Prevalence and Incidence rates

Prevalence rate = Number of persons with a characteristic / Total population .1

Prevalence rate is: All people with a health condition in a given population at a given point in time

Period prevalence rate = prevalence rate over a period of time

Incidence rate = Number of new cases of a disease / total population at risk per unit of time .2

Incidence rate is: All new cases of a disease during a given time

Prevalence Rate

The prevalence rate of a disease has the following characteristics?

*It measures all of the current cases of the disease in the community

* It depends on the duration of the disease process
* It depends on the incidence of the disease
* It can be used to determine the health care needs of a community
* P = I X D ; where P = Prevalence rate, I = Incidence rate, D = Duration of the disease.

Prevalence rate is equal to Incidence rate in case of diseases with short duration or highly fatal such as Rabies.

Occupational Health

Occupational health is concerned with the effects of work hazards on the health of workers.

Magnitude of the problem:

There has been a growing interest in environmental and occupational medicine since the end of the 19th century, as the industries began their steady climb to the "high-technology age" of today, with a rapid proliferation of new industrial materials, new production methods and new commercial products that lead to great adverse effects on the environment and on human health.

Workers are now exposed to a wide range of occupational diseases, work related diseases and work injuries.

Occupational disease: It is a disease that results from exposure to a specific causative agent in a work place. This causative agent could be: identified, measured and eventually controlled. (e.g. Silica→ silicosis; lead fumes → lead poisoning).

Work-related disease: It is an existing disease that may be aggravated, accelerated or exacerbated by work place hazards. (e.g. musculoskeletal disorders, diabetes, bronchial asthma).

Work injury: It is an injury arising out or in course of employment (e.g. mechanical, thermal, chemical injuries …….etc).

The most distinguishing feature of occupational hazards is that, in principle, they are preventable; therefore, there is an important role that can be played by occupational health professional both in early detection of work related medical problems and in taking appropriate measures to prevent them, as they constitute a great drain on national resources.

Occupational Medical Program: is a program that deals with the health of workers in relation to their work.

Objectives:

- **To protect workers against work place health hazards.**
- **To facilitate placement of workers.**
- **To assure adequate medical care and rehabilitation of workers with occupational disease or injury.**
- **To assist in measures of personal health maintenance.**
- **To protect the general environment of the country as far as practical.**

Activities of the program:

1) **Maintenance of a healthful work environment: The industrial safety engineer should perform periodic inspections of the different departments of the factory and evaluate the work environment in order to detect health hazards and provide basis for appropriate recommendations to improve control measures.**

2) **Pre-placement (pre-employment) examination: Each worker is subjected to a pre-placement examination before joining a new job. It helps in suitable placement of workers according to their physical, mental, and emotional capacities, so that workers can perform with an acceptable degree of efficiency and without endangering their own health and safety or that of others.**

Health assessment includes the following:

- **Personal, family and medical history.**
- **Detailed past occupational history.**
- **Complete physical examination.**
- **Laboratory investigations and screening tests which seem appropriate for the nature of the job.**

Objectives of pre-placement examination:

- **Proper placement of workers according to their medical and physical abilities to perform their job efficiently.**
- **Put a base-line of the health status of the workers at the start of work to be used in compensation claims.**

Proper job placement of workers clearly results in better performance, less absenteeism, lower accident rates, less chance of aggravating existing diseases and probably a longer production working life.

3) Periodic Medical Examination:

Periodic health evaluations are performed at appropriate intervals to:

- **Detect any early evidence of illness attributable to his employment e.g. early detection of occupational diseases.**
- **Evaluate control measures of the firm.**

Frequency of the examination will vary in accordance to:

- **The severity of exposure.**
- **The individual findings on each examination, thus, some exposed workers receive periodic examination every 6 months, while in other cases annual or biannual tests may be adequate.**

These periodic examinations concentrate on detection of effects of the present work, thus it includes:

*** Present history.**

*** Physical examination.**

*** Specific investigations according to exposure hazards (e.g. X-ray for those exposed to silica, blood count for those exposed to radiation).**

4) Diagnosis and treatment:

It is done for occupational and non-occupational diseases and injuries.

Diagnosis of occupational diseases depends on:

a) History: an occupational history of exposure to a hazardous agent or process.

b) Physical examination: symptoms and signs of the disease must coincide with the documented manifestations of the occupational disease.

c) There is improvement of manifestations away from work and aggravation on getting back to work.

d) Workplace environment: samples and measurements taken from the workplace environment should indicate that the causative agent is present in a sufficient concentration to produce the disease.

e) Surveying: reveals that workers under the same work condition may have similar manifestations.

Emergency care and treatment should be given immediately in the workplace and facilities for rapid transfer to specialists and hospitals should be available.

5) Immunization programs:

Immunization procedures should be available to workers who are exposed to significant hazards against which immunizations are available (e.g. anthrax, tetanus, tuberculosis, hepatitis …etc).

