Workplace Safety and Health Guidelines

Diagnosis and Management of Occupational Diseases

WSHCOUNCIL

Tripartite Alliance for Workplace Safety and Health

Foreword

Work serves as a vital source of income, fosters a sense of purpose and continually enhances our knowledge and skills. At the heart of all work activities are humans, who form the crucial nexus between machines, transactions, paperwork and professional interactions. By prioritising and maintaining the health of workers, workplaces can experience significant increases in productivity, improved employee satisfaction and ultimately, better business outcomes.

The key to preventing occupational diseases (ODs) is to start right at the source, from implementing upstream risk control measures in workplaces, to raising capabilities of the medical community in detecting and diagnosing ODs and placing workers under regular occupational health surveillance.

Doctors play an important role in detection and diagnosis. They are required to fulfil their professional duties and legal responsibilities, so that ODs are duly reported under the Workplace Safety and Health (WSH) Act and affected patients receive timely compensation under the Work Injury Compensation Act. To aid medical practitioners, we have revised and updated the WSH Guidelines on the Diagnosis and Management of Occupational Diseases.

In this revision, we have expanded the chapters by adding details on the conditions and diagnostic criteria for occupational cancers, occupational infectious diseases, noise induced hearing loss, pesticide and fumigant poisoning, and work-related musculoskeletal disorders and others.

Your role as a doctor in recognising and managing ODs is of utmost importance as early detection and intervention can minimise or even prevent morbidity and disability arising from these diseases. Your medical contributions can also result in the protection of other employees who may be exposed to similar risks.

We extend our partnership to all doctors, to play their role in achieving our shared vision of 'A Healthy Workforce in Safe Workplaces; A Country Renowned for Best Practices in Workplace Safety and Health.'

I would like to thank the experts, medical practitioners who have contributed and all who have given their valuable feedback to the development of this revised guide. I hope that you will find this guide a convenient and practical resource in your clinical practice.

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Contents

1. Diagnosis, Reporting and Compensation of Occupational Diseases in Singapore

The term occupational disease (OD) covers any disease contracted as a result of an exposure to risk factors arising from work activity[1]. Risk factors include exposure to physical, chemical, biological, ergonomic or psychosocial hazards at the workplace.

Doctors have a legal responsibility to report ODs under the Workplace Safety and Health (WSH) Act. In Singapore, ODs listed in the Second Schedule of the WSH Act are reportable (refer to Appendix C). The three most common ODs reported in Singapore are noise-induced hearing loss, work-related musculoskeletal disorders and occupational skin diseases.

It is important for doctors and healthcare professionals to recognise occupational and work-related diseases among their patients. This is to prevent them from being disabled by the disease and to ensure workers can claim rightful compensation. When ODs are reported, investigations will be triggered to establish causality so that preventive measures can be put in place.

1.1 Diagnosis of Occupational Diseases

As some ODs may have a latency period, the diagnostic process should include:

- Taking a detailed occupational history and medical history.
- Conducting clinical examinations for specific symptoms and signs of ODs.
- Conducting relevant investigations, including review of safety data sheets (SDS) and workplace evaluation to document exposure, and exclude non-occupational causes.

[1] International Labour Organisation (ILO), P155 - Protocol of 2002 to the Occupational Safety and Health Convention, 1981 (No. C155).

1. **A detailed occupational history includes the following:**
   - Work process (e.g. work tasks, tools used)
   - Exposure to hazards (e.g. dust, fumes, radiation, chemicals, biological, physical hazards)
   - Route of exposure (e.g. inhalation, skin, ingestion)
   - Frequency, duration and intensity of exposure
   - Use of PPE (e.g. gloves, respirators)
   - Presence of engineering controls (e.g. process enclosure, local exhaust ventilation, general ventilation)
   - Work organisation (e.g. work hours, breaks, overtime)

2. **Correlate the occupational history with symptoms in relation to:**
   - Onset and duration of symptoms (temporal relationship between exposure and onset of symptoms)
   - Aggravating factors
   - Effects of off-days and holidays
   - Other workers affected

3. **Ask for the work history and for the relevant information for previous jobs held.**

1.2 Reporting of Occupational Diseases

All registered medical practitioners are required to report any of the ODs listed in the Second Schedule of the WSH Act (Appendix C) within 10 days from the diagnosis of the disease under the WSH (Incident Reporting) Regulations. All notifications should be made via the electronic notification system at www.mom.gov.sg/ireport.

Non-compliance with the reporting requirements may result in a fine of up to $10,000 for first offence, and up to $20,000 and/or an imprisonment for a second or subsequent offence.

A robust notification system provides important data for understanding the extent and depth of the occupational safety and health status in Singapore. It helps the authorities to identify persons and industries at risk to better manage workplace hazards and identify new and emerging ones.

Doctors can refer an employee or platform worker (PW) to Occupational Medicine (OM) specialist clinics for further investigation if they are unsure if a disease is work-related. A list of OM Specialist clinics is available on the Ministry of Manpower's (MOM) website.

Upon diagnosing an OD, doctors should inform the employer or platform operator (PO) of the diagnosis in writing, so that they can fulfil their obligations to notify MOM. It is also good practice for doctors to inform the employee/PW suffering from ODs about (i) keeping their employers or POs updated on their diagnosis and management, and (ii) the employers' and POs' obligations to report to MOM.

When making an electronic notification to MOM, doctors should have the following information and documents ready for upload: (i) diagnosis of the OD, (ii) employer's or PO's name, (iii) employer's or PO's address, (iv) worker's occupation, and (v) relevant memos and investigation reports.

1.3 Compensation for Occupational Diseases

Under the **Work Injury Compensation (WIC) Act**, employers and POs are liable to pay compensation:

- For diseases if an employee or PW contracts any of the ODs listed in the Second Schedule of the Act, or
- Contracted a disease arising from exposure to biological or chemical agents at work that is not specified in the Second Schedule.

The WIC Act provides a low-cost and expeditious alternative to making a claim. Unlike common law, WIC Act is a no-fault system. To be eligible for compensation, the employee or PW only needs to show that the OD arose out of and in the course of employment/work.

The WIC Act covers all employees under a contract of service regardless of salary, age and citizenship except for (i) independent contractors and the self-employed, (ii) domestic workers, and (iii) uniformed personnel including members of the Singapore Armed Forces, Singapore Police Force, Singapore Civil Defence Force, Central Narcotics Bureau and Singapore Prison Service.

Employees and PWs can claim compensation under the WIC Act or through a civil claim against his employer or PO, but not both. In other words, the claimant will not be able to claim from both the WIC Act and common law.

The WIC Act allows employees and PWs who contracted an OD to claim the following compensation from their employers or POs:

- Medical leave wages for employees or income loss compensation for PWs when issued with medical leave (for employees and PWs) or light duty (for employees only), due to the OD
- Medical expenses, including hospital bills, medication and other charges, due to the OD
- Lump sum compensation for permanent incapacity and death

Doctors are required to refer to the MOM's "Guide to the Assessment of Traumatic Injuries and Occupational Diseases for Work Injury Compensation" when assessing the amount of permanent incapacity for the various injuries and diseases. Further information on the calculation of compensation benefits is available on MOM's website.

1.4 Other Relevant WSH Legislation

Under the WSH (Medical Examinations) Regulations, workers employed in occupations with exposure to certain hazards are required to undergo specific medical examinations. The medical examinations and tests help to detect workers with ODs or overexposure early and ensure that workers remain fit for such work. These medical examinations are conducted by Designated Workplace Doctors (DWDs) registered with MOM.

1.5 Further Reading

1. Workplace Safety and Health Act
2. Work Injury Compensation Act
3. WSH (Incident Reporting) Regulations
4. WSH (Medical Examinations) Regulations
5. MOM Webpage – Workplace Safety and Health
6. Guide to the Assessment of Traumatic Injuries and Occupational Diseases for Work Injury Compensation

2. Occupational Diseases

2.1 Compressed Air Illness and Barotrauma

2.1.1 Introduction

Compressed air illness (CAI) and barotrauma can occur in workers who may be exposed to work environments where there are:

- Significantly increased or decreased ambient pressure or,
- Rapid changes in such pressures

CAI can be further classified into decompression illness (DCI) and dysbaric osteonecrosis. DCI is due to the absorption of inert gases during the compressed air exposure. Mild cases (Type 1) present with rashes, niggles and pain. More serious cases (Type 2) present with respiratory, cardiovascular and central nervous system symptoms. Dysbaric osteonecrosis may result from chronic long-term exposure damaging the long bones of the hips and shoulder joints.

Barotrauma can affect the air-filled spaces in the body, e.g. sinuses, ear and lungs, and is due to acute pressure changes leading to injury to the ears, sinuses and other air-filled cavities in the body.

These conditions can occur in divers, as well as workers in tunnelling projects, who work in environments with compressed air. Cases of CAI from tunnelling projects in Singapore are usually seen at company appointed doctors.

2.1.2 Clinical Presentation

CAI may present with joint pains, rashes and other symptoms relating to respiratory and nervous systems. Severe cases may develop intravascular coagulation as the excessive gas triggers the coagulation cascade. 98% of DCI occurs within 24 hours, and should be suspected if the worker presents with symptoms and had been working in compressed air environment. In cases of barotrauma, pain and bleeding can occur over the affected site. See Table 1 for details on presentations of CAI and barotraumas.

Table 1: **Clinical presentations of compressed air illness (CAI) and barotrauma**

Condition		Presentation
Compressed Air Illness (CAI)	**Type1 Decompression Illness (DCI)**	Joints - Acute pain around major joints Skin - Urticarial and bluish-red mottling - Itch
	Type 2 DCI	Neurological - Vertigo, pins and needles, headache, paraesthesia, hypoaesthesia - Ataxic gait, hyperreflexia, Babinski's sign, paralysis and weakness of limbs, seizures, vomiting, visual loss or visual field defect, incontinence, impaired speech, tremors and coma Respiratory - Chest tightness, substernal pain, severe coughing, dyspnoea - Pulmonary oedema and shallow respiration
	Dysbaric osteonecrosis	- Usually asymptomatic unless there is joint involvement resulting in pain and limitation of movement - Condition may take months or years to develop
Barotrauma	**Sinus barotrauma**	- Acute pain over sinus and/or nasal area(s) - Post nasal bleeding
	Middle ear barotrauma	- Otalgia, hearing loss - Haemorrhagic exudates or burst eardrum accompanied with hypoacusis, tinnitus, vertigo when cold water enters middle ear
	Inner ear barotrauma	- Severe vertigo - Hearing loss in the affected side
	Pulmonary barotrauma	- Chest pain, breathlessness, unconsciousness, - Subcutaneous emphysema

Risk factors for compressed air illness include:

- Significant medical history: e.g., obese and older workers may be at higher risk, cardiac conditions such as right to left cardiac shunting etc.
- High pressures: e.g. pressures more than 1 bar (greater risk with higher pressures)
- Long working hours in compressed air, for example, those working > 4 hours
- Failure to follow proper decompression procedures
- Multiple entries into compressed air environment, e.g. supervisors and engineers
- Repetitive underwater dives
- Lack of acclimatisation, for example, new workers, or workers returning from illness or long leave
- Heavy manual work in free air between shifts/dives
- Consecutive days of compressed air work (e.g. more than 6 days of CAW), or diving without a rest day

Risk factors for barotrauma include:

- Medical unfitness, e.g. those with pre-existing medical conditions workers, those who are unable to clear their ears or who have chronic diseases of the ear, air passages or the lungs (see also risk factors for compressed air illness above)
- Infections affecting the ear or upper respiratory tract
- Sudden changes of pressure
- Failure to follow proper compression procedures
- Smoking
- Coughing
- Breath-holding

2.1.3 Differential Diagnosis

Exclude non-occupational causes with similar symptoms such as those due to:

- Degenerative joint diseases
- Strains and sprains
- Infections
- Spinal cord compression e.g. tumour
- Multiple sclerosis
- Guillain-Barre syndrome
- Myasthenia gravis

2.1.4 Diagnostic Criteria of Work-Relatedness

A detailed occupational history of exposure to hyperbaric (high pressure) or hypobaric (low pressure) environment is critical. In addition, supporting documents of repeated dives or rapid ascents for divers and compressed air workers will aid in determining the diagnosis of CAI and/or Barotrauma.

2.1.5 Investigation to Establish Work-Relatedness

I. Take an occupational history to establish if there is exposure to a high or low pressure environment. The following work processes may involve the use of or exposure to compressed air environment:
   - Tunnelling works in compressed air environment, e.g., cutter head inspection, and replacement of tunnel boring machine cutting tools.
   - Caisson work on riverbeds.
   - Pressurisation checks, e.g., in aircraft.
   - Work in medical lock or recompression chambers.
   - Underwater diving.

II. Correlate the exposure history with individual symptoms and investigation results such as sinus, chest and long bone x-rays, audiograms (refer to Table 1).

    Ask for the worker's logbook and dive computer which will detail records of his work in compressed air environments or diving activities.

    Ask the company for exposure records which will have information on pressures used, workers' registers and barographs.

2.1.6 Management

Treatment for CAI usually involves the administration of 100% oxygen and recompression in a hyperbaric chamber according to specific treatment protocols. Medications may be used to alleviate pain and swelling.

Treatment for barotrauma may involve the use of antihistamines, decongestants, analgesic drugs and antibiotics depending on the severity of the case. Sinus x-rays, audiometric tests and tympanograms may be useful for further evaluation and management of the condition.

All cases should preferably be referred to the Appointed Medical Practitioner[2] or to a hyperbaric/diving physician for assessment and management. Contacts of the Appointed Medical Practitioner can be found on the worker's identity disc or logbook.

For assistance, contact the hyperbaric treatment centre at:
- Singapore General Hospital (SGH) Hyperbaric & Diving Medicine Centre: 6321 3427

[2] Under the WSH (Construction) Regulations, the Appointed Medical Practitioner is a doctor with the required qualifications, training and experience to manage workers for conditions associated with compressed air work, and is a registered Designated Workplace Doctor with the Ministry of Manpower.

I. Advice to workers

- Strictly follow suspension period to avoid further exposure.
- Undergo assessment by the Appointed Medical Practitioner for fitness to return to compressed air work.
- Upon return to compressed air work, follow advice on the contraindications of entry into compressed air environment, such as colds (e.g. running nose, blocked nose), sore throat, earache or chest infection.
- Follow acclimatisation procedures.

II. Advice to companies

- Review the workplace risk assessment and put measures in place to follow proper compression and decompression procedures.
- Review the need for compressed air and if lower pressures could be used.
- Notify MOM via WSH Incident Reporting of workers diagnosed with CAI or Barotrauma (CAI and Barotrauma are notifiable and compensable diseases).
- Find out more about requirements for medical examinations under the WSH (Medical Examinations) Regulations.

2.1.7 Further Reading

1. Workplace Safety and Health (Medical Examinations) Regulations
2. Workplace Safety and Health (Construction) Regulations
3. Workplace Safety and Health Guidelines: Compressed Air Works – Prevention of Compressed Air Illness and Barotrauma
4. Workplace Safety and Health Guidelines: Statutory Medical Examinations

2.2 Diseases Caused by Excessive Heat

2.2.1 Introduction

Diseases caused by excessive heat (heat disorders) can result from prolonged exposure to excessive heat causing the body's cooling mechanism to break down, especially in unacclimatised workers. A rise in the core body temperature may lead to temporary or permanent disturbances in bodily functions, which may be further aggravated by strenuous physical activity.

Heat acclimatisation involves the gradual increase in duration of exposure to performing moderate work under hot weather. New workers would need at least one to two weeks to adjust to the local weather conditions and should not start working at full workload in a hot environment upon arrival. Heat acclimatisation can take the form of a gradual increase in daily exposure to the hot working environment for up to 14 days.

[2] Under the WSH (Construction) Regulations, the Appointed Medical Practitioner is a doctor with the required qualifications, training and experience to manage workers for conditions associated with compressed air work, and is a registered Designated Workplace Doctor with the Ministry of Manpower.

2.2.2 Clinical Presentation

Heat disorders can be viewed as a continuum of illnesses ranging from mild presentations such as heat syncope and heat cramps to more severe presentations including heat exhaustion and heat stroke (refer to Table 2).

Table 2: Clinical presentations of heat disorders

Condition	Presentation
Heat syncope	- Transient loss of consciousness, dizziness, lightheadedness
Heat cramps	- Painful muscle cramps or spasms (most often in the legs)
Heat exhaustion (If untreated, has the potential to develop into life-threatening heat stroke)	- Core temperature usually in the range 37.7 °C to 40°C - Profuse sweating, cool clammy skin, tachycardia, hyperventilation - Nausea, vomiting, diarrhoea, headache, dizziness, light-headedness - Abdominal cramps - Fatigue, weakness and inability to continue strenuous physical exertion - Normal mental state and stable neurological status
Heat stroke (Fatal if not treated promptly)	- Core temperature usually above 40°C - Hot and dry skin - Central nervous system (CNS) changes include dizziness, drowsiness, confusion, irritability, aggressiveness, apathy, disorientation, loss of bladder and bowel functions, fainting, seizures and even coma - Cardiovascular deterioration - Multi-organ failure

Predisposing factors include:

- Older workers above 65 years old
- Pregnant workers
- Overweight or obese workers
- Workers newly assigned to outdoor work
- Lack of acclimatisation to working in hot environments, such as new or returning workers from countries with a cold climate
- Returning from long leave of more than one week
- Currently unwell or recovering from illness (e.g. respiratory infection, diarrhoea)
- Personal risk factors such as chronic diseases (e.g. heart disease, diabetes), medication use, or history of heat disorders
- Other risk factors include poor hydration, highly motivated worker*, alcohol consumption^ and fatigue

* Highly motivated workers may ignore the early signs of heat stress and continue to work until heat stroke occurs.

