CLINICAL FINDINGS

There are three types of decompression sickness as described below. The type and severity of symptoms will depend on the age, weight, and physical condition of the pacient, the degree of physical exertion; the depth or altitude before decompression; and the rate and duration of decompression.

TYPE 1. Acute pain, usually around a major joint, may be incapacitating and cause the patient to assume a stoped posture ("the bends"). Pain may begin immediately after decompression or up to 12 hours later.

TYPE 2. Symptoms and signs of central and peripheral nerve damage may include vertigo, "pins and needles", Babinski's sign, paralysis of weakness of the limbs, headache, seizures, vomiting, visual loss or visual field defects, incontinence, impaired speech, tremor, and coma.

TYPE 3. It is characterised by aseptic necrosis of bone (osteonecrosis) which frequently involves the head or shaft of the humerus and less often the lower and of the femur and the tibial head.

COMPRESSION SICKNESS

When athmospheric pressure is increased, internal gases become compressed, usually with little effect.

The only common form of compression sickness is **barotitis**. This can occur with descent of an aircraft from a high altitude, which causes a relative vacuum in the middle ear space if the auditory tube is already obstructed due to allergies or upper pain or a foggy feeling in the ears, dizziness, tympanic membrane may appear inflamed and retracted or ruptured.

Barotitis can be prevented in people at risk by avoiding high-pressure exposures or, for short exposures, by using decongestants. Barotitis is usually self-limiting but can be treated with decongestant nose drops, a nasal vasoconstrictor inhaler, or use of Valsalva's maneuver.

1.3.4. INJURIES DUE TO RADIATION

INJURIES DUE TO INFRARED RADIATION

Infrared radiation covers of the electromagnetic spectrum between visible and RF radiation. It has wavelengths between 750 and 3 million nm and is composed of three spectral bands -A, B, C- which begin at 750 nm, 1400 nm, and 3000 nm, respectively.

Infrared radiation is given off from any object having a temperature greater than absolute zero.

Acute, high-intensity exposure to wavelengths shorter than 2000 nm can cause thermal damage to the cornea, iris or lens.

Thermal injury to the skin can also occur, but, it is usually self-limited and results in an acute skin burn with increased pigmentation.

Exposure to infrared radiation has been associated with cataract formation, particularly among glassblowers and furnace workers.

Injuries can be prevented by shielding heat sources, using protective eye and skin wear, and monitoring exposure levels.

INJURIES DUE TO VISIBLE RADIATION

Visible radiation (light) covers the portion of the electromagnetic spectrum between infrared and ultraviolet radiation and the wavelengths between 400 and 750 nm.

The eye is the most sensitive target organ, with damage resulting from structural, thermal, or photochemical light-induced reactions.

The retina is the wavelengths of 440-500 nm (blue light), which cause a destructive photochemical reaction. Blue light is responsible for solar retinitis (eclipse blindness) and may contribute to retinal aging and to senile macular degeneration, which can result in visual field defects.

Because the lens normally filters out wavelengths between 320 and 500 nm, it provides some protection of the retina from blue light.

Short bursts of high-intensity light can cause heat-induced flash blindness, in which the temporary visual loss and afterimage are due to bleaching of visual pigments. As the light intensity and exposure duration increase, the afterimage persists longer.

Insufficient lighting or reflected light (glare) can cause asthenopia (eye strain), visual fatigue, head-ache, and eye irritation. These problems are more likely to occur in people over the age of 40 years. Symptoms are transient, and there is no indication that repeated episodes lead to ocular damage.

Contrast from surrounding light sources on areas of lesser intensity has led to complaints of asthenopia associated with video display terminal use. This can usually be corrected by decreasing surrounding light intensities, using antiglare filters, and adjusting the contrast of the light on the screen.

INJURIES DUE TO ULTRAVIOLET RADIATION

Ultraviolet (UV) radiation covers the portion of the electromagnetic spectrum between visible radiation and ionising radiation and has wavelengths are devided into three bands -A,B and C- with the A and B bands representing the longer wavelengths and producing most of the biologic effects.

Wavelengths shorter than 200 nm are biologically inactive; they can exist only in a vacuum or an inert gas atmosphere and are absorbed over extremely short distances in air. Wavelengths of 200 - 290 nm are absorbed primarily in the stratum corneum of the skin or the cornea of the eye, while the longer wavelengths can affect the dermis, lens, iris, or retina.

Because UV radiation has relatively poor penetration, the only organs it affects are the eye and skin.

Eye injury is caused by thermal action from pulsed or brief high-power exposures, and skin damage more commonly by photochemical reactions (including toxic and hypersensitivity reactions) from brief high or extended low-power exposures.

The thermal effects of protein coagulation and tissue necrosis are rapid in onset.

The effects of chronic exposure include accelerated aging of the skin, characterised by loss of elasticity, hyperpigmentation, wrinkling and telangiectasia.