6) Medical records: It is important that a good medical recording system is maintained in any occupational health program. Every employee should have an accurate and complete medical report from the line of his first medical pre-placement examination. The records must be detailed enough to provide adequate information about job placement, health maintenance, rehabilitation and compensation.

7) **Health education and counseling: Occupational health personnel should educate employees about personal hygiene, health maintenance and prevention of occupational diseases and injuries.**

There are many situations during which health education can be done:

- **During periodic medical examination.**
- **On reviewing laboratory test results.**
- **During treatment.**
- **At a time of specific enquiry by employees.**
- **On a request of work group.**
- **On introduction of a new process or a new hazardous material.**

Successful health education will improve safe working habits and will reduce both the rate of lost time and that of minor accidents.

Staffing and Organization: The occupational health team includes the following personnel:

- **Industrial physicians.**
- **Occupational health nurse.**
- **Industrial safety engineer.**

Role of industrial physician:

The industrial physician is the key person in the occupational health team. His duties can be summarized in the following points:

- **Perform the pre-placement medical examination.**
- **Perform the periodic medical examination.**

- **Emergency treatment of injuries.**
- **Diagnosis and treatment of occupational diseases.**
- **Rehabilitation of diseased workers.**
- **Assessment of degree of disability following occupational diseases and injuries and calculate the required compensation.**
- **Treatment of mild non-occupational conditions.**
- **Referral of chronic non-occupational diseases to a specialist.**
- **Health education.**

Role of occupational health nurse:

An occupational health nurse should have special training in interviewing and dealing with workers. The duties of an occupational health nurse are:

- **Initiation of treatment in emergency situations.**
- **First aid treatment of minor injuries.**
- **Helping the physician during pre-placement and periodic medical examination.**
- **Keeping medical and environmental records.**
- **Health education.**

Industrial safety engineer:

His duties include:

- **Recognition, evaluation and control of workplace hazards.**
- **Discovering unsafe machinery.**
- **Engineering control (e.g. barrier guards, warning signs, enclosure).**
- **Supervise material handling equipments.**

- **Investigate accidents.**
- **Fire protection.**

Occupational Hazards

The following is a broad classification of different hazards that may be encountered in a workplace.

I- Chemical hazards: Chemicals (organic or inorganic) in a workplace which are able to produce harm to the human body may take the form of gas, vapor, fume, dust, mist, smoke, fog, smog, …etc. These chemicals (e.g. lead, mercury, cadmium, silica, asbestos….etc) are capable of causing diseases in different systems of the body.

II- Physical hazards: Exposure to adverse physical environment can harm the health of exposed workers. E.g. exposure to noise, extreme temperatures (hot or cold), changes in atmospheric pressure, vibration, radiation …etc.

III- Biological hazards: Exposure to living organisms in the workplace can lead to increase risk of infection with certain diseases in exposed workers e.g.:

a) Health care workers (physicians, nurses, laboratory technicians... etc) have high risk of developing certain diseases e.g. Hepatitis B, Tuberculosis, AIDS…etc.

b) Non-Health care workers (e.g. farmers, butchers, veteranians) have the risk of catching zoonotic diseases such as brucellosis, anthrax, histoplasmosis …etc.

IV- Mechanical hazards: In almost all factories workers deal with moving objects and vibrating tools at different speeds, in addition they carry and manipulate heavy objects. Exposure to these circumstances among others, could lead to accidents, musculoskeletal disorders…..etc.

V- Psychosocial hazards: The mental and physical condition of the worker as well as his abilities to function and produce properly can to a great extent be affected by his relationship with other workers and supervisors, and his ability to accept and comply with factory system and regulation.

I-Chemical Hazards

The following are sound occupational diseases due to exposure to hazardous chemical contaminants in the workplace:

1) Chronic lead poisoning:

Inorganic lead is not only one of the most important heavy metals used in industry but also an environmental pollutant which has a deleterious effect on human health.

Sources of Exposure:

a) Occupational Exposures: Exposure to lead in different industries such as storage batteries, rubber, paints, ink, ceramic and pottery industries.

b) Environmental Exposures: It occurs by drinking water running in old lead pipes or inhaling automobile exhaust (tetraethyl lead is added to gasoline as antiknock agent).

Metabolism: After being absorbed in the human body through ingestion or inhalation, more than 90% is carried by the blood in the erthrocytic membrane and the rest is carried out by the plasma.

Lead is then distributed in many organs as liver, spleen, lungs, and kidneys.

Excretion of lead occurs continuously in the stools and urine even after stoppage of exposure.

After a time, lead is redistributed and deposited in the bone cortex in the form of lead sulfate and becomes less mobilized.

Clinical Picture: Chronic lead poisoning (plumbism) manifests itself by multiple and variable symptoms and signs which depend on the degree of exposure, the type of exposure and individual susceptibility.