^Alcohol intake may reduce a worker's heat tolerance and can cause dehydration.

2.2.3 Differential Diagnosis

Exclude non-occupational causes with similar symptoms such as those due to:

- Other causes of unconsciousness or syncope, such as stroke, hypoglycaemia.
- Other causes of increased body temperature, such as fever due to infections, metabolic abnormalities (e.g. thyroid storm) and toxidromes (e.g. neuroleptic malignant syndrome).

2.2.4 Diagnostic Criteria of Work-Relatedness

There is an occupational history of physical work in a hot environment, especially an unacclimatised worker. Ask for reports on air temperature, humidity and air movement/ventilation, or Wet Bulb Globe Temperature (WBGT) which may show an increased risk for environmental heat stress.

2.2.5 Investigation to Establish Work-Relatedness

I. Take an occupational history to establish if the worker is exposed to physical work in a hot environment. Work involving strenuous physical activity will increase the metabolic demand of the worker and increase the metabolic heat generated. Impervious clothing and personal protective equipment (PPE) can limit air movement and the cooling effect of sweating. The following workers are at a greater risk of developing heat illnesses:
    - Construction workers
    - Workers who work with steel
    - Oven and furnace operators
    - Shipyard workers
    - Landscaping and agriculture workers
    - Indoor workers in foundries, commercial kitchens, laundries, boiler rooms, food and beverage manufacturing factories
    - New workers or workers returning from countries with a cold climate
    - Workers returning from long leave (more than one week)

- Workers recovering from prolonged illness
- Workers newly assigned to work in a hot environment

II. Ask the company for environmental records, such as air temperature, humidity, air movement/ventilation which are useful measures of heat stress in the environment. The Wet Bulb Globe Temperature (WBGT) is an example of a composite index for environmental heat stress.

2.2.6 Management

Treatment will depend on the type of presentation. This may range from rest and the replacement of fluids and electrolytes in heat cramps, to immediate and aggressive cooling efforts to reduce core body temperature in heat exhaustion and heat stroke.

I. Advice to workers

Workers with heat syncope and heat cramps should drink enough water before returning to work. They should seek treatment if the condition worsens (i.e. progressing to heat exhaustion or heat stroke).

Workers with heat exhaustion and/or heat stroke should be referred to Emergency Departments of public hospitals for further evaluation and treatment.

II. Advice to companies

- Notify MOM via incident report (www.mom.gov.sg/ireport).
- Review the workplace RA and put in place control measures to reduce exposure by adjusting work schedules for heavy work to the cooler parts of the day and providing shaded areas for work and rest.
- Ensure new workers are acclimatised to work in hot weather.
- Provide sufficient water intake and rest breaks.
- Ensure that workers are trained to recognise symptoms of heat stress and report illnesses early.
- Have an emergency response plan to manage incidents of heat stress.

2.2.7 Further Reading

1. Diagnostic and exposure criteria for occupational diseases – Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010)
2. WSH Guidelines on Managing Heat Stress in the Workplace.
3. MOM Website – Heat stress measures for outdoor work
4. MOM Website – FAQs on Heat stress measures for outdoor work
5. Sorensen, C., & Hess, J. (2022). Treatment and Prevention of Heat-Related Illness. The New England journal of medicine, 387(15), 1404–1413.
6. Epstein Y, Yanovich R. Heatstroke. N Engl J Med. 2019 Jun 20;380(25):2449-2459. doi: 10.1056/NEJMra1810762. PMID: 31216400.

2.3 Occupational Cancers

2.3.1 Introduction

Cancer is one of the leading causes of death in Singapore. Occupational cancer is cancer caused by exposure to confirmed human carcinogens in the workplace or from work. Confirmed human carcinogens are substances, mixtures and exposure circumstances classified by the International Agency for Research on Cancer (IARC) as Group 1 agents (IARC considers the evidence as sufficient, although of varying strength of association or relative risk).

IARC classifies the agents into five groups:
Group 1: Carcinogenic to humans
Group 2A: Probably carcinogenic to humans
Group 2B: Possibly carcinogenic to humans
Group 3: Not classifiable as to its carcinogenicity to humans
Group 4: Probably not carcinogenic to humans

2.3.2 Clinical Presentation

The clinical and histopathological features of occupational cancers are no different from cancers arising from non-occupational causes. It is not surprising that occupational cancers are under-recognised and therefore not reported. The challenges in recognising occupational cancers include:

- The clinical and pathological findings of occupational cancers are no different from cancers not due to work.
- There could be multifactorial causes for cancers including significant non-work risk factors associated with these cancers.
- Recognition requires a knowledge of the occupational carcinogens associated with the particular cancer and the local occupational exposure situations and industries. Refer to Table 3 for more information.
- The diagnosis is dependent on obtaining reliable exposure information from a well-taken occupational history that begins with the first job.
- Because of the long latency period, the patient may not know or recall what, where and when exposure has occurred. The employers may no longer be available.
- Generally, the duration and intensity of exposure to the carcinogen must be sufficiently high to cause cancer. Such information may not be available.

2.3.3 Differential Diagnosis

Identify and evaluate any risk from non-occupational causes for the specific type of cancer from the family, medical and social history. These may include:

- A strong family history (genetic predisposition).
- Previous episodes of cancer and any radiotherapy (radiation-induced cancers have been reported following radiotherapy).
- Life-style factors, for example, smoking, alcohol, diet, excessive exposure to the sun during outdoor leisure activities.
- Use of medications, such as oestrogens (breast, ovarian and uterine cancer) and tamoxifen (uterine and endometrial cancer).

2.3.4 Diagnostic Criteria of Work-Relatedness

While a complete and reliable occupational history of exposure may be difficult to obtain, it is critical for the diagnosis. Doctors should have a high index of suspicion that a cancer could be occupational (particularly if it is rare, seen in a younger person and there is a cluster of cases).

General diagnostic criteria for occupational cancers:

- Confirmed clinical and histological diagnosis of cancer of primary origin.
- Occupational exposure to a confirmed human carcinogen.
- Cumulative occupational exposure to the agent is sufficient to cause the particular cancer.
- Appropriate latency period for the particular cancer.
- The probability of the cancer being work-related is "more likely than not" (>50%), after comparing the risk from work with the relevant non-work risks.

2.3.5 Investigation to Establish Work-Relatedness

I. Take a complete and chronological occupational history to establish if there was any exposure to confirmed human carcinogens from any of the previous or current work. It is helpful to ask leading questions, e.g. asking a patient with malignant mesothelioma whether he has ever worked in an asbestos cement factory, shipbuilding/ship repair or construction (e.g. demolition or renovation), or any maintenance work involving insulation materials. Refer to Table 3 for the carcinogens and associated exposures.

II. Obtain the Safety Data Sheet (SDS) from the employer. This provides details on hazardous substances present, including their health effects and recommended preventive measures.

III. Obtain records of exposure monitoring for the specific agent from the employer or workplace (if available). Such records provide definite evidence of exposure. Reference to published exposure levels for a similar type of work process or industry are also helpful. The American Conference of Government Industrial Hygienists (ACGIH) publishes a list of threshold limit values (TLV) of various agents, some of which are specific for the prevention of cancers. These levels are useful in evaluating the risk for developing the particular cancer. In general, the higher the exposures, the higher the risk for developing the specific cancers.

IV. Check that the period from the first exposure to the carcinogen to the development of the cancer falls within the latency period for the specific cancer. The latency period can range from two to 50 years, depending on the type of cancer.

V. Evaluate the contribution from non-occupational causes

Example: Evaluate the contribution from smoking:

- Smoking is the most important risk factor for lung cancer and bladder cancer and should be evaluated for its contribution. It also has a synergistic effect with asbestos in increasing the risk of lung cancer.

VI. Weigh the contribution from the occupational versus the non-occupational risk factors (using lung cancer as an example):
- The main determining factor in confirming an occupational cancer is that there is sufficient occupational exposure to the established human carcinogen.
- When the exposures are sufficient to double the relative risk of cancer, it can be considered as an occupational cancer regardless of the risk from smoking.
- When TLV is exceeded (where it is meant to prevent cancer), there is an increased risk for cancer compared to the unexposed population. Compare this risk with that from smoking (never, light, moderate or heavy smoker) or other known non-occupational risk factors.
- Cases can be referred to an Occupational Medicine doctor or other appropriate specialist for assessment and management.

2.3.6 Management

I. Advice to workers
- Proper medical management and follow-up including possible return-to-work.
- Suspension from further exposure to the carcinogen.

II. Advice to companies
- Review the workplace RA by monitoring the exposure levels of the carcinogen in all relevant work processes and review the existing control measures.
- Implement effective control measures to reduce the exposure to as low as possible through engineering controls, limiting access and the use of PPE.
- Eliminate exposure by replacing the carcinogen with a safer substitute or enclosing the entire process, where feasible.
- Implement a regular air-monitoring and medical surveillance programme to monitor exposures and detect any early health effects or at-risk workers.
- Educate and train all workers on the risk of exposure, the health effects and the preventive measures to be taken.
- Implement relevant complementary risk reduction programmes e.g. smoking cessation (for lung or bladder cancers).
- Keep a register or a list of all workers exposed to the carcinogen.
- Notify MOM via incident report (www.mom.gov.sg/ireport).

Table 3: **Occupational cancers, carcinogens and associated exposures and diagnostic guidance**

Cancer	Carcinogen	Exposure Situations and Occupations at Risk	Diagnostic Guidance
Bladder	Aromatic amines (benzidine, 4-aminobiphenyl, b-naphthylamine, 4-chloroortho -toluidine)	- Manufacture of tyres and other rubber products - Manufacture and use of pigment and dyes containing these agents	- High cumulative exposures - Latency: 20 to 50 years - Smoking is an independent risk factor
	Ionising radiation	- Use of radioactive isotopes or irradiation equipment e.g. X-ray machines - Workers performing non-destructive testing (NDT) - Radiologists, radiographers and radiology technicians - Health care workers exposed to frequent imaging procedures - Research and laboratory workers handling radioactive materials	- High cumulative exposures - Smoking is an independent risk factor - For solid tumours caused by radiation, latency is usually >5 years
Leukaemia	Benzene	- Refineries and petrochemical industry - Bulk storage of petrol and related products - Laboratory and testing work involving the use of benzene - Work involving handling of fuels containing benzene (e.g. mechanics, petrol pump attendants)	- High cumulative exposures - Associated with acute myeloid leukaemia - Latency <10 years
	Ionising radiation	- Use of radioactive isotopes or irradiation equipment e.g. X-ray machines	- High cumulative exposures

Cancer	Carcinogen	Exposure Situations and Occupations at Risk	Diagnostic Guidance
		- Workers performing non-destructive testing (NDT) - Radiologists, radiographers and radiology technicians - Radiation oncology technicians - Health care workers exposed to frequent imaging procedures - Research and laboratory workers handling radioactive materials	- Check for accidental high exposure situations - Associated with Acute Lymphocytic Leukaemia, Acute Myeloid and Chronic Myeloid Leukaemia - Latency ≥2 years
Liver (Hepatocellular cancer)	- Hepatitis B - Hepatitis C	- Occupationally acquired (e.g. sharps injury) chronic hepatitis B and C infection - Healthcare and laboratory workers with risk of blood borne exposure to the pathogen	- Documented occupationally acquired chronic Hepatitis B or C infection - Latency ≥10 years - Exclude non-occupationally acquired hepatitis B and C infection - Excessive alcohol consumption and smoking are independent risk factors
Occupational Liver Angiosarcoma*	Vinyl chloride monomer (VCM)	- Manufacture of polyvinyl chloride (PVC) from VCM - Storage of VCM - Sampling and analysis of VCM	- High cumulative exposures for ≥10 years - This is a rare cancer - Latency: 10-40 years

Cancer	Carcinogen	Exposure Situations and Occupations at Risk	Diagnostic Guidance
Lung	Asbestos	- Manufacture of asbestos-containing materials (ACM) - Demolition and renovation of buildings containing asbestos materials - Shipbuilding and repair where asbestos is used for insulation of boilers and pipes - Insulation workers doing maintenance in boilers, refineries, ships and plants	- High cumulative exposures sufficient to cause Asbestosis i.e. lung cancer case with asbestosis - Synergistic risk with smoking - Latency ≥ 10 years
	Silica	- Construction workers exposed to grinding, hacking and cutting of materials containing high crystalline silica content - Manufacture and installation of engineered stone/composite stone (e.g. for counter tops) - Granite quarry workers	- High cumulative exposures sufficient to cause Silicosis i.e. lung cancer case with silicosis - Smoking is an independent risk factor - Latency ≥ 10 years
	Hexavalent chromium	- Chrome electroplating - Welding of stainless steel and high chromium alloys (Higher exposures with manual metal arc welding and in enclosed spaces) - Manufacture of pigments and dyes containing chromium	- High cumulative exposures - Smoking is an independent risk factor - Latency ≥ 10 years
	Arsenic	- Use and handling of arsenic containing pesticides	- High cumulative exposures

Cancer	Carcinogen	Exposure Situations and Occupations at Risk	Diagnostic Guidance
		- Use and handling of arsenic containing pigments and paints - Treatment and working with wood treated with arsenic compounds	- Smoking is an independent risk factor - Latency ≥ 10 years
	Ionising radiation	- Use of radioactive isotopes or irradiation equipment e.g. X-ray machines - Workers performing non-destructive testing (NDT) - Radiologists, radiographers and radiology technicians - Radiation oncology technicians - Health care workers exposed to frequent imaging procedures - Research and laboratory workers handling radioactive materials	- High cumulative exposures - Smoking is an independent risk factor - Latency ≥ 10 years
Malignant Mesothelioma* (pleural or peritoneal)	Asbestos	- Manufacture of asbestos-containing materials (ACM) e.g. asbestos cement products - Demolition and renovation of buildings containing asbestos materials - Shipbuilding and ship repair where asbestos is used for insulation of boilers and pipes - Insulation workers doing maintenance in boilers, refineries, ships and plants	- Low intermittent or indirect exposures to asbestos is sufficient - The majority of malignant mesothelioma cases are associated with asbestos exposure - Latency 15 to 50 years

Cancer	Carcinogen	Exposure Situations and Occupations at Risk	Diagnostic Guidance
Bone **Breast** **Colorectal** **Stomach** **Thyroid**	Ionising radiation	- Use of radioactive isotopes or irradiation apparatus e.g. X-ray machines - Workers performing non-destructive testing (NDT) - Radiologists, radiographers and radiology technicians - Radiation oncology technicians - Health care workers exposed to frequent imaging procedures - Research and laboratory workers handling radioactive materials	- High cumulative exposures - Check for accidental high exposures - For solid tumours caused by radiation, latency is usually > 5 years
Skin*	Ultraviolet (UV) radiation	- Outdoor work - Outdoor workers in construction, marine, ports, quarries and farms - Gardeners, road workers, roof workers	- The UV exposure should be significantly higher than that of the general population - Has evidence of chronic cutaneous UV damage - Single or multiple lesions of Squamous cell carcinoma (SCC) or Basal cell carcinoma (BCC) located on occupationally sun-exposed areas

Cancer	Carcinogen	Exposure Situations and Occupations at Risk	Diagnostic Guidance
	Ionising radiation	- Use of radioactive isotopes or irradiation equipment e.g. X-ray machines - Workers performing non-destructive testing (NDT) - Radiologists, radiographers and radiology technicians - Health care workers exposed to frequent imaging procedures - Research and laboratory workers handling radioactive materials	- High cumulative exposures - Non-melanoma skin cancers: Squamous Cell Cancer (SCC) or Basal Cell Cancer (BCC)
	Arsenic	- Use and handling of arsenic containing pesticides, pigments and paints - Treatment and working with wood treated with arsenic compounds	- High cumulative exposures - Usually multiple lesions and on sun-protected areas - Non-melanoma skin cancers: SCC, SCC in situ or BCC
	Polycyclic hydrocarbons in tar, pitch, bitumen, creosotes, mineral oil (including paraffin) and soot	- Aluminium production - Steel and iron foundries - Coke production and gasification - Petroleum refining	- High cumulative exposures - Non-melanoma skin cancers: SCC or BCC

* These occupational cancers are notifiable under the Second Schedule of the WSH Act.

2.3.7 Further Reading

1. International Agency for Research on Cancer (2019). IARC Monographs on the Identification of Carcinogenic Hazards to Humans
2. Sisko Anttila, Paolo Boffetta (2014). Occupational Cancers
3. Finnish Institute of Occupational Health (2013). Memorandum from the Occupational Cancer Working Group 2013
4. Guide to Occupational Diseases. National Board of Industrial Injuries. February 2015, 10th edition
5. American Conference of Governmental Industrial Hygienists (ACGIH). TLV/BEI Guidelines Noise-Induced Hearing Loss

2.4 Noise-Induced Hearing Loss

2.4.1 Introduction

Noise-induced hearing loss (NIHL) is the irreversible sensorineural hearing loss caused by damage to the nervous structures of the inner ear such as the cochlea, organ of Corti, auditory nerve due to prolonged exposure to excessive noise at work. NIHL is one of the leading occupational diseases (ODs) in Singapore.

