13. Threshold Limit Values of Hazardous Substances

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1 INTRODUCTION

Over the centuries man has devised many ingenious ways and means of utilizing the earth's natural resources. But in doing so he has created a highly complex industrialized environment which often exerts a detrimental effect on his own health and welfare. His working environment has often been contaminated with numerous noxious gases, dust, mist and fumes. The U.S.A. National Institute of Occupational Safety and Health (NIOSH) already has 64 300 entries in its "Registry of toxic effects of chemical substances". This represents 15 000 discrete substances with synonyms.[1]

Furthermore, in 1976 the World Health Organization (WHO) reported that the number of toxic chemicals used in industry in different parts of the world already amounted to 600 000. In addition some 3 000 new ones are put into production every year.[2]

Many physical variables in the occupational environment may also be detrimental to the safety and health of the worker. Amongst these are exposure to noise, vibration, ionizing radiation, microwaves, non-ionizing radiation, atmospheric pressure changes and extremes of temperature, humidity and ventilation. Any of these factors may not only impair the worker's health but may result in discomfort, lowered efficiency and increased accident risk.

With a few exceptions, such as radioactive substances and carcinogens, air contaminants are usually harmless at low concentrations to the majority of people. The average air concentration of a substance to which workers may be repeatedly exposed day after day without adverse effect forms a hygiene standard known as a threshold limit value (TLV).[3] For certain substances excursions beyond the TLV's may be tolerated, provided that the exposure is kept within a limited time interval.

2 ABSORPTION OF HAZARDOUS SUBSTANCES

The human body, on exposure to hazardous substances, will absorb such compounds by way of three routes;

(a) through the lungs by inhalation,

(b) through the skin by contact,

(c) through the alimentary tract by ingestion.

A particular route may take priority depending on the physical form and properties of the contaminant.

2.1 Absorption of gaseous contaminants through the lungs

In the occupational environment the respiratory route is probably the most important as the worker inhales the air in which he works over the entire workshift. Not all the inhaled air becomes available for absorption into the bloodstream, because of the anatomical "dead space" occupied by the tracheobronchial tree. However, 2,7 m^3 of air may be available for absorption by those who have a sedentary occupation, whereas some 6,7 m^3 of air will reach the alveolar region of a workman doing moderately heavy work during an 8-hour workshift.[4]

Gaseous contaminants are absorbed into the alveolar blood according to Henry's Law, which states that the mass of a gas that dissolves in a definite mass of liquid with which it is in contact, at a given temperature, is directly proportional to the partial pressure of that gas, provided there is no chemical interaction between the gas and the liquid.[5]

Applied to circulating blood, the coefficient of distribution can be calculated from the simple equation:[7]

$$\delta = \frac{C_1}{C_2}$$

where C_1 = concentration in the fluid phase (blood)

C_2 = concentration in the vapour phase (air) as dependant on the partial pressure of the contaminant, both in mol/m^3. (A mol of any substance is a quantity of the substance, its mass of which in grams is numerically equal to the molecular mass of the substance)

As the respiratory tissue in the lungs has been estimated to have a surface area of about 70 m^2 the equilibrium between the blood in the lungs and the alveolar air is reached very rapidly.[6]

For a substance such as acetone, which is readily absorbed, δ = 330. This means that a saturation level in blood equal to 330 times the concentration in alveolar air may be reached. Thus, the capacity of the lung to absorb acetone is very high. The molecular mass of acetone is 58,05 and its TLV is 2 400 mg/m^3. This corresponds to

$$\frac{2\,400}{58{,}05 \times 1\,000} = 0{,}041 \text{ mol/m}^3 \ (= C_2)$$

and the theoretical blood-acetone concentration (C_1) is thus

$$C_1 = 330 \times 0{,}041 = 13{,}5 \text{ mol/m}^3$$

If all the acetone at TLV were absorbed from the acetone-contaminated air reaching the alveoli (2,7 – 6,7 m^3 in one 8-hour shift) some 6,5 to 16 mg of acetone would be absorbed during one 8-hour shift.

Provided the total amount absorbed during a particular shift does not exceed the above amounts, higher "instantaneous" concentrations may be tolerated. Such excursions are however, not limitless and in the case of acetone the American Conference of Governmental Industrial Hygienists (ACGIH) does allow an excursion beyond the TLV of up to 3 000 mg/m^3, but then only for 15 minutes.

Any gas or vapour which is breathed in tends to pass through the lungs into the bloodstream to be distributed throughout the body. However, the ultimate amount of material absorbed by the blood and the accumulation of foreign gas or vapour in the body depends on a number of factors, such as the concentration in the air, the solubility of the material, the length of exposure, the rate of breathing and of circulation and whether or not the material is biochemically reactive. Differences in barometric pressures, as experienced in the mining industry between surface and underground work, may thus have a significant effect on the absorption of air contaminants. As the maximal oxygen intake of miners has been found to increase linearly with an increase in barometric pressure,[7] the rate of breathing and hence the absorption of air contaminants at the surface will differ from that underground.

2.2 Absorption of particulate contaminants through the lungs

Dusts, fumes, mists and smokes may also be absorbed via the respiratory route depending on the quality, form and particle size of such particulate matter. The process of their accumulation, distribution and elimination is much more complicated than with gases. Nevertheless, lethal quantities may dissolve and enter the circulation by absorption from the respiratory tract, or they may be absorbed by ingestion after being swallowed, subsequent to their deposition in the nose and throat.

The deposition of particulate matter in the respiratory tract is discussed in Chapter 12 under the sections dealing with dust.

2.3 Absorption through the skin

Many gaseous, liquid and solid contaminants of the air may be absorbed through the intact skin. The skin is a multi-layered protective cover, which includes a lipidic membrane which is perforated by hair roots and follicles that penetrate deeply into the outer skin and subcutaneous tissue. Toxic materials may thus enter the skin:

(a) through the lipidic membrane (transepidermal penetration) which is easily achieved by lipid-soluble substances such as pesticides and organic solvents;

(b) through penetration via hair roots and follicles (transfollicular).

In addition a skin injury provides a direct access route for all substances, and different areas of the skin have different absorption rates.

Water and most electrolytes do not penetrate the skin in significant amounts but organic compounds such as alkaloids, phenols, oxalic and salicylic acids and esters and lead acetate are absorbed in appreciable amounts. Also nicotine and strychnine are readily absorbed but their salts are not. Slight amounts of hydrogen sulphide and dangerous amounts of hydrogen cyanide may be absorbed from contaminated air. Nitrobenzene, dinitrobenzene, nitrotoluene and nitroglycerine are readily absorbed from skin contact with these materials or their solutions. Benzene, toluene, xylene, chlorinated hydrocarbons and other fat solvents are also absorbed to a degree. Thus vapours of substances such as butyl cellusolve, carbon tetrachloride and methanol are readily absorbed through the intact skin.

