CHAPTER 26

Diseases caused by ionizing radiation

Properties of the causal agent

Ionizing radiation comprises those forms of radiation that, upon interaction with matter, give rise to particles of opposite electrical charges (ions). Ionizing radiation (both natural and artificial) is of two types: electromagnetic and corpuscular. The former is characterized by a frequency range of more than 3.0×10^{15} Hz (wavelength less than 1.0×10^{-17} m) and energy per photon range (eV) of more than 1.2×10^{1} (X-rays, gamma-rays). Corpuscular radiation comprises alpha and beta particles, electrons, protons, deuterons, neutrons, etc.

Occurrence and uses

Ionizing radiation has always been a part of man's natural environment (cosmic rays, natural radioelements). Today, man-made ionizing radiation is widely used in industry, agriculture, medicine and scientific research. The sources of ionizing radiation are either high-energy electrical devices (X-ray machines or particle accelerators such as betatrons) or radionuclides (natural or artificial). Radionuclides are used either fully encapsulated (sealed sources) or as tracers added directly to the study systems (unsealed sources).

Occupations involving exposure to ionizing radiation

Persons at greatest risk of exposure to ionizing radiation include: uranium miners and mill workers, nuclear reactor and atomic energy plant workers, industrial radiographers (including those doing field work involving pipeline welding), certain health personnel (radiologists), workers employed in the production of radionuclides, scientists using radioactive material for research, and luminous dial painters.

Mechanism of action

The absorption of radiant energy by the tissues leads to adverse biological effects. The amount of radiant energy absorbed by the tissues depends on the wavelength of the radiation and the energy,

size, and charge of the particles. X-rays and gamma-rays of short wavelength and neutron particles travel a long distance in the air and penetrate deep into the tissues before being absorbed (having transferred their energy to the media they travel through). On the other hand, X-rays of long wavelength and alpha-particles travel only a few millimetres in the air, and are absorbed by thin layers of matter (tissues of less than a few millimetres' thickness). The unit of absorbed dose of radiation is the gray (Gy) (1 Gy = 1 J/kg = 100 rad).

Exposure to ionizing radiation occurs in two ways: externally and internally.

(1) *External exposure* to radiation occurs from sources localized outside the body. The effects of external exposure depend on the penetrating power of the radiation: it is mainly the outer integument that absorbs most of the radiation of a low penetrating power, whereas highly penetrating radiation reaches deep-seated tissues and organs.

(2) *Internal exposure* is caused by radioactive substances that have entered the body. Radioactive substances enter the body mainly by inhalation (radioactive dusts, vapours, or gases), although entry by ingestion (swallowing of contaminated food, water, etc.) and skin penetration (particularly through wounds in case of accidents) may also be significant. The mechanisms of absorption, distribution, biotransformation, and excretion of radionuclides are the same as those of their non-radioactive counterparts. The only difference is in residence time, which, in the case of radioactive substances, depends on both metabolism and radioactive decay of the radionuclide.

Assessment of exposure

Environmental assessment

Geiger–Müller counters and scintillation counters are used for the detection of the presence of ionizing particles and the determination of the concentration of radioactive isotopes in the body, respectively. They are used also for the detection of radioactive contamination of surfaces or skin. If they are used for the measurement of the absorbed dose or of the rate of absorption, they must be calibrated for the particular type of radiation energy being measured.

Dosimeters used for measurement of ionizing radiation include: (*a*) ionizing chambers, film strips, or thermoluminescent devices. They are most suitable for personal monitoring of the doses received by the individuals who carry them on their suits in all work locations. Usually, pocket dosimeters (ionizing chambers) are used for screening purposes, whereas film dosimeters are used for regular surveillance.

Possible air pollution by radioactive substances is assessed by first passing the polluted air through appropriate sorbents (for gases and vapours) or filters (for particles) and then measuring the radioactivity of the sample collected.

Biological assessment

The methods for the biological assessment of exposure to radioactive substances are the same as those used for the biological assessment of exposure to their non-radioactive counterparts. They are used only when internal contamination with radioactive substances is suspected. The biological specimen (urine, blood, faeces, expired air, etc.) is selected in relation to the target organ of the toxic substance in question. Radioactivity is measured directly or after separation of the radioactive substance by radiochemical methods. There is also the possibility of measuring the content of some radionuclides directly by whole-body counters, scintillation detectors, etc.

Clinical effects

The harmful effects of irradiation may be somatic or genetic. Somatic effects develop directly in the irradiated individual, while the genetic effects appear in his or her progeny. The possible genetic effects resulting from occupational exposures are to a great extent still unknown.

Radiation effects may be either stochastic or non-stochastic. Stochastic effects are those whose probability of occurrence, though not necessarily their severity, is considered to be a function of the dose received; the dose in such cases is assumed to be without threshold. Thus, with increasing dose, the incidence of stochastic effects increase in the exposed population. It is accepted that genetic and carcinogenic effects are stochastic. The risk of increased incidence of malignant tumours induced by radiation is the main stochastic somatic risk.

The term non-stochastic is applied to somatic effects for which the severity of the effect varies with the dose and for which there is a threshold value. Examples include cataract, non-malignant skin damage, suppression of haematopoiesis, and damage to gametes leading to loss of fertility.

