advanced cardiac life support (ACLS).
3. Post-resuscitation support (PRS).

Basic life support consists of:

1. Provision of a patent upper airway (A = airway).
2. Ventilation (B = breathing).
3. Well-maintained circulation of blood by closed chest cardiac compressions (C = circulation).

The ABCs of BLS can be instituted by any trained person without the need for specialized equipment. Regardless of the time from cardiopulmonary arrest to initiation of CPR, >6 minutes of closed chest cardiac compression is associated with increased neurological morbidity.

Provision of a Patent Airway

The aim of providing a patent's airways is to relive obstruction due to the tongue relaxing against the posterior pharynx. This is best relieved by head tilt-chin lift method.

Ventilation

Mouth to mouth breathing can provide adequate alveolar ventilation. Current recommendation: on initiating BLS, 2 breaths are given and then continued at a rate of 10 to 12 per minute. A single rescuer administering CPR should pause and give 2 breaths after each 15 chest compression (2 : 15). In two rescuers CPR, a single breath is to be given after every fifth chest compression (1 : 5). Exhalation can occur during the next compression.

Circulation

Circulation is provided by closed chest compressions and to be effective must be performed correctly. The most accepted theory (proposed) to explain how closed chest compressions cause blood flow through the circulatory system is 'cardiac pump mechanism' theory. This mechanism postulates that compression of the ventricles between the sternum and spine results in an increase intraventricular pressure, closure of the mitral and tricuspid valves and ejection of blood into the lungs and aorta. During the relaxation phase, negative intrathoracic pressure caused by expansion of the thoracic case, facilitates blood return and aortic pressure results in aortic valve closure and coronary perfusion.