2.4 Absorption by way of ingestion

Absorption by way of ingestion, other than by accident, is rare and is therefore usually not

considered a form of occupational exposure. But whenever basic rules of personal hygiene are ignored, exposure due to ingestion following contamination of hands, food, beverages, cigarettes, etc., will occur.

3 EFFECTS OF TOXIC SUBSTANCES

As inhalation appears to be the most prominent route of absorption of hazardous substances, the industrial hygienist is primarily concerned with air-borne hazards. The effects of toxic substances are therefore discussed here mainly in terms of airborne hazards.

Atmospheric contaminants which enter the lungs may exert their action locally or may affect one of several organs or tissues after being absorbed into and transported by the bloodstream. Insoluble particulate matter may permanently deposit in the lungs to be either relatively inert or to result in an incapaciting or even lethal condition generally known as pneumoconiosis.

Local effects of atmospheric contaminants occur at the site of contact with the tissue and usually develop in three degrees: irritation, oedema and inflammation. In severe cases necrosis of the tissue may develop. General toxic effects may be primary, secondary or tertiary, stimulative or inhibitive, with a reversible or irreversible injury. Exposure to two or more effects simultaneously may have synergistic or antagonistic effects.

Prolonged exposure to moderate concentrations of toxic substances may eventually produce intoxication symptoms or syndromes resulting from toxic effects at the molecular-cellular level. In cases of occupational exposure the toxic effects will depend on:

(a) the concentration of the toxic substances or mixture of substances in the atmosphere at the workplace;
(b) the duration of exposure; and
(c) the physiological and psychological condition and individual susceptability of exposed persons.

3.1 Haber's Law

The dose (D) provoking a toxic effect may theoretically be represented by Haber's Law:[8]

$D = c.t$ where
c = concentration of toxic substance in the work atmosphere and
t = duration of exposure.

The effect (E) is then given by:

$E = k.c.t$ where
k = Haber's constant which is an index of toxicity.

3.2 Dose-effect relationship

The toxic effect produced varies with the dose-effect relationship (Figure 13.1) and depends on the individual contaminant. Three main relationships are common:

(a) the effect is linearly proportional to the dose;
(b) the effect becomes increasingly larger as the dose increases. This relationship is represented by a hyperbolic curve, or by a non-linear continuously increasing function. At some value the increased effect for a further minute increase in dose becomes prohibitive. This may be considered to constitute a "ceiling value" which never should be exceeded; or
(c) the effect is "all-or-none" and has a distinct threshold value. Here the dose-effect relationship may be expressed by a sigmoid curve.

3.3 The distribution of toxic substances in the human body

Toxic substances entering the bloodstream may be transported to various cells and tissues of the human body as dissolved in the plasma, bound to haemoglobin or erythrocytes or to the surface of erythrocytes or their constituents or bound to various plasma fractions. Electrolytes are usually

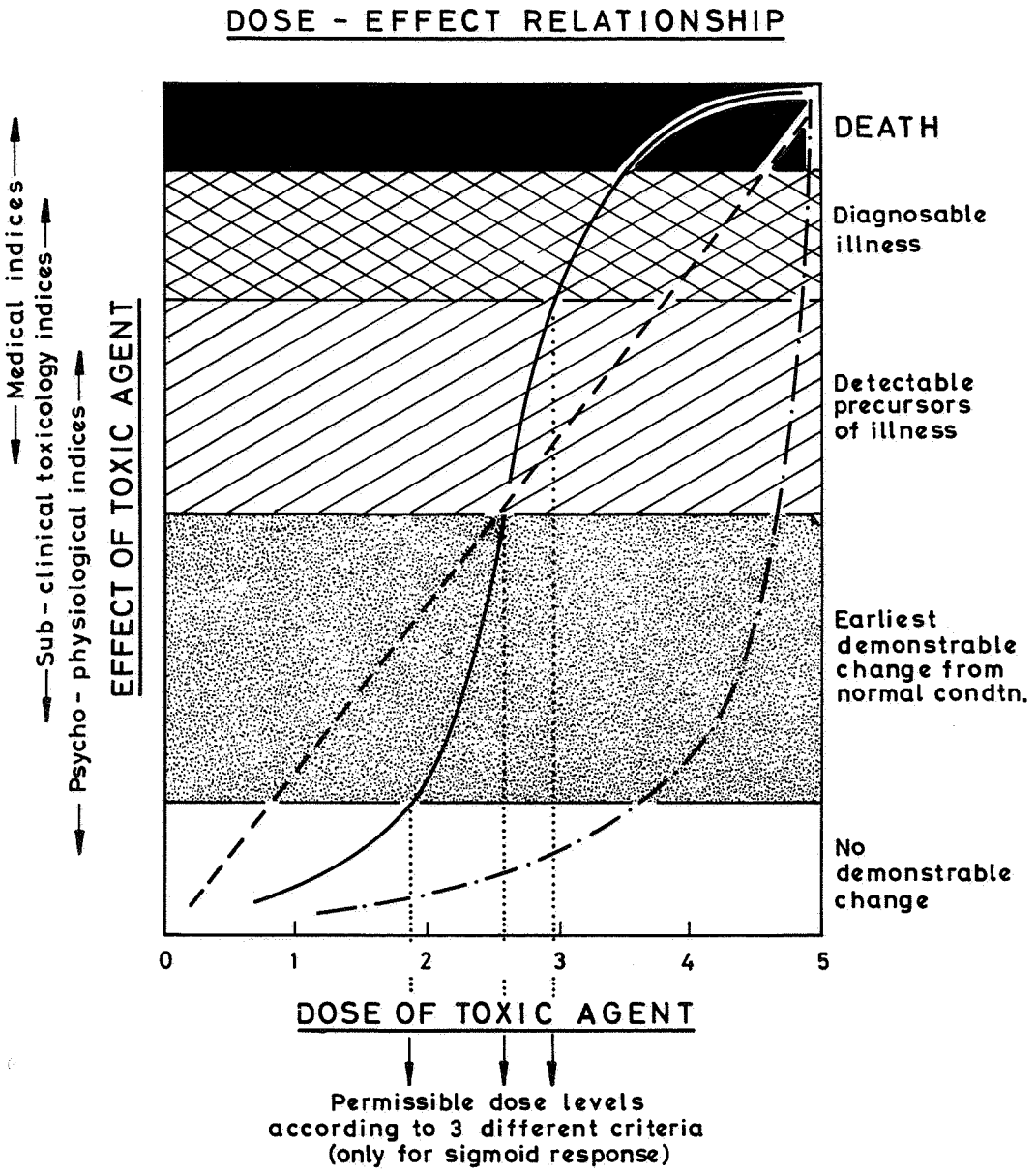

Figure 13.1 The physiological dose-effect relationship for toxic agents
1. Effect is directly proportional to dose: linear relationship (– – –)
2. Effect becomes larger with increasing dose: hyperbolic (—·—·—)
3. Effect is "all-or-none": Sigmoid (————)

transported in the form of ions in the plasma. Alternatively substances may be hydrolysed to form colloids, be suspended in the blood or to form complexes with plasma organic acids.