In addition, studies have suggested an association with Diabetes Mellitus, ischaemic heart disease, cardiovascular disease, stroke, anxiety and depression with increasing noise exposure including from non-occupational sources.

2.4.2 Clinical Presentation

NIHL develops insidiously over a long period of time. In the early stages, the person would usually be asymptomatic and may not notice hearing loss until he is unable to hear high pitched sounds, such as the electronic beep of a mobile phone. Some persons may experience high-pitched intermittent tinnitus, and in about 20% of cases, it may become continuous. There may also be a gradual loss of clarity in perceived speech, resulting in difficulty in speech discrimination.

2.4.3 Differential Diagnosis

Before arriving at a diagnosis of NIHL, other causes of hearing loss must be excluded such as:

I. Conductive hearing loss. The conditions include:
   - Infections, e.g. otitis media
   - Ear wax
   - Cholesteatoma
   - Otosclerosis
   - Ossicular chain abnormalities
   - Blast injuries/acoustic trauma*
   - Middle ear barotrauma
   - Trauma, such as head injury*

II. Sensorineural hearing loss. The conditions include:

- History of hearing loss since childhood congenital deafness may be associated with maternal infections such as rubella, cytomegalovirus, herpes simplex infections, maternal Diabetes Mellitus, toxaemia during pregnancy and prematurity.
- Familial hearing loss/deafness (genetic factors)
- Childhood illnesses, such as:
  - Measles, mumps, chickenpox, influenza
  - Others - encephalitis, meningitis, cerebral abscesses, hyperbilirubinaemia etc
- History of head injury*
- Inner ear barotrauma
- Ototoxic drugs and chemotherapy, such as:
  - Prolonged use of streptomycin, gentamycin, neomycin
  - Chemotherapy such as platinum-based drugs
- Ototoxic chemicals, such as solvents (toluene, xylene), metals (lead, arsenic), gases (carbon monoxide and hydrogen cyanide) and pesticides (organophosphates and paraquat).
- History of therapeutic irradiation especially to the head and neck regions
- Presbycusis
- Meniere's disease
- Brain tumours, e.g. acoustic neuroma (vestibular schwannoma)
- Excessive noise exposure from non-occupational sources, e.g. nightclubs, concerts, use of headphones and use of personal music devices, recreational use of powered tools, recreational use of firearms.

** Depending on the nature and mechanism of the injury, patients may have both conductive and sensorineural hearing loss.*

2.4.4 Diagnostic Criteria of Work-Relatedness

To make a diagnosis of noise-induced hearing loss, it is important to establish that there is:

- Prolonged exposure to excessive noise at work, and
- Hearing loss typical of noise exposure, i.e. Bilateral, generally symmetrical sensorineural hearing loss in a pure tone audiogram, and
- Non-occupational causes of hearing loss have been excluded.

Table 4: Severity of NIHL and diagnostic criteria

Severity of NIHL	Diagnostic Criteria
Early NIHL	- Prolonged occupational exposure to noise for 5 years or more **AND** - Bilateral sensorineural hearing loss on a pure tone audiogram and average hearing level (AHL) for air conduction at 1, 2 and 3 kHz is below 50 dBA in the better ear.
Advanced NIHL	- Prolonged occupational exposure to noise for 10 years or more **AND** - Bilateral sensorineural hearing loss on a pure tone audiogram and AHL for air conduction at 1, 2 and 3 kHz is 50 dBA and above in the better ear.

Cases that do not meet the above criteria would require further investigations and follow up to ascertain the diagnosis.

2.4.5 Investigation to Establish Work-Relatedness

2.4.5.1 Occupational History

- Take a detailed chronological occupational history to establish prolonged exposure to excessive noise in the workplace.
- Obtain details of the noisy processes that worker is involved in/exposed to, or types of noisy equipment that he handles or is nearby (refer below for common examples) and the industry sector that he worked in. Consider asking the worker if they need to raise their voice during conversations at work which may indicate a noisy work environment.

I. Excessive noise is deemed as exposure to noise levels that exceeds:

- The permissible exposure limit (PEL) for noise specified in the Schedule of the WSH (Noise) Regulations.
- An equivalent sound pressure level of 85 dB(A) over an 8-hour workday, where the noise is at a fluctuating sound pressure level.
- A peak sound pressure level exceeding 140 dB(C).

II. Noise exposure is likely high in the following industry sectors and subsectors:
- Manufacture of food, beverage and tobacco products
- Marine sector (shipbuilding and ship repair)
- Manufacture of fabricated metal products and metalworking services (metal working)
- Manufacture of non-metallic mineral products
- Manufacture of pharmaceuticals and biological products
- Manufacture of refined petroleum and chemical products
- Manufacture of rubber and plastic products
- Manufacture of wood products and furniture
- Construction and landscaping sector
- Waste collection, treatment and disposal activities, and recovery of materials
- Logistics and transport

III. Common sources of noise at the workplace include:

 **Use of Hand/ Powered Tools**	 **Process**	 **Equipment/ Machinery**
Air Gun	Abrasive blasting	Compressors
Brazing	Blending/mixing	Conveyor systems
Buffing	Blow moulding	Crushing
Cutting	Casting	Die cut machine
Chipping	Deburring	Gear drive
Drilling	Drumming	Generators
Forging	Drawing	Lathe machine
Gouging	Extrusion	Mechanical press
Grinding	Foundry operations	Miling machine
Hammering	Gas cutting	Motors/pumps
Hydro jetting	Injection moulding	Pneumatic ejector
Nail gun	Lamination	Power press
Polishing	Machining	Punching
Riveting	Melting/smelting	Shearing machine
Sawing	Sanding	Stamping machine
Spraying	Test cell	Turning machine
		Vibratory bowl

IV. Clinical examination
Carry out an auroscopic (otoscopic) examination. In the case of NIHL, the findings should be normal.

V. Review workplace Noise Monitoring Reports (NMR) to assess any excessive noise exposure. These are also known as occupational hygiene noise monitoring reports.
   - Where available, obtain current and past workplace NMRs from the employer, covering the worker's employment period. Workplace noise levels may change over time and noise monitoring is typically conducted every three years.
   - Review the sections of the NMR showing the table of machine noise levels and table of personal dosimetry results respectively for any results that are relevant to worker's work with noise levels at or above 85dBA (machine noise) and dose of more than 100% (personal dosimetry).
   - Look for the worker's or his colleague's personal noise exposure assessment (personal dosimetry) to correlate with the occupational history.
   - As noise levels at workplaces could change over time, consider requesting for such reports for the last 10 years or relevant duration based on the occupational history.

2.4.5.2 Audiometric Testing (Audiometry)

Prior to carrying out an audiometric test or pure tone audiometry, the worker should be advised to avoid exposure to loud noise (both occupational and recreational sources) for at least 16 hours to avoid temporary threshold shift and inaccurate results.

Should the annual audiometry be abnormal, the Designated Workplace Doctor should consider reviewing the person within three months or earlier to obtain an occupational history, conduct a clinical examination, and carry out a repeat audiometry. Compare past serial audiograms if available, to assess the trend in the hearing status over the years and ascertain the diagnosis.

2.4.6 Diagnosis of Noise-Induced Hearing Loss

The following diagnostic criteria for NIHL are used:

1. There is definite prolonged occupational exposure to excessive noise.
2. Pure tone audiometry (PTA) findings:
   i. There is bilateral sensorineural hearing loss (>30dB) with depression of hearing thresholds typically at the 4 kHz frequency, and which may extend to the 6 kHz frequency (V-notch pattern). In some persons, the depression may commence at the 3 kHz frequency or may be the only abnormality in the early stages. The pattern of hearing loss is often similar in both ears.
   ii. With continued exposure to excessive noise, the hearing loss extends to the mid and lower frequencies, and the workers may find difficulty in hearing normal conversation. As he is unable to hear his own voice clearly, he would also tend to speak loudly.
   iii. In general, cases of NIHL will show a gradual decline in the hearing thresholds over many years, hence it would be useful to view past audiograms (serial audiograms) as part of the assessment of hearing loss.
   iv. With the passage of time, the worker may also develop presbycusis in his more senior years, and the audiogram may reflect both an NIHL pattern with presbycusis.

Should the pattern of hearing loss not typical of NIHL be found, the DWD should exclude other diagnoses and carry out relevant investigations.

If there is difficulty in ascertaining the work-relatedness of the hearing loss, consider referring to:

I. An Occupational Medicine (OM) specialist for assistance in ascertaining the work-relatedness of the hearing loss after other causes of hearing loss have been excluded, and for assistance in assessing the percentage permanent incapacity if relevant; or
II. An Ear Nose, and Throat (ENT) Specialist to exclude other causes of hearing loss and for assistance in assessing the percentage permanent incapacity if relevant.

2.4.7 Reporting of NIHL

Cases diagnosed with early and advanced NIHL are notifiable and compensable, and doctors are required to notify MOM via iReport (www.mom.gov.sg/ireport). Doctors should also inform the employer of the diagnosis so that the employer can file the notification and comply with their statutory duties.

2.4.8 Management

The objective is to prevent further hearing loss in the worker and reduce the noise exposure to other workers.

I. Advice to workers

- Consistent and proper use of hearing protectors while working in noisy environments (see Appendix B for type of hearing protectors and tips on proper donning and maintenance).
- Attend the annual audiometric examinations to monitor progression of the hearing loss.

II. Advice to companies

- Review the workplace RA and put control measures in place to reduce exposure to noise, e.g. reducing noise from machines via engineering control measures, locating noisy machines away from workers etc.
- Educate workers on the health effects of noise exposure and proper use of hearing protectors.
- Provide workers exposed to excessive noise with adequate and suitable hearing protectors that are certified with noise ratings and ensure that they wear them correctly and consistently (see Appendix B for proper selection of hearing protectors).
- Ensure that workers exposed to excessive noise attend pre-placement and annual audiometric examinations.
- Implement a hearing conservation programme.
- Notify the worker diagnosed with NIHL to MOM via iReport (NIHL is a notifiable and compensable occupational disease).

Note: Both the treating doctor and the employer are required to notify the MOM of the worker's diagnosis of NIHL.

Work Injury Compensation

Workers diagnosed with severe hearing loss deemed to be occupational in nature i.e. advanced NIHL, may be eligible for lump sum compensation under the Work Injury Compensation Act. Should the doctor be asked to assess the worker's permanent incapacity, reference can be made to the chapter on "Assessment of Hearing" in the "Guide to the Assessment of Traumatic Injuries and Occupational Diseases for Work Injury Compensation" available on the Ministry of Manpower's website.

2.4.9 Notes

2.4.9.1 Note on Audiometric Testing

Under the Workplace Safety and Health (Medical Examinations) Regulations, employees who are exposed to excessive noise are required to undergo pre-placement and annual audiometry to screen for hearing loss. Such audiometry should be carried out by trained industrial audiometric technicians, and Designated Workplace Doctors are required to examine the persons and certify their fitness to work. The acoustic environment should be in accordance with the guidance stated in the Workplace Safety and Health Guidelines: Statutory Medical Examinations.

Industrial audiometric technicians can carry out basic audiometry. Should severe hearing loss be detected, additional audiological testing may be required. The patient should be referred to an ENT or OM practice with ENT support for diagnostic audiometry by an audiologist to assess the severity of hearing loss. The work-relatedness of the hearing loss can be evaluated by the OM physician. The acoustic environment of diagnostic audiograms should be in accordance with the Guide to the Assessment of Traumatic Injuries And Occupational Diseases for Work Injury Compensation (GATIOD).

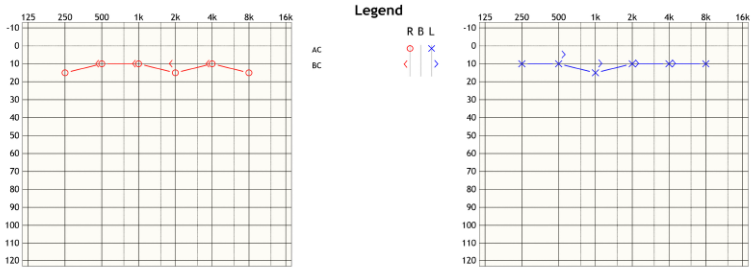
Bone Conduction at 6 and 8 kHz

In general, due to limitations of the audiometer, bone conduction is not carried out at 6 and 8 kHz, as this may lead to unreliable interpretation.

2.4.9.2 Examples of Commonly Encountered Hearing Patterns in Audiograms

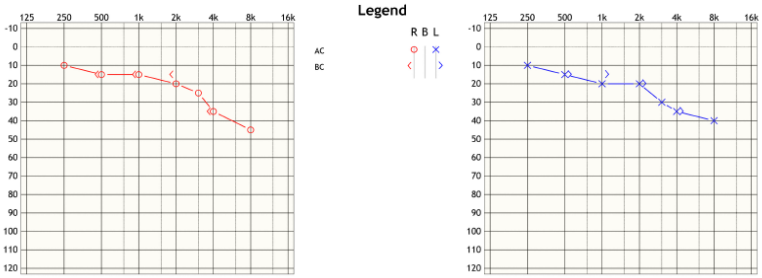
Examples of commonly encountered hearing patterns that you may encounter in audiograms are given below.

1. Normal Hearing



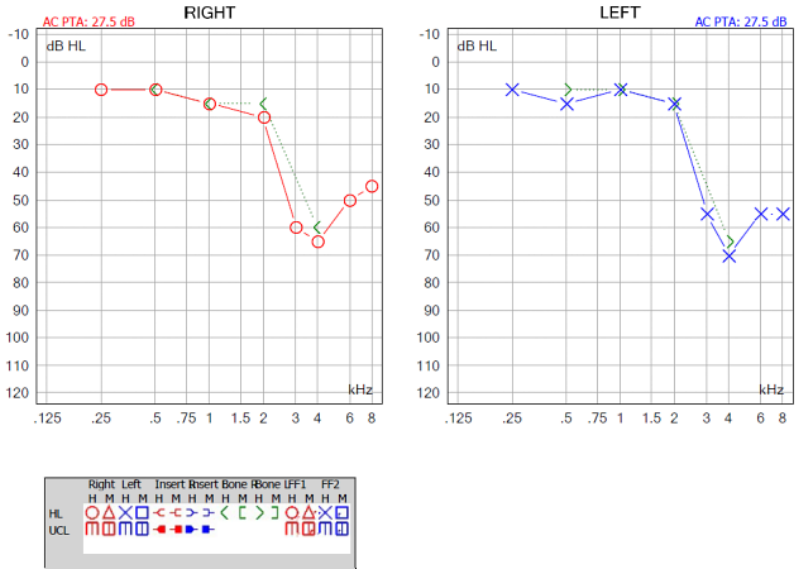
Normal hearing in both ears, air conduction (AC) and bone conduction (BC) within normal range (25dBA or better).

2. Mild bilateral sensorineural high frequency hearing loss (SNHL)



Hearing in the low frequencies are normal, mild sensorineural hearing loss (HL) in the high frequencies; AC and BC mild HL thresholds (30-45dBA).

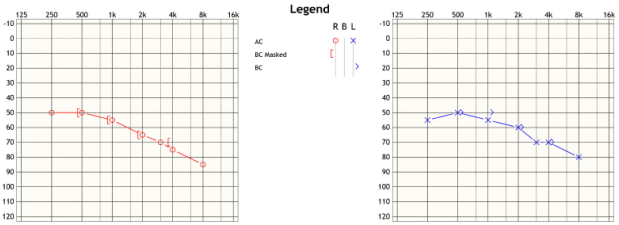
2A. Mild bilateral sensorineural high frequency hearing loss (SNHL) suggestive of NIHL



Hearing thresholds in the mid and lower frequencies are normal, and sensorineural HL at 4 kHz ("V notch" pattern) and hearing improves at 6 and 8 kHz. The depression in the threshold may extend to the 6 kHz frequency. In some persons, the HL may commence at the 3 kHz frequency or may be the only abnormality in the early stages. The pattern of hearing loss is often similar in both ears.

This pattern of SNHL is often seen in the early stages of NIHL.

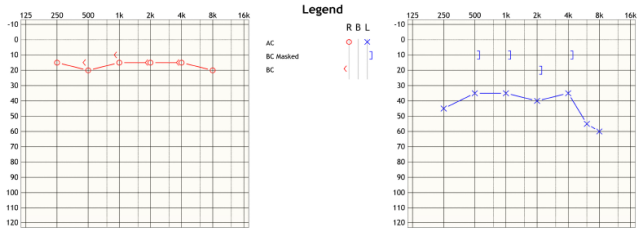
3. Moderate to severe bilateral sensorineural hearing loss (SNHL)



Moderate (45-65dB) to severe (65-85dB) thresholds in both ears for both AC and BC.

With continued exposure to excessive noise, hearing levels in the lower and mid-frequencies are also depressed and there is worsening of HL in the high frequencies. This pattern of hearing loss is also seen in other conditions, hence further information such as duration of occupational noise exposure, nature of noisy work processes and evaluation of serial audiograms are required to make a diagnosis of NIHL.

4. Conductive hearing loss



Conductive hearing loss means that the bone conduction is better than air conduction resulting in an "air bone gap".

