

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES



Occupational Safety and Health Branch
Labour Department

This Guidance Notes is prepared by
the Occupational Safety and Health Branch,
Labour Department

This edition February 2006

This Guidance Notes is issued free of charge and can be obtained from offices of the Occupational Safety and Health Branch, Labour Department. It can also be downloaded from website of the Labour Department at http://www.labour.gov.hk.eng/public/content2_9.htm. Addresses and telephone numbers of the offices can be found in website of the Department at <http://www.labour.gov.hk/eng/tele/osh.htm> or call 2559 2297.

This Guidance Notes may be freely reproduced except for advertising, endorsement or commercial purposes. Please acknowledge the source as "Guidance Notes on the Diagnosis of Notifiable Occupational Diseases", published by the Labour Department.

If you have any complaints about unsafe workplaces and practices, please call the Labour Department's occupational safety and health complaint hotline at 2542 2172.

Information on the services offered by the Occupational Safety & Health Council can be obtained through their hotline 2739 9000.

**Guidance Notes on
the Diagnosis of Notifiable
Occupational Diseases**

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Preface

Under section 15 of the Occupational Safety & Health Ordinance, medical practitioners are required to notify occupational diseases to the Commissioner for Labour. The notification must be in writing and on a form approved by the Commissioner. It must be lodged as soon as practicable.

The notification of an occupational disease may initiate a chain of events often including the investigation of the index case, active scrutiny of other workers to identify other cases, the recommendation of specific preventive measures, follow-up evaluation of the effectiveness of preventive measures being taken at the workplace, etc.

All notifiable occupational diseases are compensable under the Employees' Compensation Ordinance, the Pneumoconiosis (Compensation) Ordinance and the Occupational Deafness (Compensation) Ordinance.

The guidance book is intended for the use of medical practitioners in the identification and diagnosis of occupational diseases listed in Schedule 2 of this Ordinance. The notes do not purport to be exhaustive, but are intended as an aid to medical practitioners dealing with these illnesses.

This book is divided into four parts: Diseases caused by physical agents; Diseases caused by biological agents; Diseases caused by chemical agents; and Diseases caused by miscellaneous agents. For ease of reference, we have put down the item number in the order of the Schedule against each disease and an alphabetical index in the end.

Our Occupational Health Service is ready to give advice and assistance regarding any aspect of notifiable occupational diseases or any other matters concerning occupational health. All medical practitioners involved in dealing with such cases are welcome to contact us via Occupational Health Service, Labour Department, 15th Floor, Harbour Building, 38 Pier Road, Central, Hong Kong, telephone at 2852 4041 or fax at 2581 2049 for further information. Medical practitioners attending patients who have been diagnosed as or are suspected to be suffering from occupational disease may refer such cases to Kwun Tong Occupational Health Clinic (telephone 2343 7133) for further investigation.

Occupational Health Service
Occupational Safety & Health Branch
Labour Department
July 1997

Preface to 2nd Edition

In this new edition, some alterations have been made to the text. Two new occupational diseases namely "Carpal Tunnel Syndrome" and "Legionnaires Disease" have been added and two diseases namely "Occupational Asthma" and "Tenosynovitis of Hand or Forearm" have been expanded after the amendments made to the list of Notifiable Occupational Disease in July 1999.

The thermal effects of non-ionizing radiation have been added to the "Radiation Illnesses" with reference to the new edition of "Notes on the Diagnosis of Prescribed Diseases" published by the Department of Social Security, U.K. The other minor alteration is the addition of the concept of sub-clinical heavy metal poisoning which is an area where biochemical evidence of functional impairment appears and where even the subjective well being may be decreased to some extent. The inclusion of this concept of poisoning is considered justified on the ground that the World Health Organization defines health as "a state of complete physical, mental and social well-being." As such, we recommend that cases, which require suspension from further exposure at work, should be notified to the Labour Department for investigation and when considered necessary, for law enforcement actions.

Occupational Health Service
Labour Department
Jan 2002

Preface to 3rd Edition

The new edition of this Guidance Notes includes two new infectious diseases, namely "Severe Acute Respiratory Syndrome" and "Avian Influenza A", that were added to the list of notifiable occupational diseases in Schedule 2 of the Occupational Safety and Health Ordinance in February 2005 with a view to monitoring the occurrence and controlling the spread of these infectious diseases at workplace more effectively.

Apart from the inclusion of the two infectious diseases, some minor alternations have also been made to the text. We hope that this guidance notes can provide medical practitioners with a better understanding of all notifiable occupational diseases and assist them to fulfil their statutory duties in notifying occupational diseases to the Commissioner for Labour.

Occupational Health Service
Labour Department
November 2005

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Content

	Page
Part I: Disease Caused by Physical Agents	
A1: Radiation Illness _____	2
A2: Heat Cataract _____	6
A3: Compressed Air Illness _____	7
A4: Cramp of Hand or Forearm _____	9
A5: Beat Hand _____	10
A6: Beat Knee _____	11
A7: Beat Elbow _____	12
A8: Tenosynovitis of Hand or Forearm _____	13
A9: Carpal Tunnel Syndrome _____	14
Part II: Diseases Caused by Biological Agents	
B1: Anthrax _____	16
B2: Glanders _____	17
B3: Leptospirosis _____	18
B4: Extrinsic Allergic Alveolitis _____	18
B5: Brucellosis _____	21
B6: Tuberculosis in health care workers _____	22
B7: Parenterally contracted viral hepatitis _____	23
B8: <i>Streptococcus suis</i> Infection _____	24
B9: Avian Chlamydiosis _____	25
B10: Legionnaires' Disease _____	26
B11: Severe Acute Respiratory Syndrome _____	27
B12: Avian Influenza A _____	28
Part III: Diseases Caused by Chemical Agents	
C1: Lead Poisoning _____	30
C2: Manganese Poisoning _____	32
C3: Phosphorus Poisoning _____	33
C4: Arsenic Poisoning _____	35
C5: Mercury Poisoning _____	37
C6: Carbon Bisulphide Poisoning _____	39

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

C7: Benzene Poisoning _____	40
C8: Poisoning by Nitro-, Amino- or Chloro- Derivatives of Benzene _____	41
C9: Dinitrophenol Poisoning _____	43
C10: Poisoning by Halogen Derivatives of Hydrocarbons of Aliphatic Series _____	44
C11: Diethylene Dioxide Poisoning _____	46
C12: Chlorinated Naphthalene Poisoning _____	47
C13: Poisoning by Oxides of Nitrogen _____	48
C14: Beryllium Poisoning _____	49
C15: Cadmium Poisoning _____	51
C16: Dystrophy of the Cornea _____	52
C17: Skin Cancer _____	53
C18: Chrome Ulceration _____	54
C19: Urinary Tract Cancer _____	55
C20: Peripheral Polyneuropathy _____	56
C21: Localised Papillomatous or Keratotic New Skin Growth _____	57
C22: Occupational Vitiligo _____	58

Part IV: Diseases Caused by Miscellaneous Agents

D1: Occupational Dermatitis _____	60
D2: Chemical-induced Upper Respiratory Tract Inflammation _____	62
D3: Nasal or Paranasal Sinus Cancer _____	63
D4: Byssinosis _____	64
D5: Occupational Asthma _____	66
Silicosis _____	68
Asbestos-related Diseases _____	69
Occupational Deafness _____	71
 INDEX _____	 72
ANNEX I: Notification Form _____	75
ANNEX II: Extract from Occupational Safety & Health Ordinance _____	76

PART I

DISEASES CAUSED BY

PHYSICAL AGENTS

A1 Radiation Illness

The occupational diseases that require notification include inflammation, ulceration or malignant disease of the skin or subcutaneous tissues, or of the bones, or blood dyscrasia, or cataract, due to electro-magnetic radiation (other than radiant heat), or to ionizing particles. Any occupation involving the exposure to electro-magnetic radiation other than radiant heat, or to ionizing particles is therefore included. Electro-magnetic waves cover all wavelengths from mile long radio waves to very short femtometre x-rays. In relation to biological effects, radiation can be categorized as ionizing and non-ionizing.

Ionizing Radiation

The harmful effects of radiation have been known since the early years of this century when a scientist, Henri Becquerel, researching this new field developed a skin burn after carrying a tube of radium in his trouser pocket. Even earlier, lung cancer had been noted in miners who worked in mines where they were exposed to large quantities of radon gas.

A wide variety of workers may be exposed to ionizing radiation. These include radiologists and radiographers in hospitals, workers in the nuclear power industry, naval and military personnel, scientists, manufacturers and users of radio-luminescent paints, industrial radiographers, aircrew and miners.

Biological Effects of Radiation

The biological effects of ionizing radiation begin with the physical absorption of energy leading to the process of ionization – the removal of electrons from atoms. This can take place in 10^{-16} seconds.

Chemical damage can occur in 10^{-10} seconds. This primarily involves the formation of free radicals from the ionization of water. These are extremely reactive and react with other molecules such as DNA causing chromosomal aberrations. They can also cause lysosomal membrane damage leading to autodigestion of the cell or mitochondrial damage. This damage takes place over seconds to hours.

Biological damage to cells, tissues and whole organisms may take place over hours to several years. This may involve immediate death of cells, cell modification leading to mutation or mitotic damage or reproductive death which is the loss of unlimited capacity for division of stem proliferative cells. It can therefore be seen that tissues which have a high turnover of cells (e.g. gastrointestinal epithelium, haemopoietic system, skin) are particularly vulnerable to radiation damage.

Stochastic & Deterministic Effects

Ionizing radiation exposure may lead to two types of health effects, namely stochastic and deterministic. A stochastic effect is an effect whose probability of manifestation increases with the dose but there is no threshold of exposure below which it is certain that the effect will not occur. An example is ionizing radiation-induced cancer. A deterministic effect is an effect whose severity increases with dose but there is a threshold level below which it is certain that the effect will not occur. An example is desquamation of skin.

Local Effects of Ionizing Radiation

A significant radiation dose to the skin can cause transient reddening followed by erythema. Alopecia may result from damage to hair follicles. Dry or moist desquamation may occur in a few weeks if the dose is high enough. High doses of ionizing radiation may produce immediate or reproductive death of cells. This means that tissues may progressively become hypovascular, hypocellular and deprived of oxygen as cells live out their life span and are not replaced. This may be severe enough to lead to radionecrosis or to a similar breakdown if the tissue suffers further insult (such as surgery or accidental wounding) and is unable to respond to the challenge of healing. These are deterministic effects.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Very high doses to the gonads may cause sterility, but a whole body dose high enough to cause such problems would result in death. Neutrons and β particles are known to be effective in causing cataracts with an incubation period of 3 to 4 years. Genetic damage has been demonstrated in laboratory animals but human studies are not conclusive.

Whole Body Irradiation

A single whole body dose of ionizing radiation above 30 Gy is sufficient to cause death within 36 hours from central nervous system syndrome. The victims display a rapid onset of severe nausea and vomiting with disorientation and coma. Death is due to failure of central nervous system conduction and cerebral oedema. There is no effective treatment.

A single whole body dose of ionizing radiation between 10 and 30 Gy also causes a rapid onset of nausea and vomiting which resolves after a few hours and is followed by an apparent recovery. Death however supervenes at between 4 and 14 days after exposure due to gastrointestinal syndrome. This is due to reproductive death of gastrointestinal stem cells of the small intestine. As cells live out their normal life span but are not replaced, the lining of the intestine becomes flat and incapable of absorption. This leads to profuse diarrhoea. Breakdown of intestinal lining allows free access of bowel contents to blood. Death is from dehydration and overwhelming infection and no treatment is effective.

Transient nausea and vomiting may be seen in some persons exposed to between 2 and 10 Gy and once again is followed by apparent recovery. This time however it is the haemopoietic syndrome which may cause death due to overwhelming infection from neutropenia and bleeding from loss of platelets. A fall in peripheral lymphocytes is seen within the first 2 days but most other blood components are seen in normal numbers until their normal life span is exhausted. Reproductive death of a large part of the haemopoietic stem cell population may be fatal within about 60 days. This syndrome is treatable if aggressive support therapy can maintain the patient until the surviving stem cells can be stimulated to produce more components. If compatible bone marrow is available, transplantation may be possible.

Malignant Disease

The induction of cancer by ionizing radiation is a stochastic effect and *the malignancies so induced are indistinguishable from naturally occurring disease*. At around 3 to 10 years following ionizing radiation exposure there is a rise in leukaemogenesis, particularly of acute leukaemias. Solid tumours tend to take longer to develop – sometimes more than 40 years. These tumours include skin, breast, lung, brain, thyroid, bone and non-Hodgkin's lymphoma. Children are more susceptible than adults and due to the latency period the development of ionizing radiation-induced tumours is reduced in those irradiated later in life. The overall risk of fatal cancer induced by ionizing radiation is about 12.5 per million population per mSv. Natural deaths from cancer are about 200 000 to 250 000 per million population.

Damage to Foetus

The foetus is particularly susceptible to damage during the period of organogenesis. The effect will depend on which particular organs are developing at the time of irradiation but the effects on the developing forebrain are catastrophic, leading to severe mental retardation. This effect is deterministic and hence a limited occupational exposure can be permitted during pregnancy. The development of childhood malignancy is also increased by foetal irradiation and as in adults this is a stochastic effect.

Dose Limitation

The International Commission on Radiological Protection (ICRP) recommends that no practice involving radiation exposure be undertaken unless it is likely to bring a net benefit, and that for any such practice protection should be optimised so that doses of ionizing radiation are kept As Low As Reasonably Achievable (ALARA), economical and social considerations being taken into account.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

The statutory annual occupational dose limit is 20 mSv to the whole body, 500 mSv to the extremities and the skin, and 150mSv to the lens of the eye. For female workers of the reproductive capacity the additional limit is 5 mSv in any consecutive 3 months' intervals. In the case of a pregnant worker, the dose limit for the foetus is 1 mSv during the pregnancy. For members of the general public, the statutory annual dose limit is 1 mSv.

Non-ionizing Radiation

Non-ionizing radiation can have various thermal effects on the body depending on the particular wavelength of the radiation concerned. The wavelengths of important non-ionizing radiations include:

Ultraviolet	200 nm – 400 nm
Visible	400 nm – 760 nm
Infrared	760 nm – 1.0 mm
Microwave	1.0 mm – 1.0 m
Radiofrequency	>1.0 m

Radiofrequency and Microwave Radiation

Occupational exposures to radiofrequency and microwave radiation could be present in workplaces where employees are near equipment for dielectric heating (used in sealing of plastics and drying of wood), physiotherapy, radio-communication, and maintenance of aerial transmitters and high-power electrical equipment.

Radiofrequency and microwave radiation cause vibration and rotation of molecules which can be sufficient to cause significant local heating of tissues. The effect is related to wavelength, power intensity, and time of exposure as well as body size, shape, its electrical properties and whether reflecting surfaces are present.

Localized thermally induced masses may appear within days after exposure and consists of interstitial oedema and coagulation necrosis. The exposed skin has a sunburn appearance. Erythema, induration, vesiculation or bullae may be present. The eye, which has a limited blood supply to dissipate localized heat, can suffer lens opacity, cataracts, corneal damage and retinal lesions.

Infrared Radiation

Infrared radiation is emitted from hot bodies. Industrial sources of significant infrared radiation include furnaces, hot metals, dehydration equipment and equipment for drying and baking of paints and surface coatings, and spot heating. It is also produced by certain lasers and medical physiotherapy equipment.

Acute, high intensity exposure to infrared radiation can cause thermal damage to the cornea, iris or lens. Exposure to infrared radiation has been associated with cataract formation, particularly among glassblowers and furnace workers. Thermal injury to the skin can result in acute skin burn with increased pigmentation.

Visible Radiation

Intense exposure from very bright short pulsed sources e.g. pulsed lasers and high intensity electronic flash lamps can induce thermal injury in the retina. Blue light (wavelength 400 to 500 nm) can also cause photochemical reaction in the retina. It is responsible for solar retinitis and may contribute to retinal aging and senile macular degeneration, resulting in visual field defects.

Ultraviolet (UV) Radiation

The ultraviolet wavelengths are divided into 3 bands:

UV-A	315 – 400 nm
UV-B	280 – 315 nm
UV-C	Region below 280 nm

Wavelengths below 295 nm are absorbed in the epidermis; longer wavelengths penetrate into the dermis. Wavelengths greater than 300 nm are transmitted through the cornea and are absorbed by the lens.

Workers can be exposed to UV radiation from both natural and man-made sources. Outdoor workers are exposed to UV radiation from sunlight. Industrial processes e.g. welding and hot-metal work can produce UV radiation. Hospitals make use of UV radiation for its germicidal properties. Other applications of UV radiation include photoengraving, illumination, sterilization of foodstuffs, lasers, medical diagnosis and treatment etc.

Exposure to UV radiation can produce photokeratitis and conjunctivitis, especially after exposure to wavelengths of 270 – 280 nm. The worker may complain of pain, tearing, intolerance to light, visual haze, and a sensation of sand in the eyes. Excessive exposure to UV radiation in the 295 – 310 nm region can result in cataracts.

The most common acute UV radiation effect on the skin is erythema (sunburn). Chronic exposure may lead to:

- (a) premalignant skin lesions e.g. actinic keratosis and keratocanthoma;
- (b) malignant skin lesions e.g. basal cell carcinoma, squamous cell carcinoma and malignant melanoma.

A2 Heat Cataracts

Certain wavelengths of electromagnetic radiation incident on the eye are significantly absorbed by the lens. These wavelengths are primarily in the infrared (A) (760 – 1400 nm) but also includes ultraviolet (B) (280 – 315 nm) and ultraviolet (A) (315 – 400 nm) and microwave (1.0 mm – 1.0 m). Increased incidence of cataracts has been found in workers such as glassblowers and blast furnace operators who have been exposed over a period of many years to these radiations.

Clinical Features

The mechanism of formation of heat cataracts is not clear but may be due to direct thermal injury or injury caused by thermoelastic expansion. Presentation is usually a painless gradual reduction of visual acuity with consequent blurring of vision. Examination reveals posterior or less typically anterior subcapsular opacities. Linear splits may develop in the outer lamellae of the anterior capsule. This contrasts with the usual appearance of age related cataracts – hardening & discolouration of the central part of the lens.

Treatment is by cataract extraction and intraocular lens implantation but visual acuity may be significantly diminished for a considerable period before surgery is undertaken. Some degree of permanent disability can be expected.