Univalent ions such as sodium, potassium, lithium, rubidium, caesium, chloride, bromine and fluorine are fairly uniformly distributed in the organism, while some accumulate in the liver (La^{III}, La^{IV}, Ce, Th), specific organs for which they possess greatest affinity (I to thyroid, U to kidneys) or are deposited in the bones (Ca, Ba, Sr, Ra, Be). Lipid-soluble substances have an affinity for fatty, adipose tissues. Thus organic solvents, inert gases and pesticides may be deposited in these tissues.[8]

3.4 General and systemic effects

A distinction between the general and the systemic effects of toxic substances can be made as indicated in Table 1.

TABLE 1. GENERAL AND SYSTEMIC EFFECTS OF TOXIC SUBSTANCES

General effects	Systemic effects
Irritation, necrosis (cell destruction) toxic irritation.	Effects on the central, peripheral or vegetative nervous system.
Hypoxaemia, hypoxia	Effects on the heamatopoietic (blood forming) system.
Allergic effects	Hepatotoxic (liver) effects
Carcinogenic effects	Nephrotoxic (kidney) effects
Mutagenic effects	Other organs and tissues
Teratogenic effects	General systemic effects.

4 CLASSIFICATION OF HAZARDOUS SUBSTANCES

It is rare for any toxic substance to have only one type of effect. With many gases and vapours the type of physiological action depends upon concentration. For instance, a vapour at one concentration may exert its principal action as an anaesthetic, while a lower concentration of the same vapour may, with no anaesthetic effect, injure the nervous system, the heamatopoietic system, or some visceral organ. It is better therefore to classify hazardous substances according to the effect(s) produced by or the toxicological properties of a particular substance, as follows:

4.1 Irritants

(a) Irritants affecting chiefly the upper respiratory tract.
EXAMPLES: aldehydes (acetaldehyde, acrolein, formaldehyde, paraform); alkaline dusts and mists; ammonia; chromic acid; ethylene oxide; hydrogen chloride; hydrogen fluoride; sulphur dioxide; sulphur trioxide.

(b) Irritants affecting both the upper respiratory tract and lung tissues:
EXAMPLES: halogens (bromine, chlorine, fluorine, iodine); chlorine oxides; cyanogen bromine; cyanogen chloride; dimethyl sulphate; diethyl sulphate; ozone; sulphur chlorides; phosphorus trichloride; phosphorus pentachloride.

(c) Irritants affecting primarily the terminal respiratory passages and air sacs.
EXAMPLES: arsenic trichloride; nitrogen dioxide; nitrogen tetroxide; phosgene.

4.2 Asphyxiants

These materials exert their effects by interfering with the oxidation of the tissues.

(a) *Simple asphyxiants* are physiologically inert gases that act principally by dilution of the atmospheric oxygen below the partial pressure required to maintain an oxygen saturation of the blood sufficient for normal tissue respiration. Where the concentration of oxygen in the inhaled air falls below the normal level of 20,95% (21,2 kPa partial pressure at 101,3 kPa atmospheric pressure and 273 K) distress occurs below 16%, unconsciousness below 11% and breathing soon stops at a concentration of oxygen below 6%.[6]

EXAMPLES: acetylene; argon; carbon dioxide; ethane; helium; hydrogen; methane; neon; nitrogen; nitrous oxide.

(b) *Chemical asphyxiants* through chemical action either prevent the blood from transporting oxygen from the lungs or prevent normal oxygenation of the tissues, even though the blood is well oxygenated.

EXAMPLES: carbon monoxide (combines with haemoglobin); cyanogen, hydrogen cyanide, nitriles (inhibit oxidation enzymes); analine, methyl analine, dimethyl analine, toluidine, (forms inactive methaemoglobin); nitrobenzene (also forms methaemoglobin but also lowers blood pressure, disturbs and finally halts breathing); hydrogen sulphide (causes respiratory paralysis).

4.3 Anaesthetics and Narcotics

These materials exert their principal action as simple anaesthesia without serious systemic effects. They have a depressant action on the central nervous system governed by their partial pressure in the blood supply to the brain.

EXAMPLES: (Arranged in the order of decreasing effects):
Acetylene hydrocarbons (acetylene, allylene, crotonylene); olefin hydrocarbons (ethylene to heptalene); ethers (ethyl and isopropyl ether); paraffin hydrocarbons (propane to decane); aliphatic alcohols (ethyl, propyl, butyl and amyl), esters (these are not particularly anaesthetic but are hydrolysed in the body to organic acids and alcohols).

4.4 Carcinogens

Various neoplastic effects may be produced by a wide range of substances. Carcinogenic substances or agents may be defined as substances which, under favourable conditions through direct or indirect action, either externally or internally, act on healthy tissue cells to cause a rapid proliferation of the cellular elements and the development of structural abnormalities known as cancer.[9]

EXAMPLES: Aromatic amines (naphthylamine, 4-aminodiphenyl, auramine, benzidine); nitro- and azo-derivatives of aromatic amines, arsenic; asbestos; benzene; beryllium; chromates (hexavalent compounds); coal tar products (anthracene oil, asphalt, coke, creosote oil, carbon black, lignite pitch, soot, tar oil); hydrogenated coal, oil and tar; nitrosamines; petroleum oil (cutting oil, diesel oil, fuel oil, grease, paraffin oil, wax); radiations; (radioactive substances, ultra-violet and x-rays); cobalt and nickel compounds are also suspect.

4.5 Mutagens

These substances produce changes in the body's genetic material: In other words, they may alter the genes and thus the chromosomes of the cells. Mutations of somatic cells may result in the death (or cancer) of these cells, but mutations of germ cells may greatly change the life of the offspring, resulting in abnormalities such as dwarfism, mental retardation, congenital blindness, fatal anaemia, etc.[9]

EXAMPLES: Ionising radiations (α-rays, β-rays, γ-rays, protons, fast neutrons, thermal neutrons): radioactive isotopes (C^{14}, Na^{24}, P^{32}, Ca^{45}, Fe^{55}, Zn^{65}, Sr^{89}, I^{131}); chemical (alkylating agents such as ethyleneimine, anti-cancer drugs such as 6-mercaptopurine, antidepressant drugs, fungicides such as captan, pesticides, chemosterilants, food additives, organic intermediates, solvents).