***General: Weakness, lassitude, sleep disturbance, musculoskeletal joint pain, neuromuscular in coordination.**

***GIT: Anorexia, constipation, abdominal colics.**

***CNS: Loss of concentration, irritability, tremor and in late cases in the form of paresis and encephalopathy.**

***CVS: Hypertension or hypotension.**

As lead pass through the blood placenta barrier affection of the fetus occurs in the form of congenital anomalies, if pregnant females are chronically exposed to lead.

Diagnosis: As in other occupational diseases the diagnosis depends upon the following criteria:

1- History of exposure to lead in one of the industries in which lead is used permanently.

2- Clinical picture:

***Symptoms and signs of organ or system**

Affected.

***Lead line (bluish black line in the gingiva) is not pathognominic to lead toxicity, as its presence depends mainly on the poor oral hygiene of most of industrial workers.**

3- Investigation:

a) Blood lead:

***Blood lead level of > 80 µg/dl is indicative of toxicity.**

***Blood lead level of 40-80 µg/dl is indicative of harmful chronic exposure.**

***Blood lead level of 52 µg/dl is indicative of stoppage of exposure to prevent toxicity.**

b) Hemoglobin: decrease Hb level < 12 gm%.

c) Basophile stippling of RBCs and decrease in their number (anemia).

d) Increase in Zn protoporphyrin as a result of altered heme synthesis.

e) Urine:

***Increase delta aminolevulinic acid (δ ALA).**

***Increase lead in urine > 80 µg/day.**

Prevention and Control:

Medical Measures:

1- Pre-employment examination to exclude those with blood, kidney, liver or neuromuscular diseases.

2- Periodic medical checkup by clinic examination and investigations to early detect lead effects before reaching the toxicity level.

Workers reaching the toxicity level must be excluded from work and subjected to treatment in the form of Ca-EDTA, BAL {British anti Lewisite chelating agents (Dimercaprol)}.

Penicillamine is the drug of choice in mild toxicity.

2) Chronic Mercury Poisoning:

Mercury is a silvery white metal, peculiar in being liquid at ordinary temperature and evaporates at room temperature.

Sources of Exposure:

Mercury compounds are distinguished into two major types:

***Organic mercurials used in pesticides and seed dressings to prevent spread of fungal seed diseases.**

***Inorganic mercurials used in manufacture of electric apparatus, mercury vapor lamps, switch gears and dry batteries, also used in medical and scientific equipments: sphygmomanometer, thermometers and barometers.**

It is used in amalgam fillings in dentistry. Mercury is used as the cathode in electric cells used in chlorine industry. Mercury is also used in explosives and antifouling paint for the ship's bottom.

Metabolism:

Mercury could be absorbed through the respiratory tract by inhalation, through gastrointestinal tract by ingestion (in workers with poor personal hygiene), also it could be absorbed through sweat pores and hair follicles in the skin.

After absorption, organic mercury is carried by the blood (RBCs and plasma in a ratio of 10:1) and can cross placenta and blood brain barrier in contrast to inorganic mercury which is carried also by the blood (RBCs and plasma in a ratio of 1:1) but can barely cross the placental barrier or blood brain barrier).

Mercury is distributed in human organs, but the kidney is considered as the chief depositary organ, nervous system comes second specially the sensory ganglia of the peripheral nerves.

Mercury excretion is via the urinary system mainly but also it is excreted through the GIT.

Considerable amount of inorganic mercury is excreted in the saliva with about 20% concentration of that in the blood.

Clinical Picture:

The onset of manifestations in chronic industrial poisoning is slow and insidious. The nervous system, the kidneys and the mouth are the mostly affected.

***Nervous System: Tremors affecting small muscles (disturbed hand writing), sensory motor polyneuropathy in the form of numbness, paraesthesia, impaired touch pressure sensation and reduced tactile discrimination. Deep reflexes are exaggerated and lastly wasting of the muscles.**

***Kidneys: Glomerular and tubular affection result in proteinuria, polyuria followed by oliguria and lastly chronic renal failure.**

***Mouth: Excretion of mercury in the saliva result in gingivitis, easily bleeding gums, appearance of bluish grey line on the gingiva resulting from combination of mercury in the saliva with hydrogen sulphide from food ferment and excessive salivation. Other organs are also affected:**

***Eye: Discoloration of the lens (mercurialentis).**

***Cardiovascular system: Arrhythmia.**

***Hematologic system: Anemia with leucocytosis.**

***Psychological system: A syndrome called "erethism" consisting of anxiety, loss of self confidence, fear and sometimes attacks of depression.**

Medical Definition of ERETHISM

"Abnormal irritability or responsiveness to stimulation"

Diagnosis:

***History of occupational exposure to mercury.**

***A positive diagnosis of mercury poisoning could be arrived at if tremors and at least two of the following manifestations are present: Gingivitis, exaggerated tendon reflexes, personality changes, excessive salivation or disturbed sleep.**

***High blood or urine mercury level is confirmatory.**

Prevention and control:

Medical Measures:

1- Pre-employment examination to exclude those with kidney, blood or neuromuscular disease.

2- Periodic medical examination: By clinical examination and urine mercury determination.

