Notes on the diagnosis of occupational diseases : prescribed under the National Insurance (Industrial Injuries) Act, 1965 (not including pneumoconiosis and allied occupational chest diseases).

DEPARTMENT OF HEALTH AND SOCIAL SECURITY

Notes on the diagnosis of occupational diseases

Revised November 1972

Prescribed under the National Insurance (Industrial Injuries) Act, 1965

HMSO 47p net

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NOTES ON THE DIAGNOSIS OF OCCUPATIONAL DISEASES

Prescribed under the National Insurance (Industrial Injuries) Act, 1965

(*Not including pneumoconiosis and allied occupational chest diseases*)

LONDON
HER MAJESTY'S STATIONERY OFFICE
1972

Foreword

THIS booklet contains revised notes on the diagnosis of occupational diseases prescribed under the National Insurance (Industrial Injuries) Act, 1965. It follows the lines of its predecessors but in addition contains a brief section on the benefits available and the determination of claims. The sections on pneumoconiosis and byssinosis have been omitted in this edition as they are now the subject of a separate booklet 'Pneumoconiosis and Allied Occupational Chest Diseases'.

As before the notes do not purport to be exhaustive and are not concerned with treatment but it is hoped that they may be found useful by General Practitioners dealing with these diseases.

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1970

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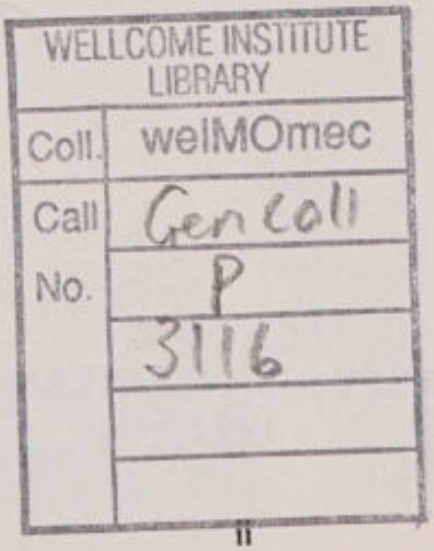

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DEPARTMENT OF HEALTH AND SOCIAL SECURITY

DECEMBER 1973

ADDENDUM TO " NOTES ON THE DIAGNOSIS OF OCCUPATIONAL DISEASES "

(Revised November 1972)

PRESCRIBED DISEASE No. 39—Papilloma of the bladder

The description of this disease has been extended to cover neoplasm of the epithelial lining of the urethra. Pages 42, 43 and 60 should be amended as follows:

Page 42 amend heading for disease No. 39 to read

39. PRIMARY NEOPLASM OF THE EPITHELIAL LINING OF THE URINARY BLADDER (PAPILLOMA OF THE BLADDER) OR OF THE EPITHELIAL LINING OF THE RENAL PELVIS, OR OF THE EPITHELIAL LINING OF THE URETER OR OF THE EPITHELIAL LINING OF THE URETHRA

Page 43 amend paragraph headed DIFFERENTIAL DIAGNOSIS to read as follows

" DIFFERENTIAL DIAGNOSIS. It is emphasised that the disease prescribed is primary neoplasm of the epithelial lining of the bladder, ureter, renal pelvis and urethra only and does not include invading growths from surrounding structures or neoplasms elsewhere. Nor does it include other conditions giving rise to frequency of micturition and haematuria even in persons thought to be at risk."

Page 60 The description of the disease as shown on page 60 should be amended to read as follows

39. Primary neoplasm of the epithelial lining of the urinary bladder (Papilloma of the bladder), or of the epithelial lining of the renal pelvis or of the epithelial lining of the ureter or of the epithelial lining of the urethra.

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Part I

1. POISONING BY LEAD OR A COMPOUND OF LEAD

Aetiology

Lead is a microconstituent of food-stuffs and is normally found in human tissue and fluids. When a worker inhales lead fume it is absorbed and an amount greater than normal will be found in the blood and urine; it cannot be too strongly emphasized that this indicates exposure to and absorption of lead only and not lead poisoning. Individuals vary in their sensitivity to lead and some may have symptoms when the blood lead level is only slightly raised, while others may have no symptoms in the presence of a grossly raised blood lead level.

Opinions differ as to what exactly is necessary before a firm diagnosis of lead poisoning is warranted, but such a diagnosis is certainly not justified unless there are present at least signs and symptoms of actual illness. Lead ingested by mouth is mostly excreted in the faeces: that fraction which is absorbed is trapped by the liver and largely returned to the intestine in the bile. Lead absorbed via the pulmonary tract on the other hand passes directly, easily, and more or less completely into the systemic circulation, by-passing the liver, so that much smaller doses absorbed thus may rapidly produce symptoms. In industry lead poisoning is almost invariably the result of inhaling lead-bearing dust or fumes, but it is important to remember that organic compounds such as tetra-ethyl lead (widely used as an anti-detonant in petrol) are absorbable through the unbroken skin. When lead is being absorbed rapidly into the body, it becomes widely distributed, and the question of whether or not it will cause symptoms of illness depends largely on the ratio between rate of absorption and rate of excretion; when absorption is slow and continuous over a long period of time, the factor of storage becomes more important. Lead is a cumulative poison which, in cases of chronic absorption, is deposited in the calcareous portion of the bones as an insoluble and harmless triple phosphate. There it may cause no symptoms, but the metabolism of lead under such conditions runs parallel to that of calcium. Conditions which favour the storage of calcium in the bones also favour the storage of lead, and conversely, stored lead is remobilised and returned to circulation by conditions, such as a depletion of the alkali reserve, which alter the reaction of the body fluids. It is for these reasons that single figures for lead excretion are difficult to interpret.

Bright metallic lead as such is not dangerous, but lead readily becomes coated, especially when heated, with a film of easily removed and easily

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reduction in haemoglobin. There may be a slight increase in the reticulocyte count. Punctate basophilia occurs but is not pathognomonic. Its appearance in a lead worker, however, should alert the observer to the possibility of plumbism.

Biochemical Abnormalities:—The interference by lead in the synthesis of haemoglobin is brought about by impeding the action of certain enzyme systems. This allows an accumulation of aminolaevulinic acid (ALA) porphobilinogen (PBG) and coproporphyrin III, which are all excreted in the urine in excess of normal. Raised levels of ALA show a constant relationship with clinical evidence of lead intoxication. There does not yet appear to be a general agreement about the significance of these biochemical abnormalities as an index of the stage where lead absorption merges into the state of lead intoxication. Most authorities hesitate to diagnose lead poisoning in the absence of signs and symptoms.

Palsy:—The commonest form of palsy in lead workers is the well-know 'wrist-drop", but nowadays it is not often seen. Lead tends to attack first muscles which are particularly subjected to fatigue, especially fatigue of an occupational nature. Paralysis, of lower motor neurone type, is preceded by muscular weakness, the poison picking out first, in the case of the upper limb, the long extensors of the middle and ring fingers, and affecting a little later the other extensors, but the weakness is not accompanied by neuritic pains; it is primarily a pure loss of muscle power. Later a brachial type of paralysis may appear, involving the supinator longus, biceps, and deltoid. 'Foot-drop' is less common.

Encephalopathy:—This formerly occurred in long continued cases of severe poisoning but it is said to be rare nowadays in persons who are exposed to metallic lead in the course of their work. However, it may occur after exposure to organic lead compounds and is characterised by mental dullness, inability to concentrate, faulty memory, tremors, deafness, aphasia, convulsions and coma.

POISONING BY TETRA-ETHYL LEAD. This substance differs from the inorganic lead compounds in that it is volatile at room temperatures, and lipo-soluble. It can therefore be readily inhaled, and on absorption it exercises a focal effect on the central nervous system. It is used in industry only as an anti-detonant in petrol, and in persons handling it in that connection it has given rise to acute poisoning of a severe and even fatal type. It is more easily absorbed than any other compound of lead in industrial use, and even a few days exposure may be sufficient to cause a rapidly fatal illness. In severe cases of poisoning the effect on the nervous system is startling; after a brief spell of indeterminate malaise there is a more or less abrupt onset of restlessness, talkativeness, excitement, and muscular twitchings, with insomnia, delusions, hallucinations, and even acute and violent mania. Typically this is accompanied by a fall in blood-pressure, a drop in body temperature, and more or less marked bradycardia. Milder cases show early fatigue,

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an involvement of the extrapyramidal motor system. There is a slight increase in the tendon reflexes, with ankle and patellar clonus, and there may be muscular twitchings varying from fine tremors to gross movements; the gait is sometimes of a peculiar high-stepping character, but alterations of the centre of gravity may produce in other cases a 'festinant' gait. In the established case the voice becomes monotonous, and there is typically a loss of the lines of facial expression, so that the patient presents a stolid, mask-like facies. This is occasionally coupled with abnormal emotionalism. Usually there is a complete absence of sensory disturbance. Blood, urine and cerebro-spinal fluid do not show any characteristic changes. Manganese dioxide is an irritant to the upper respiratory tract and an increased risk of pneumonia which is slow to recover has been reported in workers exposed to it. The underlying pathology is not clear, but permanent pulmonary changes are thought not to occur.

DIFFERENTIAL DIAGNOSIS. The diagnosis is not easy, but a history of exposure to manganese will of course help. The symptoms may simulate progressive bulbar paralysis, amyotrophic lateral sclerosis, post-encephalitic Parkinsonism, multiple sclerosis or progressive lenticular degeneration.

Prognosis

In a mild case some improvement may follow removal from further exposure, but in the established case the prognosis is unpromising as regards recovery. The degenerations produced by manganese may not, however, curtail life, for cases of gross poisoning have become long-lived cripples.

3. POISONING BY PHOSPHORUS OR PHOSPHINE OR POISONING DUE TO THE ANTI-CHOLINESTERASE ACTION OF ORGANIC PHOSPHORUS COMPOUNDS

Aetiology

The element phosphorus as extracted from the rock exists in two allotropic forms:

(i) RED PHOSPHORUS, which is amorphous and does not readily ignite; it is used for making 'safety' matches and as a starting point for other preparations, and is for all practical purposes, non-toxic.

(ii) YELLOW (OR WHITE) PHOSPHORUS, which is a waxy solid which ignites spontaneously on contact with air giving off fumes of elemental phosphorus and its oxides. This is the poisonous form the handling of which carries a risk of severe burns, and the fumes of which are usually responsible directly or indirectly for the few cases of phosphorus poisoning seen. It is used as a rat poison, in the elaboration of incendiary war material, and in the production

of non-ferrous alloys. It may also be met within chemical works in which it is converted into red phosphorus prior to further processing. Various compounds of phosphorus (v. infra) are also poisonous.

Diagnosis

ACUTE POISONING. This follows ingestion of yellow phosphorus. Absorption is slow and symptoms tend to be delayed. The main symptoms and signs are abdominal pain, vomiting, depression and general weakness, followed after an interval of days or weeks by toxic jaundice and possibly haemorrhages from the mucous membranes. The course of a fatal case is very similar to that of acute yellow atrophy of the liver.

CHRONIC POISONING. The classical lesion in chronic poisoning is 'phossy jaw', a condition in which, as a result of periostitis following ulceration of the gums, with dental sepsis or tooth extraction as predisposing causes, necrosis of bone occurs. The fulminating condition seen during the second half of the last century is now a thing of the past but a milder form of the disease is still encountered. Typically the necrosis is followed by secondary pyogenic infection, abscess formation and possible fistulae. The secondary infection may respond to treatment but the bone necrosis continues with the formation of sequestra weeks later.

Removal of those sequestra is followed by slow healing. Pain is variable but may be severe.

Pain and the failure of an extracted tooth socket to heal in a worker who has been exposed to yellow phosphorus during the last two years should suggest the possibility of an underlying phosphorus necrosis of bone.

PHOSPHINE is sometimes encountered in the production of acetylene from impure carbide and may therefore be met with as a constituent of crude acetylene gas. It is generated from phosphides used in self-igniting distress signals at sea, and may arise accidentally during the conversion of yellow phosphorus into red phosphorus and its derivatives. Its toxicity is of the same order as that of hydrocyanic acid, with a rapidly fatal issue in acute cases. In subacute and chronic cases there are haemorrhages in the lungs, pulmonary oedema, and fatty degeneration of the organs. The clinical picture resembles poisoning by arsine (q.v.) except that phosphine has no haemolytic action. An accurate diagnosis cannot be made without knowledge of the possiblility of exposure to phosphine.

POISONING BY ANTI-CHOLINESTERASE ACTION OF ORGANIC PHOSPHORUS COMPOUNDS. These organic phosphorus compounds are used extensively as insecticides in greenhouses, orchards and fields. Absorption occurs rapidly through the intact skin with only

minor local irritation. Absorption may also occur from inhalation and ingestion. The effect of these chemicals is to inhibit the action of the enzyme cholinesterase present in red blood cells and motor nerve end-plates. The action is cumulative and a toxic concentration may be built up by repeated slight exposures.

Early symptoms are non-specific and may include anorexia and nausea (characteristically increased by smoking or taking food), giddiness, drowsiness, diarrhoea and fatigue. Contricted pupils may be present at this stage. Within a few hours muscular twitching, cramps, incontinence of urine and faeces, coma, convulsions, paralyses and signs of pulmonary oedema may develop. The severity of the symptoms depends on the amount of the substance absorbed.

Prognosis

The prognosis depends on the type and severity of the poisoning. Recovery tends to be slow after most forms of phosphorus poisoning. Obvious jaundice probably means permanent liver damage. Poisoning by the organic phosphorus cholinesterase inhibitors has been reported as ending fatally in a short a time as one hour. The inhibition of cholinesterase rapidly becomes irreversible and recovery depends on its regeneration which may take several weeks. A delayed paralysis of the limbs may occur about three weeks after the onset of symptoms and following apparent recovery.

4. POISONING BY ARSENIC OR A COMPOUND OF ARSENIC

Aetiology

Arsenical poisoning occurs in industry in two distinct forms, the symptoms of which bear little or no resemblance to each other, or to those commonly associated with the use of arsenic for criminal purposes. The first form, an acute and serious type, arises from inhalation of arseniuretted hydrogen ('arsine') gas. The second, which is the more common and chronic type, results from absorption into the body, usually by inhalation over a long period, of dusts of arsenical compounds.