Acute effects

Whole-body irradiation. Irradiation of the whole body with over 1 Gy of penetrating radiation in a single exposure or over 1–2 days results in the so-called acute radiation syndrome, which is characterized by cell damage and death in the exposed tissues. Under current occupational conditions, such exposures are extremely rare, and are seen only in cases of accidents. Radiation doses of 0.05–0.25 Gy may not give rise to any symptoms, though there may be chromosomal damage detectable in peripheral lymphocytes. Exposure to higher doses of up to about 1 Gy does not lead to any symptoms, but in some individuals a small decrease in the number of white blood cells and platelets may be observed compared with pre-exposure values, especially if pre-exposure values were established on a group basis.

Doses of 1–2.5 Gy produce prodromal symptoms (nausea, vomiting, fatigue) and early haematological changes, particularly early lymphocytopenia and transient leukocytosis. After a latent phase, radiation-induced suppression of bone marrow leads to leukopenia, thrombopenia, and anaemia. Haemorrhage and infection are common. The ID_{50} in untreated subjects is about 3 Gy. Doses above 4 Gy cause severe acute radiation syndrome with gastrointestinal complications.

Local irradiation. Accidental exposure of parts of the body—usually of the hands in occupational exposure—is more frequent than whole-body irradiation. Immediately after irradiation, or within a few minutes of it, erythema develops, which is the primary skin reaction and which may be rather mild in the case of low-level exposure. Then, before the development of a second phase of symptoms, there is a latent period, the length of which is inversely related to the size of the dose—i.e., the higher the dose, the shorter the latent period. The second phase includes hyperaemia and oedema. From this stage onwards the course of the symptoms varies depending on the degree of exposure. If the exposure has been below 10 Gy, hyperaemia and oedema gradually diminish, but hyperpigmentation or depigmentation may develop. If the exposure has been greater than 20 Gy, the second phase develops after a shorter latent period, and is followed by the development of vesicles and ulcers. In cases of very high exposure (25–50 Gy), hyperaemia and oedema are followed by necrosis.

Chronic effects

Chronic radiation sickness may occur in individuals who have been repeatedly exposed to ionizing radiation over a long period (several years) during which the total cumulative dose has reached at least 1.5–4 Gy.

Mild forms are revealed by the presence of slight neuroregulatory disorders (slight imbalance of the autonomic nervous system, tendency towards arterial hypotension, tachycardia and sinus arrhythmia, dyskinesia of the intestine and the biliary tract, general excitability) and moderate and unstable leukopenia. In more advanced cases, the changes become more severe and stable. There is a further progression of autonomic nervous disorders, inhibition of the secretory function of the stomach, the appearance of clinical and electrocardiographic indications of dystrophic changes in the myocardium against a background of persistent arterial hypotension, signs of microstructural changes in the central nervous system, ovarian dysfunction (hypomenorrhoea and oligomenorrhoea) in women, bone marrow hypoplasia with persistent leukopenia (both granulocytopenia and lymphocytopenia), and less persistent thrombocytopenia. The development of anaemia is an unfavourable prognostic sign. In chronic radiation sickness caused by radioactive substances incorporated in the bone tissue, ostealgia may develop.

Chronic radiodermatitis may develop when the total radiation dose to the skin reaches at least 20–30 Gy. The manifestations are paraesthesia, disturbances of sensation, pain, itching, dryness of the skin, smooth lines on the palms and the surfaces of the terminal phalanges, and moderate dystrophy of the finger nails. After cumulative doses of the order of 40 Gy, painful cracks and focal hyperkeratosis and congestive hyperaemia appear. Late radiation ulcer may develop after cumulative doses of 50 Gy or more; this condition has a protracted course and the healing capacity of the body is distinctly reduced. Radiation carcinoma of the skin sometimes develops at injury sites.

Eye cataract may develop following exposure of the eye lens to large doses of X-rays, gamma-rays, and particularly, neutrons. This condition is characterized by the formation of a subcapsular radiation cataract at the posterior pole of the lens. While neutron radiation may induce lens opacities at a dose of only 0.5 Gy, doses of 6 Gy or more of X-rays are required at a single exposure to produce a similar effect. Eye cataract may also develop after long-term exposure, but the accumulated dose must be considerably higher. In its early stage, it can be easily distinguished from senile cataract. However, this is no longer possible if the nucleus of the lens is also affected.

Delayed effects

Carcinogenesis. A variety of cancers have been observed following exposure to ionizing radiation. The following types of cancer are related to occupational exposure:

Skin cancer (squamous cell) usually occurs after chronic radiodermatitis. Medical and dental practitioners who have worked in the past with radioactive substance, without adequate protection are most liable to such cancers.

Bone tumours are seen in radium dial painters and medical personnel responsibile for radium therapy. Radium accumulates in the bone matrix.

Leukaemias (myelogenous) occur mainly in radiologists.

Lung carcinomas are found in uranium and pitchblende miners. The causative agents are radon and its daughter products.

The period between exposure to ionizing radiation and the development of cancer may vary from a few years to several decades. It is not known whether a threshold dose exists below which cancers do not occur. Exposure limits for radiation exposure are based on the assumption that there is a linear, non-threshold relationship between dose level and the development of cancer. Epidemiological studies indicate that there is an increased incidence of the above type of cancers in occupational groups exposed to cumulative doses of several Gy. However, at lower doses and lower dose rates, the statistical probability of tumour development is so small that changes in cancer incidence under present working conditions (exposure levels) have not been found to be statistically significant in most cohort studies.

Exposure–effect relationship

The exposure–effect relationship has already been discussed under different preceding headings.