A3 Dysbarism including Decompression Illness, Barotrauma and Osteonecrosis

These condition may result from work undertaken at elevated pressure. This may include diving, work in compressed air environments such as pressurised caissons and tunnels or to staff of recompression chambers. It may also occur in aircrew as a result of ascent to high altitudes in unpressurised aircraft.

The weight of the earth's atmosphere exerts a pressure of about 100 kilopascals (kPa) at sea level. This is often expressed as 1 atmosphere absolute (1 ATA). At the top of Mount Everest the pressure is about 50% of this. In seawater for every 10m descended, the pressure increases by 1 atmosphere. Atmospheric air is composed of about 20% oxygen and 80% nitrogen. This means that at sea level the partial pressures of air are 0.2 ATA O₂ and 0.8 ATA N₂ but at 40 metres of sea water they are 1.0 ATA O₂ and 4.0 ATA N₂.

Barotrauma

This results from the damage to gas filled rigid walled spaces as a result of change of gas volume related to the pressure. It may arise during descent (compression barotraumas) or during ascent (decompression barotraumas).

Compression Barotrauma

This principally affects the ears and is due to a failure of the Eustachian tube to allow air entry and equalise middle ear pressure. Symptoms are of pain in the ear and if descent continues, trauma to the tympanic membrane may result, ranging from diffuse erythema to frank haemorrhage and perforation. Attempts to equalise pressure by a forced Valsalva manoeuvre may result round or oval window rupture with a sudden onset of unilateral deafness.

Other sites include

- Sinus blocked ostia may present as pain with epistaxis.
- Dental gas pockets due to caries or poor fillings may cause pain.
- Face mask failure to equalize pressure may cause facial oedema and subconjunctival haemorrhage.

Decompression Barotrauma

The most important site for decompression barotraumas is the lung. The cause may be pulmonary pathology (resulting in air trapping) or breath holding during ascent. There may be chest pain, shortness of breath, dyspnoea, cough or mild haemoptysis, change in voice tone or subcutaneous emphysema.

In some case there may be a frank pneumothorax as alveolar gas escapes to the pleural space. The symptoms are commonly of a sharp pain, worse on inspiration, and shortness of breath. There is mediastinal shift to the affected side, a hyperresonant percussion note and reduced breath sounds and movement on that side and cyanosis. It may become a tension pneumothorax on ascent with tracheal shift away from the affected side, cyanosis, shock and unconsciousness. Death may result if the pressure is not relieved by chest drain insertion.

The most catastrophic effect of pulmonary barotraumas is arterial gas embolism in which gas from a ruptured lung enters the pulmonary circulation and travels to the left heart via the pulmonary vein. It causes an 'air lock' in whichever artery it ends up in and symptoms depend on the target organ deprived of blood. In the case of the brain the presentation is similar to that of a cerebrovascular accident and in the heart similar to myocardial infarction.

Decompression Illness

Decompression illness results from the presence of bubbles of gas in the body tissues. At depth gas dissolves in the body tissues but as decompression occurs, the solubility of the gas falls. If the rate of decompression exceeds the capacity of the lungs to remove gas, then bubbles will form in the tissues. The presentation will depend on the location and quantity of bubbles. Some examples are:

- Joints dull ache not usually made worse by movement
 often insidious onset & poorly localized
 no classical signs of inflammation
 shoulder and knee often affected
- Neurological may be multifocal and subtle
 CNS and PNS may be involved
 paraesthesia, anaesthesia, loss of co-ordination
 visual, auditory symptoms
 disorientation, drowsiness, unconsciousness, convulsions
 sphincter control may be impaired
- Audiovestibular vertigo, tinnitus, nystagmus, loss of hearing, tinnitus

Decompression illness may manifest from 10 minutes to 2 weeks after exposure. More severe illness usually presents earlier. All persons suspected of having decompression illness should be given 100% oxygen and referred for recompression therapy without delay.

Dysbaric Osteonecrosis

This condition is an aseptic necrosis of bone which is seen in some persons working at raised barometric pressure. It is more common in compressed air workers than in divers. It is very rare in those divers who do not work at below 50 metres of seawater. The underlying pathology is unclear. Lesions may be in the subchondral region or in the shaft.

Subchondral (or juxta-articular) lesions may progress to articular collapse and if present in the head of femur or of humerus are sufficient to justify removal from the compressed air or diving environment. Shaft lesions are usually relatively innocuous, but occasionally are the site of malignant change. Symptomatic presentations are rare as it is usual practice that significantly exposed workers undergo regular radiological examination.

A4 Cramp of the Hand or Forearm due to Repetitive Movements

This condition has been known by a variety of names such as writer's cramp and telegraphist's cramp and involves episodes of spasm accompanied by tremor and pain in the affected hand or forearm in workers with an occupation entailing prolonged periods of handwriting, typing or other repetitive movements of the fingers, hand or arm.

Clinical Features

The pathology is unknown and the condition may be diagnosed by complaints of painful spasm triggered by repetitive movements. The condition takes some time to develop and there may be no physical findings on examination. Other organic causes of similar symptoms must be excluded. The prognosis is variable; some cases recover with rest but others have recurrences on return to their occupation.

A5 Subcutaneous Cellulitis of The Hand (Beat Hand)

This disorder may be seen in workers engaged in manual labour involving severe or prolonged friction or pressure on the hand. It involves bruising of the subcutaneous tissues by work involving frequent jarring of the hand with subsequent traumatic implantation of foreign material. This may subsequently become infected. Softer skin, as seen in persons unaccustomed or returning after absence to such labour is more vulnerable, as is skin subject to maceration by wet conditions.

Clinical Features

The signs and symptoms are of acute inflammation, most commonly of the palm and palmar aspects of the fingers and thumb. There may be oedema of the skin. If infection is present it may spread to affect neighbouring bursae or tendon sheaths. The infection is treated in the standard manner for acute cellulitis and the prognosis is variable.

A6 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)

This condition may be seen in workers who spend considerable amounts of time in a kneeling position. Examples include coal miners, roofers and carpet fitters.

Clinical Features

Bursitis may begin with chronic inflammation of the prepatellar bursa with haemorrhage. Infection may supervene. Subcutaneous cellulitis is due to chronic trauma followed by infection of the subcutaneous tissues. Classical signs of acute inflammation are seen. Softer skin, as seen in persons unaccustomed or returning after absence to such labour is more vulnerable, as is skin subject to maceration by wet conditions.

Treatment is as standard for acute cellulitis.

A7 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)

This condition has similar aetiology to that of 'beat hand' and 'beat knee'.

Clinical Features

Inflammation begins in the area of the olecranon. Classical signs of inflammation are present and cellulitis may spread to the upper arm or forearm. Movement of the elbow joint is painful.

Treatment is as standard for acute cellulitis.

A8 Traumatic Inflammation of the Tendons of the Hand or Forearm or of the Associated Tendon Sheaths

Several distinct conditions are included in the above. All are associated with repetitive movements of the hand and wrist. There may occur in various occupations fulfilling the above criteria, but in recent times have been particularly associated with extensive keyboard use.

1. De Quervain's Stenosing Tenovaginitis (de Quervain's disease)

In this condition there is thickening and stenosis of the fibrous tendon sheath around abductor pollicis longus and extensor pollicis brevis. Symptoms and signs include visible swelling, snuffbox tenderness, and crepitus on thumb movement. Pain on ulnar deviation of the clenched fist with the thumb folded into the palm may be present (Finkelstein test). The condition may resolve with rest, but local steroid injection or surgical decompression of the sheath may be required in some cases.

2. Stenosing Tenovaginitis of flexor tendons (trigger finger)

In this condition thickening and constriction of the mouth of a fibrous digital sheath interferes with the free gliding of the contained flexor tendon. There is tenderness at the base of the affected finger and locking of the finger in full flexion. A nodule can be felt at the base of the affected finger or thumb and the finger or thumb clicks when actively moved.

3. Tenosynovitis

This is a disorder of the tenosynovium and affects the long extensors and less commonly the long flexors of the fingers at the wrist. Presentation is with pain, particularly on resisted movement, localised swelling, tenderness and crepitus. The lesion is localised to the synovial sheaths and the wrist. Most cases respond to rest and non-steroidal anti-inflammatory drugs, but some require local steroid injection.

4. Peritendonitis Crepitans

This condition has a similar presentation to tenosynovitis but the site of the lesion is the musculotendinous junction above the limit of the tendon sheaths in the forearm. Most cases respond to rest and ultrasound.

5. Lateral Epicondylitis (Tennis Elbow)

In this condition the patient complains of pain in the elbow when using the hand and contracting the extensors of the wrist and fingers. There may be pain at rest. Tenderness is found at the common attachment of the extensor muscles of the forearm to the lateral epicondyle of the humerus. Resisted active contraction of the extensor muscles of the fingers or wrist provokes pain at the elbow. Significant morbid anatomical features have not always been demonstrated, but it may be that tears may occur in the collagen fibres attaching the muscle to the bone which do not heal due to repeated loading. Many treatments have been attempted but success has been elusive. These include NSAIDs, elbow bands, avoidance of contraction under load, physiotherapy, local steroid and anaesthetic injections and surgical release.

6. Medial Epicondylitis (Golfer's Elbow)

This is a similar condition to tennis elbow, except that it affects the common attachment of the flexor muscles of the forearm to the medial epicondyle of the humerus. Tenderness is often less well localised than in tennis elbow and the results of treatment are often less satisfactory.

A9 Carpal Tunnel Syndrome

The carpal tunnel is formed by the flexor retinaculum anteriorly and the distal row of the carpus posteriorly. The median nerve and the nine flexor tendons pass through it. The syndrome is that of median nerve compression and there are a wide variety of causes of which the following are examples: rheumatoid arthritis, gout, fractures, osteophytes, ganglia, acromegaly, amyloidosis, myxoedema and pregnancy. The occupational causes under this category are limited to the **use of vibrating hand tools**. Carpal tunnel syndrome arising as a complication of tenosynovitis for workers employed in occupations which involve repeated movements of the wrist and fingers are usually considered as a complication of '*Traumatic Inflammation of the Tendons of the Hand or Forearm or of the Associated Tendon Sheaths*' for the sake of compensation.

Clinical Features

The classical presentation is of pain or paraesthesia in the distribution of the median nerve (first three digits and the lateral half of the fourth), but the localisation may be poor and affect any part of the arm. The pain is often worse at night and may be relieved by the patient shaking his wrist or hanging his hand over the edge of the bed. If untreated it may increase in severity developing more frank neurological symptoms such as loss of co-ordination, numbness and weakness of the muscles innervated by the median nerve such as abductor pollicis brevis and opponens pollicis. In the late stages there may be obvious thenar muscle wasting.

Increased paraesthesia produced by percussion of the nerve at the carpal tunnel (Tinel's sign) or 1 minute of maximal flexion of the wrist (Phalen's sign) are present in around two thirds of cases.

Laboratory Diagnosis

Nerve conduction studies are useful in the diagnosis of carpal tunnel syndrome. The earliest sign is a slowing of sensory nerve conduction velocities in the median nerve at the carpal tunnel. Conduction in the ulnar nerve can be used as a control and can aid in differential diagnosis. Nerve conduction is also useful in the assessment of response to treatment.

Treatment

Mild occupational cases will respond to suitable modifications of work practices in combination with the use of wrist splints. Non-steroidal anti-inflammatory drugs are of no proven benefit in the long term. More severe cases may need local injection of corticosteroids but this should not be performed more than three times due to the risk of delayed tendon rupture. The definitive treatment is surgical division of the flexor retinaculum which may be done by open or endoscopic means.

PART II

DISEASES CAUSED BY

BIOLOGICAL AGENTS

B1 Anthrax

Anthrax is a potentially life threatening but rare bacterial infection affecting humans and certain types of animals and can pose a threat to workers who process hides, animal hair, bone products and wool, and to veterinarians, agricultural workers, slaughtermen and butchers handling infected animals, most commonly cattle but also goats, horses, sheep and pigs. The organism concerned is *bacillus anthracis* which can survive in the form of spores for considerable periods in soil or animal remains and thus can also endanger persons working on potentially infected sites such as those of old tanneries or pasture lands. Spores of *B. anthracis* gain entry via skin abrasions, by inhalation or by ingestion of infected of infected meat (uncommon in developed countries).

Cutaneous Anthrax

After incubation period of 2 to 7 days pruritis develops at the site of the inoculation followed by the appearance of a papule. This vesiculates, leaving a black, necrotic centre. Smaller vesicles and oedema may surround the lesion and lymphangitis may also be present. Constitutional symptoms may be slight or absent, but in some severe cases an over whelming septicaemia may ensue.

Pulmonary Anthrax

This usually begins with non-specific symptoms of upper respiratory tract infection followed by increasing dyspnoea, pyrexia, respiratory failure and septic shock within 3 to 5 days. If untreated death results in a few days, clinical signs may include pulmonary oedema and congestion with frothy blood stained sputum.

Gastrointestinal Anthrax

Severe gastroenteritis is followed by prostration, coma and death unless treated appropriately.

Diagnosis

This is by history of contact with infected animals or materials and by the clinical appearance. Laboratory confirmation is by demonstration of the organism in blood, sputum or swabs of lesions. Fluorescent antibody techniques permit rapid identification of the organism.

Treatment

Intravenous penicillin is the treatment of choice. Doxycycline or ciprofloxacin are alternatives in penicillin allergic patients. Appropriate supportive therapy may be required. Vaccination for workers at high risk is recommended.

B2 Glanders

Glanders is a very rare infectious disease caused by *Malleomyces mallei* (formerly known as *Pfeiferella mallei*). It is primarily a disease of equine species and can be transmitted to humans working with infected animals. Occupations include cavalry soldiers, farmers, veterinary surgeons, horse trainers, grooms, ostlers, blacksmiths, jockeys and knackermen. The disease has become extremely rare since the replacement of the horse by motor vehicles as the main means of transport and the limitation to ceremonial duty of horses in cavalry regiments. It is now almost unknown in horses in developed countries. The organism is present in the nasal and other secretions of infected animals and gains entry via inhalation, ingestion, intact mucous membranes or skin abrasions. Acute and chronic forms of the disease are known.

Acute Glanders

This disease is characterised by an acute onset of pyrexia, generalized pain and shivering, followed by ulceration of the nasal mucosa with a fetid blood stained discharge which rapidly progresses to necrosis of the nasal septum and ulceration of the pharynx, larynx and mouth. The skin of the nose and face becomes red and the lymph glands swollen. A pustular eruption may extend over the face and limbs. High pyrexia, rigors, vomiting and diarrhoea with a rapid feeble pulse and dry brown tongue can progress to delirium, coma and death. Nodules known as 'farcy buds' may form along the lymphatics and become pustular. They can rupture and form deep ulcers. In these cases the disease is known as farcy. Most cases of acute glanders are fatal within 10 days but occasionally mild cases recover in about 3 weeks.

Chronic Glanders

Both chronic glanders and chronic farcy are much less common than the acute forms, the lesions being less severe due to the relative inactivity of the organism. It is a long and extremely painful illness with acute exacerbations occurring over many years. Lesions may remain localised but more commonly they involve many parts of the body forming deep ulcers of the nose and many subcutaneous abscesses. Drainage of the abscesses leaves sinuses which heal slowly and are intensely painful. Many patients die from secondary infection.

Diagnosis

Diagnosis of human glanders is difficult but a history of contact with horses may suggest the diagnosis. In acute cases the symptoms may suggest pneumonia or typhoid fever. The organism can be demonstrated in lesions. Syphilis and tuberculosis must be excluded.

B3 Infection by *Leptospira*

All leptospira are now classified into one species, *Leptospira interrogans*, containing over 100 serotypes, the most important of which to humans are *Li. hardjo* (cattle), *Li. icterohaemorrhagiae* (rats) and *Li. canicola* (dogs).

Li. hardjo is usually acquired by humans from the urine of cattle. All persons exposed to contact with cattle are potentially at risk, including farmers, dairy workers, veterinary surgeons and abattoir workers. Infected cattle may be asymptomatic.

Li. icterohaemorrhagiae is usually acquired from water which has been contaminated with rat urine. The risks are highest in areas which have a high population of rats and after rain following a dry period has flushed dried urine out of rat runs. Sewage and garbage disposal workers, farmers, fish farmers, dockworkers, inshore divers and water sports enthusiasts are among those at risk.

Li. canicola may affect persons coming into contact with dog urine such as kennel workers or veterinary surgeons. The organism may also infect cattle.

Clinical Features

Li. hardjo usually presents as a flu-like illness of short duration but headache is a prominent symptom and may persist for several weeks as may lethargy and malaise. Very rarely there may be meningitis and even more uncommonly hepatic or renal failure which may cause death.

Li. icterohaemorrhagiae can cause a rapidly progressive multi-system disease (Weil's disease) which starts as a feverish illness with pyrexia, headache, myalgia and vomiting. Jaundice may be present but often does not occur in the early stages. Haemorrhages may occur in the conjunctiva or elsewhere. Patients with severe illness may present with septicaemic shock or in a toxic state with evidence of meningitis and hepatitis. Renal failure or pneumonia may follow. The mortality rate is around 20% but those who survive the acute stage usually make a complete recovery.

Treatment

Severely ill patients should be treated with parenteral penicillin G. Milder infections may be treated with oral amoxicillin. Treatment should not be delayed to await laboratory results.

Laboratory Diagnosis

Blood culture may be positive in severe cases during the first week and urine culture from the first to sixth week. The earliest serological response, IgM, takes 5 to 6 days to reach a level that is diagnostic by the ELISA method.

B4 Extrinsic Allergic Alveolitis

Extrinsic Allergic Alveolitis (EAA) is sometimes known as hypersensitivity pneumonitis and is a granulomatous inflammatory reaction caused by a specific inflammatory response to various inhaled organic dusts.

Organic dusts which cause EAA may be microbial spores which grow in vegetable matter or animal proteins, both avian and mammalian. The particles are sufficiently small to allow penetration into and retention within the alveoli and are poorly degradable and therefore able to persist in the lungs for long periods. The disease usually occurs amongst those exposed to very high doses of inhaled particles. Examples of EAA include Farmer's Lung (the most prevalent occupational EAA, caused by exposure to the spores of thermophilic actinomycetes which grow when hay, straw or grain are harvested damp and stored without drying or in enclosed or poorly ventilated buildings), Bird Fancier's Lung, Mushroom Worker's Lung, Bagassosis and Malt Worker's Lung. The clinical features of all types are alike and depend on the pattern of exposure and the severity and nature of the individual response.