It has as yet not been shown conclusively that ionizing radiations or chemicals have ever caused human mutations leading to the birth of genetically defective children i.e. who would pass on such mutations. These may, however, be unknown because of the complex problems involved in carrying out mutagenesis research in man and also because mutations are generally recessive. But as genetic mutations in bacteria, viruses, moulds, higher plants, fruit flies, mice, hamsters and rats have been irrefutably demonstrated these substances should be strongly suspected until such time as more conclusive evidence becomes available.

4.6 Teratogens

These compounds produce abnormal foetuses in pregnant women. The effect occurs after conception, while the organism is still unborn. They have no effect on the body's genetic material, as mutagens have. The effect is thus not hereditary.

EXAMPLES: X-ray exposure of the pelvis of pregnant women; German measles (blindness and deafness); thalidomide; carbon monoxide; some steroids (strongly suspect); chemicals having been reported to be teratogenic in mice, rats, chicks or rabbits, penicillin, streptomycin, sulphanilamide, quinine, nicotine, strontium, lead, selenium and thallium.

4.7 Systemic Poisons

Numerous organic as well as inorganic poisons may be encountered in the mining industry.

EXAMPLES: Halogenated hydrocarbons (cause injury to one or more of the visceral organs); materials damaging the haematopoietic system (benzene, phenols and to some degree toluene, xylene and naphthalene); nerve poisons (carbon disulphide, methyl alcohol, thiophene); heavy metals (Pb, Hg, Cd, Sb, Mn, Be, etc.); non-metallic inorganics (compounds of As, P, Se, S and fluorides).

4.8 Hazardous particulate matter

Particulate matter other than systemic poisons may also have incapaciting effects on the human body.

EXAMPLES: Fibrosis-producing dusts (silica, asbestos), inert dusts (carborundum, carbon, emery); dusts causing allergic reactions (pollen, wood, resins and many organic dusts); irritants (acids, alkalis, chromates, fluorides); bacteria and other micro-organisms (anthrax, etc.).

4.9 Biotransformation of toxic substances

Within the healthy, living organism, numerous biotransformations may occur. From the toxicological viewpoint these changes may be advantageous (detoxication) but also deleterious (lethal synthesis) depending on whether a less toxic or more toxic metabolite is synthesized. Metabolic changes may include oxidation, reduction, conjugation with hydrocarbons, amino acids or sulphur compounds or, methylation and acetylation. These changes may result in various effects.[9]

Detoxication mechanisms are not clear but it is obvious that the organism is trying to remove unrequired material by decomposition or by conversion to compounds which are more readily excretable. These processes enable man to tolerate limited amounts of toxic materials and they must therefore be taken into consideration when permissible occupational exposure levels are assessed or laid down.

5 THRESHOLD LIMIT VALUES

The maximum concentration of an air-borne hazardous substance to which nearly all workers may be repeatedly exposed day after day without adverse health effects is referred to as the threshold limit value (TLV).[3]

Of the Western countries, the USA is probably one of the world's leaders in determining TLVs. The ACGIH has to date published TLVs for some 650 industrial materials.[3] The evidence on which

each TLV is based is published in "Documentation of Threshold Limit Values". Information collected for one substance may run into hundreds of printed pages. The limits laid down by the ACGIH are clearly stated to be mere guides in the control of health hazards and should not be used as fine lines between safe and dangerous concentrations. Their main use is in the vital communication between physicians or toxicologists and the engineers who have to design control equipment as well as for the hygienist who is to monitor the occupational environment.

TLVs are based on the best available information from experimental animal studies, and industrial experience. The basis on which values are established may differ from substance to substance depending on whether protection against health impairment or freedom from irritation, narcosis, nuisance or other forms of stress are to be considered.

The actual TLV concentration which is to be applied will depend on the criteria accepted for its evaluation. One of the following criteria may be used as basis[9] (See Figure 13.1):

(a) prevention of the earliest demonstrable change from normal behaviour;

(b) keeping of levels below detectable precursors of illness;

(c) prevention of diagnosable illness.

TABLE 2. THRESHOLD LIMIT VALUES (TLV), EXCURSION LIMITS AND SHORT TERM EXPOSURE LIMITS FOR SOME HAZARDOUS GASES, FUMES AND VAPOURS WHICH MAY BE ENCOUNTERED IN THE MINING INDUSTRY. (*cf.* ACGIH TLVs, 1979).

	Compound	Time Weighted Average (TLV.TWA) (mg/m^3)	Excursion Limit (mg/m^3)	Short Term Exposure Limit for a time weighted average of 15 minutes (TLV.STEL) (mg/m^3)
	Mercury vapour	0,05	0,15	0,15
	Chromic acid	0,05	0,15	—
C	Vanadium fumes (V_2O_5 as V)	0,05	—	—
	Vanadium dust (V_2O_5 as V)	0,5	1,5	1,5
	Silver metal	0,1	0,3	—
	Lead fumes (as Pb)	0,15	0,45	0,45
	Copper fumes (as Cu)	0,2	0,6	—
	Ozone	0,2	0,6	0,6
	Uranium (as U)	0,2	0,6	0,6
	Arsenic (as As)	0,5	1,5	—
C	Trinitrotoluene (TNT)	0,5	—	—
	Sulphuric acid	1	3	—
C	Nitroglycerine (skin)	2	—	—
C	Potassium hydroxide	2	—	—
C	Sodium hydroxide	2	—	—
	Chlorine	3	6	9
C	Formaldehyde	3	—	—
	Iron oxide fumes (as Fe)	5	10	10
C	Manganese (as Mn)	5	—	—
	Welding fumes	5	10	—
	Zinc oxide fumes (as Zn)	5	10	10
	Sulphur dioxide (SO_2)	5	10	15
C	Nitrogen dioxide (NO_2)	9	—	—
	Magnesium oxide fumes (as Mg)	10	20	—
	Hydrogen cyanide (skin)	11	—	16
	Hydrogen sulphide (H_2S)	15	22,5	27
	Nitric oxide (NO)	30	45	45
	Carbon monoxide (CO)	55	82,5	440
	Acetaldehyde	180	225	270
	Turpentine	560	700	840
	Carbon dioxide (CO_2)	9 000	—	18 000

The stringent hygiene standards being applied in the USSR are such because the first criterion is used there[10]. In countries adopting the third criterion TLV values will be higher. Table 2 lists the TLVs for some hazardous substances which may be encountered in the mining industry.

The ACGIH lists three categories of threshold limit values[3]:

(a) threshold limit value – time weighted average (TLV.TWA)

(b) threshold limit value – short term exposure limit for a time weighted average of 15 minutes (TLV.STEL) and

(c) threshold limit value – ceiling (TLV.C)

The term "Threshold limit value" may thus be considered to be a collective term for these three categories (Figure 13.2).