Prognosis

Acute effects of radiation caused by a dose up to 4 Gy have a favourable prognosis if they are treated properly.

The absence of early symptoms may indicate that the radiation dose did not exceed 1 Gy. Changes in the peripheral blood (extent and persistence of lymphocytopenia, high initial neutrophilic leukocytosis) during the first day are typical of heavier exposure (radiation dose exceeding 4 Gy). Early changes in arterial pressure and pulse rate, hyperthermia, and hyperbilirubinaemia occur in the extremely severe form of injury (radiation dose approximating to 10 Gy). Liquid stools for a short time during the first day are also an indication of severe irradiation of the intestines (radiation dose of the intestinal region exceeding 6–8 Gy). Finally, the appearance of erythema on the first day points to a high (>4 Gy) local or whole-body irradiation.

Acute local injury resulting in ulcers and necrosis may lead to deformation and atrophy. In such cases it may become necessary to amputate the affected body part.

The prognosis of chronic radiation sickness is in general favourable and the manifestations improve or at least remain stable after the exposure has been terminated. Eye cataract and radiodermatitis are, however, irreversible. The prognosis of malignancies does not differ from that of tumours of other etiology.

Differential diagnosis

Some of the clinical manifestations of radiation effects (symptoms of acute exposure, early stages of cataract, acute and chronic radiodermatitis) have rather typical features. When there is clear evidence of radiation overexposure, the diagnosis can be made with certainty. The association between the other less specific or non-specific symptoms (particularly bone marrow suppression and cancers) and occupational exposure can be considered in individual cases only on the basis of probability. If there is evidence of heavy occupational exposure in the past, the probability that the cancer is related to occupational exposure is greatly increased.

Susceptibility

Post-radiation overexposure and diseases affecting organs sensitive to ionizing radiation may increase susceptibility. In some countries, work

involving exposure to ionizing radiation is not permitted if the individual is suffering from any of the following conditions: haemoglobin less than 120 g/litre for women and 140 g/litre for men; persistent changes in the composition of the peripheral blood; haemorrhagic diathesis; mental disorders and diseases of the peripheral nervous system with pronounced functional inadequacy; drug addiction; obvious forms of neurosis; malignant tumours and precancerous diseases; organic diseases of the internal organs with frequent acute attacks; stage II hypertension; visual acuity below 0.6 for the better eye and below 0.5 for the other eye; and disseminated skin diseases.

There is no evidence of differences in susceptibility between men and women, but since the fetus may be more sensitive pregnant women need to be protected from ionizing radiation.

Health examinations

Preplacement examination

The preplacement examination should include a medical history and a physical examination, with special attention to the skin, eyes, and the respiratory system (in case of possible exposure to radioactive aerosols). A blood count should also be done.

Periodic examination

In medical terms, the periodic examination is the same as the preplacement one, and as a rule it should be carried out once a year. Additional examinations may also be necessary (e.g., after over-exposure or after radioactive contamination).

Workers whose exposure exceeds 30 % of the annual exposure limit require more detailed surveillance (including regular blood counts and tests of neurovascular function). Results of such examinations provide useful background information in treating accidental over-exposures and in detecting the presence of any conditions contraindicating further work involving ionizing radiation exposure. Exposures slightly higher than the exposure limits do not usually cause any detectable clinical injury and therefore clinical and medical observations are not considered appropriate for routine monitoring.

Case management

Acute radiation injuries require specialized treatment. After recovery, further exposure to ionizing radiation should be avoided. Persons who have recovered from chronic radiation sickness need not necessarily be transferred from their jobs if the working conditions have been improved in the meantime and if the radiation levels are well below the exposure limits.

If in any worker the established annual exposure limit is exceeded and there are no detectable adverse effects, the worker should still be removed temporarily from radiation exposure in order to permit his average cumulative dose to fall below the established limit.

In the case of external contamination with radionuclides, the contaminant must be removed as soon as possible in order to prevent internal contamination. Internal contamination requires specialized treatment to speed up the elimination of the radionuclide from the body.

Control measures

Exposure to radiation from both sealed sources (e.g., X-ray equipment, nuclear power plants) and unsealed sources (e.g., radioactive chemicals in medical and industrial laboratories) can be controlled by: (*a*) reducing the duration of exposure; (*b*) maintaining a safe distance between the worker and the source of radiation (the intensity of radiation varies inversely with the square of the distance from the radiation source); and (*c*) shielding the radiation source with materials that absorb ionizing radiation (e.g., lead). It is recommended that all radiation sources should be kept covered with shields, and personal protection devices (such as lead-rubber gloves and aprons) should always be used during work involving radiation exposure.

In the case of unsealed sources of ionizing radiation, there is an additional danger of internal contamination; good personal hygiene should therefore, be strictly enforced. The control measures for different radioactive substances vary from the simple application of general rules of safe laboratory work to completely enclosing work processes, depending on the physical state of the substance (solid, liquid, gaseous), the amount, and chemical and radioactive toxicity.

Exposure limits for ionizing radiation have been recommended jointly by ILO, WHO, IAEA, and NEA.[1] For the control of stochastic effects, the annual *effective dose-equivalent*[2] *limit* is 50 mSv (5 rem). For non-stochastic effects, the annual *dose-equivalent limit* for individual organs and tissues is 500 mSv (50 rem), except for the eye lens, for which the limit is 150 mSv (15 rem). In terms of radiation dose, the internal exposure limits are the same as those for external exposure. The limits on intake may be calculated for individual radioactive substances on the basis of their radioactivity.