Clinical Features

Two distinct and overlapping forms are recognised, acute and potentially reversible and chronic irreversible.

Acute EAA follows exposure to antigen in high concentrations. The symptoms do not occur during the period of exposure, but several hours later when breathlessness, fever, malaise, myalgia and headaches develop. In the absence of further exposure the symptoms may start to resolve within 48 hours but may persist for around a week. If exposure is continuous, the symptoms become more severe and may include significant weight loss.

Chest auscultation may be normal but in some cases scattered inspiratory crackles or squeaks may be heard. Chest radiographs may be normal in some cases but in others may show a variety of abnormalities including a 'ground glass' pattern, micronodular or nodular shadowing (particularly of lower zones) or patchy consolidation from merger of nodules. Changes may take 4 to 6 weeks to resolve.

On lung function testing, total lung capacity (TLC), residual volume (RV), vital capacity (VC) and forced expiratory volume in 1 second (FEV₁) are reduced but the FEV₁/FVC ratio is maintained or increased. Transfer factor for carbon monoxide (Tl_{co}) and gas transfer coefficient (K_{co}) are both reduced. In severe cases arterial PO₂ may be sufficiently reduced for patients to be clinically cyanosed. The blood neutrophil count and ESR are usually increased. Lung function usually improves for about 4 to 6 weeks after avoidance of exposure but may continue to do so for up to 6 months.

Chronic EAA is distinguished by the development of irreversible pulmonary fibrosis. It may arise as a result of repeated episodes of acute EAA or as a result of chronic exposure to antigen at levels insufficient to provoke acute symptoms but large enough to cause progressive pulmonary damage (typical presentation for bird fancier's lung). The main symptom is breathlessness on exertion.

Finger clubbing is unusual and scattered inspiratory crackles and squeaks may be audible on auscultation. There may be considerable weight loss. Chest radiography shows linear shadows, honeycombing, and lung shrinkage, especially in the upper lobes, with compensatory dilatation in the lower lobes.

Lung function changes are similar to those of acute EAA but little improvement can be expected on avoidance of further exposure to antigen. Pulmonary hypertension may develop in those patients with widespread pulmonary fibrosis and some may present at this stage.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Diagnosis

This depends on:

1. Identification of a source or potential source of antigen.
2. Clinical, radiological and functional changes.
3. Demonstration of precipitating antibodies (precipitins) to the causal antigen in the patient's serum.

Treatment

Episodes of acute EAA should be treated by removal from source of antigen and high dose oral prednisolone until there has been maximum improvement in lung function and resolution of CXR changes. This may take up to 6 months. Progressive fibrosing disease should be treated by avoidance of exposure to source of antigen, regular oral steroids, supplemented by immunosuppressants such as cyclophosphamide or azathioprine if required.

B5 Infection by Organisms of the Genus *Brucella*

Brucellosis is a potentially severe bacterial disease caused by organisms of the genus *Brucella*. *B. abortus* causes abortion and infertility in cattle and can be contracted from contact with tissue, blood, urine, vaginal discharges, aborted foetuses and placentae of infected animals. Farmers, dairy workers, veterinary surgeons, slaughterhouse workers and butchers are at potential risk. *B. melitensis* affects goats, *B. ovis* affects sheep, *B. suis* affects pigs, and *B. canis* affects dogs. Routes of entry include ingestion, aerosol contamination of the lips, nose and eyes, inhalation of aerosols or through broken skin. Transmission may also occur by eating contaminated meat or dairy products.

Clinical Features

The symptoms of brucellosis are variable and non-specific and the incubation period may be anything from one week to seven months. The most frequent symptoms are fever, often with rigors, generalized aches and pains, headaches, anorexia and fatigue. Classically the fever is said to be intermittent or undulating but this pattern is not always present. The acute attack usually subsides in 2 to 3 weeks with or without treatment. Chronic disease is often associated with intense fatigue.

No single physical sign is invariably present. Lymphadenopathy, splenomegaly and hepatomegaly are often observed, as is arthralgia, particularly of the spine and knees. Patients often present with low back pain and systemic complaints such as fever, loss of appetite, apathy and depression. Radiographs may show bone destruction after about 3 months but radioisotope scans may reveal abnormalities at an earlier stage. *Brucella* meningitis often has a gradual onset with the patient having been unwell with headache and apathy for several weeks.

Laboratory Diagnosis

Blood and tissue cultures are diagnostic but may take up to 6 weeks to become positive. Laboratories should be forewarned of the possible diagnosis to avoid the material being discarded prematurely. Serology is helpful in many cases, but persons with occupations such as farmers, veterinary surgeons and slaughterhouse workers may have a high titre of brucella agglutinins without symptoms of disease. In cases of equivocal serology, liver biopsy which shows non-caseating granulomata may provide additional support for a diagnosis of chronic brucellosis.

Treatment

The World Health Organisation currently considers that the treatment of choice is doxycycline 200 mg/day and rifampicin 600 mg/day for 6 weeks.

B6 Tuberculosis in Health Care Workers

This is a prescribed disease in workers whose occupation involves close and frequent contact with a source of *Mycobacterium tuberculosis* infection, e.g. health care workers, research workers, laboratory workers, pathologists and post-mortem workers. Infection may result in disease within a few months of acquisition or it may lie dormant for many years if the initial infection was contained by the activation of cellular immunity. Uncommonly the disease may become disseminated.

Clinical Features and Laboratory Diagnosis

This disease should be suspected in employees at risk, who present with low grade fever, night sweats, fatigue, anorexia, weight loss, and a persistent productive (purulent or blood stained) or non-productive cough. If the chest X-ray shows a typical apical cavitating lesion, the patient is likely to produce infected sputum which can be shown to contain *Mycobacterium tuberculosis* on microscopic examination or culture. Tuberculin testing is of little value as employees are likely to have been immunised with BCG.

B7 Parenterally Contracted Viral Hepatitis in Health Care Workers

The hepatitis B virus (HBV) is of great importance to health care workers and those who handle human blood or blood products as it is comparatively readily transmitted during surgical procedures and through needlestick injuries.

Clinical Features

The incubation period lasts usually for about a month but can be up to 5 months and is followed by a preicteric or prodromal phase with malaise, nausea, vomiting, diarrhoea, anorexia and headaches. Fever is usually more severe than that of hepatitis A and there may be upper abdominal discomfort. There are often few physical signs – the liver may be tender but is not enlarged initially.

In about 25% of patients an immunological syndrome may be seen, consisting of urticaria or a maculopapular rash and polyarthritis affecting the small joints. Arteritis and glomerulonephritis are sometimes seen.

After about 2 months the patient becomes icteric (though some never do) and symptoms often improve with decreased malaise and improved appetite. The liver is moderately enlarged and the spleen is palpable in about 10% of patients. The stools are pale and the urine dark.

The majority of patients recover completely but fulminant hepatitis leading to death occurs in about 1%. Some patients develop chronic hepatitis which may lead to cirrhosis or hepatocellular carcinoma and others become asymptomatic carriers.

Laboratory Findings

HBsAg appears in the blood from about 6 weeks to 3 months after an acute infection and then disappears. HBeAg rises early and then declines rapidly. Anti-HBc is the first antibody to appear and high titres of IgM anti-HBc suggest an acute and continuing viral replication. It persists for many months and may be the only serological indicator of recent HBV infection when HBsAg has disappeared and anti-HBs (which appears late and indicates immunity) is not detectable.

Persistence of HBsAg indicates chronic infection or a carrier state. HBeAg persists and correlates with the development of chronic liver disease. When anti-HBe develops (seroconversion), the HBeAg disappears and there is usually a rise in ALT. HBV DNA suggests continual viral replication.

Treatment

There is no specific treatment apart from symptomatic therapy and it is strongly recommended that all employees at risk be vaccinated.

B8 *Streptococcus suis* Infection

Streptococcus suis is a group R streptococcus which causes septicaemia in pigs. Occupations most at risk are those which involve handling pigs or meat from pigs, such as pig farmers, butchers, cooks, meat processors, meat inspectors and slaughterhouse workers. The main route of entry of the organism is through abraded or cut skin though the respiratory route may be more common in pig farmers.

Clinical Features

The disease usually presents as typical acute pyogenic meningitis with a rapid onset of severe headache, neck stiffness, vomiting and pyrexia, typically preceded by up to a week of cough, flu-like symptoms and anorexia. The condition may be fatal if not treated promptly.

Treatment

Large doses of intravenous penicillin G usually give a good response. Vancomycin is a suitable alternative in patients allergic to penicillin. Some patients have suffered permanent hearing loss and vertigo after recovery. The most effective means of prevention is strict personal hygiene. Hands should be frequently washed with hot water and soap. Skin inoculation can be prevented by wearing thick rubber gloves. In case of wounds sustained while handling pork, prompt first aid treatment with antiseptics is recommended.

B9 Avian Chlamydiosis

Chlamydiae are Gram-negative bacteria which lack their own respiratory system and are therefore obligate intracellular parasites. The infective form is almost metabolically inert and may survive in dust and dried urine and faeces for a long time. *C. psittaci* strains are found among many mammals and birds. Humans are not generally susceptible to infection by mammalian strains with the notable exception of the ovine abortion agent. Infection of pregnant ewes causes abortion and premature delivery and pregnant women coming into contact with infected animals or products of conception may suffer a severe and sometimes life-threatening septicaemic illness with fever, vomiting, hypotension, headache, foetal death and abortion.

Avian disease is widespread in caged, wild and exotic birds and in poultry, particularly ducks and turkeys. Sources of infection are faeces, nasal discharge, and contaminated feathers. Particular infectivity is associated with aerosols of faeces such as are produced during poultry evisceration.

Occupations at risk include pet shop or aviary workers, taxidermists, demolition or construction workers involved in roof demolition or repair, poultry farmers, poultry process workers, veterinary surgeons, meat inspectors and feather and down processors.

Clinical Features

The incubation period is usually from 4 to 15 days and the onset may be sudden or insidious with chills, fever, malaise, headache, sore throat, myalgia and arthralgia. Cough productive of mucoid and occasionally blood stained sputum is often a feature. Pneumonia is often a complication but endocarditis and hepatitis are much less common. Crackles are usually present on auscultation of the chest but unequivocal signs of consolidation are unusual. Chest radiography typically shows interstitial pneumonitis with patchy or streaky shadowing. Death may occur in older workers, but most appropriately treated cases proceed to complete recovery.

Laboratory Diagnosis

The diagnosis may be confirmed by serological testing, but treatment should not be delayed till the results are available.

Treatment

Tetracycline 500 mg four times daily for 2 weeks is the treatment of choice. Erythromycin 500 mg three times daily for 3 weeks is also effective. A second course may be required in the event of relapse.

B10 Legionnaires' Disease

This disease is named after an outbreak of pneumonia which resulted in the deaths of 29 delegates at a conference held in a Philadelphia Hotel in 1976. The causative organism is *Legionella pneumophila* which is one of a group of bacteria which are widely found in nature in wet surroundings. *Legionella pneumophila* was not recognised as an important cause of lobar pneumonia for a long time due to the unknown special requirements to culture it, but it has been found in retrospective analyses from stored serum samples from pneumonia cases from the 1940's.

Legionella pneumophila prefers a growing temperature of about 35°C and has often been known to colonise cooling towers and hot water systems. The bacteria may be drawn into air conditioning systems by intakes which are near cooling towers from which they are discharged as aerosols or may be distributed as aerosols from hot water systems via shower heads. Colonisation of whirlpool spa baths or jacuzzis has been known. Cases may be sporadic or epidemic. Occupational cases derive from persons working on fresh water cooling systems or hot water service systems.

Clinical Features & Laboratory Diagnosis

Legionnaires' disease manifests as a lobar pneumonia which is difficult to distinguish from more common pneumonias such as those caused by pneumococci, mycoplasmae or rickettsiae. It often begins as a flu like illness with fever, malaise and myalgia and proceeds to cough (with little sputum), dyspnoea, and sometimes severe anorexia, marked confusion and coma. Diarrhoea and vomiting are common and renal failure may develop. Compared to many other forms of pneumonia, the patients with Legionnaires' disease may be more confused and hyponatraemic and usually have a worse prognosis. The diagnosis can be facilitated by the presence of antigen in lung secretions, sputum culture on special media or a fourfold rise in antibody titre to *Legionella pneumophila*.

Legionella pneumophila can also cause a less serious illness known as Pontiac fever. It normally manifests as a flu like illness, usually of short duration, but some of those with high titres of antibody to *Legionella pneumophila* reported persistence of lethargy, shortness of breath and difficulty with concentration for several years. It has been postulated that Pontiac fever is caused by the soluble antigen rather than the viable whole organism leading to Legionnaires' disease.

Treatment

The antibiotic of choice for the treatment of Legionnaires' disease is erythromycin with the addition of rifampicin in severe cases. Tetracycline may also be used. *Legionella pneumophila* is not affected by cephalosporins or penicillin. The mortality of the condition is about 20%, the majority of deaths being among the elderly or heavy smokers.

Prevention

Meticulous cleaning and maintenance of cooling towers, hot water systems, spas and air conditioning plants is required. Hot water storage should be at above 60°C, at which temperature *Legionella pneumophila* is destroyed.

B11 Severe Acute Respiratory Syndrome

Severe acute respiratory syndrome (SARS) is a viral respiratory infection caused by a coronavirus, called SARS-associated coronavirus (SARS-CoV). It first appeared in Southern China in November 2002. During November 2002 through July 2003, a total of 8,098 people worldwide became sick with SARS that was accompanied by either pneumonia or respiratory distress syndrome and 774 of them died.

SARS is predominantly transmitted through close person-to-person contact, especially via respiratory droplets produced when an infected person coughs or sneezes. Droplets spread can take place when droplets from the coughs or sneezes of an infected person are propelled a short distance (generally up to 3 feet) through air and deposited on the mucous membranes of the mouth, nose, or eyes of persons who are nearby. The virus can also spread when a person touches a surface or object contaminated with infectious droplets and then touches his or her mouth, nose, or eyes. Occupations at risk include health care workers, research workers, laboratory workers, pathologists, post-mortem workers and funeral services workers.

Clinical Features & Laboratory Diagnosis

Patients with SARS usually begin with a high fever ($>38.0^{\circ}\text{C}$), which is sometimes associated with chills, headache, malaise, muscle pain or even diarrhoea. At the onset of illness, some patients may also experience mild respiratory symptoms. At 2 to 7 days, the patients may develop a dry, non-productive cough that may be accompanied by or progress to hypoxia. In 10-20% of cases, patients may require mechanical ventilation. Most patients develop pneumonia. Symptoms can be more variable among elderly patients.

Several laboratory tests can be used to detect SARS-CoV. A reverse transcription polymerase chain reaction (RT-PCR) test can detect SARS-CoV in clinical specimens such as blood, stool and nasal secretions. Serological testing also can be performed for SARS-CoV antibodies which can be detected from around 10 days after onset of the illness. Virus cultures are not routinely used for patients suspected of having SARS and it must be conducted under Biosafety Level (BSL) 3+ laboratory conditions.

Treatment

The SARS-CoV is being tested against various antiviral drugs to see if an effective treatment can be found. There is no vaccine currently available.

B12 Avian Influenza A

Avian influenza A viruses usually infect birds. The highly pathogenic H5N1 strain was first isolated from birds (terns) in South Africa in 1961. Like all avian influenza A viruses, H5N1 virus circulates among birds worldwide, is very contagious among birds, and can be deadly. It is known previously to infect birds only, but 18 and 2 human cases were documented in Hong Kong in 1997 and 2003 respectively. Since late 2003, outbreaks of Avian Flu in chickens and ducks have been reported in several Asian countries and cases of human infection have also been reported. The infected poultry or birds shed the virus in their saliva, nasal secretions and feces. Most cases of H5N1 infection in humans have resulted from contact with infected poultry or contaminated surfaces. Transmission between humans is very inefficient. Apart from H5N1, human infection with H9N2 and H7N7 strains have also been reported. Nevertheless, the disease caused by these strains is usually less severe than H5N1 infection.

Occupations at risk are those who involve handling of infected poultry or birds or their untreated products, such as pet shop workers, poultry farmers, poultry handlers and veterinary surgeons. Research and laboratory workers may also contract the disease from handling of the infective materials at work.

Clinical Features and Laboratory Diagnosis

The initial clinical presentation of H5N1 infection is similar to that of other influenza infection, typically with high fever, malaise, myalgia, sore throat and cough. Gastrointestinal symptoms such as vomiting, diarrhoea or abdominal pain are not uncommon. The infection may run a rapidly downhill course complicated by viral pneumonia, respiratory distress syndrome and multiple organ failure. Severely ill patients often show deranged liver function and sometimes impaired renal function. The clotting times may also be prolonged. Myocarditis or encephalitis is not a recognized feature of the disease.

Laboratory diagnosis depends upon the demonstration of the virus and/or a rising antibody titre. Virus culture, reverse transcription polymerase chain reaction (RT-PCR), immunofluorescence using monoclonal antibody and serological tests are available. Several commercial rapid diagnostic tests are also available but their sensitivity may not always be optimal. For virus culture, it should be conducted under Biosafety Level (BSL) 3+ laboratory conditions.

Treatment

The H5N1 virus that has caused human illness and death and is currently infecting birds in Asia is resistant to amantadine and rimantadine, that are commonly used for treating influenza. Thus, neuraminidase inhibitors e.g. oseltamivir and zanamivir are the drugs of choice in the treatment of H5N1 infection. Currently, there is no vaccine to protect humans against the H5N1 virus.

PART III

DISEASES CAUSED BY

CHEMICAL AGENTS

C1 Poisoning by Lead or a Compound of Lead

Although lead poisoning is uncommon in developed countries, worldwide it remains the most common of occupational poisonings. Workers in many industries may be exposed to lead. A few examples are:

- Primary Smelting – extraction of lead from its ore
- Secondary Smelting – recycling of scrap lead (mainly from car batteries etc.)
- Manufacture of batteries, paints, colours, rubber products, glass, lead compounds etc.
- Application of lead paints and glazes
- Demolition work involving oxy-acetylene cutting of lead painted metal
- Firearms instructors
- Production of leaded petrol and cleaning of its storage tanks

The rate of absorption of any particular lead compound is related to its solubility in body fluids. The primary route of entry is by inhalation but poor personal hygiene may lead to ingestion. Some organic lead compounds such as tetra ethyl lead are absorbed through the skin. Lead in the blood is mostly bound to haemoglobin, but this represents only about 2% of the total body burden as most is in the skeleton. Blood lead has a biological half-life of about 30 days and is the most commonly used index of lead exposure but only reflects recent uptake. Urinary lead excretion may be used to monitor organic lead exposure.