Arseniuretted hydrogen is generated in industry almost always accidentally; its production occurs when nascent hydrogen is released in the presence of arsenic. Such an accident is liable to occur wherever dilute sulphuric or hydrochloric acid is used in the processing of ores or alloys etc. containing, or contaminated with arsenic, but particularly in extracting mineral ores or residues, 'pickling' (acid-cleaning) metals, clearing acid-tanks of sludge, manufacturing electric accumulators, etc. Arseniurretted hydrogen may also be evolved in dangerous concentrations by the action of water on the arsenides of alkali metals (common contaminants of metallic drosses and residues) and in many other situations—including chemical laboratories.

The solid arsenical compounds are met with in smelting processes, in chemical works in which they are made, and in various industrial and other processes in which they are used. For example, arsenious oxide ('white arsenic') is used as a weed killer, as an insecticide, in the preservation of hides, skins and furs, and in glass-making; sodium arsenite is used in sheep dips; copper aceto-arsenite (emerald or Paris green), lead arsenate, and calcium arsenate are used as insecticides and fungicides in orchards. Cupric arsenite (Scheele's green), at one time used as a dye in wall-paper, etc. and capable of releasing dimethylarsine, a potent arsenical gas, is now seldom met with.

Diagnosis

POISONING BY ARSENIURETTED HYDROGEN. This gas is a subtle and powerful haemolytic agent causing haemoglobinuria, anaemia, and haemolytic icterus. Mild cases, after a latent period of a few hours to a day or more, show varying degrees of nausea, headache, shivering, exhaustion, giddiness, epigastric pain, and vomiting—all of sudden onset. In more severe cases the lag period may be reduced to six hours or less, after which haemoglobinuria, the first indication of a serious degree of haemolysis, appears. In a bad case the urine may rapidly assume a dark red colour so great is the destruction of blood cells. Within twenty-four hours or so these is a haemolytic jaundice, with pain and tenderness in the liver region, and by the third day anaemia, so marked that the red cell count may fall below 1,000,000 per c.mm, with a corresponding fall in haemoglobin. There is severe kidney damage, with albumin and casts in the urine, which may become scanty as the condition progresses. In a case which goes on to recovery progress is slow. The haematuria ceases, but albumin may persist in the urine for many days, and anaemia may last for many weeks. In more severe cases the patient drifts into a typhoid state, death from anuria being a common termination. The mortality rate in industrial cases which have come to light has been in the region of 30 per cent.

POISONING BY SOLID COMPOUNDS. Solid arsenical compounds are usually met with in industry in a state of fine subdivision, and these powders which are also light, are prone to 'fly' during drying, packing and handling generally. For this reason poisoning from absorption of solid compounds is largely the result of inhalation of dust. At the same time the dust alights on exposed skin and mucous membranes, and there acts as a local irritant, causing inflammation or ulceration. Thus, when such lesions are seen in an arsenic worker, it is sometimes difficult to say at once whether they are the result of local action only, or of systemic poisoning.*

LOCAL ACTION. Arsenical dust alighting on the skin, especially where there are creases or wrinkles or where the surface is moist as in the axillae, or around the genitalia, tends to set up dermatitis which may assume an

* *Note*: Where a claimant suffers from affections of the skin or mucous membranes of the upper respiratory tract thought to be due to the local action of arsenic, he should be treated, if the examiner is satisfied that there are no indications of systemic poisoning, as suffering from either No. 41 or 42 on the list of Prescribed Diseases and not from poisoning by arsenic.

acneform or eczematous aspect and which if untreated is prone to advance to ulceration. Such lesions are commonly seen on the face, where the edge of a respirator causes friction on a skin surface which is creased, warm and moist. They may be accompanied by evidence of simultaneous damage to mucous membranes, such as conjunctivitis, blepharitis, rhinitis, pharyngitis, and laryngitis. Hoarseness is sometimes a prominent symptom. Ulceration of the nasal septum, not infrequently going on to perforation is generally painless and does not result in deformity, for only the cartilaginous part of the septum is involved.* Arsenic may occasionally act as a carcinogen causing a squamous-celled carcinoma of the skin. This is dealt with under Prescribed Disease No. 23(c).

GENERAL ACTION. On absorption, by whatever route, arsenic tends to cause mainly gastro-intestinal symptoms (vomiting, abdominal cramp, and purging) but chronic cases generally show skin lesions as well, which may be due, at least in part, to the systemic poisoning; possible manifestations are dermatitis, ulcers, scleroderma, bronzing, trophic changes in the nails, loss of hair, etc. Arsenical bronzing in slight cases is best seen in the eyelids, temples, neck and nipples; in more marked cases there may be extensive bronzing of the trunk. Peripheral neuritis used to be cited as a complication, but it is probable that when this occurs there are additional factors such as alcoholism in operation.

DIFFERENTIAL DIAGNOSIS. In poisoning by arseniuretted hydrogen, mild cases are liable to be missed altogether, or diagnosed as influenza, or food-poisoning. In the more severe cases arsenic may be detected in the urine for several weeks after the accident. In cases of long-continued absorption of small quantities of arsenic in industry, the amounts found in the body fluids are so small that they usually offer little help in diagnosis, although on occasion amounts significantly greater than normal can be detected in the hair and nails. A single observation in a given individual, however, is not very informative.

5. POISONING BY MERCURY OR A COMPOUND OF MERCURY

Aetiology

Mercury and its compounds may enter the body in dangerous amounts via the alimentary tract, or though unbroken skin or mucous membranes, or may be inhaled in the form of vapour or dust. Contact with the skin or mucous membranes may or may not result in local inflammatory effects according to the material involved. Risk of poisoning arises commonly in the following occupations: mercury mining and recovery of metal from the ore; use of metallic mercury in the manufacture of thermometers, barometers, and electric meters; associated mercury distillation processes, manufacture of salts of mercury; the use of the latter as disinfectants, in antifouling paints, etc., or as carotting agents in making fur-felts and the like; the use of mercury amalgams in water-gilding; the manufacture and use of fulminate of mercury in

* See note on page 8.

explosives factories; and the manufacture and use of organic compounds of mercury as fungicides in seed-dressings, etc.

Diagnosis

In industrial poisoning resulting from exposure to metallic mercury or to the dust of inorganic mercurial compounds, the earliest symptoms may be the gradual development of a sallow colour with frequent headaches and vague dyspepsia. The characteristic features are gingivitis accompanied by sialorrhoea, muscular tremors, and some degree of mercurial 'erethism'. The gums become swollen and tender, bleed easily and may ulcerate; the teeth become loose and later may drop out. In more chronic cases most of the teeth will have been lost, while any remaining will usually be blackened and markedly eroded. Intention tremors, at first very fine and affecting the fingers, eye-lids, lips, tongue, etc. develop early; these may later spread elsewhere accompanied by or superceded by coarser movements which become exaggerated on attention being drawn to them. Alcoholism favours the development of tremors. Mercurial erethism is a peculiar form of abnormal timidity reminiscent of the extreme shyness of the nervous juvenile, and often accompanied by insomnia and an anxiety state accompanied by vasomotor disturbances. Later there may be emotional upsets, characterised notably by depression, apathy and drowsiness. Less frequently such cases show psychotic changes, or suffer from hallucinations and loss of memory, going on to marked dulling of the intellectual faculties.

A brownish coloured reflex from the anterior capsule of the lens of the eye may be seen on examination with the slit-lamp. This gives rise to no interference with vision and is not necessarily accompanied by other signs of mercurialism. It is of value only as an indication of prolonged absorption of mercury.

RENAL DAMAGE. There is evidence that the nephrotic syndrome may develop in workers exposed to mercury. It is accompanied by a high level of mercury in the urine and recovery usually takes place when the patient is removed from the hazard. Transient albuminuria may occur in some workers. Mercury is excreted in the urine, the normal daily amount being 5–10 microgrammes. This level is exceeded in those who are occupationally exposed. There is no relation between the level of mercury in the urine and the onset of signs and symptoms of poisoning, but workers who excrete more than 300 microgrammes of mercury in twenty-four hours may develop manifestations of chronic mercury poisoning. However even where mercury is excreted at a high level, poisoning should not be diagnosed in the absence of signs and symptoms.

ORGANIC MERCURY. In cases of poisoning described as following exposure to the dust of organic mercurial compounds used as seed-dressings, the nervous symptoms and signs have tended to be more pronounced, and psychotic symptoms less so. Such cases are described as showing tremors, ataxia, dysarthria, and marked constriction of the

visual fields, but having no stomatitis, sialorrhoea or erethism. It should be noted that local contact with these compounds may also cause a severe vesicular dermatitis.*

FULMINATE OF MERCURY. This is a skin irritant which, however, does not readily give rise to systemic mercurial poisoning. Occasionally fulminate workers show excessive salivation, but other symptoms of undue absorption are uncommon. Fulminate dermatitis ('fulminate itch') starting as an erythema of exposed parts, accompanied by intense itching, swelling and oedema, may assume later a papular, vesicular, or pustular form. Fulminate, if lodging in folds or cracks of the skin, may cause painful necrotic lesions ('powder holes') which later may become circular punched-out ulcers which may penetrate deeply. Fine fulminate dust also causes marked irritation of the mucosa of eyes, nose and throat.*

Prognosis

Mercurial dermatitis usually clears up in a week or two with suitable treatment; 'powder holes' may be more resistant. Systemic poisoning, resulting from the absorption of inorganic compounds, may cause many weeks of disablement; when due to the absorption of organic compounds there may be residual permanent disablement. There are no specific sequelae.

6. POISONING BY CARBON BISULPHIDE

Aetiology

Carbon bisulphide is a volatile solvent once much used in the vulcanising of rubber but more often met with now in the manufacture of artificial silk and cellophane. It is used to a small extent in the manufacture of fine chemicals, pharmaceutical products, and waterproof cement, and is occasionally sold as an insecticide. It is a powerful narcotic which acts also as a general nerve poison, affecting in some cases mainly the higher centres of the brain, and in others mainly the peripheral nervous system. Toxic effects in man are usually the result of exposure to fumes, but absorption through the skin is possible. Women and young persons, and those of a neuropathic diathesis appear to be more susceptible than others.

Diagnosis

In cases of acute poisoning, usually the result of inhaling fumes in high concentration, the early symptoms are similar to those of mild alcoholic intoxication, but soon the initial euphoria gives place to headache, giddiness, dyspnoea, vomiting, palpitations, and perhaps

* *Note*: Where a claimant suffers from affections of the skin or mucous membranes of the upper repiratory tract thought to be due to the local action of mercury, he should be treated, if the examiner is satisfied that there are no indications of systemic poisoning, as suffering from either No. 41 or 42 on the list of Prescribed Diseases and not from poisoning by mercury.

precordial and abdominal pain. On continued exposure, excitement passes into delirium and coma, with death from respiratory failure. In more chronic cases there are marked metabolic changes, with gastro-intestinal disturbances and recurring bouts of headache, giddiness, difficulty in concentrating, paraesthesias in the limbs, and general 'nervousness'. In some cases psychotic changes are prominent, usually in the form of extreme irritability, defects of memory, and broken sleep. In this type of case there may be aberrant sexual impulses and, later, lessened libido. These symptoms may later merge into confusion, delirium, hallucinations and a condition of manic-depressive insanity. In others a Parkinsonian syndrome predominates, with tremor of the face, tongue and hands, slow speech, and an unsteady gait; reflexes are exaggerated, there is muscular weakness and Rombergism, and Babinski's sign may be present. In old standing cases a polyneuritis, with some loss of muscle power in the limbs and paralysis of single muscles may occur. Ocular changes may include diminution of visual acuity, contraction of the visual field, abolition of the corneal reflex, abnormalities of the pupillary reflexes, and optic neuritis. Blood changes, other than some secondary anaemia, are not common.

Prognosis

Cases of polyneuritis may recover completely if removed from further exposure, but recent opinion inclines to the view that the prognosis is less favourable than was formerly believed to be the case; some cases improve only slightly, while a few continue to deteriorate. Cases of psychosis, except perhaps those showing evidence of serious degenerative changes such as Parkinsonism, usually recover rapidly after cessation of exposure.

7. POISONING BY BENZENE OR A HOMOLOGUE

Aetiology

Benzene, one of the products of the destructive distillation of coal-tar, is a colourless, freely volatile solvent with a distinctive odour and a narcotic action. It must be carefully distinguished from *benzine* which is a distillate of petroleum, and which is very much less toxic. Crude benzene, such as is commonly used in industry under the name of 'benzol' of 'benzole'. contains as impurities varying amounts of the homologues of benzene (toluene and xylene are the only ones important in industry) and may also contain carbon bisulphide, phenol, and other coal-tar derivatives. Similarly, the homologues themselves when in crude form (the 'toluol' and 'Xylol' of industry) may, like some 'naphthas', contain variable quantities of benzene, and such mixed solvents owe their toxicity almost wholly to their benzene content. Benzene is sometimes handled in industry on a large scale in closed systems in which it is used, recovered and re-used as part of a continuous process; such systems if well designed and maintained are usually quite safe. More often, benzol is used in open systems, as a solvent, e.g. in the

extraction of oils, alkaloids, etc., and in the manufacture of coloured inks, dyes, paints, cellulose lacquers, varnishes, paint removers, rubber, and artificial leather. Benzol is well known as a degreasing and cleaning agent, and its careless use for such purposes may involve heavy exposure to fumes, since benzene itself, (and to a lesser extent its homologues, toluene and xylene) volatilises freely at room temperatures. Benzene is absorbed mainly by inhalation of the vapour, but it can also be absorbed through the skin. It is outstanding among industrial solvents in that it may have a serious toxic action on the bone marrow.

Diagnosis

ACUTE BENZENE POISONING. In its initial stages acute benzene poisoning gives rise to a feeling of euphoria, or merely to headache, giddiness, nausea, and vomiting; in more serious cases excitement, with a sensation of constriction in the chest, convulsive movements, and paralysis may give way, as intoxication increases, to delirium, coma, and death from respiratory failure. Such cases are usually the result of accidental occurrences involving isolated exposures to high concentration of fumes. Toluene and xylene have a similar but lesser toxic effect.

CHRONIC BENZENE POISONING. This is the commoner form of the poisoning seen in industry. It is essentially an affection of the haemopoietic system, the point of attack being the bone marrow. In making a diagnosis it must be remembered that reliance cannot be placed on clinical symptoms alone, for these may be scanty, and, at least in the early stages, they are not commensurate with the degree of damage done to the blood system; chronic benzene poisoning may, in fact, be relatively far advanced before symptoms of obvious illness appear at all. The principal early symptoms and signs are lassitude and muscular weakness, pallor, mild digestive disturbances, giddiness, and in women amenorrhoea or menorrhagia. The later symptoms are those of a severe anaemia, with extreme pallor, muscular weakness, haemorrhages from mucous membranes, and purpuric haemorrhages in the skin. An acute diagnosis of chronic benzene poisoning can be made at an early stage only by a full blood examination. The blood picture is variable, but a fairly constant finding is diminution in the total number of white corpuscles; neutropenia in particular is often an early feature, but in some cases there is an initial leucocytosis. A diminution in the total number of red corpuscles without a corresponding decrease in the haemoglobin level, and hence a colour index greater than 1.0 are also sometimes found. The terminal phase is usually one of aplastic anaemia, with the bone marrow showing marked aplasia, but cases of death from leukaemia have also been recorded.