5.1 Time-weighted average (TLV.TWA)

It is found in practice that the concentrations of air-borne contaminants may vary between wide limits within any one 8-hour work shift. For the assessment of worker exposure limits the average concentration of the contaminant should thus be used. The value to which nearly all workers may repeatedly be exposed for a normal 8-hour workshift (or a 40-hour workweek) constitutes the time weighted average (TWA) limit and is a concentration expressed in either parts of vapour or gas per million parts of contaminated air by volume at 25 °C and 101,3 kPa pressure (p.p.m.)* or in approximate milligrams of substance per cubic metre of air (mg/m^3).

Time weighted averages (TWA) permit excursions above the TLV for short periods. The amount by which they may be exceeded without injury to health depends on factors such as[8]:

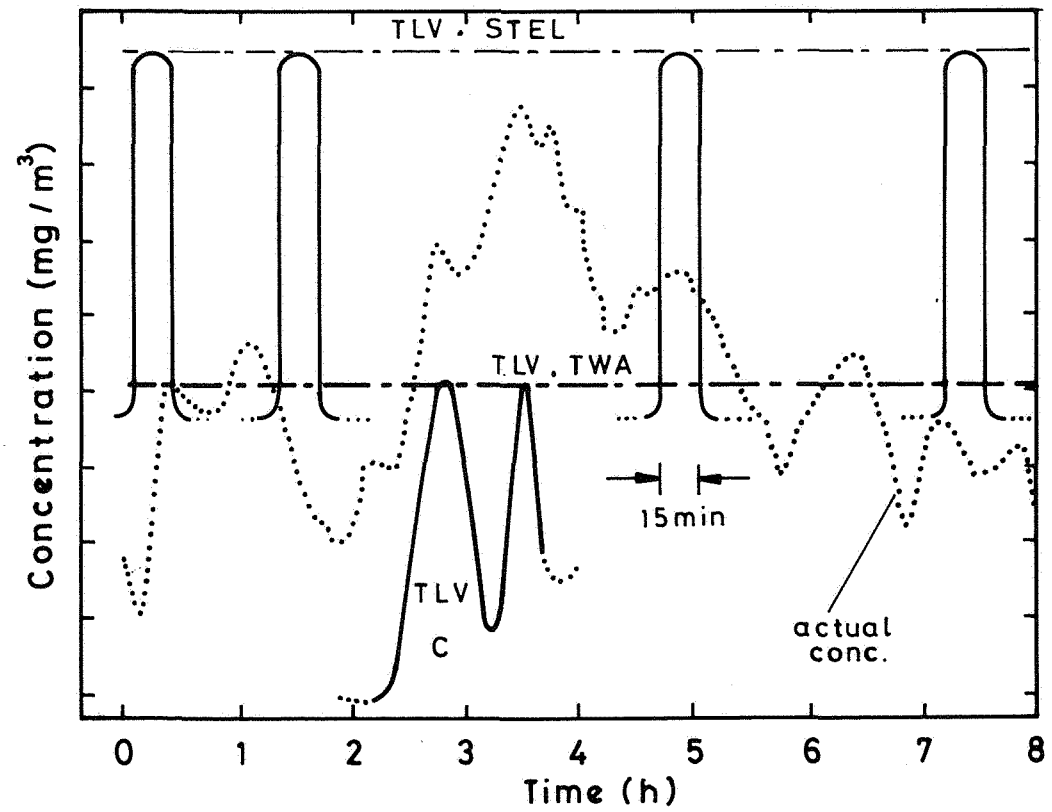

Figure 13.2 The three categories of Threshold Limit Values: time weighted average (TLV.TWA), short term exposure limit (TLV.STEL) and ceiling value (TLV.C)

(a) the nature of the contaminant;
(b) whether very high concentrations, even for short periods, produce acute poisoning;
(c) whether the effects are cumulative;
(d) the frequency with which high concentrations occur; and
(e) the duration of such periods.

For all substances for which no ceiling value has been set, the following excursion factors may be applied[3]:

for TLV of 0 – 1 (ppm or mg/m^3) excursion factor = 3
for TLV of >1 – 10 (ppm or mg/m^3) excursion factor = 2
for TLV of >10 – 100 (ppm or mg/m^3) excursion factor = 1,5
for TLV of >100 – 1 000 (ppm or mg/m^3) excursion factor = 1,25

These excursions are permitted only if the TLV.TWA is not exceeded, and the number of times the excursion may be exceeded is also governed by conformity with the TLV.TWA.

The cumulative exposure dose of a hazardous substance, by application of Haber's Law (see above), can be calculated from the formula:

$$E_{cum} = c_1 t_1 + c_2 t_2 + c_3 t_3 + \ldots c_n t_n$$

The average cumulative exposure dose is then

$$E_{av\text{-}cum} = \frac{E_{cum}}{T}$$

$$\text{Thus } E_{av\text{-}cum} = \frac{1}{T} \sum_{n=1}^{n} c_i t_i$$

Where c_i = air concentration of substance during any period of time t_i (in ppm or mg/m^3)
t_i = the duration (hours) of the exposure at the concentration c_i and
T = the total time of exposure (i.e. $T = t_1 + t_2 + t_3 + \ldots t_n = \Sigma\, t_i$).

EXAMPLE:

A diesel loco-driver hauls ore for 5 hours per day in a haulage in which the average concentration of carbon monoxide (CO) is 35 mg/m^3. Refuelling requires 10 minutes per day in a bay in which the CO concentration is 270 mg/m^3. His daily inspection requires 15 minutes and is carried out in an underground workshop in which the concentration of CO is 420 mg/m^3. If he spends the rest of his 8-hour shift in travelling and waiting places in which the CO concentration is neglible, his cumulative exposure dose per day will be:

$$E_{cum} = 35 \times 5 + 270 \times {}^{10}/_{60} + 420\ {}^{15}/_{60}\ \text{mg.h/m}^3$$
$$= 325\ \text{mg.h/m}^3$$

His average cumulative daily exposure dose is then

$$E_{av\text{-}cum} = \frac{325}{5 + {}^{10}/_{60} + {}^{15}/_{60}} \quad \frac{\text{mg.h}}{\text{m}^3} \cdot \frac{1}{h}$$

$$= 60\ \text{mg/m}^3$$

The equivalent exposure E_{eq}, i.e. time weighted exposure (or concentration*) for an 8-hour shift is then obtained from:

$$E_{eq} = E_{av\text{-}cum} \times \frac{T}{8}$$

*It has been recommended that the unit ppm should not be used in the SI unit system[11].

*Where the concentration of the air contaminant varies with time the average concentration of any time period may also be calculated according to the above formula, provided the total period is divided into time period intervals sufficiently small for the concentration to be practically constant for each individual time interval.

where

T = total time of exposure to the toxic substance (hours), and
$T = \Sigma\, t_i$ (as above).