Workers suffering from mercury toxicity must receive chelating agents in the form of BAL and N-acetyl 1 D penicillamine for mobilization of mercury from the target organs, copper and zinc supplements are essential.

Engineering Measures:

1- Replacement of mercury if possible.

2- Local exhaust suction of mercury vapor.

3- Environmental monitoring of mercury vapor.

Personal Measures:

1- Wear of mask as a personal protective measure.

2- Health education about sources of Hg exposure and ways of prevention and control.

3- Byssinosis:

Bissinosis has been described in all countries of the world where cotton, flax, and hemp are spun or processed. A significant prevalence of byssinosis has been reported from Spain, Egypt and the Netherlands during the past 4 decades.

Occupational Exposure:

Workers involved in cotton and flax spinning especially during bale opening and during carding of cotton or flax (i.e. removing impurities from cotton and flax aligning its fibers).

N.B. cotton dust consists of broken cotton fibers bacteria fungi and minerals.

Pathogenesis:

It is a form of occupational asthma, but the exact mechanism responsible for bronchospasm is not certain.

Many theories have been put forward to explain the air way changes:

a) Pharmacologic theory: where cotton dust has a direct effect on mast cells in the bronchial tree leading to release of histamine and thus broncho-constriction.

b) Allergic theory: where an antigen-antibody reaction takes place, precipitating IgG antibodies were detected in exposed workers and a type III reaction has been postulated.

c) Bacterial endotoxin theory.

d) Fungal enzymes theory.

Diagnosis:

1- Occupational history of exposure to cotton, flax, or hemp dust for about ten years.

2- Symptoms and signs: Acute symptoms consist of chest tightness and breathlessness and sometimes cough which develop during the afternoon of a work day, although in severe cases they occur a few hours after starting work in the morning.

Clinical Grades: Prefixed by "C"

***Grade C'/2: Occasional chest tightness on the first day of the working week (Saturday dyspnea).**

***Grade CI: Chest tightness and/or difficulty in breathing on every first day of the working week (Saturday dyspnea).**

***Grade C2: Chest tightness and/or difficulty in breathing on the first day and other days of the working week.**

***Grade C3: Grade C2 symptoms accompanied by evidence of permanent respiratory disability from reduced ventilatory capacity.**

In C3 although some relief may be experienced when they leave the industry, this is incomplete and respiratory disability is permanently established. The first three grades are reversible.

3- Investigations:

a) Chest X-Ray: Byssinosis is not characterized by any abnormality of the chest radiography.

b) Lung functions: spirometry reveals a variable obstructive pattern of impairment (diagnosed by a fall in FEV1% i.e. FEV1/FVC is less than 75% of the predicted normal value) which is reversible in early stages of disease and become fixed later on in the advanced stage (C3).

Prevention:

a) Medical Measures:

1. Pre-employment examination: In addition to respiratory history, physical examination, spirometry and chest x-ray, details of atopic family history, personal history of allergy and asthma should be included for each case.

2. Periodic examination: It should be done every year using forced spirometry. If a significant decrease occurs the worker should be transferred to a less dusty area. However, during the first month of employment the workers FEV1 should be recorded before and after six hours of commencing his shift on the first day of working week. If a significant decrease occurs he should be transferred to a less dusty area.

Engineering Measures:

a. Dust control by:

*** Spraying of ripening cotton with bactericides and fungicides.**

***Treatment of raw cotton with gaseous hydrogen chloride or acetic acid as acids has been found to inactivate the active component in cotton bracts and dust.**

b. Personal Protective Equipment (PPE); i.e. masks.

4) Pneumoconiosis (or dusty lung):

This term is usually restricted to conditions where a permanent alteration in lung architecture is detected due to the inhalation of mineral dust (e.g. silica, asbestos, coal).

A) Silicosis:

Definition: Silicosis is the parenchymal lung disease caused by inhalation of particles of crystalline silicon dioxide (SiO2) (Synon: Quartz, Silica).

Occupational Exposure:

Silicon dioxide is widely deposited in the rocks that make up the earth's surface. Industrial activities that involve cutting, polishing or shearing rocks are thus all potential sources of respirable silica, these include mining, tunneling, quarrying and stone cutting. Industrial use of sand, which is largely composed of quartz, can lead to exposure to high concentrations of respirable silica, especially the use of sand for abrasive blasting. Sand is also widely used in foundry work glass blowing and pottery making.

Types: There are three different forms of silicosis, roughly related to the intensity of exposure to respirable silica:

***Chronic silicosis: Is defined as radiographic abnormalities that are first noted 15 years or more after the onset of exposure.**

***Accelerated silicosis: Resembles the chronic disease but occurs 5 to 15 years after the onset of exposure to high concentrations of silica.**

***Acute silicosis: Occurs within 5 years of the onset of exposure, it is always caused by massive exposure, and is clinically and pathologically quite different from the other two forms.**

Pathogenesis: The mechanism by which silica dust produces the characteristic fibrotic lesions in the lung is still not known. The most accepted theory is that tissue injury from silica particles is initiated by the interaction between silica crystals and alveolar macrophages. Macrophages stimulated by silica secrete factors that are chemotactic for other macrophages and neutrophils that stimulate fibroblasts to proliferate and lay down collagen.