The audiogram shows the following findings:
Left ear:

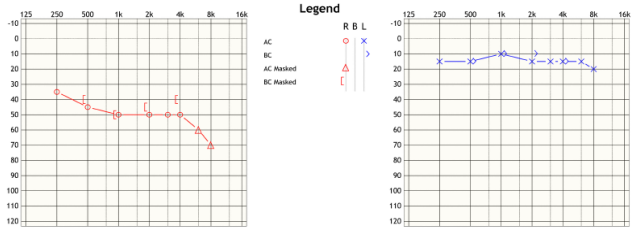
- Mild to moderate conductive hearing loss:
  - The left AC thresholds are in the mild to moderate HL range.
  - BC thresholds are normal; resulting in a significant air bone gap in the AC and BC thresholds (usually 15 – 20 dBA or more) across multiple frequencies.
- Note that the remaining sensorineural hearing thresholds might be normal or also abnormal.

Right ear:

- The right ear hearing is normal.

This can be correlated clinically with a Rinne's and Weber's tuning fork test.

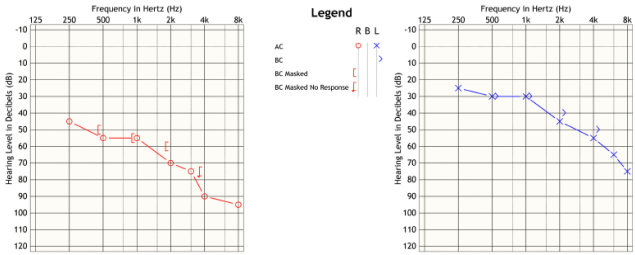
5. Unilateral hearing loss



Unilateral means that the hearing is abnormal in only one ear; the abnormality can be due to sensorineural, conductive, or mixed hearing loss pathologies.

This audiogram depicts left normal hearing, and right mild to severe SNHL.

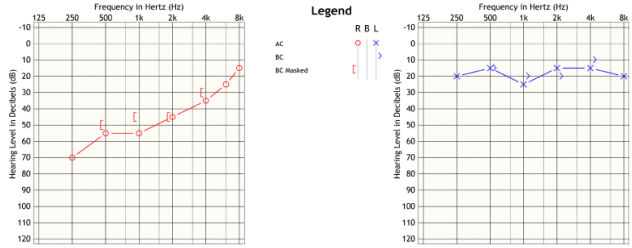
6. Asymmetrical hearing loss



Asymmetrical hearing loss usually refers to hearing loss in both ears, but one ear is significantly worse than the other with at least a 20dB difference between 3 consecutive frequencies (taking the worse ear's AC threshold compared to the better ear's AC/BC threshold). The abnormality can be due to sensorineural, conductive, or mixed hearing loss pathologies. Patients with asymmetry typically require further investigations (e.g. MRI/CT scan) to rule out cerebellopontine angle tumours such as vestibular schwannoma.

This audiogram depicts left mild to severe sloping SNHL, and right mild to profound sloping SNHL.

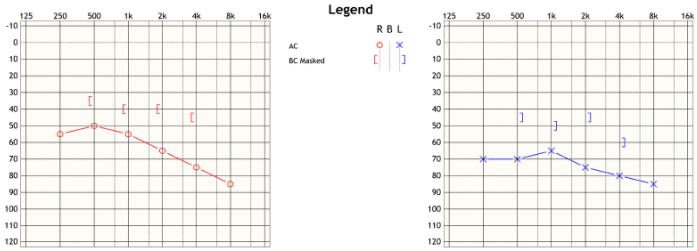
7. Low frequency hearing loss



This usually refers to the low frequency thresholds being worse than the higher frequencies and is sometimes referred to as rising hearing loss (as opposed to sloping).

This audiogram depicts a right normal to severe low frequency/rising SNHL (with left normal hearing). This is commonly seen in Meniere's disease.

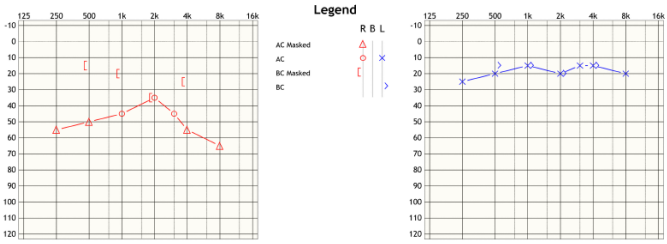
8. Mixed hearing loss



This usually refers to the presence of both a conductive and sensorineural hearing issue; both AC and BC thresholds are abnormal, and a significant air bone gap is also present. The condition of a mixed HL can be found on any type of audiogram (unilateral, asymmetrical, or symmetrical hearing loss).

This audiogram depicts a right moderate to severe mixed HL, and left severe mixed HL; naming convention will only refer to the AC thresholds to describe the degree of the loss.

9. Possible otosclerosis



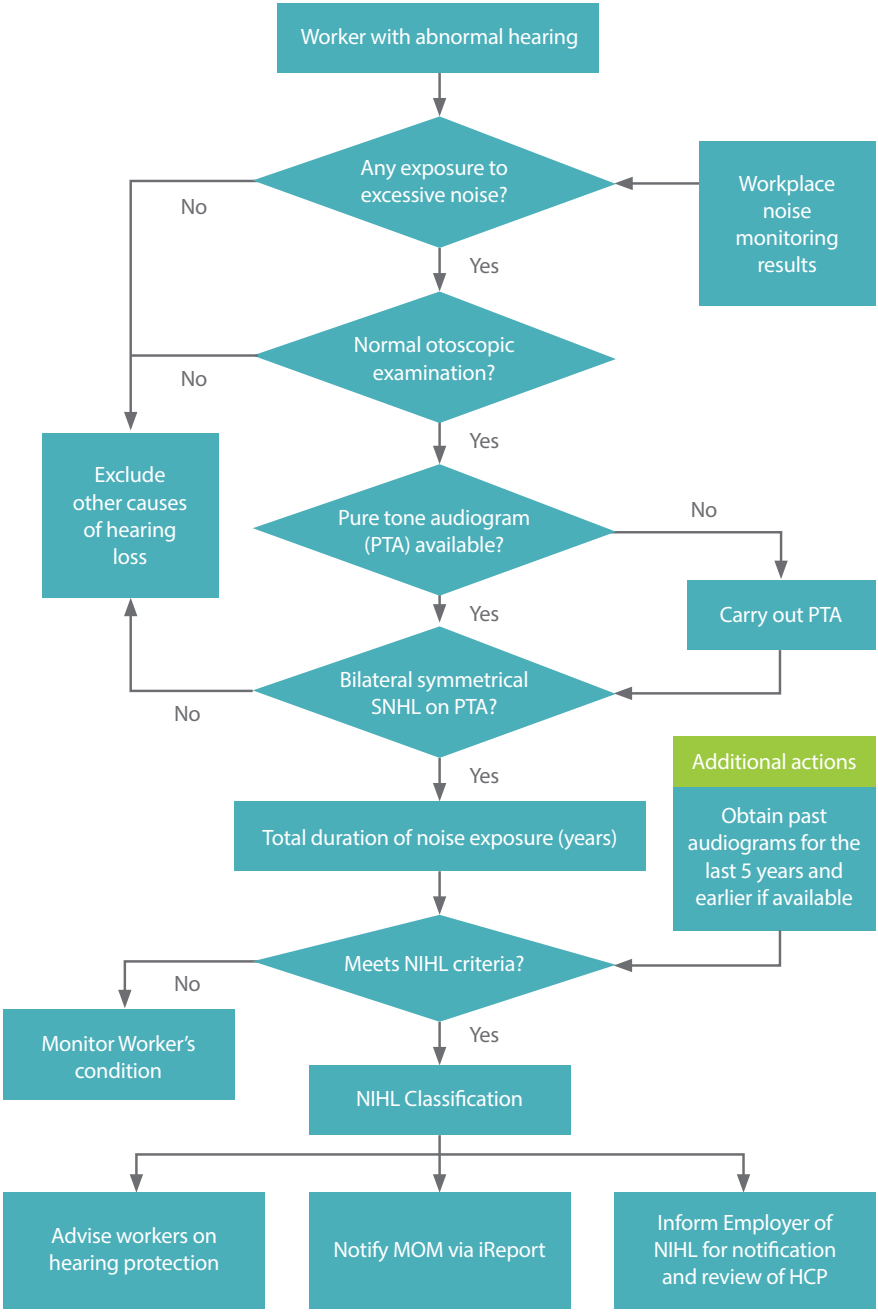
Otosclerosis usually results in a conductive component of hearing loss due to the pathology of the middle ear ossicles. The audiogram is sometimes characteristic of a Carhart's notch at around 2kHz (a notch is where a particular region of the audiogram breaks/diverges from the general shape of the whole audiogram, resulting in a triangular "cut").

This audiogram depicts a mild to moderate mixed HL due to otosclerosis; the left ear is normal.

The WSH Council would like to thank the Department of Otorhinolaryngology at Tan Tock Seng Hospital for the sample audiograms.

The flow chart in Figure 1 would help in the evaluation of a worker with abnormal hearing.

Figure 1: Flow chart for workers with abnormal hearing



2.4.10 Future Reading

1. Diagnostic and exposure criteria for occupational diseases: Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010). Edited by Shengli Niu, Claudio Colosio, Michele Carugno, Anil Adisesh. First published 2022. ISBN 978-92-2-035682-1
2. Current insights in noise-induced hearing loss: a literature review of the underlying mechanism, pathophysiology, asymmetry, and management options. Le et al. Journal of Otolaryngology - Head and Neck Surgery (2017) 46:41
3. Workplace Safety and Health Guidelines: Statutory Medical Examinations
4. Ministry of Manpower Webpage - Enhanced workplace health surveillance (WHS+)
5. Medical assessment for Work Injury Compensation: A Guide To The Assessment Of Traumatic Injuries And Occupational Diseases For Work Injury Compensation (GATIOD)
6. Workplace Safety and Health (Medical Examinations) Regulations
7. Workplace Safety and Health (Noise) Regulations
8. Workplace Safety and Health (Incident Reporting) Regulations
9. Work Injury Compensation Act 2019
10. WSH Guidelines: Hearing Conservation Programme
11. Singapore Standard SS 549:2009 Code of practice for Selection, use, care and maintenance of hearing protectors
12. List of Occupational Medicine Specialist clinics on the MOM website

2.5 Occupational Infectious Diseases

2.5.1 Introduction

Occupational infectious diseases are illnesses caused by exposure to biological and infectious agents in specific workplaces. Examples of such workplaces are: workplaces handling biological agents, biomedical laboratories, healthcare settings and veterinary health settings where workers care for patients or animals with infectious diseases.

Workers who are exposed to or handle infectious biological materials in laboratories, research facilities or healthcare settings are at risk of occupational infections. Workers who provide healthcare services and have close and/or frequent exposure to patients suffering from infectious diseases are also at risk.

2.5.2 Clinical Presentation

Patients with infectious disease may initially present with non-specific symptoms such as fever, malaise and headache. They may also present complications resulting from the infection. Uncommon and emerging infectious diseases may also pose diagnostic and management challenges.

If unsure, doctors should consider consulting an Infectious Diseases specialist, the National Centre for Infectious Diseases or the Communicable Disease Agency for further information such as clinical presentation, diagnosis, or management of specific infectious diseases.

There is also a need for vigilance and careful history taking to actively seek out sources of occupational exposures when evaluating patients for infectious diseases. The successful control of occupational infections is dependent on the early recognition and prompt diagnosis of the condition by attending doctors and controlling the source of infection and preventing transmission. The clinician should also be alert for potential clustering of cases within similar occupational groups or at specific workplaces.

2.5.3 Differential Diagnosis

Exclude non-occupational sources of infection such as:

- Imported infections - Check for travel history and activities, including contact history, exposure to any infectious source and vaccination records, if any. Check for residence in a high-burden country.
- Endemic infections – Check for sick household members and other close contacts in a non-occupational setting (e.g. social and voluntary settings), previous instances of the same infection, hobbies and pets.

2.5.4 Diagnostic Criteria of Work-Relatedness

To categorise an infection as occupational, a clinical diagnosis and laboratory confirmation (where available) are required. It is also crucial to obtain clear occupational history that details the exposure or contact with the infectious agent at the workplace. This includes obtaining history that suggests possible exposure to the infectious agent during high-risk situations at work or during prolonged and/or frequent contact with the infectious agent. It is also important to demonstrate a temporal relationship between the exposure and infection that corresponds with the incubation period of the specific disease.

Where facilities are available, genome sequencing or similar tools can be used to establish the link between the source of infection, e.g. index case and the affected worker(s).

2.5.5 Investigation to Establish Work-Relatedness

Take a detailed occupational history focusing on the employee's job nature and work processes, and how contact with the infectious agent could have occurred. The history should include:

I. Whether there was a specific exposure incident that occurred such as:
   - Spills or splashes
   - Injuries, such as sharps injuries
   - Conduct of high-risk activities resulting in the generation of aerosols such as bronchoscopy, intubation etc.

II. Any prolonged close and/or frequent contact with the potential infectious source, such as in caring for sick patients or animals.

III. Any prolonged or frequent exposure to environmental elements, including soil contact (e.g. during landscaping) or contact with water fixtures (e.g. plumbing or sanitation work).

IV. Any clusters of co-workers with the same infection linked to a common source at the workplace or in similar occupational groups. Establish if there are any clusters at the workplace (two or more cases linked by epidemiological features of time, place and person).

V. Use of appropriate PPE.

VI. Exclude:
   - Non-occupational causes, such as imported infections from overseas travel and presence of similar infections among family members or close contacts.
   - Presence of past exposure for certain long latency infections e.g. Tuberculosis.

Carry out laboratory and relevant investigations to confirm the diagnosis and/or identify the specific infectious or biological agent affecting the worker. Where a disease may be endemic in the region, additional tests may be required to show that the infection is recently acquired due to exposures at work. Where available, carry out further confirmatory laboratory tests to link the source of infection to affected worker(s).

Should the source of infection or risk factors from work remain unclear, consider carrying out a workplace visit and assessment to ascertain the risk factors at work, and/or the likely source of infection. Additional tests can be taken during the workplace visit such as environmental or clinical sample (e.g. from other potentially exposed or infected persons) for microbial testing. This is particularly important for emerging infectious diseases.

Evaluate that the likelihood of the infectious disease is work-related by:

I. Correlate the findings with the clinical presentation, supporting investigations, the incubation period from exposure to onset of disease, and the mode of transmission.

II. Take into consideration the common reservoirs of infection, incubation period, modes of transmission, and correlate this with the site of infection/organ affected.

III. Where available and applicable, compare results of pre-placement and current investigations to determine if there is a temporal relationship between exposure and onset of infection (e.g. Baseline Chest X-ray for Tuberculosis infections, baseline hepatitis status for occupations with high exposure risk).

IV. Where facilities are available, carry out confirmatory laboratory tests to link the source of infection to affected worker(s).

Table 5 gives a list of selected infectious diseases, the main routes of transmission, incubation period, source of infections and at-risk occupational groups.

Notification of cases of confirmed occupational infectious diseases is required under the WSH Act and is mandatory for those arising in healthcare, research and laboratory settings. All notifications should be made electronically at www.mom.gov.sg/ireport.

Medical practitioners and laboratories should also notify the Ministry of Health (MOH) of suspected emerging infectious diseases, bioterrorism agents and events of public health significance. Please refer to MOH's Guidelines on the Notification of Infectious Diseases in Singapore.

Occupational infectious diseases are compensable under the Work Injury Compensation Act.

Latent TB is not infectious and does not cause symptoms. It is not reportable under the WSH Act.

Table 5: **Occupational infections and route of transmission, incubation period, source of infections and at-risk occupational groups**

Disease	Main Routes of Transmission	Incubation Period	Source of Infections	Exposure Situations/At-risk Occupational Groups
Anthrax+	- Direct contact with infected animals including their carcasses and tissues or contaminated animal products, or with anthrax spores in soil. - Inhalation of aerosolised spores.	- Cutaneous anthrax: One to seven days (range of one to 17 days) - Inhalation anthrax: One to 43 days (up to 60 days)	- Spores of Bacillus anthracis in animals, animal products, tissues or any materials or substances containing anthrax spores or anthrax bacteria. - It is found naturally in soil and commonly affects domestic and wild hoofed animals such as cattle, sheep, goats, camels and antelopes.	- Veterinary and wildlife workers handling infected animals. - Butchers, workers who process meat, hides, hair wool or bone of untreated animals.
Avian Influenza*	- Close contact with infected poultry and other animals (e.g. mammals) (live or dead), secretions and excrement from infected animals, or contaminated environments (such as poultry markets). - Consumption or exposure to infected animals' undercooked or unprocessed products (e.g. unpasteurised milk).	One to 10 days (up to 17 days for A(H5N1)) Refer to MOH or other international bodies for latest updates.	- Primarily from infected poultry or birds. The virus is shed in the saliva, mucus and faeces of birds. - Sporadic human infections have been reported in the United States associated with dairy cattle exposures. - The virus has been found in the raw milk, lungs, muscle and udder tissue of infected dairy cows.	- Poultry workers carrying out mass culling of infected birds, workers at live animal markets or dairy cattle farms. - Contact with contaminated surfaces.