A. Ocular exposure to UV wavelengths shorter than 315 nm (especially wavelengths of 270 nm, to which the eye is most sensitive) can cause **photokeratoconjunctivitis.**
B. **Cataracts**: Cataractogenesis has been attributed to both photochemical and thermal effects of intense exposure to UV wavelengths of 295-320 nm and usually appears within 24 hours.
C. **Other eye injuries**: The lens protects the retina from the effects of UV wavelengths shorter than 300 nm, but damage to this iris and retina is posible if individuals with aphakia are exposed to these wavelengths.

Two lesions of the bulbar conjunctiva have associated with repeated exposures to UV radiation: pterygium (a benign hyperplasia) and epidermoid carcinoma.

D. **Erythema**: Absorbed UV radiation reacts with photoactive substances present in the skin and 2-24 hours later causes erythema (sunburn), the most common acute UV effect. Erythema is most severe following exposure to wavelengths of 290-320 nm and may be accompanied by edema, blistering, desquamation, chills, nausea, and rarely circulatory collapse.

E. **Photosensitivity reactions**: Two types of acute photosensitivity reactions of the skin can occur following exposure to UV radiation: phototoxic (non-allergic) and photoallergic reactions.

- **Phototoxic reactions** are much common and frequently occur in association with use of medications such as griseofulvin, tetracycline, sulfonamides, thiazides, and preparations containing coal tar or psoralens.

  Phototoxicity may exaggerate or aggravate the effects of some systemic diseases, including lupus erythematosus, dermatomyositis, congenital erythropoietic porphyria cutanea tarda symptomatica, pellagra, actinic reticuloid, herpes simplex, and pemphigus foliaceus. Photosensitivity reactions may be characterised by blisters, bullae and other skin manifestations.

  Exposure to UV wavelengths above 320 nm after skin contact with furocoumarin-producing plants as celery can cause phytophotodermatitis. A mild phototoxic reaction causes pigmentary changes along the patern of points of contact, while bullae may result from a more severe inflammatory reaction.

- **Photoallergic reactions** to UV radiation occur in association with bacteriostatic agents and perfume ingredients, which cause skin irritation, erythema, and blistering.

F. **Premalignant and malignant skin lesions**

Premalignant lesions associated with chronic exposure to UV radiation include actinic keratosis, keratoacanthoma, and Hutchinson's melanosis. Malignant lesions associated with exposure are basal cell carcinoma, and malignant melanoma.

Hazardous UV wavelengths are thought to be between 256 and 320 nm.

UV radiation also promotes carcinogenesis following exposure to some chemicals, including those found in tar an pitch.

Increased risk for premalignant and malignant lesions occurs in fair-skinned individuals and in those who have repeated sunburn or tan poorly. Patients with a history of xeroderma pigmentosum are at greater risk for malignant melanoma.

PREVENTION

Outdoor workers should be instructed to use sunscreen and protective clothing, and persons at increased risk because of preexisting medical conditions or excessive exposure should be examined periodically for the presence of premalignant or malignant lesions.

IONISING RADIATION INJURIES

The two most significant health responses to ionising radiation are the acute radiation syndrome that follows a brief but massive exposure and the chronic effects that are due to a brief high-dose exposure or to high cumulative exposures.

Because of the ubiquity of ionising radiation in our environment, the effects of long-term low-dose exposures are more difficult to pinpoint, but clusters of illnesses have been found near nuclear test sites and in association with some occupations.

Ionising radiation is emitted from radioactive atomic structures as energised particles (alpha, βeta, proton and neutron particles) that impart energy through collision with other structures or as high-energy electromagnetic X-rays or gamma rays.

The different forms of ionising radiation vary in natural source, energy, frequency and penetrability, but they all share the ability to ionise incident materials and exist at the highest energies and frequencies of the electromagnetic spectrum.

External biologic exposure to X-rays, gamma rays, and proton and neutron radiation results in high absorption, whereas beta particles penetrate at all.

Internal exposure to alpha or beta particles by inhalation, implantation, or ingestion can result in serious acute or delayed injury.

If radioactive contamination is suspected, decontamination procedures should be followed scrupulously during all phases of patient management.

ACUTE RADIATION SYNDROME

Acute radiation syndrome is due to brief but heavy exposures of all or part of the body to ionising radiation.

In radiation disrupts chemical bonds, which causes molecular excitation and free radical formation. Highly reactive free radicals react with other essential molecules such as nucleic acids and enzymes, and this in turn disrupt cellular function.

.The clinical presentation and severity of illness are determined by the dosage, body distribution, and duration of exposure. Tissues with the most rapid cellular turnover are the most radiosensitive: reproductive, hematopoietic, and gastrointestinal tissues.

CLINICAL FINDINGS

Abnormal laboratory findings may be seen at any dose over 25 cGy.

For doses of 100-400 cGY, symptoms begin within 2-6 hours and may last up to 48 hours.

For doses of 600-1000 cGY, syptoms begin within 2 hours and later merge into the illness phase.

Doses of 1000-3000 cGy can cause immediate gastrointestinal symptoms and massive fluid, blood and electrolyte loss resulting from denudation of the gastrointestinal mucosa.