Clinical Picture: In chronic silicosis, the development of small nodular opacities is not associated with symptoms or signs, late in the course of the disease when fibrosis is advanced or Progressive Massive Fibrosis (PMF) has developed, the patient may complain of breathlessness, chest tightness, and dry non-productive cough. If hemoptysis occurs it is always suggestive of

Tuberculosis as patients with silicosis have an increased susceptibility to tuberculosis mycobacterium infection.

Signs: In advance cases, lung expansion may be decreased by 20 or 30 percent and remains equal on both sides.

Diagnosis: The diagnosis of silicosis is based on the following:

1. Occupational history of exposure to silica dust for several years (usually 10-15 years).

2. Chest radiography: Radiological changes are present before the appearance of symptoms or clinical signs. Chest radiographs usually show multiple small nodules distributed throughout both lung fields, but more marked in the upper zones. Hilar lymph nodes may be enlarged and may contain outer rims of calcium giving a pathognominic feature called

"Egg-shell calcification".

In patients with PMF, large masses are usually seen in the upper lobes, often symmetrically distributed around the hilar regions in the so called "angel's wing" distribution. In these patients compensatory emphysema is also common.

3. Respiratory functions: Pulmonary function tests are usually normal in patients with "simple silicosis", they are not helpful in establishing the diagnosis. Pulmonary function tests are useful in evaluating and quantifying pulmonary impairment in symptomatic workers. When abnormalities are found they are usually those of a restrictive pattern due to the fibrotic changes in the lung parenchyma.

Prognosis: Silicotic fibrosis will progress even after cessation of exposure to silica dust. Silicosis may be complicated by chronic bronchitis, emphysema,

Cor-pulmonale and tuberculosis. Lung cancer is not proved to be a complication of silicosis.

Treatment: There is no proven effective treatment for any form of "silicosis", once a worker has "silicosis", there is no treatment that will affect the course of the disease, and its rate of progression will vary from case to case and continue even after cessation of exposure. However, complications of silicosis can be treated.

The major hope for reducing the prevalence of silicosis lies with prevention. Industrial processes that generate respirable free silica should be enclosed or modified. Dust containing silica should never be swept dry, and workers should always use personal respiratory protective devices for unavoidable short-term exposures.

B) Asbestosis:

Definition: The term "asbestosis" means the response of the lung parenchyma to inhaled asbestos fibers.

Occupational Exposures: Worldwide use of asbestos increased dramatically throughout most of this century. Asbestos fibers are widely used in shipbuilding, construction insulating and brake manufacturing as they are highly resistant to heat, acid and chemical degradation.

Pathogenesis:

Pleural affection: Asbestosis can cause localized or diffuse areas of a cellular pleural fibrosis that are usually bilateral and primarily on the parietal pleura.

Asbestosis is indistinguishable from other causes of pulmonary fibrosis, except for the presence of asbestos fibers. A small percentage of asbestos fibers become coated with hemosiderin and form asbestos bodies. Presence of asbestos bodies in the sputum is an evidence of exposure to asbestos but it does not imply disease. Malignant pleural mesotheliomas may be present and lung cancer can be of any cell type.

Asbestos fibers appear to cause tissue injury by simulating alveolar macrophages to secrete cytotoxic materials, inflammatory cell chemo attractants and factors that stimulate fibroblast proliferation. Because of their

durability, individual fibers can repeatedly stimulate macrophages for many years without being degraded. This helps to explain the continued progression of asbestos induced disease after cessation of exposure.

Clinical Manifestations:

Asbestosis presents as do other forms of pulmonary fibrosis, with dyspnea that is initially most prominent with excretion and is often associated with cough. Bibasal rales are a common finding and clubbing can occur.

Diagnosis:

1. A positive history of exposure to asbestos fibers (10-15 years).

2. Chest radiograph: It reveals linear and irregular opacities that are most prominent in the lower lung fields. Pleural plaques are often found.

3. Respiratory functions: In patients with early asbestosis, ling function studies may be normal, later a progressive restrictive pattern is noticed.

Treatment: There is no proven effective treatment for asbestosis. The major strategy for prevention is worldwide elimination of asbestos use and replacement with synthetic substitutes that appear to be considerably less toxic. Continuing exposure to asbestos presently in use needs to be minimized by use of engineering controls, personal protection (PPE) and public education.

Physical Hazards

Exposure to adverse physical environment can harm the health of exposed workers. E.g. exposure to extreme temperatures (hot or cold), changes in atmospheric pressure, vibration, radiation, exposure to noise…..etc.

I. Exposure to Extremes of temperature:

A. Hyperthermia (Exposure to Heat)

Occupational Exposure: Workers in many occupations may be exposed to heat e.g. smelters, steelworkers, glassblowers, furnace operators, farmers, fishermen and construction workers.