Toluene and xylene, homologues of benzene, are in their pure state much less toxic than benzene, but in their crude forms (toluol and xylol) they must be regarded as potentially toxic agents, with an action similar to, but less marked than, that of benzene.

Prognosis

Removal from exposure at an early stage of chronic benzene poisoning is usually followed by recovery, but there are good grounds for protecting persons so rescued from any further exposure to benzene or its homologues. In cases of chronic poisoning, with unequivocal signs of damage to the bone marrow, the prognosis is unfavourable.

8. POISONING BY A NITRO-, OR AMINO- OR CHLORO-DERIVATIVE OF BENZENE OR OF A HOMOLOGUE OF BENZENE, OR POISONING BY NITROCHLOR-BENZENE

Aetiology

Nitro- and amino-derivatives of benzene and of its homologues are most 'intermediates' from which more complicated—but usually less toxic—chemical substances (generally dyes or explosives) are elaborated. The principal nitro-derivatives are nitrobenzene, the dinitrobenzenes and trinitrobenzene, the nitrotoluenes, dinitrotoluenes, trinitrotoluenes and trinitroxylenes. Closely related to these but not true 'nitro-derivatives' are tetryl, the nitrochlorbenzenes and the dinitrochlor-benzenes. The important amino—derivatives are aniline and the toluidines, xylidines, phenylene diamines and toluylene-diamines. Closely related to these but not true 'amino-derivatives' are methyl and dimethyl aniline, ethyl and diethyl aniline, benzyl aniline, the nitro-anilines, dinitroanilines, chloranilines, nitrochloranilenes, and the diphenylamines.

The true nitro- and amino- derivatives of benzene and of its homologues are all in greater or lesser degree capable of being absorbed through the skin as well as through the alimentary and respiratory tracts. The rapidity of their absorption through lungs and skin, and therefore to some extent their immediate toxicity in industry, is dependent largely on their physical state and their volatility; for example aniline, a volatile liquid, is much more rapidly toxic than the less volatile solid trinitrotoluene. Acute cases of poisoning by these substances are usually the result of a single exposure to a high concentration of fumes, or of gross contamination of clothing, the result of an accident; chronic poisoning, more insidious in onset and likely to be progressive, results from long-continued slower absorption via the lungs or skin.

The chloro- derivatives of benzene and its homologues are not likely to give rise to trouble in industry with the exception of monochlor-benzene and the ortho- and para- isomers of dichlorbenzene. These substances are used in the dyeing and chemical industries, as constituents of lacquers, as solvents and cleansing agents, as fumigants, disinfectants, and as constituents of wood preservatives. They are seldom encountered in the pure state and may be mixed with more toxic substances such as benzene and carbon bisulphide.

Diagnosis

Although nitro- and amino-compounds vary considerably in toxicity, there are certain symptoms common to most cases of acute poisoning by these substances. In a light case of poisoning, the only symptoms may be those of anoxaemia, due to methaemoglobinaemia, viz, flushed face perhaps with slight cyanosis, throbbing headache, giddiness, muscular weakness and dyspnoea. In more serious cases there is marked cyanosis with a greyish-lilac colour, and nausea, vomiting, colic and maybe collapse. In the worst cases, which may be fatal, there are deep cyanosis and extreme prostration, cold clammy skin, low blood-pressure, thready pulse, and fluttering respiration. Symptoms due to acute poisoning may persist for many days and be followed by others indicating a variable degree of blood destruction and regeneration. Examination of the blood at this stage may show anaemia, polychromasia either diffuse or punctate, and the prescence of normoblasts.

In cases of more chronic poisoning, the result of slight absorption over a long period, a degree of secondary anaemia with perhaps slight cyanosis or a mild haemolytic jaundice may be the only signs detectable, but often there are other symptoms more specifically related to the particular compound concerned. Thus long-continued absorption of dinitrobenzene may result in cachexia with mental as well as physical degeneration; trinitrotoluene tends to cause a toxic gastritis and hepatitis which may go on to severe toxic jaundice, symptoms of which may appear after only a few weeks' exposure. More often jaundice is the result of several months' continued absorption, and occasionally it may first appear, following a latent period, some weeks after exposure to risk has ceased. Trinitrotoluene may also cause a typical aplastic anaemia, and this too may make a belated appearance after exposure to risk has ceased. Some aminocompounds have a irritant effect on the bladder mucosa,* while others, like some of the nitro-compounds, are skin irritants; paraphenylene diamine (diaminobenzene), for example, is a common cause of dermatitis in the fur trade. Toluylene-diamine on the other hand is noted more as a powerful haemolytic agent.

The nitrochlorbenzenes have a similar systemic toxic effect as the corresponding nitrobenzene. However, dinitrochlorbenzene, while resembling dinitrobenzene in its systemic effects, is much more irritating to the skin and in addition may cause sensitivity.†

Mono- and dichlorbenzene are narcotics and central nervous system poisons of considerable potency. Their effect is similar to *acute* benzene poisoning. Headache, dizziness, stupor and difficulty in micturition

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* *Note*: Primary neoplasms of the epithelial lining of the bladder are dealt with under Disease No. 39.

† *Note*: Where a claimant suffers from affections of the skin or mucous membranes of the upper respiratory tract thought to be due to the local action of a nitro-, amino- or chloro-derivative of benzene or of nitrochlorbenzene, he should be treated, if the examiner is satisfied that there are no indications of systemic poisoning, as suffering from either No. 41 or No. 42 on the list of Prescribed Diseases and not from No. 8.

have been reported as resulting from prolonged exposure to monochlorbenzene. Chronic effects of poisoning by dichlorbenzene need not be expected.

Prognosis

Complete if somewhat slow recovery is the rule in cases of acute poisoning by nitro- or amino- compounds, but in cases of chronic poisoning not only is convalescence prolonged but recovery may be incomplete. The mortality rate in cases of toxic jaundice is in the region of 30 per cent and it is probable that cases which recover are left with some permanent liver damage. Similarly when once aplastic anaemia is established it is unlikely that the bone marrow will ever recover completely.

9. POISONING BY DINITROPHENOL OR A HOMOLOGUE OR BY SUBSTITUTED DINITROPHENOLS OR BY THE SALTS OF SUCH SUBSTANCES

Aetiology

The dinitrophenol ('DNP') met with in industry is the 1:2:4 isomer, a yellowish-white crystalline material with a slight odour of phenol. Used in 1914–1918 as an explosive it is now used as a dye intermediate, and as a wood preservative. It sublimes readily and is freely soluble in warm water and various organic solvents. It has caused acute (and fatal) poisoning as a result of absorption by mouth, and of exposure to fumes, and it is probable that in some of these cases skin absorption also played a part.

The only homologue of dinitrophenol of medical importance is dinitro-ortho-cresol ('DNOC' or 'DNC'). This substance, also a yellow solid, and explosive, is extensively used for agricultural purposes as a combined insecticide, ovicide, and fungicide. It is put up as a dust, wash or solution in oil, and its manufacture, mixing, etc, and use (often in pressure sprays) have given rise to a number of cases of illness, some of them fatal.

An example of a substituted dinitrophenol is dinitro-butyl-phenol (dinoseb), also used as an agricultural insecticide.

Diagnosis

Slight poisoning by these substances causes only transient stomach upset, with pale furred tongue, anorexia, vomiting, headache, and vertigo. There may be mild jaundice and albuminuria. With more prolonged or intensive exposure the symptoms are more characteristic; there are profuse sweating especially at night, fever, thirst, rapid pulse, marked dyspnoea, a very high basal metabolic rate, and consequent rapid loss of weight. The patient is pale, with slight cyanosis (and often tremor) of the lips, and complains of a feeling of constriction in the chest.

With these substances the fraction not dealt with by the liver is excreted unaltered via the skin, and this may cause in cases of poisoning a canary-yellow staining of covered and moist areas, which provides a clue to the true nature of the illness. The very high metabolic rate engendered upsets the heat-regulating mechanism, and massive dosage may cause extreme hyperpyrexia with collapse and death from heat-stroke. There are no characteristic post-mortem changes.

DIFFERENTIAL DIAGNOSIS. Other conditions associated with an abnormally high basal metabolic rate can usually be distinguished by the presence of additional diagnostic signs; in this case there are no such signs, except perhaps staining of the skin, and the identification of amino- or possibly diamino-nitrophenol (or -nitrocresol) in the urine.

Prognosis

Except in the most severe cases of poisoning cessation of exposure to risk is usually sufficient to ensure an eventual uncomplicated recovery, but progress is slow.

Sequelae

Cases of cataract with delayed onset, and of agranulocytosis have been recorded as following long-continued absorption of dinitrophenol and dinitro-ortho-cresol.

10. POISONING BY TETRACHLORETHANE

Aetiology

Tetrachlorethane is a colourless liquid resembling chloroform, but much more toxic. It is used in industry as a solvent for cellulose acetate in the manufacture of cinema films, artificial silk, artificial pearls, adhesives, polishing waxes, safety glass, shoe cements, etc, and to a small extent in the rubber industry. It is non-inflammable, and has been used also as a fire-extinguisher.

This solvent normally enters the body via the respiratory tract, in the form of fumes, and habitual exposure to the fumes for periods as short as three months has resulted in fatal illness.

Diagnosis

On absorption, tetrachlorethane exercises a focal effect on both the nervous system and the liver, tending to cause acute yellow atrophy of the latter organ, and/or a toxic polyneuritis.

1. THE GASTRO-INTESTINAL OR HEPATIC SYNDROME. There may be early loss of appetite, fatigue, persistent headache, abdominal pain, and vomiting. After a few days to a few weeks, during which accurate diagnosis may be difficult, jaundice is liable to appear, at first only slight, with constipation and clay-coloured stools, but

later more pronounced, with vomiting, albuminuria, and enlargement of the liver, with local tenderness. Later still in a severe case there are somnolence, delirium, convulsions, and possibly epistaxis, haematemesis, and ascites before death supervenes.

2. THE NERVOUS SYNDROME. There are at first numbness and tingling of the fingers and toes, with tremor of the hands, and twitching of the facial muscles. Later there may be weakness or paralysis of the interossei muscles of the hands and feet, and possibly also of the flexors and extensors of the fingers.

Prognosis

The prognosis in both types of case is serious. In the hepatic form there may be extensive destruction of liver cells, with fatty changes in heart muscle and kidneys. In the polyneuritic form recovery is long-delayed.

11. POISONING BY TRI-CRESYL PHOSPHATE

Aetiology

Tri-cresyl phosphate (or 'lindol') is an odourless, oily liquid used as a plasticiser in the manufacture of plastics, waterproof materials, and flexible cable-coverings, etc. Only the ortho-isomer is believed to be toxic, but since this isomer is difficult to isolate from the others it is likely to be present in most preparations used in industry. The toxic action of tri-cresyl phosphate when ingested (e.g. through using it as a cooking fat) has been proved in several incidents which have involved many cases of illness and a number of deaths (described sometimes under the title 'jake paralysis'); it is also readily soluble in fats and oils, and absorption through the skin is known to occur; it seems probable that poisoning may also result from the inhalation of fumes, since the liquid volatilises freely at the temperatures to which it is subjected in industry. Tri-cresyl phosphate produces irreversible inhibition of pseudo-cholinesterase. It causes the cells of the anterior horn of the spinal cord and motor nuclei of the brain stem to undergo chromatolysis with degeneration of the pyramidal tracts and demyelination of peripheral nerves.

Diagnosis

Poisoning becomes apparent first as a transient stomach upset of varying severity which clears up more or less completely in a day or two. One to three weeks later, stiffness, tingling, and numbness of the toes, and probably of the calf muscles, draw attention to a muscular weakness which soon develops, in a moderately severe case, into a flaccid paralysis affecting both flexors and extensors. There is an early loss of ankle jerks, and the plantar reflex is absent; there may be some loss of cutaneous sense and joint sense. In mild cases, only the feet are affected, but in others the hands may become involved in a week or two, with tingling, numbness, and muscular weakness going on rapidly to flaccid paralysis

of the flexors and extensors of the wrists and fingers. Occasionally the thigh and trunk muscles may be affected. In some cases there may be evidence of an upper motor neurone lesion.

DIFFERENTIAL DIAGNOSIS. This toxic neuropathy, affecting as it does chiefly the motor nerves serving the limb muscles, clinically resembles acute anterior poliomyelitis, and cannot readily be distinguished from it unless the occupational history provides a clue. The occupational history will help to exclude also toxic agents such as lead which may be responsible for peripheral neuritis.

Prognosis

Recovery is slow. Foot-drop and a consequent high-stepping gait may continue for a year or more with little change, but there may be gradual improvement for several years. In a number of recorded cases, however, there has been some degree of permanent residual paralysis.

12. POISONING BY TRI-PHENYL PHOSPHATE

Cases of alleged poisoning by tri-phenyl phosphate (which is also used as a plasticiser) are not well documented, but the few descriptions available suggest that this substance possesses a toxicity similar to that of tri-cresyl phosphate (v. supra). The concurrence of recorded opinion, however, is that tri-phenyl phosphate is much less toxic than tri-cresyl phosphate.

13. POISONING BY DIETHYLENE DIOXIDE (DIOXAN)

Aetiology

This is one of the solvents. It is a colourless, moderately volatile liquid which at one time had a limited industrial use as a solvent for cellulose. It has also been used for degreasing wool, but its use is more or less restricted now to the manufature of polishing compounds and cosmetics. It may be used as a paint stripper, and as a preserving agent. Poisoning occurs as a result of absorption by inhalation, but the substance is capable of being absorbed through the skin.

Diagnosis

The fumes of diethylene dioxide in low concentration have a mildly irritant effect on the mucous membranes of the eyes, naso-pharynx, and throat, causing conjunctivitis with dimness of vision, symptoms of coryza, and cough; but with continued exposure these symptoms may subside and so fail to act as an adequate warning. When this occurs headache, vertigo, drowsiness, anorexia, nausea, and vomiting may follow with, later still, a toxic hepatitis accompanied by abdominal and lumbar pain. In cases of prolonged exposure to toxic concentrations a

haemorrhagic nephritis may supervene, going on to anuria and death from uraemia.