Pregnant women should not be subjected to annual exposures exceeding 30% of the dose-equivalent limits.

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[1] Basic safety standards for radiation protection. Vienna, International Atomic Energy Agency, 1982 (Report of an advisory group jointly sponsored by IAEA/WHO/ILO/NEA).

[2] The *dose-equivalent* is the product of the absorbed dose (in Gy) and the quality factor of the type of ionizing radiation (e.g., 1 for X-rays, 10 for neutrons, and 20 for alpha-particles). The unit of dose-equivalent is the sievert (Sv), and 1 Sv = 1 J kg = 100 rem. The *effective dose-equivalent* corresponds to the mean dose-equivalent in a tissue modified by a weighting factor reflecting the detriment from stochastic effects.

CHAPTER 27

Occupational skin diseases[1]

Properties of the causal agents

The number of possible causal agents is very large, and only major classes of common hazards can be mentioned here.

Physical agents. These include pressure or friction, weather conditions (wind, rain, frost, sun), heat, radiation (ultraviolet, ionizing), and mineral fibres.

Chemical agents. These are further divided into four categories:

(*a*) primary irritants—acids, alkalis, lipid solvents, detergents, metallic salts (of arsenic, mercury, etc.), etc;

(*b*) sensitizers[2]—metals and their salts (chromium, nickel, cobalt, etc.), compounds derived from aniline (*p*-phenylenediamine, azodyes, etc.), aromatic nitroderivatives (trinitrotoluene, etc.), resins (particularly monomers and additives such as epoxyresins, formaldehyde, vinyls, acrylics, accelerators, plasticizers), rubber chemicals (vulcanizers such as dimethyl thiuram disulfide, antioxidants, etc.), drugs and antibiotics (e.g., procaine, phenothiazines, chlorothiazide, penicillin, and tetracycline), cosmetics, turpentine, plants (e.g., primula and chrysanthemum), etc;

(*c*) acnegenic agents—chlorinated naphthalenes and bifenyls, mineral oils, etc; and

(*d*) photosensitizers—anthracene, pitch, aminobenzoic acid derivatives, chlorinated aromatic hydrocarbons, acridine dyes, etc.

Biological agents. Several microorganisms (microbes, fungi), skin parasites, and their products also cause skin diseases.

Occurrence and uses

Most of the possible agents occur in industrial work. Exposure to weather conditions is common among those who work in the open air e.g., agricultural workers and sailors). Some of the chemical agents may occur as undesirable contaminants of other chemicals or industrial

[1] This chapter includes skin diseases caused by physical, chemical or biological agents not included under other items.

[2] Almost all chemicals can act as sensitzers. The chemicals included here are some of the more potent ones.

byproducts (e.g., chromium in cement production and tetrachlorobenzodioxine in pentachlorophenol synthesis).

Occupations involving exposure to agents causing skin diseases

Some of the most exposed groups are: agricultural workers (weather conditions, plants, zoonotic agents, pesticides, antibiotics or other animal food additives, etc.); construction material production workers (cement, etc.); construction workers (cement, mineral fibres, paints, plastics, etc.); chemical production workers (types of exposure according to the substances present); electroplaters (degreasers, acids, metallic salts); dyers; glass-reinforced plastics production workers (mineral fibres, resins); painters (dyes, metallic salts, solvents, etc.); workers in engineering industries (cutting oils or lubricants, etc.); health personnel (drugs, antibiotics, local anaesthetics, disinfectants, etc.); animal dealers; and butchers (zoonotic agents).

Mechanisms of action

Physical agents cause direct mechanical, thermal or radiation injury to the skin. Similarly, most of the irritants damage skin directly by: (*a*) altering its pH; (*b*) reacting with its proteins (denaturation); (*c*) extracting lipids from its outer layer; or (*d*) lowering skin resistance. Reaction-producing skin allergy is mostly a delayed type of hypersensitivity reaction. The sensitizing agent combines with protein in the epidermis to form a hapten-protein complex, against which antibodies are produced. Acnegenic agents block the sebaceous glands and ducts, causing local inflammation. Photosensitizers increase the sensitivity of skin to ultraviolet radiation.

Assessment of exposure

Occupational skin diseases result from direct contact of the skin with the causal agent. Thus, work history and clear evidence of the presence of the agent in question in the material handled by the worker is essential for the assessment of exposure.

Clinical effects

Primary irritant contact dermatitis is the most frequently encountered occupational dermatosis. The acute form is characterized by erythema, oedema, papules, vesicles or bullae, localized as a rule on the hands, forearms or face. A skin patch test with the causal agent can induce

and confirm irritative effects and the etiology of the dermatosis. The manifestations of chronic irritant contact dermatitis are similar to those of many other dermatoses, such as chronic allergic contact eczema, hyperkeratosis, etc. Whereas the causal agent of acute irritant dermatitis is usually obvious, in chronic dermatitis the etiological factor is mostly inconspicuous. The lesions are often caused by detergents, weak alkalis, organic solvents, or less potent or diluted chemicals so that their effect is delayed, i.e., it appears only after the resistance capacity of the skin has been exhausted.

In addition to their direct effects, primary irritants can render the skin vulnerable to infection and injury, particularly injury by sensitizing agents.