Disease	Main Routes of Transmission	Incubation Period	Source of Infections	Exposure Situations/At-risk Occupational Groups
B Virus (herpes B, monkey B virus, herpesvirus simiae, and herpesvirus B)	Direct or indirect exposure to infected monkey's bodily fluids or tissues through monkey bites or scratches, sharps injuries or cuts or mucosal contact.	Three to seven days up to a month	- Infected primates especially macaque monkeys. Virus can be found in saliva, faeces, urine, or brain or spinal cord tissue. - The virus can also be found in cells from an infected monkey in the laboratory.	Primate laboratory workers, wildlife or veterinary staff handling and/or caring for macaque monkeys and who/ whose: - Are bitten or scratched by an infected monkey. - Eyes, nose, mouth or broken skin come into contact with the tissue or bodily fluid of an infected monkey. - Have a needle stick injury by a contaminated syringe - Have been scratched or cut on a contaminated cage or other sharp-edged surface - Are exposed to the brain (especially), spinal cord, or skull of an infected monkey.
Chickenpox (Varicella)	Respiratory secretions (inhalation). Direct contact with vesicle fluid of skin lesions.	14 to 16 days (range 10 to 21 days)	- Varicella-zoster virus in vesicle fluid or skin lesions of acute varicella or herpes zoster. - Infected secretions from respiratory tract of infected patients.	- Healthcare workers caring for and managing infected patients - Childcare workers, teachers handling or caring for infected children.

Disease	Main Routes of Transmission	Incubation Period	Source of Infections	Exposure Situations/At-risk Occupational Groups
Glanders⁺	- Inhalation of aerosolised respiratory secretions containing Burkholderia mallei. - Direct contact infected animals or their bodily fluids.	One to 14 days	- *Burkholderia mallei* bacterium found in tissues or bodily fluids of infected horses. - It also affects donkeys and mules and can be naturally contracted by other mammals such as goats, dogs, and cats.	Veterinary staff, caretakers of horses caring for or managing horses or other infected animals who may inhale infected aerosols or dust contaminated by infected animals or whose broken skin or mucous membranes come into direct contact with infected tissues or bodily fluids from horses.
Acute Hepatitis B*	Direct contact with contaminated bodily fluids such as blood and/or organs, or contaminated surfaces through broken skin, mucosal membrane.	Typically 90 days (range 45 to 180 days)	Hepatitis B virus (HBV) in blood and bodily fluids of infected persons and/or organs, or contaminated surfaces.	Healthcare workers, laboratory personnel, biological waste handlers handling infected tissues, bodily fluids or contaminated sharp objects.
Acute Hepatitis C*	Direct contact with contaminated bodily fluids such as blood and/or organs or contaminated surfaces through broken skin, mucosal membrane.	Typically 45 days (range 14 to 180 days)	Hepatitis C virus (HCV) in blood and bodily fluids of infected persons and/or organs, or contaminated surfaces.	Healthcare workers, laboratory personnel, biological waste handlers handling infected tissues, bodily fluids or contaminated sharp objects.

Disease	Main Routes of Transmission	Incubation Period	Source of Infections	Exposure Situations/At-risk Occupational Groups
Leptospirosis†*	Contact with the urine, fluids or tissues of infected animals or via contaminated soil, vegetation, waters.	Five to 14 days (range two to 30 days)	Leptospira bacteria can be found in renal tubules and genital tracts of infected animals and are shed in the urine, amniotic fluid or placental tissue, which could then contaminate surrounding soil, vegetation and waters. Animals that can be infected by various serovars of Leptospires include rats, swine, cattle, dogs and racoons.	Farmers, animal husbandry workers, fishery and aquaculture workers, abattoir workers, butchers, drainage and sewerage workers, road sweepers, veterinary staff, cleaners, waste and refuse collectors, construction workers.
Measles*	- Respiratory (inhalation of droplets). - Direct contact with nasal or throat secretions.	14 days (range seven to 21 days)	Measles virus in nasal and throat secretions of infected persons.	Unvaccinated or partially vaccinated healthcare workers caring for and managing infected patients, unvaccinated or partially vaccinated childcare workers, caregivers and teachers handling or caring for infected children. Despite the measles vaccine's high effectiveness of 97%, breakthrough cases may occur and should be considered when evaluating clinically compatible illnesses.

Disease	Main Routes of Transmission	Incubation Period	Source of Infections	Exposure Situations/At-risk Occupational Groups
Melioidosis*	Respiratory (inhalation of contaminated droplets or airborne particles such as soil dust). Ingestion of contaminated water. Direct contact of skin wounds with contaminated soil or water.	One to 21 days (Average of nine days) Many years may elapse between presumed exposure and appearance of clinical disease.	*Burkholderia pseudomallei*, a gram-negative bacteria found in soil and water.	- Construction workers, miners, agriculture/ farm workers (e.g. rice farmers), military personnel who have exposure to contaminated water. - Laboratory workers handling isolates of *B. pseudomallei*.
Nipah virus infection*	Direct contact with infected animals (such as bats and pigs) or their bodily fluids or excretions.	Four to 14 days (range four to 45 days)	- Infected pigs and their bodily fluids. Fruit bats (Flying foxes) are a reservoir of infection. - Persons infected with Nipah virus.	- Abattoir workers, pork vendors, veterinary staff who handle infected pigs or their carcasses. - Healthcare workers who care for or treat persons infected with Nipah virus.
Rubella*	- Respiratory (inhalation of droplets). - Direct contact with respiratory secretions.	14 to 17 days (range 14 to 21 days)	Rubella virus in nasal and throat secretions of infected persons.	Unvaccinated or partially vaccinated healthcare workers caring for and managing infected patients, unvaccinated or partially vaccinated childcare workers, caregivers and teachers handling or caring for infected children.

Disease	Main Routes of Transmission	Incubation Period	Source of Infections	Exposure Situations/At-risk Occupational Groups
Severe acute respiratory syndrome (SARS)*	- Respiratory (inhalation of droplets). - Direct contact with respiratory secretions. - Indirect contact with objects contaminated by respiratory secretions.	Two to seven days (up to 14 days), can be variable.	In infected persons, SARS-CoV can be found in respiratory, bodily fluids and excretions of infected persons.	Healthcare workers caring for and managing infected patients, laboratory workers handling infected material.
Tetanus*	Direct contact through puncture wounds or penetrating injuries.	Three to 21 days (average of eight days)	*Clostridium tetani* spores in the environment or soil or fomites contaminated with animal or human faeces.	Workers who have increased risk of traumatic or puncture injuries especially if they have contact with soil, sewage and/or domestic animals, such as agriculture, farm and outdoor workers especially with history of cuts/wounds.
Tuberculosis* ***[Active TB]***	Airborne (inhalation of airborne particles containing *Mycobacterium tuberculosis*).	Weeks to years	*Mycobacterium tuberculosis* expelled in nasal and throat secretions when people with active disease speaks, coughs or sneezes. The bacteria may also be found in other bodily fluids and in a variety of tissues.	- Healthcare workers with: • Prolonged, close and frequent contact while caring for and managing infected patients with active TB. • Carrying out cough inducing or aerosol generating procedures. - Morticians handling infected corpses, laboratory workers handling infected material containing *M. tuberculosis*.

* These diseases are legally notifiable under the Infectious Disease Act. All notifications should be made via the electronic notification system (CD-LENS). Please refer to the Ministry of Health's latest circulars for further details on accessing the notification system.

+ These diseases are notifiable under the Second Schedule of the WSH Act.

2.5.6 Management

Treatment is directed at the specific infection that has been established. In some cases, it is essential to administer antibiotic administration at the earliest signs.

I. Advice to workers

- Advise on personal hygiene and use of appropriate PPE such as surgical mask or appropriate respirators, impervious gloves, face shields, impervious gowns, and adherence to work protocols and good safety practices to reduce spread of infection where appropriate.
- Stress on the importance to attend follow-up appointments to monitor disease progression and evaluate the need for hospitalisation.
- Advise on when he can return to work based on the infectious period of the specific infection.

II. Advice to companies

- Isolate the worker immediately to reduce the risk of transmission of infection depending on the specific infection.
- Maintain a record of workers falling sick and look out for secondary cases among co-workers.
- Review workplace RA and put in place control measures to reduce exposure (e.g. prevention of needlestick injuries), engineering controls such as local exhaust ventilation, safe work procedures, vaccinations for vaccine preventable diseases, and use of appropriate PPE.
- Notify MOH and MOM where applicable.
- Discuss on the need for specific immunisation for high-risk personnel or workplaces.
- Advise to put in place a policy and procedure for infection control such as work restriction of infected workers and/or allowing time away from work during periods of illness, promoting good hygiene practices, use of personal protection and a system for monitoring sickness absence.

Also consider using protective pharmaceuticals such as vaccines or chemoprophylaxis.

2.5.7 Further Reading

1. National Centre for Infectious Diseases, Singapore. Diseases and Conditions
2. National Centre for Infectious Diseases, Singapore. Communicable Diseases Control Handbook
3. U.S. Centers for Disease Control and Prevention
4. CDC Yellow Book: Health Information for International Travel
5. American Public Health Association. Control of Communicable Diseases Manual 21st edition, published 2022
6. Communicable Diseases Surveillance in Singapore 2019 - 2020 published by MOH, Singapore
7. Aw TC, Blair I, Babcock HM. Occupational Infections. Infectious Diseases. 2017;647-655.
8. World Health Organisation (WHO). Occupational Infections.
9. Haagsma, J, Lugman T, Heederik DJ, Havelaar AH. Infectious disease risks associated with occupational exposure: a systematic review of the literature. Occup Environ Med. 2012, 69:140-146.

2.6 Occupational Lung Diseases

2.6.1 Introduction

An occupational lung disease is a lung condition caused by or aggravated by work. In Singapore, the common occupational lung diseases reported are occupational asthma, silicosis, asbestosis and malignant mesothelioma. Occupational or environmental factors that play a role in the aetiology, whether causal or contributory, should be identified for proper management of the case.

This section focuses on recognition and prevention of lung diseases which are caused by specific work-related exposures. For more detailed information on the clinical presentations, diagnosis and medical management of specific respiratory diseases, please consult with a respiratory medicine physician. For occupational lung cancer and malignant mesothelioma, please refer to the chapter on "Occupational Cancers" and for occupational lung infections, refer to the chapter on "Occupational Infectious Diseases".

2.6.2 Clinical Presentation

Acute presentations include rhinosinusitis, laryngitis, acute asthma, bronchitis, pneumonitis and pulmonary oedema. Chronic presentations may include bronchitis, bronchiolitis, lung fibrosis and cancer. Persons with occupational lung diseases may present with respiratory symptoms such as cough, sputum production, shortness of breath, chest tightness, wheezing and chest pain.

Physical examination may reveal crepitations, rhonchi, finger clubbing. However, often the worker may also be asymptomatic and present due to an abnormal chest x-ray on routine screening. In many cases, there may be a lack of physical signs on clinical examination.

2.6.3 Differential Diagnosis

Identify and evaluate non-occupational risks or causes of the lung disease by taking a detailed family, medical and social history. These may include:

- A personal and strong family history of asthma or allergy (although the presence of pre-existing asthma does not preclude the diagnosis of occupational asthma).
- Non-occupational exposures to allergens causing asthma or hypersensitivity pneumonitis.
- Non-occupational exposures to tuberculosis.
- Other causes of lung fibrosis (previous lung infections, idiopathic causes, drugs etc).
- Smoking history (heavy and prolonged smoking may be the cause of the impaired lung function or chronic obstructive pulmonary disease (COPD)).

2.6.4 Diagnostic Criteria of Work-Relatedness

General diagnostic criteria for occupational lung diseases:

- History, clinical findings, investigations and disease progression is consistent with the diagnosis.
- Occupational exposure to the specific causative agent.
- Clear exposure-effect time relationship (e.g. occupational asthma).
- Cumulative occupational exposure to the agent (e.g. for silicosis or asbestosis).
- Appropriate latency period (for silicosis or asbestosis).
- Exclusion of non-occupational causes (e.g. other causes of diffuse radiological opacities).

2.6.5 Investigation to Establish Work-Relatedness

A comprehensive occupational exposure history should be taken in every case. Radiological examination is useful in diagnosing pneumoconiosis and a lung function test will give an indication of the severity of the disease. Demonstrating an exposure-effect relationship is key to the diagnosis of occupational asthma and hypersensitivity pneumonitis. In addition, supporting documents of results of personal or workplace exposure monitoring will aid in determining the diagnosis of work-related lung diseases. The clinician should always be alert to the clustering of cases with similar occupational groups or in specific workplaces.

I. Take a complete, detailed and chronological occupational history to establish any relevant occupational or environmental causes. Refer to Table 6 for the common causative agents and the occupations at risk. Correlate the exposure history with the clinical findings and investigations.
   - For diseases such as acute respiratory reactions to irritant chemicals or gases, the exposure-onset relationship is typically very clear.
   - Workers with occupational asthma are usually able to give a history of worsening symptoms at work and improvement when away from work. This observation can be objectively documented with the serial peak expiratory flow monitoring over periods at work and away from work. Even low exposures can trigger an asthmatic attack in a sensitized individual.
   - For diseases with long latency (e.g. silicosis, asbestosis, malignant mesothelioma), the key is documenting the past exposures many years ago, starting from the first job from the history. For silicosis or asbestosis to develop, the exposures to crystalline silica or asbestos must be high and prolonged. Refer to Table 6 for the clinical features, radiological and spirometry findings in the common occupational diseases.

II. When evaluating an OD, it is important to obtain information from the employer regarding the worker's exposure to potential hazards in the workplace. These include:
   - Safety data sheet (SDS): Provides details on hazardous substances present, including their health effects (e.g. respiratory sensitisation, carcinogenicity) and recommended preventive measures.
   - Personal protective equipment (PPE): Information on the type of protective gear such as respirators (e.g. powered air-purifying respirator (PAPR), half-face, full-face respirators), protective gloves and coveralls. Ensure that the filter used is appropriate for the specific hazard, e.g. using particulate filters for dust such as respirable crystalline silica or chemical cartridges for vapours, to provide effective protection.
   - Exposure monitoring records: Documents the levels of exposure to assess whether these levels exceed the PELs.

III. Exclude non-occupational causes, pre-existing and predisposing factors. The exclusion of other causes of diffuse radiological opacities is important in making the diagnosis of silicosis or asbestosis.

2.6.6 Management

Treatment would depend on the specific disease. Cases of occupational asthma would improve with treatment, e.g. inhaled bronchodilators and/or steroids and following removal from the specific triggering exposures in the workplace. In most cases of occupational asthma, the worker would need a permanent change from his current job, and this has implications on their career and job prospects. This should be discussed with the worker and the management.

I. Advice to workers

- Provide proper medical management and education on the management of their condition and preventive advice.
- Follow up with doctors to evaluate progress and look for complications.
- Stop smoking as this often aggravates the conditions, lung function or increases the risk for other diseases.

II. Advice to companies

- Review the workplace RA by monitoring the exposure levels of the hazardous agent in all relevant work processes and review the existing control measures.
- Eliminate exposure by replacing the hazardous agent with a safer substitute or enclosing the entire process, where feasible.
- Implement effective control measures to reduce the exposure as low as possible through engineering controls, limiting access and the use of PPE.
- Implement a regular air-monitoring and medical surveillance programme to monitor exposures and detect any early health effects or susceptible workers.
- Educate and train all workers on the risk of exposure, the health effects and the preventive measures to take.
- Implement relevant complementary risk reduction programmes, e.g. smoking cessation.
- Keep a register (list) of all workers exposed to the hazardous agent (e.g. crystalline silica or asbestos).
- Notify MOM at www.mom.gov.sg/ireport.

Table 6: **Occupational lung diseases, causative agents, occupations at risk and diagnostic guidance**

Disease	Causative agents and occupations at risk	Diagnostic and management guidance
Occupational Asthma*	Common causative agents include isocyanates (e.g. toluene diisocyanate, hexamethylene diisocyanate etc) used commonly in polyurethane systems (e.g. foam cushions, paints, varnishes, glues), grain dust, wood dust, soldering fumes, welding fumes, acid anhydrides, amines, antibiotics, animal proteins (e.g. laboratory animal allergy), disinfectants (e.g. glutaraldehyde). Given the many known causative agents and the widespread use of these agents, occupational asthma can occur across many occupational settings and industries.	Evidence of asthma Clinically documented asthma; positive bronchodilator response; positive methacholine or histamine challenge test; diurnal variation>20% on peak expiratory flow rate (PEFR) monitoring. Evidence of work-relatedness Improvement of symptoms when away from work; serial PEFR showing asthmatic pattern at work and improvement when away from work. Specific causative agent Serial PEFR documenting work-relatedness and exposure to only one known asthma causing agent at work or positive specific bronchoprovocation test to the causative agent. Early diagnosis and removal from the causative agent are associated with better outcomes. Impact on career and job prospects has to be considered.
Acute Respiratory Reactions	These can be respiratory irritants such as gases (e.g. chlorine, ammonia, sulphur dioxide, ozone, nitrogen oxides, phosgene), metal fumes (e.g. mercury, cadmium), acid fumes, chemicals (isocyanates, acetaldehyde). Exposures occur during accidental leakage or spillage, during maintenance work	Highly soluble gases act on the upper respiratory tract, e.g. epiglottic edema from ammonia or bronchospasm within minutes. Moderately soluble gases affect both the upper and lower respiratory tract (e.g. chlorine, SO2) causing upper respiratory tract irritation and bronchoconstriction.