Prognosis

The prognosis depends on the degree of permanent damage to the liver and kidneys, and indirectly therefore on the severity of the exposure.

14. POISONING BY METHYL BROMIDE

Aetiology

Methyl bromide is a colourless, extremely volatile liquid, boiling at 4.5°C, above which temperature it is a gas, three times as heavy as air, with a faint, slightly sweet smell. It is used in this country mainly as a fire-extinguisher (sometimes mixed with carbon tetrachloride), and in this connection it is to be noted that where a fire has been incompletely extinguished by methyl bromide the decomposition-products of the latter, particularly bromine, may be liberated. It is used also in pest control (rodents, fleas, bugs etc.), and for fumigating warehouses, flour mills, railway food vans, greenhouses, and even soil. In the USA it is used also as a refrigerant. It is highly toxic, even in very low concentrations, but has a markedly delayed action, causing congestion and oedema of the lungs, injury to the vascular system resulting in haemorrhages, and degenerative changes in the liver and kidneys. Exposure may dull the sense of smell so that workers handling it may have no warning that they are in danger.

Diagnosis

In high concentrations the gas is markedly irritant to the upper respiratory tract, and thus the exposure is not usually prolonged. If it is, death is likely to result within 48 hours from pulmonary oedema. With even a brief exposure to a high concentration there may be headache, smarting of the eyes, cough, loss of appetite, abdominal discomfort, and numbness of the feet. These symptoms mostly pass off in a few days, but peripheral paraesthesia may persist for as long as three months, and delayed acute nephritis may appear. With longer exposure to lesser concentrations the initial symptoms are malaise, smarting of the eyes, nausea, etc., but after 2–16 hours interval, delayed symptoms begin to appear; there are then blurring of vision and occasionally amblyopia, symptoms such as slow and indistinct speech, giddiness, staggering gait, etc., (suggestive of alcoholism) and apathy, drowsiness, and tremor. There may be paresis of the lower extremities and epileptiform attacks, and delayed but severe uraemia has been recorded.

Even short exposures to low concentrations, although not disabling, tend to cause transient anorexia, nausea, headache, and pain in the quadriceps.

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exposed skin surfaces,* or a focal effect on the liver, on which the higher compounds at least appear to exercise a highly selective action which results in acute necrosis and consequent toxic jaundice. Several fatalities, each the result of acute yellow atrophy of the liver, have been recorded among persons exposed to the fumes of chlorinated naphthalene waxes. There is some evidence that simultaneous exposure to the fumes of carbon tetrachloride (which is a solvent of these waxes) increases the likelihood of liver damage.

Prognosis

It is dangerous for a worker who has shown evidence of even mild liver damage through exposure to chlorinated napthalene to return to an occupation involving further similar exposure.

16. POISONING BY NICKEL CARBONYL

Aetiology

This is a heavy, colourless, freely volatile liquid prepared and used only in the course of refining nickel. It is highly toxic when inhaled as a gas, its toxicity being at least five times that of carbon monoxide. It is probable that on being inhaled the vapour passes through the pulmonary tissue into the blood stream unchanged, and that the pulmonary epithelium is severely damaged by such passage. In industrial practice, nickel carbonyl gas, when encountered, is almost always mixed with a larger quantity of carbon monoxide.

Diagnosis

Symptoms of poisoning are both immediate and delayed. Between these there is frequently a quiescent period of twelve or more hours during which recovery from early symptoms may be so complete as to encourage a patient to return to work.

IMMEDIATE SYMPTOMS. Exposure to low concentrations of vapour may cause only persistent dull headache. At higher concentrations giddiness, nausea, and maybe vomiting are added; the gait becomes unsteady, and unconsciousness may supervene. At this stage the clinical picture is likely to resemble closely that of carbon monoxide poisoning.

DELAYED SYMPTOMS. Dyspnoea is the main feature. There is a short, irritating, unproductive cough, and the typical signs of widespread pulmonary congestion appear; the cough becomes worse, but produces only a little frothy sputum; cyanosis increases, and the pulse, already very rapid, may become thready, and the patient obviously very gravely ill. The chest symptoms tend to get worse for about six days, during which signs of cardiac failure may appear; they then slowly regress. Backache with pain in the loins is a common complaint.

* *Note*: Where a claimant suffers from chlor-acne thought to be due to exposure to chlorinated naphthalene, he should be treated, if the examiner is satisfied that there are no indications of systemic poisoning, as suffering from No. 42 on the list of Prescribed Diseases and not from poisoning by chlorinated naphthalene.

DIFFERENTIAL DIAGNOSIS. A clear history of the possiblity of exposure to the gas is essential for accurate diagnosis, and the presence in the blood during the first few days of illness of nickel and carboxy-haemoglobin will clinch matters. Traces of nickel may be detectable in the urine also during the first few days after exposure.

Prognosis

The immediate prognosis depends on the apparent degree of pulmonary damage. In non-fatal cases complete recovery in a reasonably short time is the rule. No specific sequelae have been recorded.

17. POISONING BY NITROUS FUMES

Aetiology

The term 'nitrous fumes' is confusing. The fumes so designated do not contain nitrous oxide, which is non-irritating, but are composed of a mixture of the two highly irritating forms of nitrogen dioxide. (The other common oxide of nitrogen, nitric oxide, being unstable is rapidly oxidised in air to the dioxide or peroxide.) These fumes which are of a brownish colour in high concentrations, are evolved when nitric acid is exposed to air, or when it reacts with organic material. They may be generated, for example, when nitric acid is spilt on a wooden floor, and when cotton is dissolved in nitric acid in the manufacture of gun-cotton. The same fumes may be given off during the combustion of material, such as celluloid and dynamite, containing a nitrous radicle, and so may be present in certain fires, and in mines and quarries after blasting. They may also be met with as a waste product in many chemical processes in which nitric acid is used, e.g., in the nitration of toluol during the manufacture of explosives. They may be formed also by the combination of atmospheric nitrogen and oxygen which occurs at high temperatures; an example is provided by the fumes from an oxy-acetylene torch, a carbon arc, or, to a less extent, the electric arc used in welding; dangerous concentrations may be built up in a short time when, for example, an oxy-acetylene torch is used, or welding is carried on, in a confined ill-ventilated space. Nitrogen dioxide may be given off silage due to reduction of nitrites following fermentation of forage, and exposed workers may get 'silo-fillers' disease', which is the name applied to poisoning occurring in this way.

Diagnosis

Nitrous fumes are the most insidious of all irritant gases. Inhalation may be followed by cough, but in many cases signs of upper respiratory tract irritation are slight and a latent period follows exposure during which the affected worker may be free of symptoms for some 2 to 20 hours and may carry on with his work and return home as if nothing were amiss. At the end of the latent interval symptoms may come on quite suddenly with tightness in the chest, breathlessness and cough with clear frothy sputum which may later become blood-stained. The

breathlessness may become marked and accompanied by cyanosis. There may be nausea, abdominal pain and vomiting and a rise in temperature. Physical and radiological examination of the chest may suggest the presence of pneumonia. The illness is due to an acute chemical bronchiolitis with pulmonary oedema which interferes with oxygen exchange. Death may occur from anoxia in a proportion of cases. In others, slow recovery sets in with gradual improvement of the symptoms and signs. Those who recover do so completely in most cases. In patients who have died, congestion and oedema of all the lobes of the lungs have been demonstrated. There is no satisfactory evidence that exposure to concentrations of nitrous fumes which are insufficient to cause acute pulmonary oedema, results in chronic ill-health.

18. POISONING BY GONIOMA KAMASSI (AFRICAN BOXWOOD)

Aetiology

Gonioma kamassi (known also in industry as 'kamassi boxwood' and 'Knysna boxwood', but different from 'Cape boxwood') is an African wood used to a small extent for shuttlemaking and for certain bushes and bearings of textile machinery. It is held to be injurious to workmen handling it on the ground that it yields an alkaloid resembling curare which acts, at least in susceptible individuals, as a general systemic poison with a focal action on the myocardium (causing marked bradycardia) and on the central nervous system. Absorption of this alkaloid is said to follow exposure to sawdust and the finer dust liberated during the machining of kamassi shuttle-blocks, etc.

Diagnosis

The most prominent symptom in susceptible persons exposed to kamassi dust is usually marked langour, with mental dullness, but complaint may also be made of recurring attacks of illness resembling hay-fever, with headache, coryza, conjunctivitis, and lachrymation, or of asthma, with retrosternal oppression and air-hunger.

19. ANTHRAX

Aetiology

Anthrax is a disease which may occur both in man and in certain animals as a result of infection with the Bacillus anthracis, which forms spores which can be killed by boiling for 10 minutes but survive for long periods in soil and in animal remains. Cattle are the main source of infection. Infection may occur in man through contact either with fresh infective material containing the bacillus (e.g., in agricultural workers, veterinary surgeons, butchers, knackers, etc.), or with dried animal products such as hides, skins, hair, wool, horns, hooves, bone-meal, etc., harbouring the spores (e.g., in dock-workers, tannery and wool workers, workers in bone-crushing and other fertiliser factories, etc.). Hides, skins

(especially dry-salted and sun-dried) and hair from the Far East, and from Siberia and parts of Africa are notoriously liable to be infected, as are wool and hair (mohair, goat, and camel hair) from the Middle East.

Infection in man may be external (cutaneous type of 'malignant pustule') or internal (pulmonary type or 'wool-sorter's disease') and, rarely, a gastro-intestinal infection.

Diagnosis

The disease manifests itself in almost every case as a grave toxaemia, with headache, shivering, muscle and joint pains, nausea, vomiting, and collapse, together with superadded local symptoms depending on the site and type of infection.

MALIGNANT PUSTULE. This is the commoner form. The term is misleading as the lesion does not contain pus. Infection takes place through cuts or minute scratches on the skin. After an incubation period of 1–4 days an irritant pimple develops. This pimple rapidly enlarges and breaks down with a black necrotic centre. The lesion may be ringed with small vesicles and inflammatory swelling; it is not markedly tender or hot but is puritic. Lymphangitis is the exception, rather than the rule, and the regional lymph glands may be slightly enlarged. Constitutional symptoms may be slight or absent until the skin involvement is severe or the infection has become disseminated. In 90 per cent of cases the pustule is situated on some uncovered part of the body, and in the case of the face and neck the intense oedema may itself be a danger to life. The diagnosis is rendered certain by discovery of the bacillus in the discharge or by its recovery after animal inoculation.

INTERNAL ANTHRAX. In these cases, even more than in external cases, the general intense toxaemia, with sudden vertigo, somnolence, dyspnoea, croup and marked prostration dominates the picture, and death may supervene before localising symptoms assist in arriving at a diagnosis. In a typical pulmonary case the appearance of wide-spread congestion and oedema or of an atypical pneumonia with frothy blood-stained sputum may focus attention on the lungs, but more often in such cases the disease is fulminating in type, and if untreated, death occurs from septicaemia in the first few days. Widening of the mediastinal shadow on the x-ray indicates haemorrhagic mediastinitis. Haemorrhagic meningitis may also occur. In gastro-intestinal cases—which are even more rare in man than pulmonary cases—severe gastro-enteritis of acute onset passes rapidly into a state of extreme prostration, followed by death in 2–4 days in the absence of appropriate treatment.

Prognosis

With modern treatment started sufficiently early the prognosis is now good in cases of malignant pustule, complete recovery being the rule.

20. GLANDERS

Aetiology

Glanders is a very rare contagious disease, caused by infection with Malleomyces mallei, which affects chiefly the equine species. Although transmissible directly from man to man, infection is acquired almost invariably as a result of direct contact with an infected animal. Horeshair is said to be a potential source of infection. The bacillus gains entry via skin wounds (including bites) or through the undamaged mucous membrane of the eye, nose, mouth or gastro-intestinal tract. There are two forms of the disease, acute and chronic.

Diagnosis

ACUTE FORM. After an incubation period of 2–3 days the acute phase, a bacteriaemia, is heralded by general malaise, headache, anorexia, nausea and articular pains, followed by the more specific symptoms of swelling and early ulceration at the site of infection, with marked lymphangitis and acutely painful local adenitis. The infection rapidly becomes generalised, the symptoms suggesting pyaemia, with enlargement of the liver and spleen and the appearance of pasty tumours which quickly suppurate and discharge, leaving painful ulcers; at this stage there is marked pyrexia, the temperature reaching a maximum usually between the sixth and twelfth days when there appears a chracteristic eruption affecting the face and mucosa of the eyes, nose and mouth. The individual lesions, appearing as red patches, rapidly become papular and then pustular, particularly in the nose where painful ulceration and destruction of cartilage and bone proceed apace, accompanied by a thick blood-stained, mucopurulent discharge. Death may supervene in 2–3 weeks or less.

CHRONIC FORM. (Farcy). This form tends to last two years or more; it comprises numerous and widespread nodular swellings which, if superficial, break down with the formation of indolent ulcers. There are similar nodular granulomata in the lungs, and it is doubtful if the infection is ever eliminated. Such cases may become acute at any time, but usually die of an intercurrent infection.

The occupation of the patient helps to suggest the diagnosis.

DIFFERENTIAL DIAGNOSIS. Other necrotic and pyaemic conditions such as syphilis and tuberculosis must be excluded.

Prognosis

The prognosis is unfavourable but as yet little is known of the response to modern antibiotics.

21 (a). INFECTION BY LEPTOSPIRA ICTEROHAEMORRHAGIAE

Aetiology

This disease (known also as leptospiral jaundice, spirochaetal jaundice, spirochaetosis icterohaemorrhagica and Weil's disease) is a febrile

infection caused by the Leptospira icterohaemorrhagiae, an organism commonly found in rats, which are the ordinary source of human infection. Such infection may be due to ingestion of food or water contaminated with the urine of infected rats; alternatively the infection may enter through the skin or through the mucous membranes of the eyes, nose or mouth. The disease sometimes occurs among men who work in places that may be rat-infested, as for example, sewer-men, miners, bargemen, wharfmen, fish and offal workers, and workers in slaughterhouses, piggeries, etc.