Heat Disorders: Five medical disorders can result from excessive exposure to hot environments (in order of decreasing severity):

1. **Heat stroke**
2. **Heat exhaustion**
3. **Heat cramps**
4. **Heat syncope**
5. **Skin disorders**

1. Heat Stroke.

*** It is a life threatening medical emergency due to failure of the heat regulating mechanism. Heat stroke becomes imminent as the core temperature approaches 41.1 C. It usually follows excessive exposure to heat.**

*** Persons at risk are non-acclimatized workers performing tasks that require strenuous exertion, the elderly or those receiving medications (e.g.anticholinergics) that compromise heat dissipation mechanism.**

*** Manifestations of heat stroke are: hot dry skin hypertension, hyperventilation, nausea, vomiting, confusion, convulsions and unconsciousness may occur.**

Prevention:

- **In occupations in which workers are exposed to excessive heat, medical evaluation is recommended to identify individuals at increased risk for heat disorders.**
- **Workers should be trained to recognize early signs and symptoms of heat disorders and should be advised of the importance of proper clothing, nutrition, and fluid intake.**
- **Environmental monitoring by a wet bulb globe thermometer (WBGT) and shaded rest areas should be available.**

Treatment:

- **Patient should be hospitalized**
- **Lowering body temperatures by all means e.g. remove clothing, entire body spraying with cool water and blowing cool air across the patient at high velocity.**
- **Give fluids**
- **Respiratory and cardiovascular stimulants.**

2. **Heat Exhaustion. Is due to prolonged exposure to heat and inadequate salt and water intake.**

- **Manifestations: Intense thirst, weakness, increased pulse rate, moist skin, core temperature 38 C. Progression to heat stroke may occur and is manifested by a rise in temperature or a decrease in sweating.**
- **Treatment:**

- **Place the worker in a shaded place**
- **Provide salt and water orally if the worker is able to swallow**
- **Intra Venous (IV) physiologic saline and solution in severe cases.**

3. **Heat cramps:**

Muscle cramps which occur during or after exercise in a hot environment, they are due to sodium depletion caused by replacing sweat losses with water alone.

- **Manifestations:**

- **Spasm that last from 1-3 minutes in the muscles in strenuous work. The involved muscle feels like hard stoned humps similar to billiard balls.**
- **Body temperature may be normal or slightly increased.**
- **Blood tests may show low sodium levels and hemoglobin concentrations.**

- **Treatment:**

- **Place the worker in a cool environment.**
- **Balance salt solution or oral salt solution consisting 4 teaspoons of salt per gallon of water.**

4. **Heat Syncope: It is a sudden fall in blood pressure associated with loss of consciousness. Sudden episodes commonly occur following work for at least 2 hours, due to cutaneous vasodilatation.**

- **Manifestations:**

- **Cool moist skin.**
- **Systolic blood pressure less than 100 mm Hg.**

- **Treatment: Recumbency, cooling and liquids (orally).**

5. **Skin Disorders:**

- **Heat rash: caused by sweat retention due to obstruction of sweat gland ducts.**
- **Heat urticaria: localized or generalized and is characterized by the presence of wheals with surrounding erythema.**
- **Burns.**

B. Hypothermia (Exposure to cold):

The condition is less serious than exposure to heat and easier to control.

Occupational exposure: Workers at risk include both indoor and outdoor workers exposed to cold such as: meat packers and others who work with freezers, construction workers, divers, mail carriers and road-maintenance workers.

Cold injuries can be categorized as systemic or localized and as freezing or non-freezing.

Systemic hypothermia: Is reduction of the body core temperature below 35 C. The onset of hypothermia is often insidious without any specific characteristics. With profound hypothermia, there is often diminished memory and absence of shivering.

- **Prevention:**

-Workers exposed to cold should be medically examined to identify those at increased risk for hypothermia.

-Workers should be trained to recognize early signs and symptoms of hypothermia and should be advised of the importance of proper clothing, avoidance of smoking, drugs or alcoholic drinks.

- A proper work schedule with adequate rest periods and heated rest facilities should be available.

- **Treatment:**

- **Re warming by external or internal techniques.**
- **Cardiac rhythm and rate should be monitored because the risk of death due to ventricular fibrillation is high with severe hypothermia.**

Localized hypothermia: It affects extremities when rapidly exposed to excessive cold.

a. **Freezing injuries. E.g. Frost bite: The cheeks, nose, ear lobes, fingers, toes, hands, and feet are the area's most likely to develop ice crystals within the tissue, resulting in localized hypothermic injury with symptoms of numbness, prickling and itching, in severe cases there may be parasthesias and stiffness. Skin is often white and edematous.**

b. **None freezing injuries.**

Chilblains: red itchy skin lesions may be initially associated with edema and blistering and then progress to ulcerative or hemorrhagic lesions which can cause scarring, fibrosis, or atrophy.