Inorganic Lead Poisoning

Patients may remain asymptomatic until the body burden of lead is sufficient to cause sudden onset and rapid progression of symptoms. Disruption of haem synthesis leads to a reduction in synthesis of haemoglobin and oxidative enzymes. Depression of aminolaevulinic acid dehydratase (ALAD) and ferrochelatase results in an increased amount of ALA which is excreted in the urine and also leads to protoporphyrin forming a metal compound with zinc instead of iron, leading to increased ZPP. ALA may competitively inhibit GABA in the brain.

The classical presentation is of abdominal pain, colic and constipation, but this is now rare. It is more common for the patient to present with non-specific symptoms of fatigue, lassitude, arthralgia, myalgia, and abdominal discomfort. The patient may have pallor and there may be some muscle weakness if there is a peripheral neuropathy. The classical sign of wrist drop is now rare as are encephalopathy and renal damage in adults. A blue line may be present on the dental margin of the gums and there may be muscle, joint and abdominal tenderness.

Red blood cells may show stippling due to basophilic granules. Blood lead concentration in the normal population is around 10 to 15 µg/dl (0.5 to 0.75 µmol/l). Male workers are normally suspended from work if the blood level is above 50µg/dl (30 µg/dl for females under 50 years of age) in 2 successive examinations and the Labour Department should be notified for further investigations. The levels of urinary ALA and blood ZPP will increase, but there is a variable lag period.

The basis of treatment is the removal from further occupational exposure and in most cases this is all that is required. The blood level falls gradually and periodic monitoring is sufficient. More severe cases may require chelation therapy, often with penicillamine or sodium calcium edetate (both require renal monitoring) or with dimercaptopropane sulphonate (DMPS) or dimercaptosuccinic acid (DMSA).

Organic Lead Poisoning

The most common form of organic lead poisoning is that by tetraethyl lead which is used as an anti-detonant in leaded petrol. It is highly volatile and may be absorbed by inhalation or by passage through intact skin. In severe cases there may be a brief period of general malaise, followed by acute onset of a toxic organic psychosis with insomnia, restlessness, excitement, delusions, hallucinations or mania. Muscular twitching, hypotension, bradycardia and hypothermia may be present. In some cases this may lead to death. Milder cases may display fatigue, lassitude, weakness, insomnia, headache, vertigo and excitement. There is nothing in the clinical picture that is pathognomonic and the diagnosis is made by establishing exposure and may be confirmed by a finding of raised urinary lead. Workers with urinary lead above the level of $150\mu\text{g/l}$ in 2 successive examinations should be suspended from further occupational exposure and the case should be notified to the Labour Department for further investigations. Blood lead is usually not significantly raised and ZPP may also be normal. Red cells do not show basophilic stippling. Symptomatic treatment (e.g. sedation) may be required. Treatment with penicillamine increases the urinary output of lead, but does not appear to speed recovery in cases of poisoning. Most patients recover completely, but some may have psychotic lapses.

C2 Manganese Poisoning

Manganese is a very hard white metal which is mainly used in the production of toughened steels. Compounds of manganese are also used in dry cell batteries, paints, varnishes, inks, matches, fireworks, fertilizers and drugs. Permanganate compounds are used as oxidizing agents and as decolourising agents and pigments in the ceramics and glass industries.

It is very poorly absorbed from the gut (more is absorbed in iron deficiency) but can be absorbed via the lungs from manganese fumes. It crosses biological membranes with difficulty and is mainly bound to red cells in the blood. Only small amounts appear in the urine as it is mostly excreted in the bile.

Clinical Features

Many compounds are irritant to skin, eyes and mucous membranes. Inhalation of manganese probably produces an inflammatory reaction in the lungs and this is probably responsible for a high incidence of pneumonia form superimposed bacterial infection in workers handling manganese ores.

The main manifestation of chronic manganese poisoning is the insidious onset of a Parkinson like syndrome. This appears in three stages

1. Workers complain of lassitude, fatigue, anorexia, apathy, headaches, and weakness of the legs, joint pains, muscular cramps and irritability. There are no specific signs.
2. Development of dysarthria. Disturbances of gait, excessive salivation, often accompanied by an organic psychosis ('manganic madness') which usually resolves when true parkinsonian symptoms appear. The disease may be reversible up to this stage.
3. Akinesia and rigidity (most pronounced in the lower limbs), muscle pains, paraesthesia and disturbances of speech. The tremor is frequently an intention tremor rather than a resting tremor as in idiopathic parkinsonism. Some degree of dystonia is often present. This stage denotes permanent damage to the CNS.

Histological Features of the Brain

In true parkinsonism, the substantia nigra is most affected, the striatum and pallidum are little affected although their dopamine concentration is much reduced. The situation is reversed in manganese poisoning.

Treatment

Removed from exposure before appearance of parkinsonian symptoms. (Some authors have reported improvements when EDTA is also administered) Treatment with L-dopa has been successful both when hypokinetic and dystonic features predominate.

Monitoring

Periodic medical examinations should aim to detect early stages of behavioural and neurological changes such as hypotonia and tremor. Workers who recover slowly from respiratory infections should be advised to avoid further exposure. Biological monitoring (blood or urine levels) can be carried out. Patients with urine manganese levels of more than 50µg/l in 2 successive examinations should be recommended for suspension from further exposure. The cases should be notified to the Labour Department for further investigations.

C3 Poisoning by Phosphorus or an Inorganic compound of Phosphorus or the Anti-cholinesterase or Psuedo-Anticholinesterase action of Organic Phosphorus Compounds

Phosphorus is widely distributed in the form of phosphate rock, chlorapatite and fluorapatite. It may be extracted as there allotropes, white (or yellow), red and black. White is the only one normally considered toxic. Phosphorus and its compounds are used in the manufacture of munitions, fireworks, explosive and incendiary devices, safety matches, chemicals, fertilizers, sugar, detergents, pharmaceuticals, animal feed, chlorinated organic compounds, insecticides, rodenticides, eletropolishing, engraving, metal cleaning, water treatment, photography, semiconductors and electroluminescent coatings.

Clinical Features

In former times white phosphorus was used to make matches and led to the condition known as 'phossy jaw' in worker employed as match dippers. Chronic exposure led to thickening of the periostium of the mandible. Tooth extraction caused by poor dental hygiene or the action of phosphorus on the tooth allowed bacterial access, followed by infection, necrosis, osteoporotic sequestra and abscesses. The mortality rate was about 20% (mostly from septicaemia) and many other workers suffered the disfigurement of partial or total removal of the mandible.

Phossy jaw may still occur in persons using white phosphorus in the manufacture of munitions, explosives and napalm. Improved dental hygiene and the use of antibiotics means that full blown cases are no longer seen, though occasional surgery to remove sequestra is required.

White phosphorus causes serious burns on contact with the skin and many inorganic compounds of phosphorus are extremely irritation to the eyes, mucous membranes and upper respiratory tract. The trichloride and pentachloride may be associated with the development of chronic bronchitis.

Poisoning by Phosphine

Phosphine is a colourless flammable gas which can auto-ignite and forms an explosive mixture with air. It is used as an intermediate in the synthesis of organophosphines and organic phosphonium derivatives. It is widely used in pure form as a dopant in the manufacture of semiconductors and is used as a fumigant against insects and rodents in sealed grain stores, grain elevators and cargo holds of grain ships. It is an unwanted by-product in a number of metallurgical reactions and may contaminate acetylene generated from impure calcium carbide.

Initial symptoms include headache, weakness, chest pain, difficulty breathing, cough and fainting. More prolonged exposure may cause nausea, vomiting and diarrhea. Pulmonary oedema may occur within 24 hours. Convulsions, coma and death may result. Although phosphine is not haemolytic, some sources of accidental exposure may also liberate arsine.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Organophosphate Pesticide Poisoning

Organophosphates function as pesticides by inhibition of the enzyme cholinesterase which breaks down acetylcholine, a neurotransmitter. Acetylcholine therefore persists at nerve endings. Inhibition can last a considerable time. Route of entry can be dermal absorption (the most common with pesticide applicators), inhalation and ingestion.

Symptoms are of cholinergic effects and include chest tightness, muscle weakness, vomiting, colic, diarrhoea, sweating, salivation and lacrimation. Signs include hypotension, ataxia, tremors, muscle fasciculation, incoordination, arrhythmias and miosis. Some patients may have dizziness, headaches, anxiety, mental confusion and restlessness. Severe cases can have convulsions, coma, respiratory failure and cardiovascular collapse.

Treatment involves withdrawal from source of contamination and first aid measures. Treatment for acute cases should be 2-4mg atropine by slow i.v. infusion and 1-2 g pralidoxime (or 250 mg obidoxime chloride) 6 hourly. Larger doses of atropine may be required in severe cases. Diazepam may be added to control fasciculations or convulsions.

Health surveillance should include determination of plasma or red cell cholinesterase before exposure and periodically during exposure. A reduction of 30% or more is regarded as indicative of overexposure and should be suspended from work with organophosphate. Depression of plasma cholinesterase may last a few weeks but that of red cells may persist for months.

C4 Poisoning by Arsenic or a Compound of Arsenic

Arsenic occurs as an impurity in the ores of copper, lead and zinc and is used in industry in both organic and inorganic forms and as the gas arsine which is used in the electronics industry. Most arsenic is presently used in pesticides and herbicides but exposure may occur in smelting and in the chemical and pharmaceutical industries. Most exposure is to the inorganic compounds of which the trivalent are generally more toxic than the pentavalent. The use of arsenical pigments such as Paris green and Scheel's green is now uncommon.

Arsenic can be absorbed by inhalation, ingestion or skin absorption. It is present as a normal constituent of certain foods such as fish and shellfish.

Arsenic has a half-life in blood of about 60 hours and is rapidly eliminated via the kidney. About 75% of a dose of inorganic arsenic is detoxified by methylation to monomethyl arsonic acid or dimethyl arsinic acid.

Acute Poisoning

Acute poisoning usually follows ingestion. The presenting symptoms are abdominal pain, profound vomiting, rice water stools, dehydration and shock. Stupor, convulsions, coma and death may follow.

Chronic Poisoning

This form of arsenic poisoning is more common and can have multi-system effects. Arsenic dust may cause a variety of reactions in the skin, particularly in skin creases or in moist areas such as axillae or genitalia. These include eczematous or follicular dermatitis, ulcerations, hyperkeratosis of palms and soles and hyperpigmentation. Certain types of skin cancer have been associated with arsenic exposure — basal cell carcinoma, squamous cell carcinoma and Bowen's disease.

Painless ulceration and perforation of the nasal cartilaginous septum due to inflammatory and erosive actions is a characteristic respiratory tract lesion.

Arsenic neuropathy usually presents as paraesthesia with a glove and stocking distribution. Loss of vibration sense may be the earliest sign. There may be pains in the calf muscles followed by muscular weakness of the legs. Arsenic encephalopathy is rare and may resolve completely.

Trivalent arsenic in particular has been associated with hepatomegaly and cirrhosis. A megaloblastic anaemia has also been reported. In addition to skin cancer, arsenic has also been linked to angiosarcoma of the liver and lung cancer.

Arsine Poisoning

Arsine is a colourless, non-irritating gas with an odour similar to garlic. It is used in the manufacture of semiconductors in the microelectronics industry but can be evolved accidentally by the action of acid on arsenic contaminated ore or by the action of water on a metallic arsenide. The gas is a powerful haemolytic agent and its effects are usually fatal if poisoning is severe enough to cause anuria. The presenting symptoms are nausea, vomiting and headache which can arise between 2 and 24 hours after an exposure of only 1 to 2 minutes. Exposure to only 3-10 ppm of arsine can produce symptoms after several hours of exposure and 25-50 ppm for 30 minutes is potentially fatal. Dark red urine (haemoglobinuria) is an early sign and abdominal pain and jaundice arise in 24 to 48 hours followed by anuric or oliguric renal failure in about 72 hours. Differential diagnoses include leptospirosis, malaria and paroxysmal nocturnal haemoglobinuria. Chemical agents capable of causing haemoglobinuria include potassium chlorate, pyrogallol acid and stibine gas.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Diagnosis is by history of exposure and elevated urinary arsenic. There is reticulocytosis and leucocytosis with elevated plasma haemoglobin. Methaemoglobin may form in the plasma and urine. The main complication is renal failure from acute tubular necrosis and treatment by exchange transfusion is required. Mild cases of haemoglobinuria may be treated by forced diuresis. Recovery from acute tubular necrosis may be followed by chronic renal failure with glomerular sclerosis, atrophic tubules and interstitial fibrosis. Damage may also occur to the liver, myocardium, nervous system and bone marrow. A chronic form of poisoning with severe anaemia, mildly elevated serum bilirubin and raised urinary arsenic has been described in workers engaged in cyanide extraction of gold and zinc smelting.

Organic Derivatives of Arsenic

These have no commercial applications but were used extensively in chemical weapons in both world wars. They are vesicant and highly irritating to mucous membranes. Their use led to the development of BAL (2,3-dimercaptopropanol) to prevent and reverse the toxic effects. BAL is also used to treat the thiol-binding effects of certain types of heavy metals.

Monitoring of Workers exposed to Arsenic

The usual method of monitoring has been the measurement of total arsenic concentration (inorganic arsenic, monomethyl arsonic acid and dimethyl arsinic acid) but this can be affected by consumption of seafood. It is advisable to ensure that worker avoids seafood for 3 days prior to urine collection. Workers with urine arsenic level of more than 50 μ g/g creatinine in 2 successive examinations should be suspended from further exposure and the Labour Department should be notified for further investigations. As arsenic is a human carcinogen, it is advisable to keep exposure levels as low as reasonably practicable.

The measurement of arsenic in hair and nails is useful in forensic science but is not applicable to occupational exposure.

C5 Poisoning by Mercury or a Compound of Mercury

The largest single use of mercury is as a liquid electrode in the electrolytic production of chlorine, but it is also used in the manufacture of fungicides, anti-fouling paints, switchgear, laboratory apparatus, thermometers, detonators, dental amalgams and batteries. Elemental mercury vaporises readily at room temperature and is readily absorbed through the lungs as are compounds of mercury. Metallic mercury is not well absorbed from the gut, but methyl and phenyl mercury are almost completely absorbed. Some organic and aryl compounds are corrosive if swallowed. Mercury compounds may also be absorbed through the skin, and phenyl and butyl salts can cause chemical burns.

Mercury, like lead, combines with and inhibits the action of enzymes containing -SH groups. Inorganic mercury is distributed almost equally between the red cells and the plasma, but alkyl compounds are heavily concentrated in the red cells. Mercury principally targets the CNS and the kidney. The blood-brain barrier is easily crossed by elemental and alkyl mercury but not so well by inorganic compounds. Mercury binds to metallothionein in the kidney and renal damage only occurs when saturation is completed.

Fulminate of mercury is used in the manufacture of detonators and ammunition and is a skin and mucosal irritant but rarely leads to clinical mercury poisoning. It may cause a dermatitis, starting as erythema of exposed parts, accompanied by pruritus, swelling and oedema and may progress to a popular, vesicular or pustular rash. Fulminate lodged in cracks in the skin may cause painful necrotic lesions which may later become circular, punched out ulcers.

Acute Poisoning

This seldom results from occupational exposure but some cases have been seen as a result of the inhalation of elemental mercury vapour. Most have been the result of deliberate or accidental ingestion. The corrosive nature of some compounds complicates the picture. There may be pain, inflammation and necrosis of the oropharyngeal mucosa, nausea, vomiting, abdominal pain and renal damage with lesions of the proximal tubule and glomerulus.

Severe cases may have acute papillary necrosis or chemical colitis with shock, oedema, tremor and ataxia. Chemical pneumonitis with cough, dyspnoea, retrosternal pain, basal late inspiratory crackles and patchy shadowing on chest X-ray may follow copious inhalation. There may be pulmonary oedema and blood stained sputum.

Chronic Poisoning

The classical signs of mercury poisoning are gingivitis, tremor and erethism.

The earliest symptoms of chronic poisoning are usually gingivitis, hypersalivation, and a bitter, metallic taste in the mouth. A blue line (like that in lead poisoning) and grey or red punctate pigmentation of the buccal mucosa are less common.

Tremor is usually slight and may be present at rest and accompanied by mild motor retardation. There is often an intentional component which may impair fine and complex movements. There may be fluctuating severity with ataxia; and in poisoning with methyl mercury, cerebellar ataxia, dysarthria and visual field defects may be apparent. The clinical picture may resemble parkinsonism, multiple sclerosis or cerebellar disease, but nystagmus is not a feature.

Psychiatric manifestations (erethism) usually present as timidity, extreme irritability, mental hyperactivity and rage. There may also be depression, memory problems, somnolence and difficulty in concentration.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Peripheral neuropathy (predominantly sensory) is most common in those with organic mercury poisoning. There may be paraesthesia of the extremities and periorally.

The most common renal effect is tubular damage, with necrosis being more common in inorganic than organic poisoning. Glomerular damage may lead to albuminuria. Inorganic poisoning may occasionally result in nephrotic syndrome.

Laboratory Diagnosis

Workers with urine mercury levels exceeding $35\mu\text{g/g}$ creatinine in 2 successive examinations should be suspended from further exposure. The Labour Department should be notified for further investigations.

C6 Poisoning by Carbon Bisulphide

Carbon bisulphide is a highly combustible, volatile liquid which was extensively used in the vulcanisation of rubber. Its use is decreasing, but it still has uses in the manufacture of rayon and cellulose fibres and as a solvent for fats lipids and resins and in laboratory analysis.

Carbon bisulphide is readily absorbed through the skin, lungs and gastrointestinal tract, but inhalation is the main route of entry with pulmonary uptake being about 40% of the inhaled concentration. In the post exposure period up to 30% may be eliminated unchanged in the breath. Biological monitoring may be carried out by measurement of 2-thiothiazolidine-4-carboxylic acid (TTCA) in the urine for up to 48 hours after exposure.

Clinical Features

Acute poisoning may give rise to intoxication followed by headache, dyspnoea, vomiting, palpitations, delirium and coma. Acute psychosis in the past has resulted in some workers committing suicide, but in others there have been manic depression, hallucinations, sexual deviancy and amnesia.