Doses exceeding 3000 cGy are lethal.

Some pathients with acute radiation syndrome pass through phases:

- Prodrome
- Latent phase
- Illness
- Recovery

A. **Prodrome**: Symptoms and signs may include anorexia, nausea, vomiting, diarrhea, intestinal cramps, salivation, dehydration, fatigue, apathy, prostration, arrhythmia, fever, respiratory distress, hyperexcitability, ataxia, headache, and hypotension. Gastrointestinal and central nervous system findings predominate.
B. **Latent phase**: The prodrome is sometimes followed by a period of relative well-being prior to the onset of illness.
C. **Illness phase**: Symptoms and sign in this phase may include fatigue, weakness, fever, diarrhea, anorexia, weight loss, hair loss, arrhithmia, ileus, ataxia, desorientation, convulsions, coma and shock. Effects are primarily hematopoietic and due to inhibition or hematopoietic stem cells. Cardiovascular collapse, pericarditis, and myocarditis have been reported. With doses exceeding 200 cGY, ther may be reproductive system effects, including sterility, aspermatogenesis, and cessation of menses. Fetal and embryotoxicity or death can also occur.
D. **Recovery phase**: The prognosis for recovery from exposures of up to 600 cGy is good when appropriate therapy is given. For higher exposures, the prognosis worsens as the dose increases. Infection and sepsis are the major causes of morbidity and mortality in cases involving exposures below 1000 cGy, in which the major impact is hematopoietic.

PREVENTION

Occupational exposure to ionising radiation should be monitored.

Environmental or area monitoring devices include the Geiger-Műller counter, ionising chamber, and scintillation detector.

Where an exposure potential occurs, shielding with lead or other effective barrier can contain emissions.

ACUTE LOCALISED RADIATION INJURIES

Exposure of isolated skin and body parts to ionising radiation will result in hair loss (doses above 300 cGy), erythema (above 600 cGy), dry desquamation (radionecrosis) (above 1000 CGY), and wet desquamation (above 2000 cGy). Pain and itching occur shortly after exposure and are followed by erythema and blister formation. In cases of severe localised burns, there may be tissue ischemia and necrosis.

DELAYED EFFECTS OF HIGH-DOSE RADIATION

Radiodermitis often occurs in association with ionising radiation therapy. The skin is dry, smooth, shiny, thin, pruritic, and sensitive, and there are signs of telangiectasia, atrophy, and diffuse pigmentation. The nails are brittle and striated.

Scaring in other tissues following high-dose exposure has led to endarteritis obliterans, intestinal stenosis, and cataracts.

Various cancers related to localised organ radioactivity have been described.

Other effects of high-dose exposure include premature aging, shortening of the life span, teratogenic (central nervous system deficit, mental retardation, microcephaly) and reproductive abnormalities.

EFFECTS OF LOW-DOSE RADIATION

Although developmental abnormalities have been associated with doses as low as 10 cGy and cancers associated with levels below 10cGy, the practical

relevance of low-dose phenomena is extremely difficult to establish not only because the cumulative average exposure for people is approximately 8-10 cGy per lifetime.

1.4. AIR QUALITY

Air is a mixture of gases. It contains different percentages of nitrogen (78%), oxygen (21%), argon (0.9%), carbon dioxide (0.033%).

Oxygen is a colorless, odorless, and tasteless gas that is only slightly soluble in water. It is the only gas used by the human body and it is essential to life. The other gases breathed from the atmosphere serve only as a vehicles and diluents for oxygen.

Health hazards: At 15-19% oxygen impaired coordination and decrease ability to work and at 10-14% creates respiratory problems. Few recorder after more than 5 minutes in less oxygen.

Exposure limits:

Deficiency: 18%

Enrichment: 23%

Nitrogen is a colorless, odorless and tasteless gas. It is chemically inert and is incapable of supporting life. At normal pressure nitrogen does not influence the human organism and an amount of about 1 liter is dissolved in the blood and the tissues.

Health hazards: Acts as asphyxiant through displacement of normal air.

Carbon dioxide is a gas produced by various natural processes such as animal metabolism, combustion, and fermentation. It is colorless, odorless, and tasteless. A person should not breathe air containing more than 0.1% CO_2 by volume.

Health hazards: Causes headache, dizziness, increases the heart rate and blood pressure and coma.

Exposure limits:

5000 ppm -15000 ppm

Argon, neon, and hydrogen have been used experimentally as diluents for oxygen in breathing gas mixtures.

Water vapors. The normal weight of water vapors in the air is considered 1-1.5%. They have a harmful effect on the human organism if a large amount is present.

1.5. OUTDOOR AIR POLLUTION

The dramatic air pollution episodes that occurred in the early part of the twentieth century in the Meuse Valley of Belgium, Donora, Pennsylvania and London, England are not likely to occur in the world today.

These episodes were due to the large scale burning of coal in the presence of "ideal" meteorologic conditions-atmospheric inversion leading to a stagnant air mass.

A clearly evident excess mortality was observed during and after these episodes.