Disease	Causative agents and occupations at risk	Diagnostic and management guidance
	or from chemical reactions (from mixing of incompatible chemicals). Exposures can be very high in spaces with poor ventilation or in a confined space (e.g. accidental mixing of bleach and acid cleaner in a toilet; leakage of ammonia used as a refrigerant on fishing boats).	Low solubility gases penetrate to the lower respiratory tract and cause pulmonary edema 6 to 24 hours later. Remove immediately from exposure and give oxygen. Overnight observation is needed to observe for delayed pulmonary edema. Following recovery some patients may develop persistent cough and non-specific bronchial hyper-reactivity (Reactive Airways Dysfunction Syndrome).
Hypersensitivity Pneumonitis	Causative agents include allergens of bacterial, fungal, serum protein, chemical and dust origin. Handling of mouldy vegetable compost (e.g. farmers, sugar cane workers, mushroom compost handlers).	Shortness of breath and non-productive cough, often sudden onset followed by fever and chills (4-10 hours after exposure). Clinically, there may be rapid breathing and fine basal crepitations. Lung function is restrictive. The chest x-ray (CXR) may show patchy infiltrates and there can be leucocytosis in the acute phase. Recognition of consistent clinical picture with temporal relationship after exposure to specific dust is key. Suspected diagnosis can be confirmed by workplace challenge or inhalational challenge in hospital.

Disease	Causative agents and occupations at risk	Diagnostic and management guidance
Byssinosis*	Acute or chronic airways disease caused by exposure to cotton dust, flax, hemp and jute. Exposure to raw cotton dust is highest near blowing and carding machines (stripper and grinders are at highest risk)	Shortness of breath and chest tightness on the first day of workweek. Lung function test may reveal cross-shift fall in FEV1 (>10%) on the first day of workweek. Transfer from exposure is indicated for those with symptoms and a post-shift fall in FEV1 and those with moderate or severe chronic airway obstruction (FEV1<60% predicted).
Silicosis*	Silicosis results from prolonged and high exposure to respirable crystalline silica dust e.g. during breaking, cutting or crushing, grinding or granite, ceramic, engineered stone. Occupations at risk include miners, sandblasters, tunnel drillers, quarry workers, foundry workers, stone carvers, jade workers, ceramic workers, construction and renovation workers, engineered stone workers (e.g. used for kitchen counter tops). Silicosis is an example of an occupational disease which has resurfaced in recent years in developed countries with the introduction of a new material "engineered stone" which has a high crystalline silica content.	Chronic simple silicosis is usually asymptomatic with normal or mildly restrictive lung function. Cases are detected because of the CXR (done for other reasons) showing bilateral diffuse nodular shadows in both lungs, more predominantly in the mid and upper zones. Occasionally, egg shell calcifications may be present (e.g. calcified hilar lymph nodes). Those with higher exposures may develop progressive massive fibrosis (PMF) with increasing dyspnea and respiratory failure. Complications include associated pulmonary tuberculosis and pneumothorax. Rarely, an acute and rapidly progressive form of silicosis occurs within months following intense exposure to very high concentrations. This can rapidly lead to PMF and respiratory failure.

Disease	Causative agents and occupations at risk	Diagnostic and management guidance
		Silicosis or exposure to crystalline silica is known to be associated with autoimmune diseases such as rheumatoid arthritis. There is an increased risk of lung cancer among silicosis cases. Crystalline silica dust exposure and silicosis increase the risk of tuberculosis. Treatment is symptomatic. Smoking cessation should be advised.
Asbestosis*	Asbestosis results from prolonged and high exposure to asbestos fibers e.g. during installation or removal of asbestos insulation materials in boilers for ships, equipment or buildings, demolition of old buildings with asbestos-containing materials such as in roofs, walls and rubbish chutes, manufacture of asbestos materials (e.g. construction materials, pipes, gaskets etc.), maintenance of friction materials (such as brake linings and clutch facings)	Shortness of breath on exertion, crepitations on lung bases, finger clubbing; CXR shows diffuse interstitial fibrosis with irregular streaky opacities in the mid and lower lung zones. Lung function shows a restrictive picture and DLCO is reduced. Pleural plaques (with or without calcification) may be present (with or without asbestosis) and is considered a marker of asbestos exposure. Asbestos exposure can cause malignant mesothelioma and lung cancer (these are covered under occupational cancer) Treatment is symptomatic and exposed workers should be advised to stop smoking to maintain lung function and reduce risk of lung cancer (synergistic effect).

* MOM is to be notified of these occupational diseases under the Workplace Safety and Health Act.

2.6.7 Further Reading

1. Workplace Safety and Health (Medical Examinations) Regulations
2. Workplace Safety and Health (General Provisions) Regulations
3. Workplace Safety and Health Guidelines: Statutory Medical Examinations
4. De Matteis S et al. Current and new challenges in occupational lung diseases. Eur Respir Rev 2017; 26: 170080
5. David Fishwick and Chris Barber. Respiratory Disorders. Textbook of Occupational Medicine Practice 5th Edition 2022
6. Susan M Tarlo and Catherine Lemiere. Occupational asthma. N Engl J Med 2014;370:640-9
7. List of Occupational Medicine Specialist clinics on the MOM website

2.7 Occupational Skin Diseases

2.7.1 Introduction

An occupational skin disease is a skin condition caused or aggravated by work. In Singapore, occupational skin diseases are the third most reported occupational disease. These conditions often result from exposure to irritants and allergens in various work environments. Prolonged contact with water or chemicals can compromise the skin barrier, leading to contact dermatitis, notably irritant contact dermatitis.

Occupational skin diseases are more frequently reported in the healthcare, manufacturing and construction industries. Common causative agents include alcohol, wetwork, latex, and coolants. Medical practitioners and employers must notify occupational skin diseases upon diagnosis under the Workplace Safety and Health Act.

2.7.2 Clinical Presentation

Occupational skin diseases may resemble non-occupational conditions, but their distribution and history of exposure can suggest a work-related cause. If the skin rash appears on parts of the body which are in contact with the offending agent, e.g. on the hands and forearms, it may be work-related. Notwithstanding, the face and other parts of the body may also be affected where there are exposures to airborne agents, e.g. oil mists and metal fumes.

The most common presentation of occupational skin disease is contact dermatitis, an inflammatory eczematous skin disease. Contact dermatitis can be divided into:

- Irritant contact dermatitis – A non-allergic skin inflammation caused by direct damage from irritants like alcohol and disinfectants.
- Allergic contact dermatitis – A delayed hypersensitivity reaction triggered by sensitisation to allergens such as nickel, rubber accelerators, and resins.

Acute presentations may include redness, swelling, blisters and oozing. Chronic contact dermatitis may present with scaly, thickened, fissured appearance and pigmentary changes. Other presentations include contact urticaria, acneiform eruptions and secondary infections.

Contact with natural rubber latex can cause contact urticaria. This condition can occur in healthcare workers. Contact urticaria may also be caused by raw seafood.

Acneiform eruptions may result from exposure to oil and grease. Frictional dermatitis and callosities may occur in workers using mechanical tools. Warty growths may develop in workers who are continually exposed to tar, pitch, bitumen, mineral oil or paraffin. If untreated, ulcers and skin cancer may develop (refer to Section 3.3 Occupational Cancers). Common sites involved are the eyelids, cheeks, chin, behind the ears, neck, arms, scrotum and thighs.

2.7.3 Differential Diagnosis

Exclude non-occupational sources of exposures that may cause or aggravate the skin rash. Ask for:

- Domestic exposures, e.g. housework – wet work and use of detergents.
- Cosmetics and jewellery – sensitivity to fragrances and nickel compounds.
- History of atopy – possibility of endogenous eczema.
- Hobbies using epoxy glues and solvents.

2.7.4 Diagnostic Criteria of Work-Relatedness

A thorough occupational history to determine the worker's work process, materials, practice and habits is essential in the diagnosis of an occupational skin disease. Please refer to Appendix A for common work processes that may lead to this condition. The onset or worsening of the rash (primary location with or without secondary spread) should be correlated to :

- Duration of employment or exposure.
- Change in the work process or use of new chemicals.
- Time relationship of the rash with work periods (usually there is some improvement when the patient is away from work).

The use of personal protective equipment (PPE) may itself be the cause of the rash (e.g. workers may be allergic to the latex gloves worn).

2.7.5 Investigation to Establish Work-Relatedness

I. Take a thorough occupational history to establish if there is exposure to the following toxic agents. Table 7 shows common skin irritants and allergens known in selected industries.

Table 7: **Skin irritants and allergens known to cause occupational skin disease across selected industries**

Industry	Irritants	Allergens
Agriculture and horticulture	Animal feeds, fertilisers, solvents, plants, oils, disinfectants, pesticides	Plants, gloves, animal feeds, pesticides, disinfectants
Chemical and pharmaceutical	Chemicals, acids, alkalis, water, detergents	Chemicals, medicaments, latex gloves and rubber masks
Construction and building	Cement, preservatives, fiberglass, solvents, oils	Cement, rubber or leather gloves and boots, epoxy resins, woods, paints
Electronics	Solvents, acids, alkalis, resins, fibreglass	Resins (epoxy, acrylates, isocyanates, formaldehyde), soldering fluxes, nickel, chromate, cobalt, gloves and finger cots
Food and catering	Vegetable and food juices, water and detergents	Food, gloves, antioxidants, preservatives
Hairdressing	Shampoos, permanent wave solutions, water	Nickel, hair dyes, fragrances, latex or rubber gloves
Healthcare	Alcohol, disinfectants, antiseptics, water	Latex gloves, rubber masks, formaldehyde, preservatives, resins
Metal fabrication	Cutting fluids, oils, coolants	Cutting fluids and oils
Shipbuilding and repair	Cutting fluids, oils, coolants	Cutting fluids and oils, welding fumes, resins
Woodworking and furniture making	Wood dust, resins, soaps and detergents, solvents, oils, turpentine	Woods, rubbers or latex gloves, resins, wood preservatives

II. Correlate the exposure history with the individual symptoms and investigation results.
- Correlate symptoms with work practices and work periods. Observing the work processes would be useful in establishing if there is personal contact with the offending agent. The relationship between onset/aggravation of rash with work periods provides a clue to its work-relatedness (the rash is likely to worsen during work and improve when off work/on leave).
- Correlate the use of PPE and the onset/worsening of rash.
- Patch testing may be useful to determine if the worker has allergic contact dermatitis.
- Prick testing can be used to determine if a worker has contact urticaria, e.g. to latex or seafood.

III. Ask the company for the safety data sheet (SDS).
- This provides additional information as to the type and toxicity of materials and chemicals handled at work.

IV. Exclude non-occupational causes, pre-existing and predisposing factors.

2.7.6 Management

The management of occupational dermatosis depends on its presentation and cause. The causative agent must be identified.

I. Advice to workers
- Minimise contact with the causative agent – wear suitable PPE, e.g. impervious gloves and/or aprons in the case of irritant contact dermatitis. Please refer to Appendix B for more information on PPE.
- For workers with allergic contact dermatitis, consider recommending that they ask for a job scope with no exposure to the offending agent.
- Maintain good personal hygiene and work practices.
- If the rash does not improve or if an allergic contact dermatitis is suspected, the worker may need to be referred to the Occupational Dermatosis Clinic at National Skin Centre or the Joint Occupational Dermatology Clinic at Singapore General Hospital for further investigation.

II. Advice to companies
- Review safety data sheets and put in place control measures to minimise workers' exposure in high-risk work processes. This may include the substitution of harmful substances with less harmful ones, provision of local exhaust ventilation, and suitable PPE, e.g. impervious gloves, aprons and washing facilities.
- Consider reassigning worker with allergic contact dermatitis to other work not involved with exposure to specific allergen.
- Notify the Ministry of Manpower via iReport (www.mom.gov.sg/ireport).

2.7.7 Further Reading

1. Workplace Safety and Health (Medical Examinations) Regulations
2. Workplace Safety and Health Guidelines: Statutory Medical Examinations
3. Ministry of Manpower Webpage – Enhanced workplace health surveillance (WHS+)
4. Diagnostic and exposure criteria for occupational diseases – Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010)
5. List of Occupational Medicine Specialist clinics on the MOM website
6. National Skin Centre website: Webpage on Occupational Skin Disease
7. Managing Skin Health at Work – published by the Society of Occupational Medicine (UK)

2.8 Poisoning: Aniline

2.8.1 Introduction

Aniline belongs to the group of aromatic amines and is a clear to slightly yellow liquid with characteristic fishy or aromatic odour.

In occupational settings, absorption can occur through the skin or inhalational routes. The main toxic effect is through the production of methaemoglobin. Aniline is a suspected carcinogen.

2.8.2 Clinical Presentation

Exposure to aniline causes a blood disorder, methaemoglobinemia, in which oxygen delivery to the tissues is impaired. This may have mild to severe consequences depending on the duration and amount of exposure and symptoms vary depending on the level of methaemoglobin. Following acute exposure, cyanosis, dizziness, headaches, arrythmia, convulsions, coma, and death may occur. Direct contact with aniline can also produce skin and eye irritation.

Long-term exposure to lower levels of aniline may cause symptoms similar to those experienced in acute high-level exposure.

2.8.3 Differential Diagnosis

Exclude other causes of methaemoglobinaemia such as:

- Hereditary causes
- Drug induced, e.g. nitrites and nitrates, phenacetin, sulfonamides, dapsone

2.8.4 Diagnostic Criteria of Work-Relatedness

A thorough occupational history of exposure and its correlation to the clinical presentation is critical. In addition, safety data sheets, supporting documents of results of personal or workplace exposure monitoring, used will aid in determining the diagnosis of work-related poisoning.

2.8.5 Investigation to Establish Work-Relatedness

I. Establish the exposure history and correlate it with the signs and symptoms presented. The main exposure situations that would raise suspicion that the worker's clinical presentation is work-related are listed in Table 8.

Table 8: **Clinical presentation and at-risk exposures to aniline**

Exposure situations/ At-risk exposures	Acute presentation	Chronic presentation
Manufacture of: - **Synthetic dyestuff and marking inks** - **Methylene diisocyanate** - **Stabilisers for rubber products** - **Polyurethane foam** - **Explosives and pesticides**	General - Headache, nausea, tinnitus, weakness, irritability, vomiting, dry throat Mucosal membranes - Lips, tongue and mucus membrane turns grey, irritation to skin, eyes and respiratory tract Neurological - Confusion, ataxia, disorientation, lethargy, drowsiness, seizures, coma Cardiovascular - Heart blocks, arrhythmias, cardiogenic shock Respiratory - Failure, paralysis, breathlessness Genitourinary - Dysuria, haematuria, renal failure	General - Loss of appetite, headache, dizziness, insomnia Haematological - Anaemia, intravascular haemolysis Cardiac, renal and hepatic damage may occur due to secondary effects of haemolysis.

II. Ask company for results of exposure monitoring and the SDS of chemicals used. Review if the chemicals handled contain aniline. If the exposure levels exceed the PELs, a work-related condition should be suspected. Please note that hand contamination and accidental ingestion are also possible sources of exposure.

Blood methaemoglobin levels should be obtained to substantiate the diagnosis and monitor the condition. Other investigations include full blood count, peripheral blood film, electrocardiogram (ECG), renal and liver function tests.

2.8.6 Management

I. Advice to workers

- Advise to stop further exposure or reassign the worker to another area without exposure to aniline till further review.
- Provide advice on good personal hygiene to reduce absorption (e.g. avoid smoking and eating with hands at the workplace).
- Advise proper use and maintenance of appropriate personal protective equipment (PPE), such as respirators, impervious gloves, PVC or rubber boots, face shields and overcoats/aprons. Refer to Appendix B for further information on PPE.
- Workers with acute exposure or with signs and symptoms of aniline poisoning should be referred to the Emergency Department at public hospitals with relevant SDS for further evaluation and management. This can be followed by referral to an Occupational Medicine Specialist.

II. Advice to companies

- Review workplace risk assessment and put in place control measures to minimise exposure.
- Conduct regular toxic substances monitoring to assess the workers' level of exposure to hazards. The frequency of monitoring required is dependent on the level of toxic substance measured as a percentage of the PEL stated in the First Schedule of the WSH (General Provisions) regulations.
- Educate workers, particularly those identified as high-risk, on the health effects of toxic substance exposure and proper use of PPE to minimise hazardous chemical exposure (see Appendix B).
- Notify the Ministry of Manpower (poisoning by aniline is a notifiable occupational disease).

2.8.7 Reading

1. Workplace Safety and Health (Medical Examinations) Regulations
2. Workplace Safety and Health (General Provisions) Regulations
3. Ministry of Manpower Webpage – Enhanced workplace health surveillance (WHS+)
4. Ministry of Manpower Webpage – Requirements for hygiene monitoring
5. Casarett & Doull's Essentials of Toxicology, Fourth Edition
6. Agency for Toxic Substances and Disease Registry – Toxic Substances Portal
7. Diagnostic and exposure criteria for occupational diseases – Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010)
8. List of Occupational Medicine Specialist clinics on the MOM website

2.9 Poisoning: Gases

2.9.1 Introduction

In occupational environments, exposure to toxic gases primarily occurs as a result of accidents. These gases can be inhaled or absorbed through mucosal surfaces, leading to a range of health effects depending on their chemical properties and exposure levels.