Allergic contact dermatitis (eczema) (both acute and chronic) has the same clinical features as non-occupational eczema. The acute form resembles acute irritant dermatitis and the chronic form is characterized by lichenification and fissuring. Patch tests are helpful in diagnosing skin hypersensitivity. (The patch test involves the application of a non-irritating concentration of the suspected allergen to a part of the unaffected skin of the patient for 24–48 hours; in positive cases, an eczematous dermatitis develops beneath the covering patch.)

Occupational acne is characterized by plugged sebaceous follicles and suppurative lesions. While the acne caused by mineral oil or tar and pitch affect only those sites of the body that come into close contact with the agent, that caused by chlorinated aromatic compounds may be more generalized.

Microtraumatic lesions are caused by natural or man-made mineral fibres (glass or other silicates), and are characterized by tiny whitish or pink papules localized on the exposed sites, particularly on the arms.

Acute solar dermatosis is regarded as an occupational disease if it is largely promoted by photodynamic substances used in the occupation—e.g., tar and tar-derived products, sulfonamides, phenothiazines, or tetracyclines.

Skin diseases caused by ionizing radiation are discussed in Chapter 26.

Skin cancers of occupational origin (squamous or basal cell carcinoma, rarely other types) do not differ from other similar non-occupational tumours (see Chapter 28). Histological examinations are useful in defining the exact type of the tumour, but they do indicate the etiology. Occupational tumours tend to occur on the skin surface most exposed to the carcinogens and develop from precancerous lesions (hyperkeratosis, papillomatosis).

The most frequent *contagious occupational skin diseases* are zoonotic diseases: dermatophytoses, candidiasis, erysipeloid, tuberculosis verrucosa, etc. (see also Chapter 29).

Exposure–effect relationship

The extent and severity of contact irritant dermatitis, acne and radiation-induced diseases vary with the degree of exposure. On the other hand, allergic contact dermatitis (eczema) can be induced even by minute amounts of allergens.

Prognosis

Irritant dermatitis, acne, and infectious diseases heal after the causative agent has been removed. (For details of radiation-induced dermatitis see Chapter 26). The prognosis of allergic eczema depends on the nature of the allergen and the duration of the affection. If the patient is removed from the exposure early and if exposure to the allergen is confined to the workplace, full recovery is the rule. However, if the allergen is also widespread in the general environment (e.g., chromium, detergents, etc.), or if secondary allergy to bacterial infection in the eczematous lesions develops, the skin affection may last throughout life.

Differential diagnosis

The clinical picture of most occupational skin diseases is similar to that of their non-occupational counterparts. The differential diagnosis is based on two principles: (*a*) proper diagnosis of the nosological entity in order to exclude immediately diseases of non-occupational origin (e.g., palmar psoriasis, hyperkeratosis, mycosis, or contact allergic eczema); (*b*) identification of the etiological agent of occupational dermatoses (e.g., differentiating contact allergic eczema due to occupational exposure to epoxy resins from eczema due to hypersensitivity to turpentine in shoe-polish used at home).

The following general guidelines should be followed in the diagnosis of occupational skin diseases:

—The clinical picture, localization, and the course of the disease must correspond fully to the established characteristics of the occupational disease.

—Occupational exposure to the harmful agent must be proved with certainty.

—There must be a reasonable time relationship between the development of the skin disease and the onset of exposure to the suspected agent (i.e., a latent period—incubation time in infectious diseases, development of sensitization in eczema).

—In contact allergic dermatitis (eczema), patch tests should give positive results and confirmation may be obtained by other types of laboratory examination.

—There should be a positive response to removal from work and re-exposure. As a rule the affection improves during absence from work (e.g., during holidays, and relapses after return to work). It should also be noted that non-occupational eczema may deteriorate at the workplace owing to possible nonspecific irritation.
—The development of a skin disease in a group of workers usually indicates that the disease is of occupational etiology.
—Any non-occupational cause of the disease must be excluded, e.g., chemicals or other allergens or irritants at home, substances encountered in leisure activities and drugs.

Susceptibility

Persons with atopy (eczema and other allergic skin diseases and also allergic affections of other organs), chronic skin affections including hyperhidrosis, seborrhoea or ichthyosis, abnormal pigmentation, and precancerous skin lesions are more susceptible than others.

Health examinations

Preplacement examination

The preplacement examination should include a medical history and a physical examination, with special attention to the skin (of the whole body) and allergies (atopy).

Periodic examination

In medical terms the periodic examination is the same as the preplacement one. Patch tests are not recommended for the screening of symptom-free subjects. Intervals between examinations usually range from 6 months to 2 years, depending on the level of exposure at the workplace.

Case management

Patients with occupational allergic contact dermatitis (eczema) almost always require transfer to another workplace free of the causal allergens. Patients with irritative dermatitis and other acute non-allergic affections should be temporarily removed from exposure, and should be allowed to return to their original jobs only after the causal agent has been controlled. Permanent transfer to another job may be necessary in the case of subjects in whom repeated temporary transfers do not lead to a complete recovery. Precancerous conditions require permanent removal from exposure to the causal agent.

Control measures

Whenever possible, strong allergens, sensitizers, and carcinogens should be replaced by less dangerous substances. Skin contact with causal agents should be limited by technical control measures; the key to effective prevention is the elimination of skin contact with them. Protective clothing, aprons, gloves or barrier creams, boots, and face masks may be necessary. Basic facilities for personal cleanliness (showers, etc.) should be provided, and their use should be encouraged or made mandatory.