For the above example on carbon monoxide:

$$E_{eq} = 60 \times \frac{5\ ^{25}/_{60}}{8}$$
$$= 41\ mg/m^3$$
$$(= TWA)$$

Thus, although the average cumulative exposure numerically exceeds the TLV.TWA of 55 mg/m^3 (Table 2) the overall time weighted average, being numerically equal to the E_{eq}, over one 8-hour workshift is in compliance with the TLV.TWA. Furthermore although the TLV.TWA was numerically exceeded, both in the refuelling bay and in the underground workshop, the short term exposure level (see below) of 440 mg/m^3 for 15 minutes, was not exceeded. This, together with the fact that the TLV.TWA was not exceeded, indicates that the exposure of the loco driver was still within acceptable threshold limit values.

If the equivalent exposure is calculated for a 40-hour workweek the following formula applies:

$$E_{eq} = E_{av\text{-}cum} \times \frac{T}{40} \text{ where}$$

T = the total exposure time over the workweek

In calculating the equivalent exposure the larger value for T must always be considered, i.e., if a six-day week is worked, or if the duration of the daily shifts vary from day to day, the formula for a 40-hour week should be used.

For compliance with the ACGIH TLVs the value E_{eq} should not exceed the value of the TWA.

It should be noted that where a worker works a shift of more than 8 hours per day (or a total of more than 40 hours per week) and he is exposed to a hazardous substance the whole time, his equivalent exposure will be more than the TLV.TWA even if the average concentration is exactly equal to the TLV.TWA**.

EXAMPLE:

A gang is engaged in clearing a section of a mine after a rockfall. The average concentration of hydrogen sulphide (H_2S) is 15 mg/m^3. If these men each work six 8-hour shifts per week, what will their equivalent exposure to H_2S be?

In this case the 8-h/day formula should not be applied, but the 40h/week formula should, since these men will be exposed to H_2S for $6 \times 8 = 48$ hours per week. As the exposure is to an average concentration of 15 mg/m^3***, $E_{av} = 15\ mg/m^3 = E_{av\text{-}cum}$.

$$\text{Thus } E_{eq} = 15 \times \frac{48}{40}$$
$$= 18\ mg/m^3$$

**It should be noted that the formulae for E_{eq} actually only apply to a linear dose-effect relationship. For an exponential or sigmoid relationship an exponential or sigmoid expression should be used for a correction factor. For most practical purposes the above are, however, satisfactory except for substances with a C-value (see below).

***It must be noted that the ACGIH TLVs may not be the same as those laid down in South Africa in terms of the Mines and Works Act (Act No. 27 of 1956). Regulation 10.6.6 of this act requires a maximum permissible concentration for H_2S of 20 p.p.m. All mines which fall within the jurisdiction of South African Laws must comply with the requirement of the Mines and Works Act and the regulations framed thereunder. (20 p.p.m. = 30 mg/m^3).

These men are exposed to an equivalent level of 18 mg/m^3 which is in excess of the TLV.TWA of 15 mg/m^3. The equivalent exposure should therefore be reduced to 15 mg/m^3. This can be achieved by reducing the work time to 40 hours per week or by applying dilution ventilation. Alternatively gas-absorbing respirators or gas masks should be worn.

5.2 Short Term Exposure Limits (TLV.STEL)

For many hazardous substances a momentary peak concentration, even beyond the TLV.TWA excursion limits, may be tolerated, provided that the exposure is for only a limited time-interval. These concentration values are designated short term exposure limits (TLV.STEL). The ACGIH lists these for 15 minute periods. TLV.STELs thus represent the maximum concentration to which a worker can be continuously exposed for a period of up to 15 minutes without suffering from irritation, chronic or irreversible tissue change, or narcosis of sufficient degree to increase accident proneness, impair self-rescue, or materially reduce work efficiency, provided that no more than four excursions per day are permitted, with at least 60 minutes between exposure periods and provided also that the daily TLV.TWA is not exceeded.

From Table 2 it can be seen that the TLV.STEL value very often equals that of the excursion limit. The above restrictions thus apply to both. For substances such as chlorine, hydrogen sulphide and carbon monoxide the TLV.STEL is higher than the excursion limit. This simply means that the excursion limit may be reached more than four times per day. The TLV.TWA, however, may not be exceeded in respect of any 8-hour shift.

The ACGIH recommends that TLV.STEL values should not be used as engineering design criteria nor be considered as emergency exposure levels (EEL) such as have been suggested for calculating risks from any large scale accidental air pollution, e.g. in the bulk handling, transport and storage of chemicals.[12]

5.3 Ceiling concentrations (TLV.C)

Certain substances such as chloroform, hydrogen chloride, nitrogen dioxide and vanadium fumes are predominantly fast acting. Even short-term exposure to high concentrations of such substances may thus have some deleterious effect on the human body. TLV.TWAs for these are thus unsatisfactory. Substances with this type of response are best controlled by a ceiling (C) limit that should never be exceeded. These limits therefore constitute maximum allowable concentrations (MACs) a term which has been confused with TLV.TWA limits. This is particularly so where the German definition (Maximale Arbeitsplatz-Konzentration, MAK) is translated literally.[13]

5.4 Threshold limit values for mixtures

When two or more hazardous substances are present and the components in the mixture have similar toxicological effects, their combined effect must be considered. The effects of the different hazards should thus be considered as additive, unless information to the contrary is available.

The TLV of a mixture is considered to be exceeded if the sum of the following fractions exceeds unity:

$$\frac{C_1}{TLV_1} + \frac{C_2}{TLV_2} + \frac{C_3}{TLV_3} + \ldots + \frac{C_n}{TLV_n}$$

Thus, for compliance

$$\sum_{n=1}^{n} \frac{C_i}{TLV_i} \leqslant 1$$

where C_i = the observed concentration (average in p.p.m. or mg/m^3) of a contaminant i and
TLV_i = the threshold limit value (in p.p.m. or mg/m^3) of the particular contaminant i.

EXAMPLE:

The atmosphere in a work place contains 450 p.p.m. of acetone (TLV = 1 000 p.p.m.) 250 p.p.m. of ethyl acetate (TLV = 400 p.p.m.) and 120 p.p.m. of methyl ethyl ketone (TLV = 200 p.p.m.)

For this mixture (total atmospheric concentration of 820 p.p.m.)

$$\sum_{n=1}^{n} \frac{C_i}{TLV_i} = \frac{450}{1\,000} + \frac{250}{400} + \frac{120}{200}$$
$$= 0{,}45 + 0{,}625 + 0{,}6$$
$$= 1{,}675$$

The threshold limit value is thus exceeded.

When there is good reason to believe that the chief effects of the different harmful substances are not additive but independent, such as for example lead fumes and sulphur dioxide, the TLV is ordinarily exceeded when at least one member of the series

$$\frac{C_n}{TLV_n}$$

has a value exceeding unity.

Antagonistic action or potentiation may occur with some combinations of atmospheric contaminants. At present such cases must be determined individually.