Diagnosis

After an incubation period of 6–12 days there is an abrupt onset of high fever, rigors, headache, muscular pains and vomiting. The face is flushed, there is intense injection of the conjunctivae, and prostration is marked. Irregular pyrexia continues for 3 or 4 days, then subsides by slow lysis over some 5-10 days, but the temperature tends to rise again towards the end of the second week, continuing thereafter irregularly mobile for about another week. Jaundice does not occur in all cases; it does so in probably 50 per cent to 60 per cent of cases, appearing on the 2nd or 3rd day of the illness. In jaundiced cases there is marked liver tenderness, and the spleen may be enlarged. At this stage herpes is common, and papular or, less often petechial rashes may appear. The urine is scanty, high-coloured, often bile-stained, and may show albumin. There is a tendency to sleeplessness, with nocturnal delirium (which may be accompanied by meningeal symptoms) and haemorrhages, usually starting as epistaxis, are common. By the time this stage is reached there is generally a well-marked polymorph leucocytosis, and the van den Bergh test, at least when jaundice is present, is directly positive. Direct infection of the meninges is said to occur in about 10 per cent of cases.

LABORATORY FINDINGS. The diagnosis is usually made in the laboratory, the leptospira being recovered from the blood during the first week, or from the urine for up to about six weeks from the 10th day. Serological tests show the presence of agglutinins in dilutions upwards of 1 in 100 or thereabouts after the 8th day, and the titre may rise as high as 1 in 30,000. A rising titre is, of course, diagnostic.

Prognosis

Mortality varies with the age of the patient and the virulence of the organism is greatest in those over 50 years of age. Most cases proceed to complete recovery. There are no special sequelae.

21 (b). INFECTION BY LEPTOSPIRA CANICOLA

Aetiology

This disease known also as canicola fever, is a similar condition to that caused by Leptospira icterohaemorrhagiae. The main vector animal is the dog although other domestic animals may on occasion harbour the

infection. The disease in the animal varies from an acute rapidly fatal illness to a symptomless carrier state similar to that of Leptospira icterohaemorrhagiae in the rat. Efforts to estimate the infection rate in dogs by serological surveys suggest that between 20 and 40 per cent of the canine population has been infected at some time in the past. The true incidence of human canicola fever is unknown. About 20 cases a year are diagnosed in this country but it is likely that a large number of cases escape diagnosis.

The mode of infection is uncertain but is probably similar to that of Leptospira icterohaemorrhagiae.

Diagnosis

The illness resembles a milder form of Weil's Disease and, as with other leptospiral infections there are no prodromata. The onset is sudden with shivering or rigors and intense and intractable headache. The temperature mounts rapidly to 103°–104°F. Signs of meningeal irritation are common and may include stiffness of the neck, a positive Kernig's sign and mental confusion. Bradycardia and lowered blood pressure are usually present. Weakness of muscles together with the meningeal involvement may give rise to a suspicion of poliomyelitis while in other instances congestion of the lungs may be a prominent symptom.

Fleeting rashes may occur but jaundice is much less common than in Weil's disease and when present seldom amounts to more than a sub-icteric tinge.

Albuminuria is almost always present but the degree of kidney involvement varies widely. An acute nephritis may develop but it is rare.

Injection of the corneal vessels occurs in about half the cases in the early stages and iritis, iridocyclitis, nystagmus, optic neuritis and the presence of opacities in the vitreous are common manifestations that may appear at any time from the acute phase to the end of convalescence.

LABORATORY FINDINGS. Leptospires may be isolated from the blood during the first weeks or from the urine after about the tenth day of the illness. The diagnosis is usually made, however, by the presence of specific antibodies in the serum. Agglutinins appear at about the end of the first week and a rapidly rising titre of agglutination is pathognomic of a recent infection.

Prognosis

The disease is self-limiting and, in the absence of pre-existing severe kidney damage, complete recovery is the rule. There are no sequelae.

22. ANKYLOSTOMIASIS

Aetiology

Ankylostomiasis or Hookworm Disease is the term applied to the presence of the nematode ankylostoma in the human intestine. The

eggs develop only after being excreted by the host, and on reaching warm moist soil. There is no intermediate host. The mature larvae make their way through the contaminated skin of a new host to the bloodstream, and emerging from the pulmonary capillaries into the alveoli, pass from there via the trachea, oesophagus and stomach to the small intestine. There the worms attach themselves securely, and about six weeks later sexual forms are mature and eggs begin to appear in the stools. In this country infection is very unlikely to occur except in a hot deep mine since the ovum requires a temperature upwards of 75°F. for its development. Once hatched, however, the larval form may survive in suitable surroundings for some months while awaiting a new host.

Diagnosis

The disease is inclined to be somewhat protean in its manifestations. However, the leading symptoms are generally those associated with a secondary anaemia. The severity depends on the weight of the original infection and on whether there has been reinfection for, since the parasite cannot complete its life-cycle in the intestine, a single infection eventually dies out. During the first week or two following infection the symptoms are variable and help little in diagnosis. The main symptoms and signs in the established case are usually anaemia, joint pains, mental and physical lethargy, dyspepsia, and oedema, with more of less interference with nutrition, and the appearance of skin rashes of vesiculo-eczematous type. The erythrocyte count may fall below 3,000,000 per c.mm. with a corresponding fall in haemoglobin. The white cell figure is variable but usually, at least in recent infections, there is an eosinophilia of 10–20 per cent. The diagnosis can be established with certainty only by discovery of the ova in the faeces.

Prognosis

In the absence of reinfection a spontaneous cure eventually takes place, although a single heavy infection may result in marked anaemia and a prolonged lowering of general vitality.

23(a). DYSTROPHY OF THE CORNEA (INCLUDING ULCERATION OF THE CORNEAL SURFACE) OF THE EYE
(b). LOCALISED NEW GROWTH OF THE SKIN, PAPILLOMATOUS OR KERATOTIC
(c). SQUAMOUS-CELLED CARCINOMA OF THE SKIN

due in any case to arsenic, tar, pitch, bitumen, mineral oil (including paraffin), soot or any compound, product (including quinone or hydroquinone), or residue of any of these substances.

Aetiology

The substances referred to above are liable to be or contain irritants which, on continued contact, exert a damaging effect on mucous

membrane and skin. When such damage becomes evident after only a brief exposure to risk, it is usually of a simple inflammatory nature only, but it may be refractory to treatment; for example in the patent-fuel industry where anthracite dust and coal-tar pitch are handled, inflammation and ulceration of the corneal conjunctiva, slow to heal, may result in permanent scarring and interference with vision. Similarly, workers in a pitch-bed are prone to suffer from acute dermatitis* of exposed skin surfaces, and may at the same time suffer from corneal complications.

Quinone and hydroquinone, used in photography and as a stabilizer and reducing agent in industry, may give rise to permanent staining of the area of the cornea exposed to light. This staining may become so dense as to impede vision. More prolonged exposure may lead to a deformation of the structure of the cornea with keratoconus and progressive astigmatism. At this stage the cornea may break down into ulceration. Quinone and hydroquinone, although skin irritants, do not give rise to neoplasms however.

Long exposure to any of the other agents mentioned may result in more chronic skin lesions, such as localised new growths of a papillomatous or kerototic nature. Such new growths, seen typically in the bituminous shale worker, and at first apparently entirely benign in nature, may, if untreated, adopt at any time the active and unregulated mode of growth which marks frank malignancy. In those engaged in extracting oil from the shale such growths commonly appear on the arms, but in users of this and other oils they may appear elsewhere, on parts more freely or more continuously contaminated, or on parts which are more susceptible or subjected to added irritation. Thus the cotton mule-spinner may develop oil cancer (squamous-celled carcinoma) on any part of the skin surface, but the classical site in his case is on the scrotum. Similarly persons habitually exposed to tar, or pitch, especially in the form of fumes and dust, may suffer from skin growths of a warty nature which, if untreated, tend later to ulcerate and assume a malignant character. The retort-man exposed to tarry fumes in a gas-works or coke-oven plant, the man in a tar-distillery, and the man using tar for net-fixing, or for making brushes or brattice cloth, or in boat-building, are all exposed to the same risk; so too are the various pitch-workers—pitch getters, pitch loaders, road-makers, labourers in patent-fuel works, optical lens makers, etc. Any of these may show after some years of exposure to risk warty growths which occur most often on the eyelids, cheeks, chin, behind the ears, on the neck, arms, scrotum, or thighs.

Where such warts appear and show degenerative changes pointing to malignancy, it is probable that the neoplastic change is regional rather than focal, for pitch warts are not uncommonly multiple, and they may recur several times in the same area of skin. But not all 'tar warts' become malignant; warts appearing within the first

* *Note*: Where a claimant suffers from dermatitis thought to be due to the local action of arsenic, tar, pitch, bitumen, etc., but does not show evidence of localised new growth or of squamous-celled cancer of the skin, he should be treated as suffering from No. 42 on the list of Prescribed Diseases.

few months of exposure to risk commonly disappear, especially if the exposure ceases, while those which appear many years later, may or may not show malignant tendencies. All of them are true neoplasms, but not all of them persist, and yet where one had already disappeared spontaneously another may appear and show almost from the beginning active and unregulated cell proliferation. Tar or pitch may thus initiate in an area of skin a neoplastic change while a man is at work, but malignant warts or progressive squamous-celled carcinoma may not appear until long after his exposure to risk has ceased. This latent period may be of some years' duration. The same time-lag occurs in the case of the man whose exposure continues over many years; the primary damage may occur in the early part of that time, but few cases of malignant ulceration of occupational origin become clinically evident under 30 years of age: commonly they occur between the ages of 55 and 65.

Neoplasms of the skin due to occupational exposure to arsenic or its compounds are not common. They may occasionally occur in workers in such occupations as the manufacture of arsenical sheep dips and insecticides. The neoplasms are nearly always multiple and a squamous-celled carcinoma of the skin may be accompanied by hyperkeratosis and multiple warts elsewhere on the body.

Diagnosis

While an accurate diagnosis of localised new growths of the skin and of squamous-celled carcinoma of the skin can sometimes be made on clinical grounds alone, it is desirable that all such cases should be referred to a pathologist.

Industrial skin cancers do not readily give rise to secondary growths, but where, in any part of the skin, a neoplastic change has once been initiated and become apparent clinically, any subsequent form of irritation may again precipitate proliferative changes of a cancerous nature.

24. [*Diseases formerly bearing this number are now covered by Diseases Nos. 41 and 42 below.*]

25. INFLAMMATION, ULCERATION OR MALIGNANT DISEASE OF THE SKIN OR SUBCUTANEOUS TISSUES OR OF THE BONES, OR BLOOD DYSCRASIA, OR CATARACT, DUE TO ELECTRO-MAGNETIC RADIATIONS (OTHER THAN RADIANT HEAT), OR TO IONISING PARTICLES

Aetiology

Electro-magnetic radiations are those radiations by which energy is transmitted without the necessity of a material medium. The spectrum of electro-magnetic radiation extends from the short X or gamma rays to the long radio waves and includes X-rays, ultra-violet rays, visible

light, infra-red rays* and short radio waves. Ionising particles are the corpuscular products of atomic disintegration, and include alpha and beta particles and neutrons.

X-rays are being increasingly used in industry for radiographic, fluoroscopic and crystalographic examinations of castings, etc. Radium and radio-active isotopes are used in 'bomb' form for the radio-graphic examination of welds at inaccessible sites. Radium, thorium, and other radio-active substances are used as constituents of self-luminous paints. Radio-active isotopes may also be used for eliminating static electricity and are being increasingly used in biochemical research. Future developments in the utilisation of nuclear energy are likely to increase the potential hazards due to electro-magnetic and ionising radiations.

Diagnosis of skin injuries

(i) ALOPECIA. Both X-rays and radium in small doses may have a depilatory action, the hair falling out about three weeks after exposure. With higher dosage there may be destruction of the hair follicles and any of the following conditions may supervene.

(ii) ACUTE INFLAMMATION. Ultra-violet rays can cause mild erythema (after a latent period of some hours) and superficial skin burns which heal readily. Burns tend to be followed by pigmentation (which may be permanent) and to result in local chronic inflammatory changes.

Both X-rays and radium may produce acute reaction in the form of erythema, or an actual burn similar to that caused by heat rays. Such a lesion may disappear in the course of a few weeks, or especially if repeated, may go on to (iii).

(iii) NECROSIS AND ULCERATION. Ulceration following X-ray or radium burns may be slow-healing and refractory to treatment. It is seldom the result of a single burn.

(iv) CHRONIC DERMATITIS. Repeated minor exposures to X-rays or less commonly, to radium, may cause a chronic dermatitis, the result of the inter-play of atrophic and regenerative forces. Clinically, there are thickening of the epidermis, hyperkeratosis, fissuring, and degenerative changes in the deeper tissues—a pre-cancerous condition. In the case of radium, especially following exposure to the Beta-rays emitted by luminous compounds, the earliest lesion commonly takes the form of a shiny redness of the skin immediately adjoining the nail-folds.

(v) MALIGNANT DISEASE. This may supervene in an area of chronic dermatitis, or may start de novo in the form of epitheliomatous warts occurring at first in an area previously damaged and later on various parts of the skin surface.

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* *Note*: It will be seen that pathological changes due to radiant heat are excluded from Disease No. 25. A claim in respect of caratact or dermatitis due to radiant heat should be dealt with under No. 26 or No. 42 respectively.

Prognosis

Lesions resulting from excessive exposure to ultra-violet rays usually heal well. Inflammation resulting from a single exposure to X-rays usually disappears without special treatment. Alopecia similarly caused may be permanent. Later skin changes from X-ray exposure, especially if appearing for the first time long after exposure has ceased, may become malignant.

Diagnosis of bone lesions

These are due, in the case of poisoning by radium, to accumulation in the bones of radium which has been either ingested or inhaled. The lesions may consist of either necrosis or malignant disease (usually sarcomatous in nature) or, less often, of rarefying osteitis with spontaneous fracture.

Diagnosis of blood dyscrasias

Exposure to X-rays or to the gamma rays emitted by radium may cause anaemia of the aplastic type. In the case of over-exposure to X-rays the earliest blood-change is usually a diminution in the number of polymorph white cells, sometimes accompanied by a corresponding decrease in the number of lymphocytes and sometimes by a relative lymphocytosis. With radium, especially when ingested or inhaled, the first signs may be those of over-stimulation of all formed elements of the blood, and in particular, an increase in lymphocytes, accompanied by the presence of abnormal or immature cells in the stained film. There is evidence that exposure to ionizing radiation may produce an increased incidence of leukaemia of most varieties except chronic lymphatic.

Prognosis

Once aplastic anaemia is fully developed the prognosis is serious, the bone marrow having permanently lost its reparative capacity, but the condition may run a protracted course after the first over-exposure.

Diagnosis of Cataract

Cataracts may be produced by exposure to X-rays, gamma rays and neutrons. As in heat cataracts (see No. 26 below) the opacities are of the posterior polar type but tend to be sharply delimited and have a multifoliate shape. The latent period in the development of cataracts following radiation depends on the magnitude of the dose. It may be as short as six months or as long as twelve years.