The WHO classification of the chronic effects of carbon bisulphide is as follows:

- Psychoses (manic depressive and disorientation)
- Polyneuropathy of lower extremities, sensory disturbances, and decrease of motor and sensory conduction velocities in peripheral nerves.
- Gastrointestinal tract disturbances such as chronic hyper- and hypoacidic gastritis and duodenal ulceration.
- Myopathy of calf muscles
- Neurasthenic syndrome in the autonomic nervous system
- Optic neuritis
- Atherosclerotic vasculoencephalopathy

With modern control systems the above effects have become very rare. Studies have demonstrated intellectual impairment and EEG abnormalities, impaired colour discrimination and a reversible relationship with coronary heart disease. There have also been links with menstrual disturbances, an increases risk of miscarriage and hypospermia and abnormal sperm morphology.

C7 Poisoning by Benzene or a Homologue

Benzene is a volatile, clear, flammable hydrocarbon which is a natural constituent of crude oil. It is the simplest of the aromatic organic compounds and was formerly isolated from the coal tar fraction of coal of distillation. It was formerly used as a degreasant, a solvent for adhesives and paint removers, in the production of plastics, inks, dyes, lacquers, varnishes, rubber and explosives and in the extraction of oils, alkaloids etc. Current use is strictly controlled. Up to 5% benzene may be found in unleaded petrol.

Clinical Features

Benzene is absorbed systemically by inhalation and skin absorption. Acute effects include headache, nausea, dizziness, narcosis and loss of consciousness. There is considerable individual variation in response.

Chronic effects of benzene exposure include depression of the bone marrow, aplastic anaemia and leukaemia. Benzene is also suspected of causing multiple myeloma. The most common type of leukaemia reported is acute non-lymphocytic myeloblastic leukaemia, but chronic myelocytic leukaemia, chronic lymphatic leukaemia and erythroleukaemia have all been reported. The latent period for the development of leukaemia after exposure to high levels is 10 years. It is classified by IARC as carcinogenic in humans.

Haemopoietic effects include leucocytopenia, thrombocytopenia and pancytopenia. In severe cases there may be fatal aplastic anaemia. Splenomegaly with haemolysis, hyperbilirubinaemia, punctuate basophilia of erythrocytes and marrow hyperplasia may occur. Haematological changes may persist for months after exposure has ceased.

Chronic neurological effects include behavioural and psychomotor changes and labyrinthine, vestibular or acoustic impairment. At high levels there may be effects on the myocardium and arrhythmias.

Workers with urine phenol levels of more than 50 mg/g creatinine in 2 successive examinations should be suspended from further exposure. The Labour Department should be notified for further investigations.

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.

C8 Poisoning by a Nitro- or Amino- or Chloro- Derivative of Benzene or of a Homologue of Benzene, or Poisoning by Nitro-chlorobenzene

Nitrobenzene is used as a solvent, as a lubricating oil and as an ingredient of polishes, perfumes and soaps. Most of it, however, is converted to aniline (an amino derivative), a starter chemical for a wide variety of products. Other nitro- derivatives are dinitrobenzene, trinitrobenzene, nitrotoluene, dinitrotoluene, trinitrotoluene and trinitroxylyene. Closely related but not true nitro- derivatives are tetryl, the nitrochlorobenzene and the dinitrochlorobenzene.

Amino derivatives include aniline, the toluidines, xylydines, phenylene diamines and toluylene diamines. Closely related but not true amino- derivatives are methyl and dimethyl aniline, ethyl and diethyl aniline, the nitroanilines, dinitroanilines, chloranilines, nitrochloranilines and the diphenylamines.

Clinical Features

Nitro- and amino- derivatives of benzene are easily absorbed via the skin and lungs. In addition to the normal effects of organic solvents such as mucosal irritation, dermatitis and CNS depression, nitro- and amino derivatives of benzene can cause methaemoglobinaemia and haemolytic anaemia. The rapidity of absorption and therefore their immediate toxicity is largely related to their physical state and volatility (aniline is a volatile liquid and is much more toxic than the solid trinitrotoluene). Acute poisoning is usually the result of a single high level respiratory or dermal exposure.

The chloro- derivatives of benzene are not likely to give rise to much trouble in industry with the exception of monochlorobenzene and the ortho- and para- isomers of dichlorobenzene. These substances are used in the dyeing and chemical industries, as constituents of lacquers, as solvents and cleaning agents, as fumigants, disinfectants and as constituents of wood preservatives. They are often contaminated with substances such as benzene and carbon disulphide.

Acute Poisoning

Although nitro- and amino- derivatives vary considerably in toxicity, there are certain symptoms common to most cases of acute poisoning. In mild cases the only symptoms may be those due to methaemoglobinaemia. The main features are those of cyanosis, most noticeably in the face and fingernails, symptoms of respiratory distress, throbbing headaches, dizziness and muscle weakness. Symptoms are related to the degree of methaemoglobinaemia and the speed of onset. In more serious cases there may be nausea, vomiting, colic and collapse. Symptoms usually occur at methaemoglobin levels of >15%. Heinz bodies in erythrocytes, diffuse or punctuate polychromasia and other features of anaemia may be seen in peripheral blood films. Extreme cases of exposure can be fatal, with patients displaying signs of shock, but in most cases slow and complete recovery can be expected.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Chronic Poisoning

The main signs of chronic poisoning are those of mild anaemia, slight cyanosis or mild haemolytic jaundice, with additional signs peculiar to the compound concerned. Examples are:

- dinitrobenzene cachexia and mental deterioration
- trinitrotoluene toxic gastritis and hepatitis with or without toxic jaundice appearing after weeks or months. Aplastic anaemia
- paraphenylene diamine dermatitis
- toluylene diamine haemolysis
- dinitrochlorobenzene Cachexia, mental deterioration, skin irritation & sensitisation
- monochlorobenzene headache, dizziness, stupor & difficulty in micturition

In the case of toxic jaundice, the mortality rate is about 30% and those that recover usually have permanent liver damage. Similarly, aplastic anaemia never results in full recovery of bone marrow.

C9 Poisoning by Dinitrophenol or a Homologue or by Substituted Dinitrophenols or by the Salts of Such Substances

Dinitrophenol is a yellowish-white crystalline material used in explosives, dyestuffs, photographic developers and wood preservatives. It sublimes readily and may be absorbed by ingestion or inhalation (and possibly via the skin). The only homologue of note in terms of occupational medicine is dinitro-*o*-cresol which is a pesticide (although another pesticide, pentachlorophenol, shares the same biological actions) and is rapidly absorbed through the skin.

Acute Poisoning

These substances act by uncoupling the chain of oxidative phosphorylation (a mitochondrial process by which cells produce adenosine triphosphate [ATP] from adenosine diphosphate [ADP]) and therefore preventing the storage of energy in the phosphate bond. This energy is dissipated by an increase in the metabolic rate.

Mild poisoning produces stomach upsets with a pale, furred tongue, anorexia, vomiting, headache and vertigo, possibly with mild jaundice and albuminuria. More severe cases may have hyperthermia, tachycardia, tachypnoea, prolific sweating, thirst and rapid dehydration. Death may ensue from heat-stroke, hepatic or renal damage. Some of the substances may be excreted unchanged through the skin, leading to yellow discoloration of covered and moist areas. Metabolites such as amino- or diamino-nitrophenol (or -nitrocresol) may be detected in the urine.

Chronic Poisoning

Cataract with delayed onset and agranulocytosis have been reported following long term absorption of dinitrophenol and dinitro-*o*-cresol.

C10 Poisoning By Halogen Derivatives of Hydrocarbons of the Aliphatic Series

The industrially important chemicals described above are mainly alkanes and alkenes where one or more hydrogen atoms has been substituted by a halogen (generally chlorine) atom. The solvents generally share toxic effects on the liver and kidney and also tend to cause depression of the central nervous system. The hepatotoxicity may cause increased levels of enzymes such as ALT or AST. Heavy drinkers may be more at risk due to ethanol induction of enzymes leading to increased production of toxic metabolites. Some studies have indicated that occupationally exposed persons may have an increased risk of developing glomerulonephritis and that once developed, continued heavy hydrocarbon exposure may lead to accelerated progression to renal failure.

Some examples of halogen derivatives of aliphatic hydrocarbons are:

Carbon Tetrachloride

At one time carbon tetrachloride was used extensively in dry-cleaning and as a fire extinguisher. It is no longer used for these purposes due to its toxicity and its use in the production of chlorofluorocarbons (CFCs) has declined for environmental reasons. It is still used as a degreasing agent and in the rubber, pharmaceutical and pesticide industries.

Carbon tetrachloride may be absorbed by inhalation or through the skin. It is stored in fat, liver, kidney, brain and bone marrow and can be detected in the breath for up to 2-3 weeks after inhalation. It can cause fatty degeneration and necrosis of hepatic tissue, and widespread damage and death of renal proximal tubular cells. Liver cancer has been reported only when cirrhosis was already present and carbon tetrachloride has been classified by IARC as a possible human carcinogen.

Acute effects of inhalation may include nausea and vomiting, confusion, albuminuria and coma. Constriction of visual colour fields, amblyopia and optic atrophy have been reported. Acute toxic hepatitis may follow within a few days of overexposure with jaundice and an enlarged, tender liver. Transaminase and serum bilirubin levels may be raised. Acute necrosis may follow in very severe cases. Diagnosis may be aided by detection of carbon tetrachloride in the breath or blood.

1,1,1-Trichloroethane

The above solvent is commonly used as a cleaner and degreaser in the engineering and electronics industries. It is also used in the furniture and upholstery industries and as a solvent and thinner for correction fluid, adhesives, paint, varnishes and inks. It is readily absorbed by inhalation, but poorly through the skin. Elimination is slow, mainly unchanged through the lungs. Acute inhalation at high concentrations depresses the CNS and may produce narcosis with dizziness, lassitude, headaches and in high concentrations coma and death. 1,1,1-trichloroethane may be detected in breath or blood and the metabolites 2,2,2-trichloroethanol and trichloroacetic acid may be identified in urine.

Trichloroethylene

The primary use of this solvent is as a metal degreasing agent, but it is also used in textile treatment, dry-cleaning, as a rubber solvent, production of inks, coatings and adhesives, PVC and fire retardants. It was formerly used as an anaesthetic.

It is readily absorbed through the skin but the main route is by inhalation. A large proportion is exhaled unchanged, but some is oxidised in the liver to chloral hydrate which is then converted to trichloroethanol (conjugated before excretion) or trichloroacetic acid. These two metabolites may be measured in urine samples towards the end of the working week as a means of biological monitoring.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

High concentrations may cause eye and nasal irritation accompanied by dizziness, headache, nausea, fatigue, visual disturbance, confusion, unco-ordination, and narcosis. Coma and death may occur in extreme cases. There has been evidence that ventricular arrhythmias are the cause of death. Consumption of alcohol after exposure may result in transient reddening of the face and neck ('degreaser's flush') in a few individuals.

In most instances the symptoms resolve within a few hours of removal from exposure, but in some cases residual headaches, psychological symptoms and nerve conduction problems have been reported. There is no strong evidence for hepatotoxicity or nephrotoxicity.

Tetrachloroethylene (perchloroethylene)

This solvent is used mainly in the dry-cleaning industry but also has uses as a metal degreasant and in the production of adhesives, thinners, cleaning agents, fluorocarbons and hexachloroethane. It is absorbed mainly by inhalation, but also through the skin. Toxicological properties are similar to trichloroethylene. It can be detected in the breath for a considerable time after exposure.

Long term exposure may lead to headache, fatigue, nausea and light-headedness. IARC classifies tetrachloroethylene as 'possibly carcinogenic in humans'.

C11 Poisoning by Diethylene Dioxide (Dioxan)

Dioxan was formerly used as a solvent for fats and cellulose and for degreasing wool. It may be encountered occasionally in the manufacture of polishing compounds and cosmetics and as a paint stripper and preserving agent. It can be absorbed by inhalation or via the skin.

Clinical Features

Diethylene dioxide fumes at low concentrations is mildly irritant to the mucous membranes of the eyes, nasopharynx and throat, causing conjunctivitis, blurred vision, coryza and cough. Greater exposure may lead to headaches, vertigo, drowsiness, anorexia, nausea and vomiting, followed by toxic hepatitis accompanied by abdominal or lumbar pain. Prolonged exposure may result in haemorrhagic nephritis leading to anuria and fatal uraemia.

C12 Poisoning by Chlorinated Naphthalene

Chlorinated naphthalene is used as a synthetic insulating wax has a higher melting point than common paraffin wax, is non inflammable and resists acid and alkali. As used in industry the wax usually consists of a mixture of chlorinated naphthalenes of which the higher members (penta- and hexachloronaphthalene) are more toxic than the lower members. They are usually melted and applied as an insulating coating to wires or used for blanking off parts of an article to be electroplated. If heated more than a few degrees above melting point it volatilises and produces fumes or particles which may be inhaled or come into contact with exposed skin. The use of chlorinated naphthalene has declined in recent years.

Clinical Features

Chloracne

This is an occupational dermatosis which principally affects the cheeks, forehead, behind the ears and less commonly limbs, trunk and genitalia. The lesions are pruritic, with pale comedones associated with pale, yellowish or flesh coloured cysts and later larger inflammatory lesions. Chloracne is not unique to chlorinated naphthalene; other chemicals causing this problem include:

- chlorinated dibenzodioxins and chlorinated dibenzofurans (may be contaminants of polychlorinated biphenyls), and
- tetrachlorodibenzodioxin (contaminant of herbicide 2,4,5-trichlorophenoxyacetic acid)

Focal Liver Damage

The higher chlorinated naphthalenes appear to exert a highly selective action upon the liver. This may result in acute necrosis with ensuing toxic jaundice. Acute yellow atrophy and death may occur. Simultaneous exposure to carbon tetrachloride fumes increases the likelihood of liver damage.

C13 Poisoning by Oxides of Nitrogen

Nitric oxide is a colourless gas which rapidly oxidises in air to nitrogen dioxide, a reddish brown gas with a pungent odour apparent at about 0.5 ppm. Both are found in nitrous fumes produced by fuming nitric acid and are formed by the combustion of fossil fuels or biomass. Although some fuels contain little or no nitrogen, nitrous fumes are formed in the region of peak flame temperature. Other sources of nitrous fumes include

- Unvented gas cookers and paraffin heaters
- Action of nitric acid on metals or on organic materials
- Welding operations
- Fermentation of silage (especially from high nitrate soils in dry conditions)
- Incomplete detonation of nitroexplosives such as dynamite, gun cotton and nitroglycerine
- Accidental burning of nitroexplosives
- Fumes from expended ammunition

Clinical Features

Nitrogen dioxide is an oxidant gas with free radical properties which hydrolyses in water to nitrous and nitric acids. These properties make it injurious to respiratory airways and lung parenchyma. Mild asthmatics are more susceptible to injury. The gas is only mildly irritant and at exposures of up to 50 ppm there may be no warning of its hazard. Exposure to as little as 25 ppm for 8 hrs may result in pulmonary oedema 5 to 48 hrs later. Any person believed to have had significant exposure should therefore be admitted to hospital and placed under observation for 48 hrs. Exposure for less than 1 hr to 100 to 150 ppm can result in fatal pulmonary oedema arising between 3 and 72 hours later after initial irritant effects of cough, headache, throat irritation, chest tightness and sweating which may resolve within 30 mins. More elevated concentrations can produce severe and immediate hypoxaemia which may be fatal. In less severe cases the delayed symptoms may be dyspnoea of sudden onset, cough, haemoptysis and headache.

Most cases recover with corticosteroid and bronchodilator therapy, but if the steroids are stopped prematurely, bronchiolitis obliterans may develop as a complication after an asymptomatic period lasting up to 6 weeks. This appears as a fine, widely scattered nodular infiltration on chest X-ray and may be fatal. Current evidence suggests that exposure to concentrations insufficient to lead to acute problems does not cause chronic disease.

C14 Beryllium Poisoning

Beryllium is a very light, hard metal. It occurs naturally as beryllium aluminium silicate, or beryl and as the lower grade ore bertrandite. Aquamarine and emerald are forms of beryl. Beryllium was formerly used in the production of fluorescent light tubes, but its main use now is the manufacture of alloys, particularly beryllium copper. It is used in some alloys in the aerospace industry and in communications components. It has the ability to slow down neutrons and is therefore used in the nuclear industry. It is difficult to handle in pure form and is generally marketed as a fine powder for sintering.

Acute Effects

Direct irritation of the respiratory tract may lead to nasopharyngitis, tracheobronchitis or chemical pneumonitis. Signs and symptoms may include dyspnoea, chest pain, cough with blood stained sputum, tachycardia and cyanosis. Radiographs show diffuse or localised infiltrations. Severe cases may develop a fatal bronchoalveolitis with extensive necrosis. Complete resolution can be expected in most cases following removal from exposure, oxygen and in some cases corticosteroid therapy.

Dermatological Effects

Implantation of beryllium subcutaneously leads to the formation of non-caseating granulomata. These lesions were common in those who cut themselves on fluorescent light tubes. A non-tender lump forms beneath the skin weeks or months after the injury and enlarges or forms satellite lesions along the lymphatic drainage routes unless excised. Peripheral nerves may be involved in some cases leading to altered sensation.

Beryllium may also act to produce a contact dermatitis which may be accompanied by periorbital oedema, conjunctivitis or upper respiratory tract involvement.

Chronic Berylliosis

Inhalation of beryllium dust may lead to the formation of pulmonary granulomata after a latent period ranging from a few months to 10-15 years or more. The presenting symptom is usually shortage of breath on exertion, followed by fatigue, weight loss chest and joint pains and cough. Signs may include hepatosplenomegaly, skin rashes, lymphadenopathy, basal crackles and finger clubbing.

The course of the disease is variable. Some patient deteriorate progressively while others remains symptomatically stable. Those presenting with radiographic changes only may develop symptoms late or never. Deterioration is indicated by increasing shortness of breath, deterioration in lung function, development of pulmonary hypertension and cor pulmonale.

Radiographic Changes

Standart chest X-ray reveals a diffuse pulmonary infiltration and hilar lymphadenopathy. Opacities may be granular, nodular, linear or mixed. 50% of patients have hilar lymphadenopathy (usually bilateral and not marked) but it is uncommon in those without parenchymal disease (in contrast to sarcoidosis). There may be evidence of collapse of lobes (usually upper) with overinflation of adjacent lung tissue, calcification in the parenchymal opacities and hilar nodes, pleural thickening, cysts and pneumothorax.