Many hazardous substances may be encountered in the mining industry. For most of these the ACGIH has recommended threshold limit values and permissible excursion limits beyond the TLVs. The ACGIH, however, maintains that the TLVs are not intended for use, or for modification for use:

(a) as a relative index of hazard or toxicity,
(b) in the evaluation or control of community air pollution nuisances,
(c) in estimating the toxic potential of continuous, uninterrupted exposures or other extended work periods,
(d) as proof or disproof of an existing disease or physical condition, or
(e) for adoption by countries whose working conditions differ from those in the U.S.A. and where substances and processes differ.[3]

5.5 Biological limit values (BLV)

Since the physiological response of individuals to certain environmental hazards can vary considerably it may often be desirable to measure a particular worker's response to a specific hazard. Thus the blood, urine, hair, nails and other body tissues or fluids or exhaled breath may be analysed to determine the amount of substance absorbed and hence to obtain some measure of the individual's overall exposure and possibly to corroborate the findings of particular clinical symptoms. In addition to the analysis for the particular contaminant, changes in the amount of some critical biochemical constituent or of the activity of some critical enzyme may be determined. The limits up to which these biological parameters may deviate from the normal condition are referred to as the Biological Limit Values (BLV). Thus, for mercury, for example, an excretion level in urine of 10 $\mu g/\ell$ is considered as normal, but if the level excreted exceeds 50 $\mu g/\ell$ this is considered excessive.[14] Many Public Health Organizations even consider "50 $\mu g/\ell$ with symptoms" or "100 $\mu g/\ell$ without symptoms" as the biological threshold limit value.

6 EXAMPLES OF HAZARDOUS SUBSTANCES

In considering the health hazards of substances which may be encountered in the mining industry, not only the raw ores mined, but intermediate as well as end-products of further refining should always be known in order that the necessary safety precautions may be taken.

Space does not permit an elaboration on all the existing hazardous materials. Only a few selected examples will therefore be discussed here.

6.1 Industrial Solvents

Numerous organic liquids are used as solvents in many processes where water is incapable of dissolving the substance at hand. These have been applied for a variety of purposes such as extraction, ore flotation, degreasing, dry cleaning and many others.

When using an industrial solvent, not only the health hazard, but also its fire and explosive properties must be taken into account. A large number of most useful solvents unfortunately are very flammable liquids. The fire risk is enhanced by their volatility. In addition flammable solvents form explosive mixtures with air, often at normal room temperatures. Where the room temperature is higher than the flash point of such liquids, very stringent safety precautions must be applied.

The halogenated hydrocarbons probably constitute the most important as well as the most widely used solvents. Some are highly toxic while others may be less so. All are, to a varying degree, powerful narcotic agents. Saturated members such as carbon tetrachloride and tetrachloroethane cause liver and kidney damage. Highly toxic phosgene may be released by some on heating. The aliphatic members are usually non-flammable.

Of the aromatic chlorohydrocarbons chlorobenzene is probably best known. It is a flammable liquid with an acute action on the central nervous system leading to unconsciousness. Certain of the lower chlorinated naphthalenes have an injurious action on the liver which may result in toxic jaundice.

Diesel fuel is also often used as a degreasing agent or for washing oily machine parts. These fuels may be regarded as a mixture of hydrocarbons but contain, in addition, related sulphur, oxygen and nitrogen compounds and trace amounts of metals such as iron, lead, copper and aluminium. The more volatile fractions have a mild anaesthetic action and may produce a severe chemical pneumonitis if inhaled into the lungs. Dermatitis from lower boiling-point products may develop, usually as the result of their defatting effect on the skin. In the mining industry the health effects of diesel fuels are also associated with the exhaust emissions of internal combustion engines. These emissions contain carbon monoxide, carbon dioxide, sulphur dioxide, nitrous oxides, polycyclic aromatic hydrocarbons (PAH), aldehydes, unburned hydrocarbons and carbonaceous particles (soot).

6.2 Fumes and vapours

A number of minerals and especially metals may become airborne in the form of metallic fumes or vapours during the ore refining process. The inhalation of these metallic compounds may bring about an acute inflammation of the lung tissue or bronchioles. The acute effects are in many ways characteristic of gassing accidents and are often classified as such, particularly where the inhaled material has been a fume. The victim is acutely ill and the clinical symptoms are similar to those of pneumonitis. The severity varies widely, the least serious possibly being that due to the inhalation of zinc oxide fumes. This is familiar under the names "zinc ague" or "brass founders' ague" and somewhat resembles an attack of malaria or of influenza. Metallic dusts or fumes of manganese, vanadium, cadmium or beryllium give rise to more severe attacks which carry an appreciable mortality. Survivors tend to recover completely, though often only after a long period of illness.

6.3 Lead

Lead poisoning (plumbism) is one of the longest known occupational diseases in the mining industry. Hippocrates (370 B.C.) was probably the first of the ancients to recognize lead as the cause of colic in a man who extracted metals[15] and many symptoms were noted long before they were ascribed to the action of lead. The mild symptoms and signs of lead poisoning are known to include tiredness, lassitude, constipation, slight abdominal discomfort or pain, anorexia, altered sleep, irritability, anaemia, pallor and less frequently, diarrhoea and nausea. The presence of a blue line in the gums and a metallic taste are useful indicators of increased lead absorption. Severe symptoms and signs include severe intermittent abdominal pain (colic), reduction of muscle power, muscle tenderness and signs of nervous disorders. Chromosomal changes have also been reported and even increased abortion rates.[16]

Inorganic lead affects the blood and bloodforming tissues.[4] This effect occurs very early and may thus be used for diagnostic purposes. The excessive excretions of intermediate metabolites as well as the concentrations of lead per se in blood and urine provide diagnostic tools for the evaluation of the degree of lead poisoning, i.e. for biological monitoring.

The following levels, known as Lane's parameters have been widely accepted:[17]

Test	Normal	Acceptable	Excessive	Dangerous
Blood lead (μg/100 mℓ)	< 40	40–80	80– 120	> 120
Urinary lead (μg/ℓ)	< 80	80–150	150– 250	> 250
Urinary Coproporphyrin (μg/ℓ)	<150	150–500	500–1 500	>1 500
Urinary δ-ALA (mg/ℓ)	< 6	6–20	20– 40	> 40

Few indicators of lead effects are completely specific but increased absorption causes a gradual change in the above.

Although some compounds of lead are water-insoluble, the hydrochloric acid in the stomach and carbonic acid in the lungs may convert insoluble compounds into soluble products.

Lead is encountered in more than 200 industries. In the gold-mining industry it is used mainly in the zinc-precipitation of cyanide-extracted gold and also in the assay of gold ores. Unless in the form of fine dust, metallic lead at normal temperatures does not constitute a high health risk but when heated to 530 °C or more, vapour of fine particle size is produced, thus resulting in an environment with an extremely high health risk. Fine dust of inhalable particle size is produced in pulverisation, blending, abrasive working and dry rubbing-down processes.