Immersion foot: is caused by combination of cold temperature and exposure to water. Initially feet are cold, swollen and waxy white or cyanotic. 2 to 3 days following removal from the cold, hypothermia occurs with severe pain, additional swelling and complications as gangrene, cellulitis or thrombophlebitis. After 10-30 days intense parasthesias may occur accompanied by cold sensitivity which may persist for years.

- **Prevention:**
  Same as for systemic hypothermia.
- **Treatment: Gradual rewarming and protecting pressure sites from trauma.**

II. Atmospheric pressure disorders (Dysbarism)

Increase or decrease in the surrounding atmospheric pressure causes compression and decompression sickness.

Decompression sickness. Occurs due to sudden shift to an environment of lower atmospheric pressure (as occurs during rapid ascent to the surface from deep sea diving or with loss of cabin pressure while flying at high altitudes).

Mechanism: decompression sickness results from mechanical physiologic effects of expanding gases and bubbles in blood and tissue. When the body is exposed to an environment of high atmospheric pressure (i.e. compression, as in tunneling or diving), it absorbs more of the inhaled gas and nitrogen concentrations increase in the tissues particularly those of the nervous system, bone marrow and fat. As the surrounding pressure decreases (i.e. during decompression, as in ascent to the surface of water), nitrogen expands and forms gas bubbles if there is insufficient time for its dissolution from tissues. Oxygen and carbon dioxide have greater fluid solubility and move easily between tissue compartments so their tendency for bubble formation is reduced.

Most cases of decompression have occurred after rapid ascent from sea depths in excess of 9 meters.

Manifestations: There are 3 types of decompression sickness. The type and severity of symptoms depend on:

- **Age, weight and physical condition of the worker.**
- **The depth before decompression.**
- **Rate and duration of decompression.**

Type I decompression sickness: Acute pain around joints, may be incapacitating and causes the patient to assume a stooped poster (The bends). It may begin immediately after decompression or within 12 hours later.

Type II decompression sickness: Gas bubbles in the central nervous system may lead to vascular obstruction and tissue infarction. Pulmonary manifestations as severe cough, dyspnea, and substernal pain due to air embolism may occur (The chokes).

Type III decompression sickness: Aseptic bone necrosis (Osteonecrosis) which frequently involves the head of humerous, femur, and the tibia head. It usually occurs 6-60 months following decompression and is asymptomatic unless there is a joint involvement which can cause a permanent impairment; it is the result of nitrogen bubble obstructing the capillaries and is reported in 50% of divers and underwater workers.

Prevention: Divers, under water workers and pilots should undergo the following:

- Screening to make sure they are in good physical condition (i.e. not overweight, with no other conditions imposing an increased risk for dysbarism, such as vascular disorders, obstructive air way disease, dehydration or recent bone fracture).

- Training in proper compression and decompression procedures.

- Education in recognizing early symptoms and signs of decompression sickness.

III. Noise

Noise has been known since the creation of earth (the great explosion). It is a health hazard created by man. With the progression of the industrial revolution and increased mechanization, deafness of workers started to become an important health problem.

The simplest definition of noise is "unwanted sound". Any vibrating body produces waves of varying amplitudes and frequencies with alternating refraction and compression. The intensity of sound which is a function of amplitude is measured in discibles (dB), the frequency of sound or (pitch) is measured in cycles per second or in hertz (Hz).

Occupational Exposure: Workers are exposed to noise in many workplaces such as in textile, plastic and petroleum industries, shipyards, construction and car manufacture.

No hearing loss occurs with lifetime exposure to noise intensity below 85 dB, so exposure to noise of intensity 85 dB or more needs protection.

Health effects of noise are classified to:

a. Non-auditory effects:

- **Increased rate of injuries.**
- **Increase rate of absenteeism.**
- **Increase rate of turnover.**
- **Arrhythmia and hypertension.**
- **Increased level of catecholamines.**
- **Interference with communication.**
- **Annoyance.**

b. Auditory effects:

- Air conduction deafness:

- **History of exposure to sudden intense noise at the work place e.g. explosion**
- **Symptoms and signs: intense ear pain with decrease in hearing acuity. The air drum is ruptured and ossicles may be dislodged. Deafness is usually unilateral.**
- **Investigation: audiogram will show unilateral conductive deafness.**

- Nerve deafness:

- **This is the most common type of hearing loss. It may progress insidiously while the worker is unaware of the situation until it reaches the speech frequencies (500 Hz, 1000 Hz, and 2000 Hz).**
- **History: prolonged exposure to continuous noise of hazardous intensities (above 85 dB).**
- **Symptoms and signs: at first hearing loss is confined to sounds of high pitch e.g. doorbell or telephone ring, later on, lower tones become involved and the worker does not hear what people are saying.**
  In the early stages hearing loss is temporary and tends to disappear after a rest period. This is called Temporary Threshold Shift (T.T.S) of hearing which is a characteristic manifestation of acoustic fatigue, and differs in individuals as there are marked differences in susceptibility to noise.
  Continued exposure of susceptible individuals leads to permanent hearing loss. This is called Permanent Threshold Shift (P.T.S) of hearing. The ear drum is usually intact.
- **Investigations: Audiogram reveals bilateral nerve deafness. The early hearing loss (either TTS or PTS) tends to begin at the frequency of 4000 Hz, forming a**

V.shaped notch which is a characteristic feature of noise induced nerve deafness. As time goes on, lower frequencies are involved to reach speech frequencies and the notch becomes deeper and wider.