Pulmonary Function

There are 3 main types of abnormality associated with chronic beryllium disease:

- A restrictive defect with reduced FVC and a normal FEV₁/FVC ratio
- An interstitial defect with normal lung volumes and air flows but with a reduced TLC
- An obstructive defect with granulomata in the peribronchial regions

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Patients with interstitial defects have the best prognosis; those with obstructive or restrictive defects often deteriorate despite steroid treatment.

Diagnosis

Chronic beryllium disease is difficult to differentiate from sarcoidosis. Both have non-caseating granulomata in the lungs and other organs but some features of sarcoidosis, including uveitis, neurological involvement and bone lesions have never been reported in beryllium disease. The Kveim test is never positive in beryllium disease and spontaneous remission of radiographic changes is very rare. The presence of beryllium in the tissues or urine would be extremely unusual in sarcoidosis. Immunological tests are not good discriminators.

The Beryllium Case Register has established the following criteria for the diagnosis of beryllium disease:

1. Establishment of significant exposure to beryllium based on occupational history and preferably the results of environmental monitoring.
2. Objective evidence of pulmonary disease and a clinical course consistent with berylliosis.
3. Radiographic evidence of interstitial fibronodular disease.
4. Evidence of impairment in pulmonary function.
5. Typical histopathological changes in biopsy specimens from the lung and/or thoracic lymph nodes.
6. Presence of beryllium in tissue specimens or urine.

Laboratory tests may show an elevation in serum gamma globulins (particularly IgA and IgG), raised ESR, hyperuricaemia, hypercalcaemia and hypercalcuria. Renal calculi may sometimes be present.

Pathogenesis

This is probably a combination of immunological or hypersensitivity reactions.

Carcinogenicity

Beryllium can cause both lung cancer and sarcomas in experimental animals. There have been reports of increased rates of lung cancer in workers with chronic beryllium disease and beryllium should therefore be regarded as a potential human carcinogen.

Treatment

Recognition and removal from exposure should be the priority. Corticosteroid therapy is effective in the early stages and is usually given for life, but does not reverse the later pathological changes.

C15 Cadmium Poisoning

Cadmium is recovered as a by-product in the smelting of zinc ores and also some lead ores and is used in electroplating, to make pigments, in batteries and as a stabiliser in plastics. Its corrosion resistance often leads to its use as a plating in civil engineering and marine structures.

It is poorly absorbed from the gut but is relatively well absorbed from the lungs. In the blood about 70% is bound in the red cells and 50% of the body burden is concentrated in the liver and kidneys. The half life in the body is about 10 years. In the tissues it is largely bound to metallothionine and is excreted as a complex with metallothionine. The rate of excretion is low unless there is renal damage.

Acute Poisoning

This most commonly follows inhalation of cadmium oxide fumes from welding or brazing cadmium or its alloys. There is usually a lag period of several hours before symptoms appear. These generally begin with a flu' like illness, followed by retrosternal pain, dyspnoea, cough, myalgia and headache. This usually lasts for 4 to 10 hours and is followed by a stage of pulmonary reaction with wheezing, dyspnoea, chest tightness, malaise, weakness, nausea and anorexia. This may last from 8 hours to several weeks. There may be fine basal crepitations and chest X-ray may show signs of bilateral pulmonary infiltration. Very severe cases may develop a fatal chemical pneumonitis with severe renal damage (bilateral cortical necrosis, tubular degeneration and glomerular infarction) as well as pulmonary oedema and alveolar metaplasia.

Chronic Poisoning

The kidney is the principal target of chronic poisoning and tubular proteinuria is the earliest sign. The appearance of low molecular weight proteins is particularly characteristic. Other evidence of tubular damage may be evident (aminoaciduria, glycosuria and phosphaturia). Calcium excretion may increase and there have been reports of renal stone formation. Some studies have also shown evidence of glomerular damage such as albuminuria.

Renal damage is unlikely to occur until a significant concentration of cadmium accumulates in the renal cortex. Most of the cadmium is bound to metallothionine and it is only the small amount of metal not so bound that causes the nephrotoxicity.

Long term pulmonary damage can also occur as a result of cadmium excretion. Some studies have shown an increase in the incidence of emphysema in cadmium workers. Lung function tests have also been reported as abnormal in cadmium workers, but there appears to be no dose-response relationship. There have also been claimed associations with hypertension, osteomalacia, prostatic cancer and lung cancer, but none of these show firm evidence.

Diagnosis of cadmium poisoning requires a history of exposure and the absence of a more common explanation of renal or pulmonary disease. An elevated urinary cadmium concentration may be used as a guide to increased exposure but cannot be used to make a diagnosis in isolation.

Monitoring

Biological monitoring is required to prevent renal damage resulting from excessive uptake. Blood levels of cadmium generally reflect exposure over the past 3 to 6 months (and are increased by cigarette smoking) and those of urine can reflect the body burden when the cadmium-binding sites in the body are not saturated and cadmium-induced nephropathy has not yet occurred. Beta-2-microglobulin level in urine can be used to assess proximal tubular damage. Workers with blood cadmium levels of more than 5 µg/l and/or urine beta-2-microglobulin levels exceeding 290 µg/g creatinine in 2 successive examinations should be suspended from further exposure. The Labour Department should be notified for further investigations.

C16 Dystrophy of the Cornea (including ulceration of the corneal surface) of the Eye

Occupational exposure to arsenic, tar, pitch, bitumen, mineral oil, soot and related substances may produce inflammation of the corneal conjunctiva. More prolonged exposure may result in inflammation or ulceration refractory to treatment which can lead to permanent scarring with interference of vision. Quinone, hydroquinone and associated products (used in the photographic industry) may give rise to permanent staining of the light exposed area of cornea. This staining may become so dense as to impede vision and the cornea may eventually ulcerate.

C17 Primary Epitheliomatous Cancer of the Skin

Occupational skin cancer has been described since 1775 when Percival Pott drew attention to soot as a cause of scrotal cancer in chimney sweeps. Since that time it has been shown that polycyclic aromatic hydrocarbons (or substances containing them such as tar, pitch, bitumen, mineral oil etc.) have an association with squamous and basal cell tumours of the skin. Chronic lesions such as localised neoplasms of a papillomatous or keratotic nature may be evident prior to the development of frank malignancy.

C18 Chrome Ulceration

Chromium is a hard silver-white metal which is used for chrome plating of steel and in the manufacture of stainless steel, and ferrochrome. Chromium compounds are used in the textile dyeing industry and as tanning agents and pigments.

Chrome Ulcers

Chromium can be absorbed through intact skin but readily enters via small scratches and abrasions. A round, hard lump may develop covered by a crust which sloughs to reveal a circular punched-out ulcer. These are only slightly painful and tend to heal spontaneously but may become secondarily infected.

Inhalation of mists containing chromium salts may lead to ulceration followed by perforation of the nasal septum.

C19 Urinary Tract Cancer

Occupational bladder cancer was first described in 1895 in workers involved in the preparation of fuchsine. Bladder tumours account for about 5% of all cancers world-wide, and tobacco consumption is the main risk in industrialised nations. It is estimated that around 20% of bladder tumours in industrialised countries are of occupational origin. Almost all are transitional cell carcinomas. The main industries implicated are rubber, dyes and gas, with textiles, leather and painting also having excess risk.

The most prominent carcinogens are aromatic amine benzidine-based dyes such as:

- 2-naphthylamine
- benzidine
- 4-aminobiphenyl

and the curing agent:

- 4,4-methylene *bis*-(2-chloroaniline)[MbOCA]

as well as the unidentified chemicals associated with

- manufacture of auramine
- manufacture of magenta

Clinical Features

The commonest presentation is of painless haematuria, but unexplained frequency, dysuria or symptoms of obstruction may occur. Urinary tract infection may be superimposed. Workers at risk should be educated to consult a doctor for any of the above problems. Under the Factories & Industrial Undertakings (Carcinogenic Substances) Regulations, all workers handling controlled carcinogenic substances should be medically examined within one month of being first so employed and periodically at 6 months interval. The medical examination should include exfoliative cytology of the urine by a laboratory of the Institute of Pathology of the Department of Health.

C20 Peripheral Polyneuropathy

Peripheral polyneuropathy is associated with exposure to n-hexane or methyl-n-butyl ketone. N-hexane is a colourless liquid hydrocarbon which occurs in industry as a component of commercial hexane mixtures or petroleum hydrocarbon products. It may be found in high concentrations in solvents for adhesives, paints and inks. Methyl-n-butyl ketone (MBK) is also a colourless liquid used as a solvent, in paints and in the printing industry.

The main route of occupational absorption is inhalation, but there is also potential for dermal absorption. A toxic metabolite, 2,5-hexanedione is probably responsible for the peripheral polyneuropathy seen in cases of chronic exposure. The neurotoxicity appears to be enhanced if there is also exposure to methyl ethyl ketone (MEK).

Acute Toxicity

N-hexane and methyl-n-butyl ketone have the usual narcotic effects on the CNS that are shared with most organic solvents.

Chronic Toxicity

Prolonged exposure (several months significant contact) may lead to paraesthesia of the extremities, or sensory neuropathy with decreased nerve conduction velocities. Even after withdrawal from further exposure, recovery is slow, taking months or years and may be incomplete.

C21 Localised Papillomatous or Keratotic New Skin Growth

Occupational skin cancer has been described since 1775 when Percival Pott drew attention to soot as a cause of scrotal cancer in chimney sweeps. Since that time it has been shown that polycyclic aromatic hydrocarbons (or substances containing them such as tar, pitch, bitumen, mineral oil etc.) have an association with squamous and basal cell tumours of the skin. Chronic lesions such as localised neoplasms of a papillomatous or keratotic nature may be evident prior to the development of frank malignancy.

C22 Occupational Vitiligo

Occupational vitiligo is indistinguishable clinically and histologically from naturally occurring vitiligo and is caused by damage to melanocytes.

Clinical Features

The affected areas can be mottled, patchy, confluent or symmetrical. Exposure to bright sunlight occasionally causes redness, irritation and pain. The diagnosis depends on the occurrence of vitiligo and a history of exposure to chemicals known to produce this change in humans. It may be localised due to contact (such as with *p*-tertutylphenol used as an adhesive in shoe repairs) or it can be widespread due to absorption via skin or lungs (such as with hydroquinone, used as a photographic developer, in the manufacture of dyes and as an antioxidant in paints and fuels). Early detection is enhanced by the use of a Wood's lamp, the light of which is absorbed by melanin and reflected from skin which contains no melanin. This can be used as a tool to screen an exposed workforce. Chemicals known to cause occupational vitiligo include paratertiary-butyl phenol, paratertiary-butyl catechol, para-amyl-phenol, hydroquinone or the monobenzyl or monobutylether of hydroquinone.

PART IV

DISEASES CAUSED BY

MISCELLANEOUS AGENTS

D1 Occupational Dermatitis

Inflammation or ulceration of the skin produced by dust, liquid or vapour (including the condition known as chloracne but excluding chrome ulceration).

The term dermatitis is synonymous with eczema. The main features of dermatitis are redness and swelling of the skin with small, fluid filled blisters, oozing in the acute state. It may be classified into two types; irritant and allergic contact dermatitis. Occupational dermatitis probably accounts for about 50% of all occupational illness.

Irritant Contact Dermatitis

This form of dermatitis is not mediated through an immune response. It may occur when the skin is subjected to damage from irritant substances with which it is in contact and may occur in an acute or chronic form. Strong irritants will produce dermatitis in almost all people exposed, but weak irritants tend to produce acute dermatitis only in susceptible persons, e.g. those with previous atopic or hand eczema. Repeated skin contact over a prolonged period with weak irritants may produce a cumulative insult irritant contact dermatitis even in those not considered susceptible.

Common occupations involved are hairdressing, cleaning, engineering (coolant oils) and construction. Cement may initially produce irritant dermatitis later complicated by allergic dermatitis from the chromate content.

The condition is largely confined to the hands and rarely involves the eyelids or genitals. Clinical features are of red, swollen, itching and sometimes painful skin with small blisters which may rupture and exude. Larger blisters may form on the palms of the hands. Classically the dorsum of the hands and fingers, the sides of the fingers and the finger webs are involved before the palms. In chronic cases painful fissures may form around the nail folds, knuckles and later the palms. Differential diagnoses include dermatomyositis, infective bacterial or fungal conditions and drug eruptions.

Allergic Contact Dermatitis

Allergic contact dermatitis is a manifestation of cell mediated immunity. Repeated contacts with an antigen are usually necessary and the allergy produced is specific to that antigen (though cross-sensitisation to chemically similar substances may occur) and is usually lifelong. Most allergens are low molecular weight compounds which can penetrate the keratin layer of the skin and skin which is damaged (e.g. by heat or degreasing) is more likely to develop allergic contact dermatitis. Common allergens include nickel, fragrances, hexavalent chromium, rubber and epoxy resin.

Allergic contact dermatitis tends to occur at the site of contact (most commonly the hands), but involvement of the eyelids is common and also of the male genitalia. With progressive exposure the eczema may become widespread. The clinical appearance is similar to that of irritant contact dermatitis.

Patch testing is often used to detect or confirm contact allergy. This is best conducted by a dermatologist with experience of the application and interpretation of the technique. Allergens are applied (often in a standard battery) in the form of patches under occlusion and are left in place and kept dry for 48 hours. They are read at 48 and at 96 hours.

Workers affected by allergic contact dermatitis will need to avoid contact with the allergen concerned. In some cases this will mean a job change, but in some cases of allergic contact dermatitis due to chromate from cement, nickel or cobalt, the condition may persist even with avoidance.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Ulceration of the skin

This condition may be caused by such chemicals as strong brine, quicklime, mercury fulminate and soda ash. A hard, raised lump is formed after penetration of the skin which breaks down in the centre to reveal a deep ulcer with round, thickened edges and a slough covered base. Sepsis is uncommon.

Chloracne

This is an occupational dermatosis which principally affects the cheeks, forehead, behind the ears and less commonly limbs, trunk and genitalia. The lesions are pruritic, with pale comedones associated with pale, yellowish or flesh coloured cysts and later larger inflammatory lesions. Chloracne is not unique to chlorinated naphthalene; other chemicals causing this problem include chlorinated dibenzodioxins, chlorinated dibenzofurans (contaminants of polychlorinated biphenyls), and tetrachlorodibenzodioxin (contaminant of herbicide 2,4,5-trichlorophenoxyacetic acid).

D2 Inflammation or Ulceration of the Mucous Membrane of the Upper Respiratory Passages or Mouth Produced by Dust, Liquid or Vapour

Almost any irritant, vapour, dust or droplet may cause an inflammatory reaction of the mucous membrane of the upper respiratory passages or mouth, but owing to natural defences most cause only transient inflammation.

The mucous membrane most likely to intercept droplet or particulate irritants is that covering the nasal septum. Chromic acid, chromates, 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 or larynx.

Clinical Features

Occupationally caused inflammation of the mucous membrane of the upper respiratory passages presents within a short time of first exposure and rarely arises for the first time after cessation of exposure. The more severe forms leading to ulceration are usually located over the cartilaginous part of the nasal septum. Ulcers are usually painless and shallow and are rarely infected. Some may be asymptomatic apart from slight bleeding. In some cases the cartilaginous septum may perforate. The bony septum is never involved.

Occupationally caused inflammation resolves rapidly on cessation of exposure. Likewise ulceration generally resolves slowly. Septal perforations are usually permanent but do not cause disability.

D3 Nasal or Paranasal Sinus Cancer

The main industries associated with this disease are wooden furniture manufacture and shoe and boot making, although chromium, nickel, mustard gas, cutting oils, coal mining and isopropyl alcohol have all been linked. The causative element in furniture manufacture is wood dust (but the exact carcinogen is unknown) which may be generated in high quantities in operations such as machine sanding. Care should be exercised to prevent inhalation of finely divided dust by engineering controls or by protective equipment. The causative agent in shoe and boot making is not known.

Cinical Features

The usual presenting features of nasal cancer are blood stained, often foul smelling, nasal discharge, nasal obstruction or facial swelling in cases presenting late. The most common tumours in furniture manufacture are adenocarcinomas of the ethmoid sinus and middle turbinates, and in shoe and boot making of mixed cell, mixed site sinus cancers. Woodworkers without carcinoma often suffer from chronic hypertrophic rhinitis, atrophic, dry nasal mucosa and nasal polyps.

D4 Byssinosis

The association of textile dust with respiratory disease was first noted by Ramazzini among flax and hemp workers and by Kay in 1831 among cotton spinners. The term byssinosis has been used since 1877 to describe the characteristic pattern of symptoms.

The aetiology and pathogenesis of the disease remain obscure and the type of fibre concerned largely determines the potential to cause disease. Cotton is the most potent, followed by flax, hemp and sisal. Harvested cotton contains a mixture of plant material including fibre, leaves, bracts and stems, bacteria and fungi. Bracts in particular have been the object of much scrutiny. The compounds involved in pathogenesis are water soluble and the risk of disease can be reduced by washing or steaming. Among the mechanisms postulated have been immunological, bacterial endotoxin activity, non-immunological release of histamine and fungal enzymes.

Clinical Features

It is rare for byssinosis to develop in the first 5 years of exposure to textile dusts and it is more usual for the process to take 20-30 years. Chest tightness and shortage of breath typically appear in the second half of the first day of the working week and are experienced most severely after the patient has returned home. The symptoms are worse after periods of absence such as holidays and affected workers may develop a productive or non-productive cough and wheezing. The incidence of bronchitis is also higher in byssinotics.

On physical examination there are no specific abnormalities in uncomplicated cases, the only signs being those of airflow limitation. Likewise there are no specific radiological features. Spirometry sometimes reveals a fall in FEV₁.

Byssinosis is traditionally Graded as follows:

- Grade 0 No symptoms
- Grade 1/2 Occasional chest tightness & respiratory irritation on first day of working week
- Grade 1 Chest tightness and/or difficulty breathing on first day of working week
- Grade 2 Chest tightness and/or difficulty breathing on first day and other days of working week
- Grade 3 Grade 2 symptoms accompanied by evidence of permanent respiratory difficulty from reduced ventilatory capacity

Diagnosis

The diagnosis of grade 1/2 to 2 rests on occupational exposure to cotton dust, a history of these clinical grades and a fall in FEV₁ during the working shift. In practice the objective evidence of FEV₁ at the time of symptoms may be difficult to obtain and the diagnosis may have to depend on the reliability of the patient's history.