CHAPTER 28

Primary epitheliomatous cancer of the skin

This chapter deals with primary epitheliomatous cancer of the skin caused by tar, pitch, mineral oil, anthracene, and compounds, products, and residues of these substances.

Properties of the causal agents

Tar (or coal-tar) is a distillation product of coal. Pitches are of different types, and may be produced by the distillation of either coal or crude petroleum. The term mineral oil refers to petroleum and other hydrocarbon oils obtained from mineral sources. Anthracene is a distillation product of coal-tar. All these substances and their cancer-producing derivatives are complex mixtures in which the actual carcinogens are probably polycyclic aromatic hydrocarbons. They also contain a number of tumour-promoting compounds.

Tar is a brownish, viscous substance, and pitch is a heavy, viscid, dark-brown material. The derivatives of these substances may be liquid, solid, or vapour. As a general rule, bitumens derived from petroleum are much less carcinogenic than tars derived from coal.

Occurrence and uses

Carcinogenic products of carbonaceous materials are present in coal-tars and pitch, heavy tar oils, certain mineral oils, products of coking operations, soot, creosote oil, crude paraffins, and shale oil and its distillation and fractionation products. These substances have many uses as indicated in the section below.

Occupations involving the risk of epitheliomatous cancer

The following workers are at risk of developing primary epitheliomatous cancer of the skin: tar distillers; coal-gas manufacturers; coke plant workers; fishing net and rope makers; briquette manufacturers; caulking material manufacturers; roofers, road builders (exposure to pitch and tar products); shale oil workers and refinery workers; match factory, naphthalene, paper industry, and munitions

workers exposed to crude paraffins; ship stokers; carbon black makers; chimney sweeps (exposure to soot); and timber picklers (exposure to creosote oil).

Mechanism of action

Long-term repeated exposure to tars and related substances appears to cause cancer, predominantly at the site of contact with the skin. Metabolic activation of polycyclic aromatic hydrocarbons appears to play some role in their carcinogenicity.

Cancer may also develop as a result of a possible synergistic effect of polycyclic hydrocarbon carcinogens and ultraviolet radiation.

Assessment of exposure

Environmental assessment

The concentrations of polycyclic aromatic hydrocarbons in coal-tars and related materials and in air samples may be used to assess the toxicity of different substances and the level of exposure to polycyclic aromatic hydrocarbons at the workplace.

Routine methods to quantify contact of such substances with the skin are not currently available.

Fluorescent substances in tars on the skin may be detected under ultra violet light. If performed, this procedure must be undertaken judiciously in order to avoid a photosensitivity reaction.

Biological assessment

Useful procedures are not currently available.

Clinical effects

Erythema accompanied by a burning sensation ("pitch smarts") usually appears within a few hours or days of exposure at the site of contact with coal-tar, pitch, etc. The burning sensation is intensified by exposure of the affected parts to sunlight. After repeated exposure over several years, thickening of the skin, hyperpigmentation, comedones (blackheads), and follicular inflammation may occur. Conjunctivitis is common, and is often accompanied by photophobia.

Long-term exposure causes chronic skin changes, which include:

—irregular areas of atrophy;
—patchy hyperpigmentation and hypopigmentation;
—wart-like papillomas (particularly on damaged skin), some of which may later develop into squamous cell cancers; and
—basal cell carcinomas and keratocanthomas.

The scrotum is particularly prone to develop squamous cell cancers following exposure to tar and soot. Skin cancers usually take 5–50 years or more to develop, and may occur even after the cessation of exposure.

Inhalation of dusts and vapours of tar, pitches, etc. has been reported to cause tumours of the lung.

Exposure–effect relationship

Owing to a paucity of data, the exposure–effect relationship is not yet established.

Prognosis

The prognosis is good if lesions are detected and treated before extensive invasion occurs.

Differential diagnosis

The pathological identity of suspected epitheliomatous cancers can be confirmed by biopsy and histological examinations. Occupational and non-occupational causes must be differentiated.

Susceptibility

Factors contributing to increased susceptibility have not yet been established with certainty.

Health examinations

Preplacement examination

A medical history and a physical examination are necessary to ensure that precancerous conditions (e.g., keratoses) or skin cancers are not present.

Periodic examination

Regular examination of skin ensures early detection and treatment of premalignant and malignant lesions. It is sufficient to carry out such examinations at one-year intervals.

Case management

Premalignant and malignant skin lesions should be treated promptly in order to avoid progression. In individuals who develop lesions, removal from further exposure must be considered, especially in the case of cancers associated with polycyclic aromatic hydrocarbons. Failing this, very careful attention must be given to control measures.

Control measures

Whenever possible, tars and related substances should be replaced by non-carcinogenic materials. Technical control measures should be applied to reduce air and surface contamination. Work clothes should be kept separate from other clothes and should be washed and changed regularly. Impervious protective clothing such as gloves, smocks, and worksuits should be used whenever necessary. Finally, workers should be made aware of the hazards associated with work involving exposure to tars, pitches, etc. and of the protective measures recommended above.

As far as possible, workers exposed to polycyclic aromatic hydrocarbons should avoid exposure to ultravoilet light during and soon after work.