Water-soluble gases such as chlorine, ammonia, sulphur dioxide and hydrogen chloride have a strong propensity to dissolve in the upper airway, leading to mucous membrane irritation. In contrast, less soluble gases such as nitrogen dioxide, phosgene, and ozone are more likely to penetrate deeper into the respiratory tract, inducing severe bronchiolitis, which may be accompanied by delayed pulmonary oedema.

Additionally, exposure to these gases may cause cardiovascular and neurological symptoms, depending on the dose and duration of exposure. This is particularly concerning in confined spaces, where poor ventilation can lead to dangerous accumulations of gases, increasing the risk of asphyxiation or acute toxicity.

2.9.2 Clinical Presentation

Acute exposure to high levels of toxic gases can lead to the sudden onset of respiratory symptoms, including coughing, wheezing, or breathlessness. Hydrogen sulphide (H_2S), commonly found in sewerage systems, is a potent respiratory toxin that can cause sudden collapse and death at high concentrations. Certain gases can impact organs beyond the lungs; for instance, carbon monoxide, an odourless and colourless asphyxiant, can also cause neurological impairment.

2.9.3 Differential Diagnosis

Exclude non-occupational causes of the presenting symptoms involving the respiratory and central nervous systems:

- Respiratory tract infections
- Narcotics
- Alcohol intoxication

2.9.4 Diagnostic Criteria of Work-Relatedness

A thorough occupational history of exposure and its correlation to the clinical presentation is critical. In addition, supporting documents of results of workplace exposure monitoring will aid in determining the diagnosis of work-related poisoning.

2.9.5 Investigation to Establish Work-Relatedness

I. Establish the exposure history to the following toxic gases and correlate it with the signs and symptoms presented. The main exposure situations that would raise suspicion that the worker's clinical presentation is work-related are listed in Table 9. Please refer to Appendix A for common work processes that may involve such exposures.

Table 9: **Clinical presentations of poisoning and at-risk exposures to toxic gases.**

Type of gas	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Carbon monoxide (CO)*	- Work involving incomplete combustion of fossil fuels, such as during operation of diesel engines in confined spaces (e.g. manholes, ship tanks, sewer systems, and boilers) and fire-fighting.	- General symptoms (headache, nausea, vomiting, dizziness, fatigue, weakness) - Neurological (confusion, disorientation, visual disturbance, syncope and seizures, cerebellar signs, coma) - Respiratory (breathlessness, pulmonary edema) - Cardiovascular (arrythmias)	- Neurological (Parkinsonism, personality disorders, memory loss, impaired mood and concentration, vegetative state, residual anoxic injury)
Carbon Dioxide (CO_2)	- Refrigerant use - Brewing and wine industries - Manufacture and use of dry ice - Greenhouses - Combustion of fuels and wood	- Neurological (drowsiness, headache, nausea, vertigo, weakness, irritability, unconsciousness) - Cardiovascular (chest pain, angina, ischaemia) - Panic attacks	- General (anorexia, lethargy) - Neurological (headache, dizziness, incoordination and ataxia).

Type of gas	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
	- Pharmaceutical processing - Arc welding - Work (e.g. cleaning) in confined spaces (e.g. manholes, ship tanks, sewer systems, and boilers) - Accidental leakage from fire retardant equipment containing CO_2		
Hydrogen cyanide (HCN)*	- Metal refining - Electroplating - Jewellery making - Film recovery - Insecticide fumigant - Laboratory work - Fumigation work	- Neurological (general weakness, confusion, bizarre behaviour, headache, sleepiness, seizures, coma) - Cardiovascular (arrythmia) - Respiratory (dyspnoea)	- General (gradual onset of weakness) - Neurological (confusion, residual anoxic injury) - Cardiac (chest discomfort, palpitations)

Type of gas	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Hydrogen sulphide (H_2S)*	- Work (e.g. cleaning) in confined spaces – manholes, shiptanks, sewer systems, and boilers) - Work in sewerage tanks (where there is decaying matter) - Laying of underground cables	- Eyes (ocular irritation) - Respiratory (upper and lower airway irritation, delayed pulmonary oedema, hypoxia, respiratory arrest) - Neurological (loss of consciousness)	- Neurological (residual anoxic injury, headaches, poor attention span, poor memory, and poor motor function)
Nitrogen dioxide (NO_2) and nitrous fumes*	- Combustion of fossil fuels (e.g. in internal combustion engines, thermal power stations, gas heaters and stoves) - Production of nitric acid, dynamite, trinitrotoluene (TNT), and fireworks - Working in Waste-to-Energy Plants - Handling semiconductor processing equipment	- Mucosal (irritation of eyes, nose and throat, cough) - Respiratory (dyspnoea, acute bronchitis, pulmonary oedema) - Cardiovascular (palpitations, tachycardia, chest pain, angina)	- Respiratory (decrease lung function, chronic bronchitis, allergic responses to inhaled pollutants)

Type of gas	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Phosgene	- Manufacture of isocyanates, polyurethane, polycarbonate resins, dyes and pesticides - Welding (when volatile chlorine compound or vapour contacts with flame or very hot metal)	- Respiratory (upper airway inflammation, reactive airway dysfunction syndrome and pneumonitis) - Cardiovascular (right heart strain, cardiovascular collapse) - Dermal (irritation) - Ocular (tearing and increased presence of blood in eye) - Haematologic (haemolysis) - Hepatic (necrosis and loss of function) - Renal (necrosis and loss of function)	- Respiratory (delayed pulmonary oedema in low doses, chronic pneumonitis)

**** Poisoning by these gases is a notifiable occupational disease under the WSH Act.***
Air monitoring: levels of the gases in air can be monitored to see if PELs are exceeded.

II. Correlate the exposure history with the individual symptoms and investigation results.

Severe acute poisoning does not usually present a diagnostic challenge in that it is related to exposure to toxic gases as a high level of exposure can be ascertained and the clinical manifestations are present. However, it may be difficult to document the specific toxic gases and its source.

For some gases, specific biological samples may be obtained to substantiate the diagnosis and monitor progress of condition (see Table 10 below).

Table 10: Biological indicators in different types of gas

Type of gas	Specific biological indicators
Carbon dioxide	Blood gas: partial pressure CO_2
Carbon monoxide	Blood carboxyhaemoglobin concentration
Hydrogen cyanide	Blood cyanide concentration
Hydrogen sulphide	Urine and blood thiosulfate levels

III. Ask company for details of work task and exposure monitoring if available. This provides additional information to substantiate the history from the worker.

IV. Exclude non occupational causes, such as respiratory tract infections, narcotics, or alcohol.

2.9.6 Management

I. Advice to workers

- Workers with signs and symptoms of poisoning should be referred directly to the Emergency Department of public hospitals with relevant safety data sheets for further evaluation and management. Cases can be followed up with an Occupational Medicine Specialist.
- Advise worker on proper use of PPE, such as respirators, impervious gloves, PVC or rubber boots, face shields and overcoats/aprons (see Appendix B).

II. Advice to companies

- The company should conduct a risk assessment and put in place control measures to reduce exposure.
- Conduct regular toxic substances monitoring to assess the workers' level of exposure to hazards. The frequency of monitoring required is dependent on the level of toxic substance measured as a percentage of the PEL stated in the First Schedule of the WSH (General Provisions) regulations.
- Educate workers on and ensure provision of suitable PPE (this could include full face masks with gas canisters, protective suits and gloves) to minimise hazardous chemical exposure (see Appendix B).
- Notify the Ministry of Manpower via iReport (www.mom.gov.sg/ireport).

2.9.7 Further Reading

1. Workplace Safety and Health (Medical Examinations) Regulations
2. Workplace Safety and Health (General Provisions) Regulations
3. Ministry of Manpower Webpage – Enhanced workplace health surveillance (WHS+)
4. Ministry of Manpower Webpage – Requirements for hygiene monitoring
5. Casarett & Doull's Essentials of Toxicology, Fourth Edition
6. Agency for Toxic Substances and Disease Registry – Toxic Substances Portal
7. Diagnostic and exposure criteria for occupational diseases – Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010)
8. List of Occupational Medicine Specialist clinics on the MOM website

2.10 Poisoning: Metals

2.10.1 Introduction

Metals are ubiquitous in the environment, with certain metals playing essential roles in physiological processes vital for sustaining life, while exposure to other metals may pose health risks. Although instances of high-dose occupational exposure to metals are infrequent, low-level chronic exposures to specific metals can lead to kidney and neurological diseases. Metals can exist in elemental, ionic and organic forms, each exhibiting distinct toxicities to various bodily systems. Workers in high-risk industries such as the aerospace or manufacturing industries may encounter metals during their occupational activities. Harmful metals can also be present as contaminants in air, water, food and soil.

2.10.2 Clinical Presentation

Acute exposures usually occur through the inhalation route and may cause pneumonitis which can progress to acute respiratory distress syndrome. Skin contact may result in dermatitis and burns. Ingestion, usually accidental, may present with gastroenteritis.

The presentation for chronic exposure is usually insidious and nonspecific. Some metals may cause multiple organs to be affected. A high degree of suspicion that the condition is work-related is when there is a cluster of cases from the same occupational group or workplace.

2.10.3 Differential Diagnosis

Exclude non-occupational causes, such as those due to:

- Sources from the environment (e.g. diet/food, especially seafood)
- Drugs (e.g. arsenicals in herbal remedies)

2.10.4 Diagnostic Criteria of Work-Relatedness

A thorough occupational history of exposure is critical. In addition, supporting documents of results of personal or workplace exposure monitoring will aid in determining the diagnosis of work-related metal poisoning.

2.10.5 Investigation to Establish Work-Relatedness

I. Establish the exposure history to the following metals and correlate with the signs and symptoms presented. Refer to Appendix A for common work processes that may lead to exposures to metals. The main exposure situations that would raise suspicion that the worker's clinical presentation is work-related are listed in Table 11.

Table 11: Clinical presentations and at-risk exposures to metals and metalloids

Common Metal	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Arsenic (As) *#	- Manufacture and use of pesticides, wood preservatives containing arsenic - Sawing, sanding and burning of wood treated with arsenical preservatives - Manufacture of semiconductors (in particular wafer production and maintenance of ion implant machines) - Manufacture and use of lead alloys, anti-fouling paints and pigments - Smelting of arsenic containing ores - Additives in animal and poultry feeds	- Fever, loss of appetite - Gastrointestinal tract (severe diarrhoea, "rice water" stools, hepatomegaly) - Lung (cough, laryngitis, mild bronchitis, and dyspnoea) - Cardiovascular (cardiac arrhythmia, cardiac failure) - Neurological (peripheral neuropathy, encephalopathy)	- Cancers (skin, lung and bladder) - Skin (increased pigmentation, desquamation, herpetic lesions about the mouth, hyperkeratoses, especially of palms and soles) - Liver (cirrhosis, hepatitis, angiosarcoma) - Lung (chronic bronchitis, basilar fibrosis) - Neurological (peripheral neuritis characterised by numbness of hands and feet, progressing to painful "pins and needles"; initially sensory, later motor) - Others (nasal septum perforation, aplastic anaemia, Raynaud's syndrome)

Common Metal	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Arsine (AsH_3)# *(Note: arsenic requires both SME for exposure and OD notification for poisoning, but arsine poisoning only requires OD notification)*	- Accidental exposures during metal refining, waste treatment and cleaning of tanks containing acid sludge - Smelting of arsenic containing metals - Manufacture of semiconductors (in particular wafer production and maintenance of ion implant machines)	- Massive intravascular haemolysis - Triad of haemoglobinuria (port-wine urine), jaundice and abdominal pain - Associated shivering, severe thirst and ECG changes	
Beryllium (Be)#	- Manufacture, fabrication or reclaiming of beryllium and its alloys in aircraft parts, automobiles, computers, sports equipment, dental bridges and mirrors.	- Lung (Acute chemical pneumonitis) - Skin and eye irritation (conjunctivitis, papular vesicular dermatitis)	- Cancer (lung) - Lung (berylliosis-chronic lung inflammation characterised by granuloma formation) - Skin (chronic granulomatous lesion)

Common Metal	Exposure situations/ At-risk exposures	Acute Presentation	Chronic Presentation
Cadmium (Cd)*#	- Waste treatment - Manufacture of cadmium-nickel batteries - E-waste recycling - Silver brazing, welding and soldering operations using cadmium-containing fillers - Plastics industry, especially with PVC compounding and use of Cd as a plastics stabiliser - Cadmium electroplating - Spray painting or removal of paints/ coatings containing cadmium by scraping or blasting - Repair and finishing in the automobile and aerospace industry (especially of landing gears with cadmium coating) - Manufacture and use of fungicides and pigments - Jewellery manufacture - Smelting and refining of Zn, Pb or Cu ores and scrap processing	- Metal fume fever with flu- like symptoms of weakness, fever, headache, chills, sweating and muscular pain. - Acute renal failure - Lungs (acute pulmonary oedema within 24 hours) - Gastrointestinal (severe irritation with nausea, vomiting and abdominal pain following accidental ingestion	- Renal (tubular and/or glomerular damage characterised by low molecular weight proteinuria, glucosuria, amino aciduria, albuminuria and reduced creatinine clearance) - Bone (osteomalacia, osteoporosis and fractures –Itai-itai disease) - Lung (Chronic pulmonary disease, lung cancer) - Others (Anosmia, anaemia, teeth discoloration)

Common Metal	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Lead (Pb)*# **- Inorganic Lead** **- Organic lead (tetra ethyl and tetramethyl)**	- Manufacture of lead- acid storage batteries - Manufacture and use of stabilisers in PVC compounding - E-waste recycling - Manufacture of solder - Burning/ welding/ cutting of lead-coated structures among shipbreakers and welders - Manufacture and use of lead-based paints - Manufacture and use of ammunition (e.g. firing range instructors) - Manufacture and use of glazes for porcelain, enamels, tiles, ceramics and pottery) - Printing industry	- Gastrointestinal (nausea, vomiting, diarrhoea or constipation and abdominal pain) - Renal (acute renal failure) - Neurological (encephalopathy, convulsions, coma, paraesthesia, pain and muscle weakness) - Mainly CNS (headache, tremor, nervousness, agitation, insomnia, troubled dreams, hallucinations, mental confusion, coma, death)	- Gastrointestinal (lead colic) - Neurological (lead palsy, wrist drop and foot drop with no sensory changes; slow mental changes such as learning difficulty may occur) - Blood (basophilic stippling, anaemia) - Burton's line (a bluish-black pigmentation at margins of gums) - Renal (Chronic nephritis and tubular degeneration) - Reproductive (spontaneous abortion, still births, subfertility and neurological damage to the foetus)

Common Metal	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
	- Cleaning of tanks containing leaded gasoline or aviation fuel - Production and transportation of anti-knock agents containing organic lead compounds - Blending anti-knock agents and raw gasoline at refineries		
Manganese (Mn)*#	- Milling of manganese ore - Manufacture of dry cell batteries (manganese dioxide) - Iron and steel industry - Manufacture of paints, varnishes, inks and dyes, fertilisers, feed additives, disinfectants and bleaching agents, glass and ceramics) - Manufacture of potassium permanganate ($KMnO_4$)	- Metal fume fever - Lung (irritation, cough, bronchitis, pneumonitis) - Minor irritation of the eyes and mucous membranes	- CNS (psychomotor, speech and gait disturbances followed by manic depressive psychosis and parkinsonism) - Lung (pneumonitis, bronchitis)

Common Metal	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
	- Welding operations with manganese coated rods - Manganese electroplating - Manufacture of matches and fireworks		
Mercury (Hg) *# - **Elemental and inorganic Hg**	- Electrolytic production of sodium hydroxide, chlorine and acetic acid as fluid cathode - Manufacture of scientific instruments (e.g. barometers, thermometers), electrical control devices (batteries, meters, switches) - Manufacture of antifouling paints and pigments - Dentistry - Plating of gold, silver, bronze and tin in jewellers - Photography and photogravure	- Irritation of the lungs, gastrointestinal tract - Circulatory collapse - Acute renal failure	- Gastrointestinal (metallic taste, burning sensation in mouth, loose teeth, gingivostomatitis, excessive salivation and nausea) - CNS (tremor, fatigue, depression, headaches) - Neuro-psychiatric (easy blushing and intense shyness, anxiety, emotional lability, irritability, insomnia) - Renal (reversible proteinuria, nephrotic syndrome)

Common Metal	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
- **Organic Hg (methyl, ethyl and phenyl Hg)**	- Laboratories (soil testing using mercury as a pressure medium, malt analysis for protein content in brewery) - Manufacture and use of certain pharmaceutical products (e.g. antiseptics, diuretics, contraceptives) - Manufacture and use of pesticides - Manufacture and use of paints and waxes (especially antifouling paints, paint preservatives) - Use as catalysts and alkylating agents in chemical industry	- As above	- Neurological (paraesthesia, concentric constrictions of visual fields, impairment of hearing, rigidity, tremor, seizures) - Others (fatigue, dyspnoea, chest and abdominal pain, vomiting)
Phosphorous[3] (white phosphorus is the most toxic) #	- Manufacture of special glass in sodium lamps - Manufacture of heads of strike matches, explosives, pyrotechnics, smoke bombs and screens	- Severe irritation of mucous membranes (throat, eyes and lung) - Sweating and weakness	- Necrosis of the jawbone (phossy jaw) subsequent distortion of face - Anaemia and leucopaenia may be observed

Common Metal	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
	- Manufacture of fertilisers containing phosphoric acid and phosphates - Manufacture of pesticides and rodenticides - Manufacture of food additives and toothpaste	- Gastrointestinal (Abdominal pain, haematemesis, diarrhoea) - Skin (severe burns which are necrotic, fluorescent under UV light and have garlic-like odour) - CNS (seizures, delirium and coma) - Acute renal necrosis - Acute hepatic necrosis - Cardiovascular collapse	- Necrosis of the jawbone (phossy jaw) subsequent distortion of face - Anaemia and leucopaenia may be observed

**** Workers exposed to these hazards are required to undergo mandatory medical examinations under the WSH (Medical Examinations) Regulations.***

 # Poisoning by this chemical is a notifiable occupational disease under the Workplace Safety and Health Act (WSH Act).

II. Ask company for results of exposure monitoring and the safety data sheet (SDS) of chemicals used. Review if the chemicals handled contain the above metals. If the exposure levels exceed the PELs, a work-related condition should be suspected. Please note that hand contamination and accidental ingestion are also possible sources of exposure.