Factors affecting the severity of hearing loss:

- **Intensity of noise.**
- **Duration of exposure to noise.**
- **Individual susceptibility.**
- **Age of the worker**

Prevention and control:

a. Medical measures: include the following:

- **Pre-employment examination: to select non risk workers.**
- **Periodic medical examination: for early detection of noise susceptible workers and early management of noise induced hearing loss.**

b. Engineering measures: noise control is the most effective measure, and this is through:

- **Design and selection of machines with low levels of noise.**
- **Replacement of noisy machine parts with less noisy ones.**
- **Proper and regular maintenance of machines.**
- **Enclosure of noisy machines if possible.**
- **Use of sound proof materials for surfaces and walls, if needed.**

C. Personal protective equipments: use of ear muffs or ear plugs. These should be appropriate for noise frequency and intensity and should be able to attenuate the intensity of noise to about 80 dB.

III. Mechanical Hazards:

Work Injuries: Occupational injuries constitute a significant proportion of the overall injury problem and are estimated to account for one sixth of all fatal injuries to persons between 18 and 60 years yearly. The direct cost of work injuries is estimated as several billions of dollars. The social burden of work injuries is difficult to be estimated. However, disabled workers, instead of being wage earners, must be supported financially. This will affect the entire family members.

Occupational injury is defined as an injury arising out of or in the course of employment. Recordable occupational injuries are those necessitating absence of one shift other than the one during which the injury occurred. The term injury is now replacing the word accident which is reserved only for unexpected, unplanned, unintended events which in this definition is rare. However, unfortunately the term accident still can be found in some references.

Injuries can be classified according to the following:

A) Intent: All occupational injuries are unintentional.

B) Nature or type of injury: This includes: sprains, strains, cuts, lacerations, punctures, contusion, crushes, fractures, drowning, burn, suffocation, amputation, enucleation, etc.

C) Body part affected: define the exact affected body part: finger, hand, wrist, arm, nose, toe, foot, etc.

D) Mechanism of injury (External cause of Injury): This includes falling from height, from the same level, struck by or against, caught in, under, or between, motor vehicle injury, rubbed or abraded, temperature extremes, overexertion, bodily reaction, etc.

E) Activity during injury: walking, sitting, running, lifting, etc.

F) Unsafe act: like none use of personal protective equipment, bad hand grips, leaning over machines, non warning of others, etc.

G) Source of injury: machines, ground, working surface, ladder, hand tool, etc.

Environment hazard: hazardous environmental conditions present during the injury like bad lighting, excessive noise, etc.

I) Place of injury: the exact place where the injury occurred is specified.

Risk Factors for Injury:

A) Machines: Unsafe guarding of machines and poor design definitely will increase the risk of injury.

B) Personal Factors:

1- Age: in the productive age group, injuries generally affect the younger. This might be attributed to lack of experience of these workers. Before the age of 60 (retirement age) the motor and sensory capabilities of the workers are not greatly affected yet.

2- Long years of experience and safety training can reduce injury risk.

3- Sex: Injuries are more common among males in general. In industry this might be due to differences in exposure risk; as few females are engaged in high risk jobs.

4- Medications: drugs such as tranquilizers, barbiturates and antihistamines increase the risk of injury as they interfere with speed of responses and performance.

5- Addiction: Hashish, opium, bango, heroin, in addition to alcohol affects time and space orientation so they increase the hazard of injury.

6- Acute illness and fatigue can also increase injury risk.

C) Environment and Social Factors:

1- Bad housekeeping and poor layout will increase injury risk.

2- High level of noise, extremes of temperature, bad ventilation, poor lighting, and excessive indoor air pollution may predispose to injury.

3- Job satisfaction and good relation among workers, supervisors, and the administration will improve the moral of workers and reduce the injury hazard.

4- Work schedule: duration and distribution of rest periods and shift work.

5- Lack of recreational and leisure activities.

Accidents Rates:

Several rates have been used to reassure accidents such as accident incidence rate and accident were designed in order to:

***Identify factors responsible for accidents.**

***Provide data for comparisons between industries and countries.**

***Evaluate preventive measures.**

Injury Control: In general, injury prevention can be classified under the following headings: Environmental measures, Engineering measures, Educational measures, and Enforcement (Legislative) measures. One practical approach for injury prevention was developed by Haddon. It is a Haddon's matrix which is composed of two dimensions: the dimension is time and the second dimension is the adopted control measure.