CHAPTER 29

Infectious and parasitic diseases

Properties of the causal agents

Exposure to live infective microorganisms and parasites and their toxic products is encountered in many occupations. The most important agents of occupation-related infectious and parasitic diseases are: (*a*) viruses (viral hepatitis, Newcastle virus disease, rabies); (*b*) chlamydiae and rickettsiae (ornithosis, Q fever, tickborne rickettsiosis); (*c*) bacteria (anthrax, brucellosis (undulant fever), erysipeloid, leptospirosis (Weil's disease), tetanus, tuberculosis, tularaemia, wound sepsis); (*d*) fungi (candidiasis and dermatophytosis of the skin and mucous membranes, coccidiomycosis, histoplasmosis); (*e*) protozoa (leishmaniasis, malaria, trypanosomiasis); and (*f*) helminths (hookworm disease, schistosomiasis).

The survival and pathogenicity of these agents in the working environment are largely determined by: physical and climatic factors (temperature, humidity, oxygen tension, soil conditions), nutritional and other requirements for multiplication, and, in the case of parasites, presence of obligatory reservoirs and vectors, which are mostly animals.

Occurrence

Work-related infectious and parasitic diseases are mostly encountered in:

—agricultural work;
—certain workplaces in warmer and less developed countries;
—hospitals, laboratories, clinics, autopsy rooms, etc.;
—work involving handling of animals and their products (veterinary clinics, slaughterhouses, meat and fish markets, etc.);
—outdoor work where animal excreta may be encountered (work in canals, rivers, ditches, sewers, docks, farm yards, construction sites, etc.).

Occupations involving exposure to infectious and parasitic diseases

Table 3 summarizes infectious and parasitic diseases encountered in different occupations.

Table 3. Summary of occupations and associated infectious and parasitic diseases

Occupation	Diseases
Agriculture, animal husbandry, forestry, trapping, and hunting	In both tropical and temperate areas: anthrax, arthropod-borne viral diseases (e.g., encephalitis, plague), coccidiomycosis, fungal infections, histoplasmosis, leptospirosis, Q fever, rabies, tickborne rickettsiosis, tuberculosis, and tularaemia In tropical areas only: arthropod-borne viral diseases (e.g., yellow fever, haemorrhagic fever), hookworm, leishmaniasis, malaria, schistosomiasis, trypanosomiasis
Construction work, land excavation, sewer work, ditching, mining	Coccidiomycosis, hookworm, histoplasmosis, leptospirosis, tetanus, wound sepsis
Meat and fish handling and packing	Bovine tuberculosis, brucellosis, erysipeloid, fungal infections, Q fever, tularaemia
Poultry and bird handling	Fungal infections, Newcastle virus disease, ornithosis
Work with hair, hides, wool	Anthrax, Q fever
Veterinarians	Tuberculosis, brucellosis, fungal infections, leptospirosis, Newcastle virus disease, ornithosis, Q fever, rabies, tularaemia
Physicians, nurses, dentists, laboratory technicians	Viral hepatitis, tuberculosis, other communicable infections
Work in warm, humid conditions, (kitchens, gymnasiums, swimming pools, etc.)	Fungal infections of the skin

Mechanism of action

Infection occurs when an unimmunized or non-resistant person comes into contact with an infective agent. The mode of entry and pathophysiology of different diseases vary considerably. While some agents are able to penetrate intact skin (anthrax, brucellosis, leptospirosis, schistosomiasis, tularaemia), others require the skin to be damaged (erysipeloid, rabies, sepsis, tetanus, viral hepatitis B) or macerated (fungal infections). Some protozoan pathogens enter the body through insect bites (leishmaniasis, malaria, tickborne rickettsiosis, trypanosomiasis). Infection may also occur by the inhalation of droplets, spores, or contaminated dust (coccidiomycosis, histoplasmosis, Newcastle virus disease, ornithosis, Q fever, tuberculosis). Agents of viral hepatitis A, diarrhoeal diseases, and enteroviral diseases

such as poliomyelitis enter the body through ingestion of contaminated food and water.

Some diseases result from an inflammatory reaction to toxins (endotoxins and exotoxins) produced by bacteria during reproduction.

Assessment of exposure

Assessment of the risk of exposure to infectious and parasitic diseases is only rarely feasible—e.g., search for encephalitis virus in ticks or for rabies virus in wild animals. However, the risk of a potential hazard in a group of workers can be assessed from:

—reports of cases of communicable or zoonotic diseases in groups of workers regarded as being at risk of contracting those diseases;
—epidemiological data on the incidence of communicable or zoonotic diseases;
—data on the prevalence of vectors and parasites;
—serological data on immunity status of workers (e.g., from serological or skin tests for tuberculosis and viral, rickettsial, chlamydial, and salmonella infections).

Clinical effects and diagnosis

Early signs of infections are seldom specific, but are usually sufficiently definite to warrant suspicion of disease if appropriate geographical and occupational factors are also present. Table 4 summarizes the early symptoms of infectious and parasitic diseases and possible corresponding diagnoses.

For full descriptions, diagnostic methods, differential diagnoses, prognoses, and treatment procedures of these diseases the reader is referred to standard medical textbooks.

Susceptibility

Individuals considered as being susceptible to infectious and parasitic diseases include: unimmunized persons; those recovering from serious systemic infections; those suffering from immunosuppression; and persons whose nutritional and general health status is poor.

Persons with renal and liver dysfunction are at increased risk of contracting leptospirosis and serum hepatitis. Minor injuries may increase the risk of skin infections, tetanus, rabies, and serum hepatitis. Wet or sweaty skin is more liable to attack by fungal infections; and anaerobic conditions resulting from puncture wounds and tissue destruction favour the multiplication of *Clostridium tetani*.