26. HEAT CATARACT

Lenticular opacities occurring in glass-workers, iron-workers, and others exposed to excessive heat have as a common factor exposure to electro-magnetic radiations in the infra-red range, and it is presumed that such radiations disturb the nutrition of the lens and cause localised

coagulation. Such cataracts are typically of posterior polar type. The age incidence varies: although much more frequent after the fourth decade, disabling degrees of cataract are not uncommon before the age of 40. The disease usually progresses slowly, and a claimant may suffer a loss of faculty long before the cataract is ripe for operation. A measure of permanent disablement is inevitable.

A claimant for benefit in respect of this disease will be referred to an ophthalmologist for examination.

27. DECOMPRESSION SICKNESS

Aetiology

Work in compressed air has to be done in works of civil engineering involving excavations in water-bearing strata or under water, as well as in occupations involving diving. Such work is effected either by single divers in diving suits, or by a group of workers in a caisson, or diving bell. A caisson consists essentially of a working chamber and a shaft communicating at the surface with an air-lock which in turn communicates with the outer air. All these receive cooled compressed air from a pipe-line. Within the caisson proper, in which the men work, and in the shaft, the air pressure must be equal to the pressure exercised by the water outside, and must therefore be raised in proportion to the depth at which work is being carried out; i.e. raised approximately one atmosphere for each ten metres of depth. In practice, the pressures used do not usually exceed 3½ atmospheres (i.e., about 35–40 lbs per sq. inch gauge pressure).

While men are being subjected to increased air-pressure in the air-lock, preparatory to descending into the caisson, slight physiological adjustments have to be made, and at this stage there may be some minor discomforts such as epistaxis, and some transient disturbance of the sense of hearing, touch, taste, or smell but it is not until later, during decompression and return to lower pressures, that decompression sickness (" caisson disease " or commonly, "the bends ") is liable to occur. A similar condition may occur as a result of very rapid ascent to high altitudes in modern high performance aircraft.

The symptoms of this ailment are believed to be due to the release within the blood and tissues, when the air pressure is reduced, of gases driven into solution when the air pressure was higher. The gases concerned are oxygen, carbon-dioxide, and nitrogen: the two former are removed very rapidly, the first by reabsorption and the second by exhalation from the lungs, but nitrogen is relatively insoluble in the body fluids and tends to collect as minute bubbles of gas which coalesce to form emboli. Nitrogen is, however, five or six times as soluble in fats and lipoids as in the body fluids, and tissues such as the nervous system and bone marrow hold proportionately more of the gas than others, releasing it in bulk when the pressure is dropped.

Diagnosis

Symptoms usually appear within the first few hours following decompression, but may not develop for twelve hours or longer. They depend in general on the location of any emboli formed, and may therefore simulate many other diseases. The most common symptom is pain in the limbs which, when mild, is known as " the niggles " and, when severe, " the bends ". Generalised itching, vertigo, nausea, vomiting, epigastric pain and dyspnoea (" the chokes ") due to emboli elsewhere, may also occur at this stage. In some cases there is involvement of the central nervous system, and symptoms such as paralysis of skeletal muscles, or of the bladder, etc. may appear. Emboli in the blood vessels of the lungs, brain or heart may be fatal. Destruction of bony tissues, an " aseptic necrosis ", has been described by several writers, occurring especially in the femur and upper arm, either in the diaphyses or epiphyses. As elsewhere, such lesions are essentially the result of infarction. Bone and joint lesions by their nature may not be noticed for some time after the incident, and there may by then be some attempts at repair, but " cystic " areas of dead bone (sometimes involving damage to articular surfaces) can often be demonstrated by X-rays long afterwards. Such lesions may result from a single over-rapid decompression. Lung cysts have been described.

Sequelae

Various sequelae, depending on the site and severity of infarction, may be met with, and may cause disablement.

28. CRAMP OF THE HAND OR FOREARM DUE TO REPETITIVE MOVEMENTS

Aetiology

This disability, known also as writer's cramp, telegraphist's cramp, twister's cramp, occupational cramp or craft palsy, is characterised by attacks of spasm, tremor and pain in the hand or forearm brought about by attempts to perform a familiar act involving frequently repeated muscular action. Muscular co-ordination necessary for the performance of fine repetitive movements breaks down and the continuation of the movements becomes impossible.

The causative factors in this condition are unknown. No structural change in the central nervous system, peripheral nerves or muscles has been demonstrated and electrical reactions are not disturbed. The cause is probably a combination of physical fatigue of muscles and nerves and an underlying psychoneurosis.

Diagnosis

The patient complains of painful spasms of the hand or forearm coming on while doing a series of familiar repetitive movements. The condition tends to develop in skilled operators and is rare in learners.

Attempts to use the other hand for the task frequently result in a recurrence of symptoms.

The condition must be distinguished from other painful and paralytic conditions of the hand and forearm such as peripheral neuritis, rheumatism, disseminated sclerosis and motor neurone disease.

Prognosis

Doubtful; the condition may last off and on for several years.

29. 30. [*Diseases formerly bearing these numbers are now included in Disease No. 28.*]

31. SUBCUTANEOUS CELLULITIS OF THE HAND (BEAT HAND)

Aetiology

This disability, as seen in industry, is the result primarily of the bruising of a devitalised cutis vera and its underlying tissues, and the implantation there, by repeated friction or pressure, of mineral particles. The condition is liable to follow frequent jarring of the hand in the use of pick and shovel, and is more likely to occur in wet working conditions, and to affect 'soft' hands as, for example, those of a man unaccustomed to manual labour or returning to it after a prolonged absence. It is usually when it is complicated by a local infection that it may become acutely disabling.

Diagnosis

The symptoms and signs are at first those of an acute inflammation affecting most commonly the palm of the hand and the palmar aspects of the thumb and fingers. There are redness, heat, and throbbing pain with a sense of fullness in the hand. At first the skin is unbroken but pits on pressure. The inflammation usually proceeds to suppuration in which nearby bursae or tendon-sheaths may become involved, and the infection if virulent may spread elsewhere.

Prognosis

In an uncomplicated case incapacity may last a few weeks. Skilled and timely surgical intervention can do much to prevent serious sequelae.

32. BURSITIS OR SUBCUTANEOUS CELLULITIS ARISING AT OR ABOUT THE KNEE DUE TO SEVERE OR PROLONGED EXTERNAL FRICTION OR PRESSURE AT OR ABOUT THE KNEE (BEAT KNEE)

Aetiology

This condition is similar in aetiology to 'Beat hand'. It is more likely to occur in those unaccustomed to working on their knees, or returning

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may be intermittent, may not be evident at examination, and may disappear altogether before the more debilitating symptoms yield to treatment.

Prognosis

Suitably treated cases of nystagmus may be restored eventually to full working capacity underground, but progress towards this end is usually slow. Recovered cases returning to work underground should not be habitually subjected again to low levels of illumination.

36. POISONING BY BERYLLIUM OR A COMPOUND OF BERYLLIUM

Aetiology

Beryllium (or glucinum) is a very light, hard, silvery-white metal formerly used in industry as a constituent of phosphorescent powders used in fluorescent lighting and neon signs, but it is used also in the preparation of metallic alloys, in the generation of atomic energy, in the manufacture of radio-valves, and as a refractory in the manufacture of crucibles and electrical porcelain. Beryllium metal, beryllium oxide, and some beryllium salts can act as irritants in contact with the skin and mucous membranes, the more soluble salts (chiefly fluorides, oxy-fluorides and sulphates) tending to cause acute inflammatory and exudative reactions while the metal itself and its less soluble salts (such as biscilicate combinations with zinc and magnesium) tend to cause chronic low-grade proliferative lesions of a granulomatous nature. The latter conditions may not become apparent until a matter of several weeks, months or even years have elapsed after exposure to risk has ceased.

Individual susceptibility to poisoning by beryllium varies within wide limits, and is probably due to an acquired sensitivity to the metal.

In industry absorption is usually the result of exposure to dust or fumes; coarse particles which are swallowed after being arrested in the nose and mouth pass through the alimentary tract with little or no absorption; on the other hand, very fine particles which are inhaled deeply into the lungs form deposits there from which beryllium may be slowly absorbed and widely distributed throughout the body.

Diagnosis

1. ACUTE INFLAMMATORY CONDITIONS:

   (*a*) DERMATITIS. Continued exposure of the skin to dust or fumes of beryllium or its salts, especially when accompanied by perspiration and friction, is liable to cause an oedematous, papulo-vesicular 'contact' dermatitis, usually localised to the part exposed and generally clearing up rapidly on cessation of exposure to risk.*

* *Note*: Where a claimant suffers from an affection of the skin or mucous membrane of the upper respiratory tract thought to be due to the local action of beryllium he should be treated, if the examiner is satisfied that there are no indications of systemic poisoning, as suffering from either No. 41 or No. 42 on the list of Prescribed Diseases and not from poisoning by beryllium.

(*b*) ACUTE INFLAMMATION OF MUCOUS MEMBRANES. Conjunctivitis, rhinitis, bronchitis and pneumonitis have been found to follow even quite short exposures (2–3 days) to beryllium-bearing dust or fumes arising from the metal, oxide, sulphate, chloride and fluoride. Cases of nasopharyngitis and tracheo-bronchitis so caused are characterised by mild pyrexia, a troublesome, unproductive cough, and possibly some dyspnoea. They are not distinguishable clinically, in the absence of an occuptional history of exposure, from analogous conditions caused by other irritants. Pneumonitis, which is more disabling, starts insidiously with only slight pyrexia, cough, substernal pain, some dyspnoea and scanty, but possibly blood-stained sputum. The pulse tends to be dis-proportionately rapid, râles are soon evident all over both lung fields, and the vital capacity is much reduced. Rapidly increasing fatigue is a prominent feature but marked apprehension may testify to a continuance of mental alertness. X-ray examination of the chest, in the early stages at least, affords little clue to the changes present, but blood examinations showing normal white counts may help to exclude infective conditions. In a persistent case, and then only several weeks after the onset, X-ray examination may reveal in succession a diffuse haziness throughout both lung fields, irregular areas of infiltration and, if the case continues long enough, discrete nodulation.

2. SUBACUTE AND CHRONIC INFLAMMATION

(*a*) GRANULOMATOSIS. Deep implantation of some beryllium salts in wounds (e.g. in wounds resulting from the breakage of fluorescent electrical fittings) may be followed, after an interval, by the development of a local benign granulomatous nodule. Similar lesions may be found post-mortem in the liver, glands and other organs in cases of long-standing absorption from the lungs.

(*b*) DELAYED PNEUMONITIS. Inhalation of the less soluble beryllium salts (notably mixtures of zinc, manganese and beryllium silicate) in a state of fine subdivision has resulted in many cases of delayed pneumonitis, with histories of exposure ranging from a few months to a few years. In this type of case the delayed onset is a striking feature, the period between cessation of exposure to risk and appearance of symptoms varying from a few months to as much as five years or more. In these cases, too, the onset is as insidious as it is delayed. Patients tend to seek medical advice in the first instance because of symptoms other than pulmonary, such as loss of weight, and fatigue. The loss of weight is progressive and may later be dramatic, but the patient's attention is meantime soon diverted to and concentrated upon a gradually increasing dyspnoea. Cough is not a prominent feature at first but may become troublesome later. Sputum, scanty and viscid, shows nothing of note on microscopic examination, and a normal white blood count confirms the absence of specific infection. Progressive dyspnoea is a distressing symptom

in a bad case, and ultimately may be painfully apparent at rest as well as on exertion. Physical signs in the chest are variable and not usually pronounced; there may be sundry adventitious sounds or merely indications of basal emphysema. The radiographic appearances vary according to the stages of the disease. In early cases only a slight generalised haziness may be seen, but later there tend to be added to this recticular shadows which may either coalesce irregullarly in the upper zones or slowly assume the picture of widespread discrete nodulation. In such cases beryllium is likely to be widely distributed in other tissues also, and, since excretion is slow, low concentrations may be detectable in the urine for very long periods.
DIFFERENTIAL DIAGNOSIS. The diagnosis of these conditions depends on a consideration of the whole picture. The occupational history, symptoms and clinical signs including radiographic appearances and the presence of beryllium in urine and tissue are important clues to the diagnosis. Chronic miliary tuberculosis, carcinomatosis, mitral stenosis, mycotic infections and the pneumoconioses should be excluded. Sarcoidosis in a beryllium worker is probably indistinguishable from chronic berylliosis.

Prognosis

Beryllium dermatitis readily clears up and may leave no residual increased sensitivity. Acute inflammations of the upper respiratory tract are usually of short duration, but an acute pneumonitis may remain active for several months. Such pneumonitis tends to progress, without exacerbations or remissions, either to complete recovery or death, the mortality rate being in the region of 10 per cent. In cases of delayed pneumonitis the outlook is less good. About a third of these cases are fatal while a further third are permanently disabled.

37(*a*). CARCINOMA OF THE MUCOUS MEMBRANE OF THE NOSE OR ASSOCIATED AIR SINUSES (*b*). PRIMARY CARCINOMA OF A BRONCHUS OR OF A LUNG

Aetiology

These diseases are held to be sometimes occupational in origin only because it has been shown conclusively that their incidence is significantly higher than average in persons who have been engaged in processing certain nickel-rich raw materials in a particular manner. The precise causal irritant has not been identified but there are good grounds for believing that the persons referred to above may be exposed at work to chemicals capable of themselves acting as carcinogenic agents or of liberating such agents in the tissues with which they come into contact.

Diagnosis

These diseases when occupational in origin are not different in any material respect from the same diseases arising from other causes. The time of their appearance does not help in differentiation since they may

appear at any age and may become manifest for the first time long after removal from the causal occupation.

CANCER OF THE NOSE. The average duration of exposure to risk before onset of symptoms has been about twenty-two years. The condition appears to start with some irritation and inflammation of the nasal passage, the actual neoplasm usually becoming evident first as a papilloma or polypus which soon shows signs of malignancy. The new growth arises most commonly from the covering of the ethmoid bone or of the middle turbinate, and tends to invade the orbit and frontal sinus.

CANCER OF THE LUNG. The average duration of exposure to risk before onset of symptoms has been over twenty years. The lesion may be either of bronchial or parenchymal origin, the early symptoms depending on the site and nature of the growth. The histology is not constant, oat-celled as well as adeno- and squamous-celled carcinomas having been described.