Smokers and others may have chronic bronchitis, evidenced by cough and sputum unrelated to periods of dust exposure. In such patients byssinosis symptoms following exposure to dust can be differentiated and an acute fall of the already impaired FEV₁ can confirm the diagnosis.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

The diagnosis of grade 3 disease rests upon the occupational history, a history of progressive development of the clinical grades of respiratory symptoms and evidence of irreversible airway obstruction.

Persons diagnosed as having byssinosis with grade 2 symptoms and about or marked fall in ventilatory capacity should be removed permanently from exposure to cotton dust.

D5 Occupational Asthma

Asthma is defined as episodic airway obstruction reversible either spontaneously or as a result of treatment. Occupational asthma can be induced by any sensitising agents which inhaled in the work environment. There are many known sensitising agents and still more are identified from time to time.

Clinical Features

The clinical features of occupational asthma are basically the same as in asthma without work connection. In typical cases there is a period ranging from days to years before symptoms appear. When sensitisation has occurred the attack may develop within minutes of exposure or more often be delayed for hours when it can occur in the evening or at night after the patient has left the workplace. Sometimes preceded by a fit of sneezing, rhinorrhoea or spasmodic coughing, classic expiratory bradypnoea ensues progressively. The attack passes off slowly accompanied by viscous expectoration. Sensitisation once developed may be permanent but when the patient is removed from exposure to the casual agent the attacks usually remit.

Causes of Occupational Asthma

Some of the more common causes are:

- Laboratory animals (especially urine of male rodents)
- Flour
- Grain dust (due to fungi, mites etc.)
- Enzymes (e.g. in biological detergents)
- Formaldehyde
- Colophony
- Di-isocyanates
- Acid anhydrides
- Complex platinum salts

Diagnosis

Distinguishing between asthma which is occupational in origin and illness which is not work related is a major problem in diagnosis. An essential step is taking an accurate and detailed clinical and occupational history. The key points in the history are:

- (a) A sensitising agent is present at work.
- (b) The patient has been exposed to that agent for a period before the asthma develops.
- (c) The condition improves away from work.

Other aids to diagnosis include:

- (a) Skin prick test: These are simple to carry out and are effective in diagnosing sensitivity to platinum salts, proteolytic enzymes and laboratory animals.
- (b) Serial Peak Expiratory Flow Measurements: Repeated frequent measurements of peak expiratory flow rates are made over a period long enough to allow observation of any consistent changes and their relationship with work. A simultaneous diary of work and leisure activities should be kept. It is useful if the period includes a few days (ideally 1-2 weeks) holiday. Although frequently used, the technique requires compliance and honesty and can be easily manipulated by patients who understand the principle.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

- (c) Immunological Tests: The range of available immunological tests is expanding. Extracts of some of the causes of occupational asthma can be used to elicit skin test reactions and some specific IgE antibodies can be identified in serum.
- (d) Bronchial provocative tests: The major indications are
- where the agent thought responsible has not previously been reliably shown to do so.
 - where the individual is exposed to more than one potential cause and future employment requires accurate diagnosis.
 - where the asthma is so severe that uncontrolled workplace exposure is not justified.
 - where the diagnosis is in doubt after other investigations.

Testing should only be carried out in hospitals by personnel experienced in the techniques and with full resuscitation facilities available. The aim should be to recreate as accurately as possible the conditions at work, in terms of the concentration of the suspected agent, temperature etc.

Control challenge tests on a different day can be made using histamine or methacholine and noting the minimum concentration needed to provoke a 20% fall in FEV₁.

Management

Accurate diagnosis and identification of the causative agent are very important. In most cases the only satisfactory management strategy is cessation of exposure. This usually means a change of employment with all the accompanied difficulties.

Silicosis

Silicosis is a fibrotic disease resulting from the inhalation of silicon dioxide. High exposures can occur in the mining, quarrying and tunneling of granite or rock with a high quartz content. Other industries at risk include sandblasting, manufacture of abrasive detergent, pottery making, brickworks, foundry work, jade polishing, etc.

Acute Silicosis

High levels of exposure can produce immediate alveolar damage due to death of alveolar cells as well as macrophages. Acute alveolitis with leakage of protein fluid may present within weeks of the start of exposure and is followed by fibrotic obliteration of the alveoli. Initial symptoms are of dry cough and shortness of breath. X-rays show patchy pulmonary oedema with the shadows progressively condensing and affected areas shrinking. Fall in gas transfer leads to cyanosis and lung function tests show a restrictive pattern. The condition is often fatal, sometimes within a few months. There is no effective treatment.

Chronic Active Silicosis

The disease is usually advanced before it becomes symptomatic. Shortness of breath is usually the presenting symptom by which time the X-ray changes are widespread. Rounded 0.5 to 1.0 cm opacities are visible in the upper zones which are nodules composed of concentric collagen layers around a quartz centre. The nodules may coalesce due to contraction of fibrous linking bands and this event usually triggers the onset of shortness of breath. In the absence of complicating tuberculosis, there may be only a non-productive cough with no physical signs. Clubbing is not a feature. Calcification may occur if survival is long enough but this is uncommon. A restrictive lesion is found on spirometry and gas transfer falls leading to cyanosis. Death is usually from cor pulmonale. Symptoms can be produced within 1 year but the more usual period is 10 to 20 years. The chances of survival are increased with late presentation. In cases of early presentation, death may occur within 5 years. The disease is progressive even in the absence of continued exposure and exposed workers may leave their occupation (with normal lung function tests and chest X-rays) and develop fatal silicosis many years later.

Chronic Inactive Silicosis

This condition may occur in workers only occasionally exposed to silica dust or to workers exposed to mildly contaminated dust. The disease may only present after a long period of exposure. The X ray picture is usually that of mixed type of pneumoconiosis and the disease may progress for around 10 years after exposure ceases. The nodules may become static and calcify.

Silicotuberculosis

In Hong Kong, the risk of persons with silicosis also developing tuberculosis is much higher than for the general population. The higher risk may be due to damage to the pulmonary lymphatic system or to impedance of the activity of macrophages in the presence of quartz dust. The usual presentation in persons with asymptomatic silicosis is with symptoms of TB such as cough, sputum and night sweats, but the onset of silicotuberculosis is much less clear in advanced silicotics. More recent soft upper zone shadows are seen against pre-existing rounded opacities. There may be haemoptysis with cavitation. Conventional chemotherapy is considerably less effective in the presence of silicosis and the combined disease has a very poor prognosis.

Asbestos Related Diseases

Asbestos

Asbestos compounds are naturally occurring metallic silicates which have crystallised into long thin particles. These particles are respirable and a high proportion of such inhaled particles are caught in the lung and few if any are cleared. They are carcinogenic after a long latent period. There are four types used commercially:

1. Chrysotile (white asbestos) is the most common form of asbestos. It has soft curly white fibres but has poor acid resistance. This type is less hazardous to health than amphiboles.
2. Amphiboles
 - Crocidolite
 - Amosite
 - Anthophyllite

These types have shorter stiffer fibres and better acid resistance than chrysotile asbestos. Due to the health hazards associated with their use, utilisation of amphiboles has largely ceased in most countries but they may be encountered in demolition and refitting work.

Exposure to high levels of asbestos fibres may occur in the production of asbestos cement products, the processing of asbestos fibres, demolition and renovation work involving removal of asbestos material, insulation work such as delagging of boilers, changing the insulation of furnaces, etc. Other exposed workers include mechanics changing brake linings, workers processing asbestos gaskets, fitters and maintenance workers in shipyards, power stations and construction workers.

Clinical Features

Asbestos warts are formed when asbestos fibres enter the skin and provoke a low grade inflammatory reaction with hyperkeratosis. They eventually shell out taking the fibres with them.

Pleural plaques are formed of hyaline whorled fibrous tissue and take about 10 years to form and another 10 to calcify. They may occur on the parietal or interlobular pleura or the diaphragm. They tend to be 0.5 to 1.0 cm in thickness and are readily visible on CT or MRI scan but are difficult to see on X-ray until calcified. They neither interfere with breathing nor become malignant.

Benign pleural effusions are uncommon and their appearance is not dose related. They are more common in younger workers with short exposures. This condition often presents with a flu-like illness, shortness of breath and unilateral dyspnoea. The effusion contains polymorphonuclear leucocytes initially but these are replaced by lymphocytes. The condition usually resolves over several months but may leave behind mild pleural thickening. In some cases the thickening continues to form a cuirass-like fixation.

Bilateral diffuse pleural thickening may be the result of heavy exposure. The costophrenic angles are obliterated and there may be pleural thickening over the diaphragm. Thickening extending up the parietal pleura may cause restriction of ventilation of the lower lobes and in severe cases may immobilise the lung. It is difficult to differentiate from asbestosis by X-ray or spirometry, but in the absence of asbestosis can be treated by pleural stripping.

Asbestosis

Patients may first present with gradually increasing shortness of breath with chest tightness and restriction of inspiration. In non-smokers there is no cough or sputum production unless the disease is advanced. With heavy pure fibre exposure the classical radiological presentation is of lower zone bilateral irregular opacities with dry end-inspiratory crackles audible in the axillary region. Changes may be seen earlier on CT scan. With continued exposure the shadows become coarser and more profuse and crowding of the airways in the lower zones may give rise to a 'birch broom' effect in the lateral view. Although there is a fall in FVC the lung function tests are non-specific. There is usually a high FEV₁/FVC ratio, but this can be masked by smoking. There is a loss of compliance and gas transfer (measured by TLco fall). Clubbing is not a consistent feature in uncomplicated asbestosis.

Primary Lung Cancer

Asbestos exposure (particularly to amphiboles) even at a level insufficient to cause asbestosis produces around eight times the normal risk of primary lung cancer. Smoking further enhances this risk. The first sign is often of clubbing in a known asbestosis case. The tumours are indistinguishable from those caused by cigarette smoking though the proportion of adenocarcinomas is higher. The prognosis is poor.

Mesothelioma

This disease is predominantly associated with amphibole exposure and has a mean latent period of over 40 years. The usual site is parietal, but some may arise in the pericardium or peritoneum. There is usually a serous or bloody effusion and malignant spread may occur to the visceral pleura or peritoneum. Inflammatory reaction may produce a cuirass of cartilaginous consistency.

The most common presentation is with shortness of breath due to pleural effusion. This may be of sudden or gradual onset. Signs are of an effusion, thickening and mediastinal shift. Lymph nodes are not enlarged and there may be minimal clubbing. Aspiration may be difficult in the presence of a cuirass and may seed tumour into and through the chest wall. Prognosis is extremely poor. Death may be from pulmonary embolism or infarction or from constriction of the lungs or mediastinal structures.

Peritoneal mesothelioma is rare and is usually associated with exceptionally heavy crocidolite exposure. Concomitant lung fibrosis is usual in these cases. The presentation is often of abdominal discomfort, change in bowel habit and weight loss and physical examination may reveal abdominal swelling, ascites and ill defined masses.

Latent Period

Due to the very long latent period of asbestos related disease, many of the patients presenting will give an occupation with no connection to asbestos work. A full occupational history will be required to ascertain the link with asbestos. The incidence of mesothelioma is still rising but will fall due to reduced use of amphiboles, though some inadvertent exposure is still likely.

Occupational Deafness

Noise Induced Hearing Loss

The effect of noise on hearing may be temporary or permanent. Temporary threshold shift (TTS) is a transitory hearing loss with a return to normal hearing. The time to recovery is variable and the degree of TTS depends on the intensity and duration of the provoking sound. It may be accompanied by tinnitus (which may become permanent if exposure is continued).

Occupational deafness or occupational noise induced hearing loss (NIHL) is irreversible permanent hearing loss which usually commences at 4 kHz but may extend to other frequencies with continued exposure. The mechanism is most likely of mechanical and metabolic damage to the hair cells of the Organ of Corti. These cells are most vulnerable in the region of 8 to 10 mm from the base of the cochlea which corresponds to the 4 kHz region.

Clinical Diagnosis

The clinical diagnosis of occupational deafness is based on a history of noise in the workplace rather than elsewhere, a clinical examination to exclude other forms of hearing loss and an audiological profile consistent with NIHL.

Audiometry

Pure tone audiometry should be conducted for each ear, usually from 125 Hz to 8kHz. The audiometer should be properly calibrated and the test conducted in a quiet place, preferably a soundproof booth. It is of value to perform air and bone testing.

The classic audiological profile of noise induced hearing loss is a dip in the audiogram at about 4 kHz with a slightly smaller amount of loss immediately above and below. It is usually symmetrical, although some exposures (e.g. rifle shooting) may result in unilateral hearing loss. Continued exposure results in the dip extending, the higher frequencies being more affected. The absence of an air-bone gap in the profiles excludes high frequency conductive loss.

Sensorineural hearing loss amounting to at least 40 dB in each ear, being due to the case of at least one ear to noise and being the average pure tone loss measured by audiometry over the 1, 2 and 3 kHz frequencies, and with 5 or 10 years history of noise exposure at work as specified in the Occupational Deafness (Compensation) Ordinance is eligible for compensation.

INDEX

A

Acid anhydrides 66
 Actinomycetes..... 19
 Agricultural workers..... 16
 Alopecia..... 2
 Amosite.....69
 Amphiboles69, 70
 Angiosarcoma 35
 Aniline..... 41, 55
 Anthophyllite69
 Aplastic anaemia 40, 42
 Arsine33, 35
 Arterial gas embolism..... 7
 Avian influenza A 28

B

Bagassosis 19
 Barotrauma..... 7, 76
 Batteries..... 30, 32, 37, 51
 Beat elbow 12, 75, 77
 Beat hand 10, 12, 75, 77
 Beat knee 11, 12, 75, 77
 Becquerel..... 2
 Bird fancier's lung..... 19
 Boilers..... 69
 Boot making 63
 Butchers 16, 21, 24

C

Caissons..... 7
 Cancer 2-4, 35, 44, 50, 51, 53, 55, 57, 63, 70, 75, 80
 Carbon tetrachloride..... 44, 47
 Cataract..... 2, 3, 4, 5, 6, 43
 Carpal tunnel syndrome 14
 Cellulitis 10-12, 77
 Chloracne.....47, 60, 61, 81
 Cholinesterase33, 34, 78
 Chromic acid.....63, 80
 Chrysotile 69
 Colophony 66
 Compressed air 7, 75
 Compression barotraumas..... 7
 Contact dermatitis 49, 60, 61
 Cooks 24
 Cooling towers 26
 Cor pulmonale 49, 68
 Cotton spinners 65
 Crocidolite 69,70

D

Dairy workers 18, 21
 Decompression illness..... 7
 Degreaser's flush..... 45
 Dental amalgam 37
 Detonators 37
 Di-isocyanates..... 66
 Diving 7
 Dry-cleaning 44, 45
 Dysbaric osteonecrosis..... 7

E

Eczema..... 60
 Electroplating..... 51
 Endotoxin 65
 Erethism 37, 38
 Explosives 33, 40, 48

F

Farcy 17
 Farmers 17, 18, 21, 16-25, 28
 Farmer's lung 19, 77
 Flexor retinaculum..... 14
 Flour 66, 82
 Formaldehyde 66, 82
 Fulminate 37, 61, 62
 Fungicide37

G

Gingivitis..... 37
 Granulomata 21, 49, 50
 Golfer's elbow 13

H

Haematuria 55
 Haemoglobinuria 35
 Haemolytic anaemia 41
 Haemopoietic syndrome..... 2
 Health care workers 22, 23, 27, 75
 Hepatocellular carcinoma 23
 Hilar lymphadenopathy 49
 Hot water systems..... 26
 Hypersensitivity pneumonitis 16

INDEX

I

Infrared 4, 6
 Ionisation 2

J

Jaundice 18, 35, 42-44

L

Laboratory animals 2, 66
 Lateral epicondylitis 13
 Legionnaires' disease 26, 75, 82
 Leukaemia 3, 40
 Lung cancer 2, 35, 50, 51, 70

M

Malt worker's lung 16
 Manganic madness 32
 Meat processor 24
 Medial epicondylitis 13
 Median nerve 14
 Meningitis 18, 21
 Metallothionine 51
 Methaemoglobin 35, 41
 Mesothelioma 70, 82
 Miners 2, 11
 Munitions 33
 Mushroom worker's lung 19
 Myeloma 40

N

Nasal septum 17, 54, 62
 Needlestick injuries 23
 Nephrotoxicity 45, 51
 Nerve conduction studies 14
 Nickel 60, 61, 63
 Nitric acid 48
 Nitric oxide 48
 Nitrobenzene 41, 42
 Nitrogen dioxide 48
 Nuclear power 2

O

Optic atrophy 44
 Ovine abortion agent 25

P

Paints 2, 4, 30, 32, 37, 56, 58
 Papillary necrosis 37
 Patch testing 61
 Parkinsonism 32, 37
 Peak expiratory flow rates 66
 Perchloroethylene 45
 Peripheral neuropathy 30, 38
 Peritendonitis crepitans 13
 Pesticides 34, 35
 Petrol 30, 31, 40, 56
 Pet shop workers 25, 28
 Phalen's sign 14
 Phosphine 33
 Phossy jaw 33
 Photographic developer 43, 58
 Platinum salts 66, 82
 Pleural plaques 69
 Pneumonitis, pneumonia .. 19, 25, 27, 28, 37, 49, 51
 Pneumothorax 7, 49
 Poultry 25, 28
 Proteinuria 51
 Provocative tests 67
 Psychoses 39
 Pulmonary fibrosis 19
 Pulmonary hypertension 19, 49

Q

Quarrying 68
 Quartz 68
 De Quervain's disease 13

R

Radiation 2-6
 Radiographers 2
 Radiologists 2
 Radionecrosis 2
 Radium 2
 Radon 2
 Repetitive movements 9, 13
 Rhinitis 63

INDEX

S

Sarcoidosis 50
 Sarcoma 35, 50
 Sensorineural hearing loss 72
 Sevese Acute Respiratory Syndrome (SARS) ... 27
 Sewage workers 18
 Shoe making, repair 58, 63
 Silage 48
 Silicotuberculosis 68
 Skin burn 2, 4
 Skin cancer 35, 53, 57
 Skin prick test 66
 Slaughterhouse workers 21, 24
 Soda ash 61, 62
 Stainless steel 55
 Stenosing tenovaginitis 13
 Sterility 2
 Stochastic effects 2