A TVL of 0,2 mg/m^3 for inorganic lead dust was initially laid down by the ACGIH, but this has been reduced to 0,15 mg/m^3. In 1977 a proposal to reduce it further to 0,1 mg/m^3 was made,[18] but this has not been accepted as yet.

6.4 Mercury

Mercury is another of the toxic heavy metals known to the ancients. Even its health hazards were known to Pliney, who, almost 2 000 years ago, described mercurialism in writing of the diseases of slaves.

In industry, mercury may be encountered as the metal in rectifiers, contact breakers or switches, or in the form of numerous inorganic compounds for a variety of applications. In the gold mining industry mercury has been used to extract virgin gold by amalgamation, for a number of years, but because of the health risk involved, very few mines still use this process.

The most insidious exposure risk related to metallic mercury probably stems from the fact that mercury is readily volatilised in air at normal temperatures.

Its concentration C in air can be calculated from:[19]

$$C = \frac{P \times 2{,}42 \times 10^4}{T} \quad \text{mg/m}^3$$

The saturation vapour pressure of mercury is given by

$$\log_{10} P = -\frac{3\,212{,}5}{T} + 10{,}169\,4$$

where P = vapour pressure (Pa)
T = absolute temperature (K)

At a temperature of 20 °C the saturation vapour pressure is thus 0,162 Pa, and the saturation air concentration will be 13,3 mg/m^3 which is 266 times the TLV of 0,05 mg/m^3 (see Table 2).

When mercury is spilled on floors and dirty tables, evaporation is facilitated by dust, which maintains minute globules and thus provides a larger air-exposed surface area. Grease tends to retard this evaporation.

Mercury salts and organic complexes are also volatile to varying degrees, but may be present in the form of air-borne dust.

Mercury may then be absorbed by inhalation or in the case of soluble salts, by ingestion. It is

generally a protoplasmic poison but inorganic compounds may cause dermatitis, vision disorders and chronic pharyngitis.[8] Occupational poisoning is usually chronic in form while acute conditions are limited to the pharyngeal area.

In chronic mercury poisoning, digestive and nervous symptoms predominate. Salivation and tenderness of the gums and mouth are usually early symptoms. Other early signs include digestive disorders, intermittent tremor, and neurotic disorders varying in intensity. Termination of exposure usually results in complete recovery.

Severe chronic forms of mercury poisoning present spectacular renal, digestive, mental and nervous disorders. The most characteristic symptom is mercurial tremor. This usually begins in the fingers, but the eyelids, lips and tongue may also be affected. The effect is best observed by studying the victim's handwriting, which becomes very irregular. In an acute form the tremor invades the hand, forearm, and so on, and may eventually reach the lower limbs so that walking becomes difficult. At this stage it is known as "hatter's shakes".

Another characteristic symptom is that of erethism which manifests itself in emotional instability. The man affected is easily upset and embarrassed, quarrelsome and loses self control. Drowsiness by day, depression, loss of memory and insomnia may occur, but hallucinations, delusions and mania are now rare.[14]

Where mercury intoxication becomes firmly established, termination of exposure may no more than alleviate the symptoms. Sweating cures and the administration of certain drugs may offer relief.

The TLV for metallic mercury vapour has been laid down by the ACGIH as 0,05 mg/m^3 and the U.S. Occupational Safety and Health Administration (OSHA) has adopted a ceiling value of 0,1 mg/m^3 for mercury and its organic compounds.[20]

Prior to 1971 the maximum acceptable urine excretion of mercury was generally accepted to be 250 $\mu g/\ell$ (at an adjusted S.G. of 1,024). However, in 1971, the National Institute of Occupational Safety and Health (NIOSH) reduced the limit to 150 $\mu g/\ell$. In 1968 this level was also accepted by the International Committee of the Permanent Commission and International Association on Occupational Health.[21]

6.5 Uranium

Uranium is one of those hazardous substances which evoke two different and totally unrelated dose-effects. One is due to its radioactive property, while the other is due to its chemical nature.

In South African gold mines the uranium concentration is known to be so low that the radioactivity presents no significant health hazard during mining operations, provided adequate ventilation is maintained. Even the concentrates in extraction plants do not pose a problem as far as the external radiation is concerned, because uranium emits mainly α-radiation at an energy level which is too low to permit penetration of the outer layers of the skin. If, however, uranium compounds become airborne and the working environment is contaminated, they may constitute a serious internal radiation hazard if inhaled.

Radioactive decay of the unstable uranium nuclei results in the formation of a chain of successive parents and daughters, one member of the series being a noble gas, radon-222, having a half-life of only 3,8 days. This gas constantly diffuses out of uranium-bearing rock while decaying to its particulate, also short-lived, daughters. These so-called daughters may attach to or be adsorbed onto dust or condensation nuclei and will thus constitute a serious internal hazard if inhaled. Radiation hazards are dealt with more fully in chapter 27.

Because uranium has a low specific radioactivity its chemical damage to the human body on inhalation, ingestion or absorption through the skin, is more likely than radiation damage. In fact, uranium and its compounds are highly toxic substances, those compounds that are most soluble in body fluids being the most toxic.

When soluble compounds are inhaled they quickly enter the blood stream and large fractions may be excreted in the urine. However, an appreciable amount may remain in the kidneys and will cause lesions, with damage to the convoluted tubules, and necrotic nephrosis. This characteristic injury to the kidneys, together with the resulting albuminuria, can be used as a sensitive and reliable indicator of uranium poisoning. Exposure may also be monitored by analysis for uranium in the

urine, the acceptable maximum excretion level being 50 $\mu g/\ell$. A level of 100 $\mu g/\ell$ should prompt immediate action.[22]

In South African extraction plants a TLV of 0,15 mg/m^3 for airborne uranium has been accepted[22] and regular checks should be performed to ensure that levels are kept within this limit.

Any occupational disease has an essential cause which can usually be identified. In the mining industry it may be difficult to point to any one particularly hazardous chemical but much information on numerous agents is available. Environmental monitoring of suspected materials should be performed regularly to identify risks before harmful effects have occurred in exposed workers. This is especially appropriate for irreversible and disabling diseases which take a long time to develop, such as the pneumoconioses and occupational cancer.

The types of preventative measures to be adopted depend on the nature of the harmful substance or agent, and its routes of absorption into the body. Built-in protection, inherent in the design of a process, is preferable to a method which depends on continual human intervention or implementation. Safe maintenance and meticulous housekeeping are as necessary as good design and installation, and the two basic principles of suppression of occupational hazards, prevention and control, are at all times to be applied.

In all endeavours to eliminate occupational health hazards in the mining industry the most important resource, man, must continually be seen as the pivot about which all considerations rotate. The area occupied by man, in any mine and especially the breathing zone, should always enjoy top priority.

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