Prognosis

In nose cases progression to a fatal termination is usually rapid. In lung cases the prognosis depends largely on the stage at which the condition is diagnosed and on the nature of any operative procedures undertaken.

38. TUBERCULOSIS

The prescribed disease tuberculosis differs in no way from that disease as ordinarily encountered. The Department will normally seek opinions on claims in respect of this disease from a specialist. Where, on examining a claimant in respect of another prescribed disease, an examining medical practitioner considers the cause of the claimant's ailment to be tuberculous in origin, no action should be taken beyond including reference to the fact in his report.

39. PRIMARY NEOPLASM OF THE EPITHELIAL LINING OF THE URINARY BLADDER (PAPILLOMA OF THE BLADDER) OR OF THE EPITHELIAL LINING OF THE RENAL PELVIS OR OF THE EPITHELIAL LINING OF THE URETER

Aetiology

Since the end of the last century it has been known that there has been an undue incidence of bladder tumours among those engaged in the manufacture of synthetic dyes. The substance first giving rise to suspicion was aniline and these tumours acquired the name of 'aniline tumours'. However it is now known that this is a misnomer and that there is no evidence to implicate aniline in the production of tumours of the bladder.

The precise carcinogenic substance is still not known but it has been shown convincingly that the manufacture of, or exposure to, the three

dyestuff intermediates alpha-naphthylamine, beta-naphthylamine and benzidine and the manufacture of the dyes auramine and magenta carry a greatly increased risk of developing tumours of the bladder, ureter and renal pelvis. There is also little doubt that there is an excessive incidence of these tumours among workers manufacturing or exposed to certain nitro or primary amino derivatives of diphenyl which may or may not be further ring substituted by halogeno, methyl or methoxy groups. The dye intermediates may be used in chemical processes other than the manufacture of dyes and may be encountered in mixtures or liberated from compounds by dissociation on heating. The use in the rubber industry of certain accelerators containing beta-naphthylamine is an example of this.

Diagnosis

These tumours, whether malignant or benign papillomata, when occupational in origin are no different clinically from comparable tumours arising apparently spontaneously among the general population. A firm diagnosis will normally require cystoscopy although a search for malignant cells in centrifuged urine may have a place as a preliminary procedure.

Tumours do not usually develop until between 15 and 20 years after the beginning of exposure to the dangerous substance. However, tumours may appear in less than 2 years or more than 45 years from the first exposure. The length of time between exposure and the development of a tumour is therefore not conclusive evidence of its occupational origin. Furthermore, this time lag does not appear to be appreciably influenced by the degree of exposure.

DIFFERENTIAL DIAGNOSIS. It is emphasised that the disease prescribed is primary neoplasm of the epithelial lining of the bladder, ureter and renal pelvis only and does not include invading growths from surrounding structures or neoplasms elsewhere. Nor does it include other conditions giving rise to frequency of micturition and haematuria even in persons thought to be at risk.

Prognosis

This depends on the nature of tumour. Benign papillomata successfully treated are not likely to give rise to incapacity of more than short duration. However local recurrence of this condition is common. The onset of malignancy carries with it a bad prognosis.

40. POISONING BY CADMIUM

Aetiology

Cadmium, commonly derived from zinc-bearing ores, is used widely as a constituent of alloys, solders, bearing metals, and the negative plates of alkaline storage batteries. It is also finding increasing favour as a protective coating for base metals, a thin film being applied by electrolytic deposition from salt solutions or directly by pressure spraying of cadmium metal drawn into wire.

Cadmium salts, which are soluble in water or in weak acids, have long been known as irritant poisons but since ingestion of these even in very small quantities produces nausea, vomiting and purging, such occurrences tend to be in the nature of isolated accidents. Chronic poisoning of a cumulative kind, the result of ingestion, is not a recognised industrial hazard. It is otherwise, however, when fumes from molten cadmium are encountered as, for example, in smelting processes, metal pouring, the manufacture of cadmium-containing alloys, spraying of atomised metallic cadmium on a base metal ('metalisation') and welding of cadmium-plated articles. Freshly produced cadmium fumes, even more than soluble salts, tend to be highly irritating to the mucous membranes of the eyes, nose and throat, but in low concentrations such fumes are respirable and continued exposure to them may give rise both to local pulmonary damage and a rate of absorption exceeding that at which this metal can be excreted. Cadmium then accumulates in various parts of the body. Inadequate control of cadmium fumes on single occasions may therefore cause 'gassing accidents' in the form of acute poisoning, with or without metal-fume fever, while repeated exposure may result in a more chronic form of ill-health.

Diagnosis

Acute poisoning may occur when the fumes of cadmium oxide, produced by heating cadmium or an alloy containing it, are inhaled. During exposure no ill-effects may be noted or there may be slight irritation of the throat and cough. This is followed by a latent period in which the patient feels nothing amiss and which may last up to several hours. Then suddenly an influenzal-like illness sets in. There is a feeling of tightness in the chest with cough and pain in the chest, breathlessness, chills, sweating, shivering, aching pains in the back and limbs, headaches and dizziness and a metallic taste in the mouth; this stage endures from 4–10 hours and is followed by a stage of pulmonary reaction during which there is severe breathlessness and wheezing, pain in chest with a feeling of constriction of the chest, cough, weakness and malaise, anorexia and nausea. There may be fever. This stage lasts from 8 hours to several weeks and may be accompanied by fine crepitations at the base of the lungs and radiological evidence of bilateral pulmonary infiltration. There may be albuminuria. There is gradual improvement and a return to normal is most cases, but death may occur as a result of severe pulmonary oedema or renal cortical necrosis. The changes in the lungs, noted in fatal cases, are pulmonary oedema, with alveolar metaplasia, and in the kidneys bilateral cortical necrosis with tubular degeneration and glomerular infarction. A case of pneumonitis due to severe acute poisoning has been followed up for three years without evidence of the development of pulmonary fibrosis (Townshend 1968).

CHRONIC POISONING. Long continued exposure to concentrations of fume, too low to cause, at the time, recognisable symptoms, is believed to produce, in a proportion of those at risk, a more insidious but eventually markedly crippling form of pulmonary damage culminating in some

cases in gross emphysema. In this type of case the early symptoms of over-exposure may be very vague: some men give a history of continued nasal and pharyngeal irritation, perhaps with recurring epistaxis and, in some cases, anosmia, but more commonly the complaint is of a more general nature—undue tiredness, dyspnoea and cough. Clinical and radiological examination of these men may show generalised emphysema apparently unconnected with bronchitis or asthma. This would appear to be a purely local effect, a result of repeated exposure to recently generated cadmium fumes in a still active phase. Whether or not this is accompanied by, or followed by, either a focal effect elsewhere or generalised systemic poisoning is more doubtful, but evidence that cadmium does, in fact, accumulate in the body as a result of absorption via the lungs is not lacking. Cadmium is excreted from the body via the kidneys and the mere finding of cadmium in the urine indicates no more than the fact of past absorption, which may not even be occupational: it is not necessarily a sign of poisoning in the ordinary sense. On the other hand, its continued excretion for up to some years after exposure to risk has ceased may be regarded as evidence of long continued accumulation. Most patients excrete protein in the urine which is mild in degree and indistinguishable on electrophoretic examination from that occurring in patients with various kinds of renal tubular disorders. There is a low albumin fraction with a predominance of alpha 2 and beta globulin. Most of the proteins present have a low molecular weight and are thought to appear in the urine as a result of defective tubular reabsorption of protein normally present in the glomerular filtrate. The presence of protein in the urine does not justify by itself a diagnosis of chronic cadmium poisoning, but certainly lends support to such a diagnosis where a cadmium worker develops an otherwise unexplained widespread emphysema. Renal stones have been reported in some cases.

Prognosis

After an acute attack of cadmium poisoning, the result of exposure to fumes, recovery is to be expected and is likely to be complete. The more insidious form of poisoning, with chronic pulmonary damage, is not likely to attract attention until it has resulted in a clinically recognisable degree of generalised emphysema. Thereafter, the damage done is likely to be progressive and so, eventually, seriously disabling.

41. INFLAMMATION OR ULCERATION OF THE MUCOUS MEMBRANE OF THE UPPER RESPIRATORY PASSAGES* OR MOUTH PRODUCED BY DUST, LIQUID OR VAPOUR

Aetiology

Almost any irritant, vapour, dust or droplet spray may cause an inflammatory reaction of the mucous membrane of the upper respiratory

* The upper respiratory passages are restricted to those of the nose, the pharynx and the larynx.

passages or mouth, but owing to the natural defences the majority cause only a transient disability.

The mucous membrane most likely to intercept droplet or particulate irritants is that covering the nasal septum. Chromic acid, chromates, bichromates, arsenic, soda ash, mercury fulminate and cement are all known to cause an inflammatory reaction of the nasal septum which may lead to ulceration and perforation. Less commonly the lesions may appear in the mouth, pharynx and larynx.

Diagnosis

Inflammation of the mucous membrane of the upper respiratory passages, if of industrial origin, usually starts within a short time of first exposure to the alleged irritant and rarely arises for the first time after cessation of exposure. The more severe affections leading to ulceration are commonly situated over the cartilaginous part of the nasal septum. The typical ulcer is shallow and painless. There may be slight bleeding but infection is rare and the ulcers may therefore be present unnoticed by the patient. Such ulcers may go on to perforation of the cartilaginous portion of the nasal septum. The bony septum is never involved.

Prognosis

Inflammation of the mucous membranes, if of industrial origin, clears up rapidly on cessation of exposure. Ulceration also heals, although the process may be slow. Perforations of the nasal septum are usually permanent but cause little or no disability.

42. NON-INFECTIVE DERMATITIS OF EXTERNAL ORIGIN (INCLUDING CHROME ULCERATION OF THE SKIN BUT EXCLUDING DERMATITIS DUE TO IONISING PARTICLES OR ELECTRO-MAGNETIC RADIATIONS OTHER THAN RADIANT HEAT)

Aetiology

The main defences of the skin against external irritants are the cornified epithelium and the glandular secretions. The cornified epithelium will withstand the action of fairly strong acids but is damaged by alkalis and sulphides. The glandular secretions normally form a fatty and slightly acid protective coating against water soluble irritants. Any thinning, breach or maceration of the cornified epithelium such as may be caused by friction, heat and excessive sweating may predispose to an inflammatory response to external irritants. Similarly the openings of sebaceous ducts and hair follicles provide a portal of entry to irritants, particularly when the irritant is fat soluble, and inflammatory changes may start at these sites. Oil folliculitis and chlor-acne provide examples of such changes. The resistance of the skin to external irritants varies with age, sex, race, colour and, to a certain extent, diet.

The causes of occupational dermatitis may be broadly divided into two groups:

Primary cutaneous irritants: These substances will cause dermatitis at the site of contact if permitted to act for a sufficient length of time in a sufficient concentration. Examples of primary irritants are strong alkalis, acids, and solvents.

Cutaneous sensitisers: These substances do not necessarily cause skin changes on first contact but effect a specific sensitisation of the skin. If a further contact occurs after an interval of about seven days or more a dermatitis will develop at the site of the second contact. Examples of common sensitisers are plants, rubber, nickel, and many chemicals.

A primary irritant may also be a sensitiser in that an initial exposure to it may so condition the skin that subsequent exposures, although in concentrations so weak that they would not have caused trouble before the first exposure, now result in dermatitis.

Chrome ulceration of the skin may be caused by chromic acid, the alkali chromates and bichromates, and zinc chromate. The penetration of one of these substances through a minute break of the skin may cause the development of a raised hard lump which breaks down at the centre, revealing a deep ulcer with rounded and thickened edges and a slough covered base. Sepsis is exceptional and the ulcers do not become malignant. Similar ulcers may be caused by anhydrous sodium carbonate (soda ash), mercury fulminate, quick lime and strong brine.

It will be noted that the description of the disease excludes dermatitis due to infecting organisms such as bacteria, fungi and animal parasites. However, an acute attack of non-infective dermatitis which subsequently becomes infected may be accepted as a sequel to the disease.

Diagnosis

In arriving at a diagnosis attention should be paid to the following points:

1. PAST HISTORY AND HISTORY OF ONSET. Occupational dermatitis usually starts within a short time of exposure to the irritant and rarely arises for the first time after exposure has ceased.

2. NATURE OF WORK. Recent changes of work or materials handled should be particularly noted. Longer hours of work leave less time for recovery and may precipitate a break-down.

3. SITE OF LESION. In all skin cases the patient should be stripped sufficiently to permit of a proper examination. An occupational dermatitis may be generalised but much more often it is restricted to the hands and arms if it is due to a liquid, or to the face, neck and arms if due to a dust or vapour. The absence of lesions on other parts of the body will often help to exclude constitutional causes.

4. APPEARANCE OF THE LESION. An acute attack of dermatitis most commonly follows the sequence–erythema–papular eruption–

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46. INFECTION BY BRUCELLA ABORTUS

Aetiology

The disease is caused by organisms of the genus brucella which are transmitted by infected animals to man. Throughout the world many kinds of animals are known to harbour various species of the organism but in Britain brucella abortus is responsible for brucellosis in man and the natural animal reservoir of this species is in cattle.

The modes of infection are by direct contact, ingestion, inhalation and accidental inoculation. Direct contact with infected cattle or their products, especially the products of gestation, is the most important means of acquiring the infection, access to the body being gained through the skin and mucous membranes, including the conjunctivae.

Infection by ingestion may take place via the alimentary tract or through the mucous membrane of the throat when untreated milk from infected animals is drunk. At the present time, however, this mode of infection is less important than direct contact. Accidental inoculation may occur among veterinarians and laboratory workers. Infection from man to man is not known to occur.

Pathology and Pathogenesis

Whatever their mode of entry into the body, the bacilli lodge in the regional lymph nodes. Here they may be destroyed, or multiply and get into the bloodstream, whence they enter reticulo-endothelial cells in various organs, and set up small foci of granulomatous inflammation.

Clinical Picture

The incubation period is from about 1 to 3 weeks. In *acute brucellosis* the onset may be sudden or gradual. There may be chills or rigors with headache and pains in the back and limbs and severe sweating and fatigue often accompanied by a cough. The temperature ranges between 100°F (37·7°C) and 103°F (39·4°C) with spikes to 105°F (40·5°C) or so. Few or no abnormal physical signs may be present on clinical examination but the spleen and peripheral lymph nodes may be palpable. A blood count usually shows a leucopenia with a reduction in the number of polymorphonuclear cells. The acute illness usually subsides quickly with recovery setting in within a fortnight. In some cases however the illness may progress through a *sub-acute* phase characterised by bouts of fever, back pain and a feeling of depression and lassitude which may last for several months. *Chronic* brucellosis may present a difficult diagnostic problem and its existence as a distinct entity has been queried by some authorities. The main symptoms suggesting the diagnosis are lassitude, depression, frontal headache and general malaise. There may be a history of a previous attack of acute brucellosis or serological evidence of infection. However in a population

which has been widely exposed to brucella abortus a considerable number of persons may show positive serological tests without ever having experienced symptoms of brucellosis and included in this group there will be those with vague symptoms of malaise, lethargy and depression and care must be taken before ascribing them to brucellosis.