T

Tanneries 16
 Taxidermists 25
 Telegraphist's cramp 9
 Tennis elbow 13
 Tenosynovitis 13, 14
 Tetrachloroethylene 45
 Tetraethyl lead 31
 Tinel's sign 14
 1,1,1-trichloroethane 44
 Trichloroethylene 44
 Trigger finger 13
 Tunnelling 68

U

Ultraviolet radiation 5

V

Veterinary surgeons 17, 18, 21, 25, 28
 Vibrating hand tools 14

W

Weil's disease 18
 Wood dust 63
 Writer's cramp 9

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

Annex I

OCCUPATIONAL SAFETY AND HEALTH ORDINANCE NOTIFICATION OF OCCUPATIONAL DISEASES

To : Commissioner for Labour

PARTICULARS OF PATIENT

Name: _____ HKID/Passport no.: _____

Male/Female* Date of birth: ___ / ___ / ___ Occupation: _____

Home address: _____

Telephone no. (Home) _____ (Office) _____ (Pager/Mobile) _____

Name and address of employer: _____

_____ Telephone no. (Employer) _____

Workplace address (if different from employer's address): _____

For Internal
use:

Code: _____

Code: _____

Code: _____

Code: _____

NOTIFIABLE OCCUPATIONAL DISEASES (Please put a tick in)

<input type="checkbox"/> 1	Radiation Illness	<input type="checkbox"/> 18	Lead Poisoning	<input type="checkbox"/> 35	Chrome Ulceration
<input type="checkbox"/> 2	Heat Cataract	<input type="checkbox"/> 19	Manganese Poisoning	<input type="checkbox"/> 36	Urinary Tract Cancer
<input type="checkbox"/> 3	Compressed Air Illness	<input type="checkbox"/> 20	Phosphorus Poisoning	<input type="checkbox"/> 37	Peripheral Polyneuropathy
<input type="checkbox"/> 4	Cramp of Hand or Forearm	<input type="checkbox"/> 21	Arsenic Poisoning	<input type="checkbox"/> 38	Localised Papillomatous or Keratotic New Skin Growth
<input type="checkbox"/> 5	Beat Hand	<input type="checkbox"/> 22	Mercury Poisoning	<input type="checkbox"/> 39	Occupational Vitiligo
<input type="checkbox"/> 6	Beat Knee	<input type="checkbox"/> 23	Carbon Bisulphide Poisoning	<input type="checkbox"/> 40	Occupational Dermatitis
<input type="checkbox"/> 7	Beat Elbow	<input type="checkbox"/> 24	Benzene Poisoning	<input type="checkbox"/> 41	Chemical Induced Upper Respiratory Tract Inflammation
<input type="checkbox"/> 8	Tenosynovitis of Hand or Forearm	<input type="checkbox"/> 25	Poisoning by Nitro-, Amino-, or Chloro-Derivatives of Benzene	<input type="checkbox"/> 42	Nasal or Paranasal Sinus Cancer
<input type="checkbox"/> 9	Anthrax	<input type="checkbox"/> 26	Dinitrophenol Poisoning	<input type="checkbox"/> 43	Byssinosis
<input type="checkbox"/> 10	Glanders	<input type="checkbox"/> 27	Poisoning by Halogen Derivatives of Hydrocarbons	<input type="checkbox"/> 44	Occupational Asthma
<input type="checkbox"/> 11	Leptospirosis	<input type="checkbox"/> 28	Diethylene Dioxide Poisoning	<input type="checkbox"/> 45	Silicosis
<input type="checkbox"/> 12	Extrinsic Allergic Alveolitis	<input type="checkbox"/> 29	Chlorinated Naphthalene Poisoning	<input type="checkbox"/> 46	Asbestos-Related Diseases
<input type="checkbox"/> 13	Brucellosis	<input type="checkbox"/> 30	Poisoning by Oxides of Nitrogen	<input type="checkbox"/> 47	Occupational Deafness
<input type="checkbox"/> 14	Tuberculosis in health care workers	<input type="checkbox"/> 31	Beryllium Poisoning	<input type="checkbox"/> 48	Carpal Tunnel Syndrome
<input type="checkbox"/> 15	Parenterally Contracted Viral Hepatitis in health care workers	<input type="checkbox"/> 32	Cadmium Poisoning	<input type="checkbox"/> 49	Legionnaires' Disease
<input type="checkbox"/> 16	Streptococcus suis Infection	<input type="checkbox"/> 33	Dystrophy of the Cornea	<input type="checkbox"/> 50	Severe Acute Respiratory Syndrome
<input type="checkbox"/> 17	Avian Chlamydiosis	<input type="checkbox"/> 34	Skin Cancer	<input type="checkbox"/> 51	Avian Influenza A

Diagnosis: Confirm/Suspect*

Date of onset of illness: _____ / _____ / _____

Follow-up of patient: Treated/Referred to hospital/Others(specify)*: _____

Other relevant information: _____

Name of notifying medical practitioner: _____

Address of notifying medical practitioner: _____

Telephone no. of notifying medical practitioner: _____

Fax no. of notifying medical practitioner: _____

Date: _____

Signature: _____

**Delete whichever is inapplicable*

Please return this form by fax (no. 25812049) or by mail to Occupational Health Service, Labour Department, 15/F Harbour Building, 38 Pier Road, Central, Hong Kong.

For details of Notifiable Occupational Diseases and their related occupations, please refer to Schedule 2 of the Occupational Safety & Health Ordinance and to the Labour Department publication "Guidance Notes on the Diagnosis of Notifiable Occupational Disease". Enquiry telephone no.: 2852 4041.

LD483 (Rev. 8.2.2005)

Annex II

Extract from Occupational Safety and Health Ordinance

15. Medical practitioner to notify occupational disease to Commissioner for Labour

- (1) If, on examining an employee or a former employee or the body of a person who was immediately before the death an employee or former employee, a medical practitioner -
 - (a) finds or suspects that the employee or former employee is or was suffering from an occupational disease specified in Schedule 2; and
 - (b) believes that the disease was or may have been attributable to an occupation specified in column 3 of that Schedule,
 the practitioner must notify the finding or suspicion to the Commissioner.
- (2) The notification must be in writing and on a form provided or approved by the Commissioner for Labour and must be lodged as soon as practicable after the conclusion is formed.
- (3) A medical practitioner who, without reasonable excuse, fails to comply with this section commits an offence and is liable on conviction to a fine at level 3.

**SCHEDULE 2
NOTIFIABLE OCCUPATIONAL DISEASES**

Item	Disease	Occupation
1.	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.	Any occupation involving exposure to electro-magnetic radiations other than radiant heat, or to ionising particles.
2.	Heat cataract.	Any occupation involving frequent or prolonged exposure to rays from molten or red-hot material.
3.	Dysbarism, including decompression sickness, barotraumas and osteonecrosis.	Any occupation involving subjection to compressed or rarefied air or other gases or gaseous mixtures.
4.	Cramp of the hand or forearm due to repetitive movements.	Any occupation involving prolonged periods of handwriting, typing or other repetitive movements of the fingers, hand or arm.
5.	Subcutaneous cellulites of the hand (beat hand).	Any occupation involving manual labour causing severe or prolonged friction or pressure on the hand.
6.	Bursitis or subcutaneous cellulites arising at or about the knee due to severe or prolonged external friction or pressure at or about the knee (beat knee).	Any occupation involving manual labour causing severe or prolonged external friction or pressure at or about the knee.
7.	Bursitis or subcutaneous cellulites arising at or about the elbow due to severe or prolonged external friction or pressure at or about the elbow (beat elbow).	Any occupation involving manual labour causing severe or prolonged external friction or pressure at or about the elbow.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

8. Traumatic inflammation of the tendons of the hand or forearm (including elbow), or of the associated tendon sheaths (Amended L.N. 147 of 1999)
- Any occupation involving manual labour, or frequent or repeated movements of the hand or wrist.
9. Anthrax.
- Any occupation involving contact with animals infected with anthrax or the handling (including the loading and unloading or transport) of animal products or residues.
10. Glanders.
- Any occupation involving contact with equine animals or their carcasses.
11. Infection by leptospira.
- Any occupation involving -
- (a) work in places which are, or are liable to be, infested by rats, field mice or voles, or other small mammals; or
 - (b) work at dog kennels or the care or handling of dogs; or
 - (c) contact with bovine animals or their meat products or pigs or their meat products.
12. Pulmonary disease due to the inhalation of the dust of mouldy hay or other mouldy vegetable produce and characterized by symptoms and signs attributable to a reaction in the peripheral part of the bronchopulmonary system, and giving rise to a defect in gas exchange (farmer's lung).
- Any occupation involving exposure to the dust of mouldy hay or other mouldy vegetable produce attributable to employment in -
- (a) agriculture, horticulture or forestry; or
 - (b) loading or unloading or handling in storage the hay or produce; or
 - (c) handling bagasse.
13. Infection by organisms of the genus brucella.
- Any occupation involving contact with -
- (a) animals infected by brucella; or
 - (b) the carcasses or any parts of those animals; or
 - (c) any untreated products derived from those carcasses or parts; or
 - (d) laboratory specimens or vaccines of, or containing, brucella.
14. Tuberculosis.
- Any occupation involving close and frequent contact with a source of tuberculosis infection that is attributable to employment -
- (a) in the medical treatment or nursing of a person or persons suffering from tuberculosis, or in a service ancillary to that treatment or nursing; or
 - (b) in attending to a person suffering from tuberculosis, where the need for attendance arises because of the person's physical or mental infirmity; or
 - (c) as a research worker engaged in research in connection with tuberculosis; or
 - (d) as a laboratory worker, pathologist or post-mortem worker, where the employment involves working with materials that are a source of tuberculosis infection; or
 - (e) in any occupation ancillary to employment in an occupation specified in paragraph (d).

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

- | | |
|---|---|
| 15. Parenterally contracted viral hepatitis. | Any occupation involving contact with -
(a) human blood or human blood products; or
(b) a source of viral hepatitis. |
| 16. Infection by <i>Streptococcus suis</i> . | Any occupation involving contact with pigs infected by <i>Streptococcus suis</i> , or with the carcasses, products or residues of pigs so infected. |
| 17. Avian chlamydiosis. | Any occupation involving contact with birds infected with chlamydia psittaci, their remains or untreated products. |
| 18. Poisoning by lead or a compound of lead. | Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes, dust or vapour of, lead or a compound of lead, or a substance containing lead. |
| 19. Poisoning by manganese or a compound of manganese. | Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes, dust or vapour of, manganese or a compound of manganese, or a substance containing manganese. |
| 20. Poisoning by phosphorus or an inorganic compound of phosphorus or the anti-cholinesterase or pseudo anti-cholinesterase action or organic phosphorus compounds. | Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes, dust or vapour of, phosphorus or a compound of phosphorus, or a substance containing phosphorus. |
| 21. Poisoning by arsenic or a compound of arsenic. | Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes, dust or vapour of, arsenic or a compound of arsenic, or a substance containing arsenic. |
| 22. Poisoning by mercury or a compound of mercury. | Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes, dust or vapour of, mercury or a compound of mercury, or a substance containing mercury. |
| 23. Poisoning by carbon bisulphide. | Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes, or vapour of, carbon bisulphide or a compound of carbon bisulphide, or a substance containing carbon bisulphide. |
| 24. Poisoning by benzene or homologue of benzene. | Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes of, or vapour containing, benzene or any of its homologues. |

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

25. Poisoning by a nitro-derivative, amino-derivative or chloro-derivative of benzene or of a homologue of benzene, or poisoning by nitro-chlorobenzene. Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes of, or vapour containing, a nitro-derivative, amino-derivative or chloro-derivative of benzene or nitro-chlorobenzene.
26. Poisoning by dinitrophenol or a homologue or by substituted dinitrophenols or by the salts of those substances. Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes of, or vapour containing, dinitrophenol or a homologue or substituted dinitrophenols or the salts of those substances.
27. Poisoning by halogen derivatives of hydrocarbons of the aliphatic series. Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes of, or vapour containing, halogen derivatives of hydrocarbons of the aliphatic series.
28. Poisoning by diethylene dioxide (dioxin). Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes of, or vapour containing, diethylene dioxide (dioxin)
29. Poisoning by chlorinated naphthalene. Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes of, or dust or vapour containing, chlorinated naphthalene.
30. Poisoning by oxides of nitrogen. Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes of, or dust or vapour containing, oxides of nitrogen.
31. Poisoning by beryllium or a compound of beryllium. Any occupation involving -
(a) the use or handling of; or
(b) exposure to the fumes, dust or vapour of, beryllium or a compound of beryllium or a substance containing beryllium.
32. Poisoning by cadmium. Any occupation involving -
(a) the use or handling of; or
(b) exposure to the dust or fumes of, cadmium.
33. Dystrophy of the cornea of the eye (including ulceration of the corneal surface). Any occupation involving -
(a) the use or handling of; or
(b) exposure to, arsenic, tar, pitch, bitumen, mineral oil (including paraffin) or soot, or any compound, product or residue of any of those substances.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

34. Primary epitheliomatous cancer of the skin. Any occupation involving -
(a) the use or handling of; or
(b) exposure to,
arsenic, tar, pitch, bitumen, mineral oil (including paraffin) or soot, or any compound, product or residue of any of those substances.
35. Chrome ulceration including perforation of nasal septum. Any occupation involving the use or handling of -
(a) chromic acid, chromate or bichromate or ammonium, potassium, sodium or zinc; or
(b) any preparation or solution containing any of those substances.
36. Primary neoplasm of the epithelial lining of the urinary tract (renal pelvis, ureter, bladder and urethra), including papilloma, carcinoma-in-situ and invasive carcinoma. Any occupation involving the production, use or handling of -
(a) alpha-naphthylamine, betanaphthylamine or methylene-bis-orthochloraniline, or disphenyl substituted by at least one nitro or primary amino group or by at least one nitro and primary amino group (including benzidine); and
(b) any of those substances if further ring substituted by halogeno methyl or methoxyl group and the salts of any of those substances and the production of auramine and magenta.
37. Peripheral poly-neuropathy. Any occupation involving -
(a) the production, use or handling of; or
(b) exposure to,
any physical form of, or any preparation or solution containing n-Hexane or methyl-n-butyl ketone.
38. Localised new growth of the skin, papillomatous or keratotic. Any occupation involving -
(a) the use or handling of; or
(b) exposure to,
arsenic, tar, pitch, bitumen, mineral oil (including paraffin) or soot, or any compound, product or residue of any of those substances.
39. Occupational vitiligo. Any occupation involving -
(a) the use or handling of; or
(b) exposure to,
paratertiary-butyl phenol, paratertiary-butyl catechol, para-amyl-phenol, hydroquinone or the monobenzyl or monobutyl ether of hydroquinone.
40. Inflammation or ulceration of the skin produced by dust, liquid or vapour (including the condition known as chloracne but excluding chrome ulceration). Any occupation involving exposure to dust, liquid or vapour, where the exposure is capable of irritating the skin.
41. Inflammation or ulceration of the mucous membrane of the upper respiratory passages or mouth produced by dust, liquid or vapour. Any occupation involving exposure to dust, liquid or vapour.

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

42. Carcinoma of the nasal cavity or associated air sinuses (nasal carcinoma). Any occupation involving -
- (a) the manufacture or repair of wooden goods; or
 - (b) the manufacture or repair of footwear or components of footwear made wholly or partly of leather or fibre board.
43. Byssinosis. Any occupation involving exposure to raw cotton dust.
44. Occupational asthma. Any occupation involving the use or handling of, or exposure to, any of the following agents which may irritate or sensitise the respiratory system -
- (a) isocyanates;
 - (b) platinum salts;
 - (c) fumes or dusts arising from the manufacture, transport or use of hardening agents (such as epoxy resin curing agents) based on phthalic anhydride, trimellitic anhydride or triethylenetetramine;
 - (d) fumes arising from the use of rosin as a soldering flux;
 - (e) formaldehyde;
 - (f) proteolytic enzymes;
 - (g) animals or insects used for the purposes of research or education or in laboratories;
 - (h) dusts arising from the sowing, cultivation, harvesting, drying, handling, milling, transport or storage of barley, oats, rye, wheat or maize, or the handling, milling, transport or storage of meal or flour made from them;
 - (i) any other sensitising agent inhaled at work. (Added L.N. 147 of 1999)
45. Silicosis. Any occupation.
46. Asbestos-related diseases (such as asbestosis and mesothelioma) Any occupation.
47. Occupational deafness. Any occupation.
48. Carpal tunnel syndrome. Any occupation involving repetitive use of hand-held powered tools whose internal parts vibrate so as to transmit that vibration to the hand, but excluding those which are solely powered by hand. (Added L.N. 147 of 1999)
49. Legionnaires' disease. Any occupation involving the repair, maintenance or service of -
- (a) cooling systems that use fresh water, or
 - (b) hot water service systems. (Added L.N. 147 of 1999)

GUIDANCE NOTES ON THE DIAGNOSIS OF NOTIFIABLE OCCUPATIONAL DISEASES

50. Severe acute respiratory syndrome

Any occupation involving close and frequent contacts with a source or sources of severe acute respiratory syndrome infection that is attributable to employment-

- (a) in the medical treatment or nursing of a person suffering from severe acute respiratory syndrome, or in a service ancillary to that treatment or nursing; or
- (b) in attending to a person suffering from severe acute respiratory syndrome, where the need for attendance arises because of the person's physical or mental infirmity; or
- (c) in identifying, detecting, tracing, isolating, detaining, supervising or surveillance of a person suffering from severe acute respiratory syndrome; or
- (d) as a research worker engaged in research in connection with severe acute respiratory syndrome, or in a service ancillary to that research; or
- (e) as a laboratory worker, pathologist post-mortem worker or funeral services worker, where the employment involves the handling of any human body or other materials that are a source of severe acute respiratory syndrome infection, or in a service ancillary to that handling. (Added L.N. 214 of 2004)

51. Avian influenza A

Any occupation involving close and frequent contacts with a source or sources of avian influenza A infection that is attributable to employment-

- (a) as a worker engaged in the handling of poultry or birds or their uncooked remains or residues, or their untreated products, that are a source of avian influenza A infection, or in a service ancillary to that handling; or (Amended L.N. 14 of 2005)
- (b) as a research worker engaged in research in connection with avian influenza A, or in a service ancillary to that research; or
- (c) as a laboratory worker engaged in the handling of materials that are a source of avian influenza A infection, or in a service ancillary to that handling. (Added L.N. 214 of 2004)