Table 4. Early symptoms of infectious diseases and possible corresponding diagnoses

Early symptoms	Possible diagnoses
Severe illness with bad headache and symptoms of nervous system involvement	Brucellosis, leptospirosis, malaria, rabies, tickborne rickettsiosis, or trypanosomiasis
Fever with respiratory problems or pneumonia	Coccidiomycosis, histoplasmosis, ornithosis, Q fever, or tuberculosis
Severe systemic symptoms preceded by a skin lesion or ulcer	Anthrax, leishmaniasis, trypanosomiasis, or tularaemia
Gastrointestinal symptoms followed by dark urine and jaundice	Viral or leptospiral hepatitis
Painful muscular spasms, particularly around the jaw[a]	Tetanus
Progressive debility and anaemia, especially if preceded or accompanied by haematuria or blood-stained diarrhoea	Schistosomiasis
Indolent itching and erythematous skin lesions	Erysipeloid (if on hands), hookworm (if on feet), or fungal or bacterial infection (if skin appears to be damaged or macerated)

[a] Note that if tetanus is not suspected at this stage it is easily missed.

Health examinations

The vast majority of persons exposed to infectious and parasitic diseases are self-employed rural workers in developing countries. Since they mostly live and work in areas that are not readily accessible, health examinations are rarely, if ever, done. However, certain groups of workers in rural areas (health workers, municipal workers, construction and mining workers, etc.) can be reached more easily and regular health examinations of such workers should be encouraged.

Moreover, in certain areas it may be possible to identify groups of rural workers at risk of contracting a particular disease; in such cases regular health examinations will be particularly useful. Whenever possible, health examinations should be promoted among rural workers.

Preplacement examination

The preplacement examination should include a medical history and a physical examination. The main objectives of this examination are: (*a*) to determine and record initial health status of the worker; (*b*) to identify susceptible persons; and (*c*) to diagnose and treat latent and active cases of infectious diseases. In occupations involving the risk of

tuberculosis (e.g., health service workers and veterinary and laboratory workers), the tuberculin test should also be done along with a chest X-ray. Depending on the geographical area and occupation, selected serological and microbiological tests may also be necessary in order to detect past or current infection. Whenever possible, all workers should be immunized against locally prevalent diseases for which vaccinations exist.

Periodic examination

In medical terms, the periodic examination is the same as the preplacement one. It involves the maintenance of medical records of febrile or infective illnesses by systematic inquiry and by repetition of previously conducted serological tests; in tuberculin-positive workers, a chest X-ray should be repeated.

In most rural workers, periodic examinations are desirable at annual intervals; in the case of health and laboratory workers, the interval should be 6 months.

Case management

Cases of infectious and parasitic diseases should be notified at once to the appropriate health authority. Specific antibiotic treatment or chemotherapy is fairly effective in most but not all diseases. The success of the treatment will depend on correct diagnosis and skilled care. Cases of tetanus should be treated urgently in well-equipped hospitals. Isolation of patients with contagious diseases may be necessary.

Control measures

Environmental

Some zoonoses can be controlled by eliminating their animal reservoirs or insect vectors. Spraying of residual insecticides may be used against mosquitos (larvae as well as adults), sandflies, and tsetse flies. Predatory fish can reduce the population of snails, that harbour schistosoma parasites, and rodent control is useful against leptospirosis. The immunization of cattle and domestic animals is an effective way of reducing the risk of brucellosis and rabies, respectively. By controlling and restricting the import of birds, domestic mammals, hides, wool, and products made from animal bone it may be possible to prevent ornithosis, psittacosis (parrot fever), and anthrax. In certain workplaces, dust suppression by exhaust ventilation may also prevent airborne anthrax and ornithosis.

Protection of workers

Health education

Exposed groups should be informed about the nature of infectious and parasitic hazards in their occupation and region. Special emphasis should be placed on personal hygiene and workers should be urged to wear appropriate protective clothing (especially boots and shoes), handle animals and animal products with care, avoid swimming and wading in contaminated water, avoid drinking unboiled milk, and protect themselves from animal and insect bites.

Specific prophylaxis

All workers should be vaccinated against tetanus, especially those working in agriculture. Other vaccines should be used as required: BCG for tuberculin-negative health workers; rabies and anthrax vaccines for veterinarians; typhus and Q fever vaccines for laboratory workers and populations in endemic areas. Chemoprophylaxis with suppressive drugs is important for workers in malarious areas. Immunoglobulins may be useful in providing passive protection in cases of injuries and in unimmunized persons at risk of developing tetanus, rabies, or hepatitis B.

Protective clothing

Gloves and boots should be worn by all persons working in mines, rivers, ditches, fields, etc. where there is risk of schistosomiasis, leptospirosis, and hookworm. Gloves and barrier creams are also useful in protecting animal and fish handlers from fungal infections and erysipeloid. Laboratory and certain health service employees may also require special protective clothing.

Codes of practice

Ideally, such codes are required for all occupations exposed to infectious agents, but at present they exist only for workers in laboratories and autopsy rooms, some groups of sewer workers, abattoir and animal product workers, and hospital staff (for protection against viral hepatitis). Codes for hospital staff include screening of blood donors, blood recipients and hospital staff for hepatitis B antigen, rules for the sterilization and disposal of instruments and contaminated materials, and the provision of separate facilities for dialysis and other surgical procedures for virus carriers.