Diagnosis

There are no specific signs and symptoms and the diagnosis rests on an evaluation of clinical and epidemiological observations aided by laboratory tests. The bacilli may be cultured from the blood in a small proportion of cases. Tests carried out on the blood serum include serum agglutination, complement fixation and the Coombes test. In the present state of knowledge it is inadvisable to lay down too rigid criteria for the interpretation of these tests the results of which must be examined in the light of all the information available about a particular case. It is well to bear in mind that in some patients with undisputed evidence of infection such as a positive blood culture serological tests may be negative while in others with no illness or history of infection the tests may be strongly positive.

Prognosis

No figures are available for cases treated with antibiotics. In most instances the disease is self-limiting and with modern methods of treatment recovery may confidently be expected.

47. POISONING BY ACRYLAMIDE MONOMER

Acrylamide ($CH_2=CHCONH_2$) is a white crystalline solid generally encountered in powder form. It is a vinyl monomer which readily undergoes polymerization and copolymerization. It is employed as a waterproofing and soil stabilizing agent, in flocculating processes and in the manufacture of paper, adhesives, fibres, dyes, pigments and leather substitutes. It is used in the preparation of photographic emulsions, gelatin substitutes, plastics, and in the making of rubber, surface coating and thickening agents.

Toxicity

The monomer of acrylamide is toxic and the polymer non-toxic. Experiments with animals have shown that the monomer is toxic whether absorbed through the unbroken skin, ingested or administered by injection. Evidence of injury to the peripheral nerves with reduction in the motor-nerve-conduction velocity in the fibres has been demonstrated in rats, cats and monkeys in which degeneration of myelin sheaths and axons and signs of severe peripheral neuropathy were also noted. In occupational poisoning the only portal of entry of the monomer appears to be by absorption through the skin. Clinical observation suggests that damage is caused to the peripheral nerves and possibly to the mid brain. As well as being a neurotoxin the monomer is irritating to the eye and is a primary irritant of the skin.

Clinical Picture

Patients complain of weakness of the limbs and unsteadiness whilst walking, numbness of the finger tips and slurring of the speech. There may be a feeling of fatigue and loss of body weight. Some individuals suffer from impairment of bladder control. Weakness and wasting of limb muscles may be discovered on examination together with evidence of sensory impairment. There may be a tremor of the limbs and trunk. The skin of the hands may be red, moist and peeling.*

Differential Diagnosis

This is from other causes of polyneuritis. The occupational history should give the clue to the diagnosis and a study of the process may be invaluable in indicating the hazard.

Prognosis

There is no specific treatment other than removal from contact with the monomer. In all cases improvement has been noted on cessation of exposure and recovery may be looked for, but may require varying periods depending on the severity of the disease.

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*When a claimant is suffering from an affection of the skin only and there is no indication of systemic poisoning he should be treated as suffering from disease No. 42 on the list of Prescribed Diseases and not from poisoning by acrylamide monomer.

Part II

NOTES ON BENEFITS

Introductory

The following paragraphs give brief information about benefits and, in relation to the diseases covered in Part I of the booklet, deal with those aspects of the way in which claims are decided which may be of interest to the medical profession. Details of the remaining prescribed diseases—pneumoconiosis (which includes silicosis and asbestosis) byssinosis, farmer's lung and mesothelioma—are contained in the booklet 'Pneumoconiosis and allied occupational chest diseases', obtainable from H.M.S.O.

Benefits payable

There are three kinds of benefit payable under the Industrial Injuries Act:

(*a*) INJURY BENEFIT, a temporary payment made so long as the insured person is incapable of work as a result of the disease during what is known as 'the injury benefit period' which cannot last for more than six months.

(*b*) DISABLEMENT BENEFIT, a payment dependent on the degree of disablement an insured person still suffers as a result of the disease when the injury benefit period ends, or when he is capable of work but suffers some disablement due to the disease. It is either a weekly pension or a gratuity and the rate depends on the assessment by a medical board of the degree of disablement.

(*c*) DEATH BENEFIT, payable to certain dependents if death results from the disease.

In addition, for a person awarded disablement benefit, there are a number of increases designed to meet particular contingencies, for example, for treatment of the disease while he is in hospital, or if he is unable as a result of the disease to return to his regular job and unable to work at a job of equivalent standard. Details of the increases of disablement benefit available are given in separate leaflets obtainable free from local offices of the Department of Health and Social Security.

How claims are decided

Claims for benefit under the Industrial Injuries Act are decided by independent statutory authorities, the first of whom is the insurance officer.

On each claim there are normally a number of questions to be decided and where, in the case of prescribed disease, some of these are medical

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before the Industrial Injuries Scheme began in July 1948, but the diseases may develop so slowly that even where cover was provided under the Workmen's Compensation Act, it is too late to make a claim under them. The diseases covered are pneumoconiosis (including asbestosis); byssinosis; epitheliomatous cancer; malignant disease of the skin due to X-rays, radium or other radio-active substances; nickel cancer; papilloma of the bladder; diffuse mesothelioma; and nasal adeno-carcinoma. The Scheme which is financed by the Industrial Injuries Fund is administered by an independent board—the Pneumoconiosis, Byssinosis and Miscellaneous Diseases Benefit Board.

Appendix

LIST OF PRESCRIBED DISEASES

The prescribed diseases (other than pneumoconiosis and byssinosis) are set out in the first column of the table below. Each disease is prescribed in relation to all insured persons who have been employed on or after 5th July, 1948, in insurable employment in any occupation set against it in the second column.

Sequelae or resulting conditions

Where a person insured against a disease is suffering from a condition which in his case is adjudged to have resulted from that disease, he is regarded for benefit purposes as if he were still suffering from the disease itself.

Description of disease or injury	Nature of occupation
Poisoning by:	Any occupation involving:
1. Lead or a compound of lead	The use or handling of, or exposure to the fumes, dust or vapour of, lead or a compound of lead, or a substance containing lead.
2. Manganese or a compound of manganese	The use or handling of, or exposure to the fumes, dust or vapour of, manganese or a compound of manganese, or a substance containing manganese.
3. Phosphorus or phosphine or poisoning due to the anti-cholinesterase action of organic phosphorus compounds	The use or handling of, or exposure to the fumes, dust or vapour of, phosphorus or a compound of phosphorus, or a substance containing phosphorus.
4. Arsenic or a compound of arsenic	The use or handling of, or exposure to the fumes, dust or vapour of, arsenic or a compound of arsenic, or a substance containing arsenic.
5. Mercury or a compound of mercury	The use or handling of, or exposure to the fumes, dust or vapour of, mercury or a compound of mercury, or a substance containing mercury.

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Description of disease or injury	Nature of occupation
Poisoning by:	Any occupation involving:
18. Gonioma kamassi (African box-wood)	The manipulation of gonioma kamassi or any process in or incidental to the manufacture of articles therefrom.
19. Anthrax	The handling of wool, hair, bristles, hides or skins or other animal products or residues, or contact with animals infected with anthrax.
20. Glanders	Contact with equine animals or their carcases.
21. (*a*) Infection by Leptospira ictero-haemorrhagiae ...	Work in places which are, or are liable to be, infested by rats.
(*b*) Infection by Leptospira canicola	Work at dog kennels or the care or handling of dogs.
22. Ankylostomiasis	Work in or about a mine.
23. (*a*) Dystrophy of the cornea (including ulceration of the corneal surface) of the eye, (*b*) Localised new growth of the skin, papillomatous or keratotic, (*c*) Squamous-celled carcinoma of the skin, due in any case to arsenic, tar, pitch, bitumen, mineral oil (including paraffin), soot or any compound, product (including quinone or hydroquinone), or residue or any of these substances.	The use or handling of, or exposure to, arsenic, tar, pitch, bitumen, mineral oil (including paraffin), soot or any compound, product (including quinone or hydroquinone), or residue of any of these substances.
24. (Now covered by diseases 41 and 42)	
25. Inflammation, ulceration or malignant disease of the skin or subcutaneous tissues or of the bones, or blood dyscrasia, or cataract, due to electro-magnetic radiations (other than radiant heat), or to ionising particles ...	Exposure to electro-magnetic radiations other than radiant heat, or to ionising particles.

Description of disease or injury	Nature of occupation
	Any occupation involving:
26. Heat cataract	Frequent or prolonged exposure to rays from molten or red-hot material.
27. Decompression sickness ...	Subjection to compressed or rarefied air.
28. Cramp of the hand or forearm due to repetitive movements	Prolonged periods of handwriting, typing or other repetitive movements of the fingers, hand or arm.
29. } 30. } (Now covered by disease 28)	
31. Subcutaneous cellulitis of the hand (Beat hand) ...	Manual labour causing severe or prolonged friction or pressure on the hand.
32. Bursitis or subcutaneous cellulitis arising at or about the knee due to severe or prolonged external friction or pressure at or about the knee (Beat knee)	Manual labour causing severe or prolonged external friction or pressure at ~~or~~ about the knee.
33. Bursitis or subcutaneous cellulitis arising at or about the elbow due to severe or prolonged external friction or pressure at or about the elbow (Beat elbow)	Manual labour causing severe or prolonged external friction or pressure at or about the elbow.
34. Traumatic inflammation of the tendons of the hand or forearm, or of the associated tendon sheaths	Manual labour, or frequent or repeated movements of the hand or wrist.
35. Miner's nystagmus... ...	Work in or about a mine.
36. Poisoning by beryllium or a compound of beryllium	The use or handling of, or exposure to the fumes, dust or vapour of, beryllium or a compound of beryllium, or a substance containing beryllium.
37. (*a*) Carcinoma of the mucous membrane of the nose or associated air sinuses. (*b*) Primary carcinoma of a bronchus or of a lung	Work in a factory where nickel is produced by decomposition of a gaseous nickel compound which necessitates working in or about a building or buildings where that process or any other industrial process ancillary or incidental thereto is carried on.

Description of disease or injury	Nature of occupation
38. Tuberculosis	Any occupation involving: Close and frequent contact with a source or sources of tuberculous infection by reason of employment:— (*a*) in the medical treatment or nursing of a person or persons suffering from tuberculosis, or in a service ancillary to such treatment or nursing; (*b*) in attendance upon a person or persons suffering from tuberculosis, where the need for such attendance arises by reason of physical or mental infirmity; (*c*) as a research worker engaged in research in connection with tuberculosis; (*d*) as a laboratory worker, pathologist or person taking part in or assisting at post-mortem examinations of human remains where the occupation involves working with material which is a source of tuberculous infection.
39. Primary neoplasm of the epithelial lining of the urinary bladder (Papilloma of the bladder), or of the epithelial lining of the renal pelvis or of the epithelial lining of the ureter	(*a*) Work in a building in which any of the following substances is produced for commercial purposes: (*i*) Alpha-naphthylamine or beta-naphthylamine. (*ii*) Diphenyl substituted by at least one nitro or primary amino group or by at least one nitro and primary amino group. (*iii*) Any of the substances mentioned in sub-paragraph (*ii*), above if further ring substituted by halogeno, methyl or methoxy groups, but not by other groups.

Description of disease or injury	Nature of occupation
	Any occupation involving: (*iv*) The salts of any of the substances mentioned in sub-paragraphs (*i*), to (*iii*), above. (*v*) auramine or magenta; (*b*) the use or handling of any of the substances mentioned in sub-paragraphs (*i*), to (*iv*), of paragraph (*a*), or work in a process in which any such substance is used or handled or is liberated; (*c*) the maintenance or cleaning of any plant or machinery used in any such process as is mentioned in paragraph (*b*), or the cleaning of clothing used in any such building as is mentioned in paragraph (*a*), if such clothing is cleaned within the works of which the building forms a part or in a laundry maintained and used solely in connection with such works.
40. Poisoning by cadmium ...	Exposure to cadmium fumes.
41. Inflammation or ulceration of the mucous membrane of the upper respiratory passages or mouth produced by dust, liquid or vapour	Exposure to dust, liquid or vapour.
42. Non-infective dermatitis of external origin (including chrome ulceration of the skin but excluding dermatitis due to ionising particles or electro-magnetic radiations other than radiant heat)	Exposure to dust, liquid, or vapour or any other external agent capable of irritating the skin (including friction or heat but excluding ionising particles or electro-magnetic radiations other than radiant heat).
43. *Pulmonary disease due to the inhalation of the dust of mouldy hay or of other mouldy vegetable produce, and characterised by symptoms and signs attributable to a reaction in the peripheral part of the broncho-pulmonary system, and giving rise to a defect in gas exchange (Farmer's lung)	Exposure to the dust of mouldy hay or other mouldy vegetable produce by reason of employment:— (*a*) in agriculture, horticulture or forestry; or (*b*) loading or unloading or handling in storage such hay or other vegetable produce; or (*c*) handling bagasse.

Description of disease or injury	Nature of occupation
44. *Primary malignant neoplasm of the mesothelium (diffuse mesothelioma) of the pleura or of the peritoneum	Any occupation involving: (*a*) The working or handling of asbestos or any admixture of asbestos; (*b*) the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos; (*c*) the cleaning of any machinery or plant used in any of the foregoing operations and of any chambers, fixtures and appliances for the collection of asbestos dust; (*d*) Substantial exposure to the dust arising from any of the foregoing operations.
45. Adeno-carcinoma of the nasal cavity or associated air sinuses	Attendance for work in or about a building where wooden furniture is manufactured.
46. Brucellosis	Contact with bovine animals infected by brucella abortus, their carcases or parts thereof or their untreated products, or with laboratory specimens or vaccines of or containing brucella abortus, by reason of employment: (*a*) as a farm worker; (*b*) as a veterinary worker; (*c*) as a slaughterhouse worker; (*d*) as a laboratory worker; or (*e*) in any other work relating to the care, treatment, examination or handling of such animals, carcases or parts thereof or products.
47. Poisoning by acrylamide monomer	Any occupation involving, the use or handling of, or exposure to, acrylamide monomer.

* *Note*: See booklet 'Pneumoconiosis and allied occupational chest diseases', obtainable from H.M.S.O.

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