[3] Phosphorous is a very reactive non-metal.

III. Carry out specific investigations such as toxicological tests or biomarkers to document worker's absorption of metals and its effects on health as listed in the Workplace Safety and Health Guidelines: Statutory Medical Examinations. Workers with biomarkers exceeding the threshold limits specified in the Guidelines should be suspended from further exposure and reviewed closely.

IV. Exclude non-occupational causes, such as from the diet or herbal products.

2.10.6 Management

I. Advice to workers

- Stop further exposure by suspension from further exposure or reassign to another area without exposure to the metal for a period of up to three months.
- Repeat the specific biological indicator at end of three months or earlier. If results improve, continue monitoring every three months or earlier till results revert to normal before returning to previous work with exposure. If not, refer to an Occupational Medicine Clinic for further investigation.
- Workers with signs and symptoms of poisoning should be referred directly to the Emergency Department of public hospitals with relevant SDSs for further evaluation and management. Cases can be followed up with an Occupational Medicine Specialist.
- Practice good personal hygiene to reduce absorption (e.g. avoid smoking and eating with hands at the workplace).
- Instruct workers on the proper use and maintenance of appropriate PPE, such as goggles, respirators and gloves (see Appendix B).

II. Advice to companies

- Review the workplace risk assessment and put in place control measures to reduce exposure to hazardous metals.
- Conduct regular toxic substances monitoring to assess the workers' level of exposure to hazards. The frequency of monitoring required is dependent on the level of toxic substance measured as a percentage of the PEL stated in the First Schedule of the WSH (General Provisions) regulations.
- Identify workers at-risk and educate them on measures to reduce exposure to hazardous metals.
- Educate workers on and ensure provision of suitable PPE to minimise hazardous metal exposure (see Appendix B).
- Notify the Ministry of Manpower (Poisoning by certain metals is a notifiable disease).
- Advice on requirements for medical examinations under the WSH (Medical Examinations) Regulations.

2.10.7 Management

1. Workplace Safety and Health (Medical Examinations) Regulations
2. Workplace Safety and Health (General Provisions) Regulations
3. Workplace Safety and Health Guidelines: Statutory Medical Examinations
4. Ministry of Manpower Webpage – Enhanced workplace health surveillance (WHS+)
5. Ministry of Manpower Webpage – Requirements for hygiene monitoring
6. Casarett & Doull's Essentials of Toxicology, Fourth Edition
7. Agency for Toxic Substances and Disease Registry – Toxic Substances Portal
8. Diagnostic and exposure criteria for occupational diseases – Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010)
9. List of Occupational Medicine Specialist clinics on the MOM website

2.11 Poisoning: Pesticides and Fumigants

2.11.1 Introduction

Pesticides are chemical compounds used to eliminate pests and vectors of diseases and protect crops. They are used as insecticides, herbicides, fungicides, rodenticides, larvicides etc, and some may have multiple uses. They are classified according to chemical classes with some being categorised across multiple chemical classes. Fumigants are hazardous chemical gases used to kill pests in food warehouses, ports and ships (vessels) and certain buildings. Use of such fumigants in Singapore is legislated by the Hydrogen Cyanide Act and its Regulations and is under the purview of the National Environment Agency. Fumigants used in Singapore include hydrogen cyanide, methyl bromide and hydrogen phosphide (also known as phosphine).

Occupational exposures to these chemicals can occur through contact (dermal/ocular) and inhalational routes, while non-occupational exposures usually occur through the dermal route and/or by accidental ingestion.

Common chemical classes of pesticides that may have human health effects are:

- Carbamates (insecticides)
- Coumarin derivatives (rodenticides)
- Nicotinoid compounds (insecticides)
- Organophosphorus compounds (insecticides and herbicides)
- Pyrethroids (insecticides)

Common exposure scenarios include:

- Production and formulation of commercial pesticides
- Mixing, preparation and application (e.g. spraying, fogging etc) of pesticides for agriculture or pest control for public health purposes
- Use of materials or objects treated with pesticides
- Entry into areas that had been treated with pesticides. This may include members of the public or household members.
- Use of pesticides to prevent biological degradation *e.g. paper manufacture, leather tanning etc)

The health effects of pesticides can be affected by the formulation of the compounds, whether as a single chemical or as a mixture of chemicals, the concentration and properties of the chemical(s), and physical state of the product. A mixture of pesticides may be used and these together with the addition of solvents, emulsifiers, surfactants, preservatives etc, can affect the toxicity of the pesticide. Such added chemicals may also have innate toxicity of its own and result in compounded adverse health effects.

Some of the common mechanisms of action of pesticides are interference with:

- Axonal nerve conduction (pyrethroids)
- Synaptic transmission (organophosphorus compounds, carbamates)
- Mitochondrial respiration (paraquat and hexachlorobenzene)
- Blood coagulation (coumarin rodenticides)

2.11.2 Clinical Presentation

The clinical presentations vary depending on the type of pesticide/fumigant used and are typically acute in nature. Exposure is mainly via inhalation and/or contact (dermal, ocular, mucosal) and occasionally by ingestion. The main organs affected are the skin, eyes, mucosae of the respiratory tract, and digestive and nervous systems.

The acute effects can range from cholinergic effects to weakness, confusion, coma and convulsions. Neurological symptoms, shortness of breath, nausea and vomiting can also occur. Chronic effects usually present as peripheral neuropathy and neuropathic effects.

For example, acute poisoning by organophosphorus and carbamate insecticides usually present with acute cholinergic crisis presenting with muscarinic symptoms (DUMBBELLS), nicotinic effects at neuromuscular junction with weakness, fasciculation and paralysis, and CNS symptoms of confusion convulsion and coma. Organophosphate-induced delayed neuropathy may develop 20 to 25 days and up to six weeks after recovery from an acute cholinergic crisis. Symptoms may continue to progress for three to six months.

DUMBBELLS is a mnemonic for the muscarinic effects of acetylcholine and stands for:
D – Diaphoresis and diarrhoea
U – Urination
M – Miosis
B – Bronchospasm and bronchorrhoea
B – Bradycardia
E – Emesis
L – Lacrimation
L – Lethargy
S – Salivation and Sweating

2.11.3 Differential Diagnosis

Exclude non-occupational causes due to:

- Other causes of neurological disorders, such as motor neuron disease, impending cerebrovascular accident, diabetic ketoacidosis.
- Gastroenteritis or food poisoning – acute pesticide poisoning may present with diarrhoea and vomiting.
- Non-occupational exposure to pesticides used in domestic settings, accidental ingestion of pesticides and exposure to nerve agents used in chemical warfare, especially in a mass casualty situation.

2.11.4 Diagnostic Criteria of Work-Relatedness

A detailed occupational history for exposure and its correlation to the clinical presentation is critical. Description of work-related processes and the need for personal protective equipment (PPE) will help in assessing exposure. In addition, supporting documents such as safety data sheets (SDS) and results of personal or workplace exposure monitoring where available will aid in determining the diagnosis of work-related poisoning.

2.11.5 Investigation to Establish Work-Relatedness

General Principles

I. Establish the exposure history to the pesticides/fumigants and correlate it with the signs and symptoms presented. The main exposure situations that would raise suspicion that the worker's clinical presentation could be work-related are listed in Table 12.

II. Correlate the exposure history with the individual's symptoms and investigation results.
- Acute poisoning does not usually present a diagnostic challenge as a history of excessive exposure is usually available and the clinical manifestations are present. However, mild cases of poisoning may not be apparent as the symptoms can be non-specific.
- For some pesticides, obtain biological samples to substantiate the diagnosis and monitor progress of condition.

III. Obtain further information from the employer.
- Ask the employer/company for details of work activities and SDSes of the pesticide/fumigant and results of workplace exposure monitoring if available. This provides additional information to substantiate the history from the worker.

2.11.6 Diagnostic Criteria for Poisoning

The range of pesticides and fumigants is wide and heterogeneous, a selection of more commonly encountered exposure conditions is presented in this chapter. Users can consult the resources at the end of this chapter for more information on other conditions attributed to pesticides or fumigants.

2.11.7 Diseases Due to Pesticide Exposure

Acute pesticide poisoning refers to the development of health effects following exposure to a pesticide that occurred recently. It can include exposure to a single high level of pesticide or to repeated exposures to pesticides with a long half-life. In addition, the presence of additional chemicals such as solvents may increase skin absorption and cause irritation to the mucous membranes, eyes and skin.

The clinical presentation of acute pesticide poisoning includes:
- Eye irritation such as stinging pain, photophobia, ulceration, conjunctival irritation.
- Skin irritation such as redness, itching, swelling, pain and blistering.
- Nose and upper respiratory tract irritation such as coughing, choking, chest tightness, shortness of breath.
- At higher concentrations, tachypnoea and chemical-induced asthma or reactive airways dysfunction syndrome (RADS) may be observed.
- In severe cases, pulmonary oedema may occur hours or days after exposure, characterised by a rapid onset of dyspnoea at rest, tachypnoea, tachycardia and severe hypoxaemia.
- Other nonspecific symptoms may include salivation, dysphagia, intense thirst, nausea, vomiting, haemorrhage, diarrhoea, and abdominal pain.

Table 12: **Clinical presentations of poisoning and at-risk exposures to selected pesticides and fumigants**

Type of Pesticide / Fumigant	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Organophosphorus compounds (Organophosphate (OP))* - **Acephate** - **Azamethiphos** - **Chlorpyrifos** - **Diazinon** - **Dichlorvos** - **Dimethoate** - **Fenitrothion** - **Fenthion** - **Malathion** - **Pirimiphos** - **Profenofos** - **Quinalphos** - **Temephos** - **Trichlorfon**	Used as an insecticide - Pest control - Farming and gardening use - Crop management - Manufacture of pesticide Routes of exposure: - inhalation - dermal - ingestion Minimum duration of exposure: few minutes. Maximum latent period: Few hours up to three days	<u>1. Acute cholinergic crisis</u> - Excess Acetylcholine effects on the central nervous system (CNS) – confusion, convulsions, coma - Excess Acetylcholine effects on muscarinic receptors (DUMBBELLS) - Diarrhoea, diaphoresis - Urination - Miosis - Bradycardia - Bronchorrhoea, bronchospasm - Emesis - Lacrimation - Lethargy - Salivation, sweating - Excess Acetylcholine effects on nicotinic receptors - Weakness - Fasciculation - Myoclonus - Confusion - Coma - Convulsion	<u>1. Organophosphate-induced delayed neuropathy (OPIDN)</u> Associated with certain OPs: phosphates, phosphonates, and phosphoramidates (e.g. dichlorvos, isofenphos, methamidophos, mevinphos, mipafox, trichlorfon, trichlornat, phosphamidon) after high exposure resulting in an acute cholinergic crisis. Occurs about six weeks after exposure <u>Neurological</u> - muscle cramps with distal paraesthesias - progressing to progressive weakness of the legs - decreased deep tendon reflexes - motor neuropathy (flaccid paralysis may progress to spastic paralysis)

Type of Pesticide / Fumigant	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
		- Various other effects secondary to additive chemical contents in OP compounds <u>2. Intermediate syndrome</u> - General weakness and respiratory failure - affects mainly muscles innervated by cranial nerves. - occurs 24 to 96 hrs after initial exposure and lasts for 1 to 2 weeks	<u>2. Chronic low exposure to OP leading to chronic OP toxicity</u> - Symmetric sensorimotor axonopathy: leg cramping to extremity weakness and paralysis. Sensory > Motor neurons with predilection for long axons. Ascending paralysis type manifestation can be confused for Guillian-Barré syndrome. Long-term effects that include neuropsychological manifestations with delays in executive functioning, attention, psychomotor speed, verbal and memory functions, coordination, and more. Diagnosis is clinical and should be based on history, physical exam findings of cholinergic toxidrome, garlic-like or hydrocarbon odour on the patient, or if presenting with neuropathies as above.

Type of Pesticide / Fumigant	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Carbamates - **Carbaryl** - **Carbosulfan** - **Fenobucarb (BPMC)** - **Methomyl** - **Propoxur**	Used as an insecticide - Pest control - Farming and gardening use, - Crop management - Manufacture of pesticide Minimum duration of exposure: few minutes. Maximum latent period: up to 24 hours	Similar to OP except that they are usually self-limiting and resolve within a few hours of stopping exposure.	
Pyrethrum and synthetic pyrethoid insecticides - **Allethrin** - **Bifenthrin** - **Bioallethrin** - **Bioresmethrin** - **Cyfluthrin** - **Cyhalothrin** - **Cypermethrin** - **Cyphenothrin** - **Deltamethrin** - **Esfenvalerate** - **Etofenprox** - **Fenvalerate** - **Imiprothrin** - **Metofluthrin** - **Permethrin** - **Phenothrin** - **Prallethrin** - **Tetramethrin** - **Transfluthrin**	Used as an insecticide - Agricultural applications - Pest control Routes of exposure: - inhalation - dermal - ingestion	<u>Skin contact</u> temporary paraesthaesia at the area of contact: abnormal cutaneous sensations such as tingling, burning, stinging, numbness, and itching <u>Neurologic (main health effects) usually with high exposure</u> - dizziness - headache - nausea - muscle twitching - reduced energy - changes in awareness - convulsions and - loss of consciousness.	

Type of Pesticide / Fumigant	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Nicotinoid compounds (Neonicotinoids) - **Acetamiprid** - **Clothianidin** - **Dinotefuran** - **Imidacloprid** - **Thiamethoxam**	Used as an insecticide - Agricultural applications - Pest control Routes of exposure: - inhalation - dermal - ocular - ingestion	<u>Typical adverse effects:</u> - ocular, dermal and oral irritation - nausea, vomiting, abdominal pain - erythema and red eye - respiratory and cardiovascular symptoms, metabolic imbalance and renal dysfunction	
Coumarin Derivatives <u>First-generation compounds</u> - warfarin - coumachlor - Coumatetralyl <u>Second generation compounds</u> (Superwarfarins) - Brodifacoum - Bromadiolone - Difenacoum - Diphacinone - Flocoumafen	Rodenticides Routes of exposure: - inhalation - dermal - ingestion	<u>Clinical manifestation:</u> - Primary manifestations are haemorrhagic, e.g. unusual bruising, nosebleeds, bleeding gums, exaggerated bleeding from cuts, exaggerated and prolonged menstrual bleeding, anaemia, melaena, haemothorax, hyphaema, epistaxis, haemoptysis, and haematuria. - Other manifestations may develop, such as weakness, dizziness, ataxia, colic, gangrene, and tachypnoea 2nd generation compounds are more potent than first generation compounds and effects may be delayed and last up to two years after initial exposure.	
Fumigants			
Hydrogen cyanide	- Fumigation of soil, crops - Fumigation in mills, warehouses, ships, freight cars - Manufacture of fumigant	<u>Clinical presentation</u> <u>Eye, skin and mucosa</u> - burns/corrosion - cough, sore throat <u>Respiratory</u> - dyspnoea, tachypnoea, wheezing - Pulmonary oedema and respiratory failure - irritant induced asthma, reactive airways dysfunction (RADS)	

Type of Pesticide / Fumigant	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
	Routes of exposure: - inhalation - dermal - ocular	<u>Cardiac</u> - tachycardia, and chest pain. Neurological - Altered mental states, confusion, convulsions & coma Death can occur in the most severe cases.	
Methyl bromide - also known as bromomethane, monobromomethane	- Fumigation in mills, warehouses, ships, freight cars - Manufacture of fumigant Routes of exposure: - inhalation - dermal - ingestion	<u>Respiratory:</u> - cough, oedema, dyspnoea - haemorrhagic lesions <u>Neurological:</u> - headache - weakness, ataxia, tremors, paralysis - seizures. <u>Skin:</u> - Erythema, oedema, and blisters on contact with liquefied methyl bromide or its vapour - conjunctivitis, erythema, and oedema of the eyelids when exposed to methyl bromide vapour	<u>Neurological:</u> - visual disturbance, ataxia, vertigo, tremor and seizures, peripheral neuropathy <u>Neurobehavioural</u> - agitation

Type of Pesticide / Fumigant	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Hydrogen phosphide (Phosphine)	- Fumigation in mills, warehouses, ships, freight cars - Manufacture of fumigant Routes of exposure: - inhalation - dermal - ingestion	<u>Central nervous system:</u> - headache, restlessness, dizziness - loss of feeling - impaired gait - trembling of the extremities during movement - double vision. Severe exposure can cause seizures and coma. <u>Respiratory:</u> - irritative effects - chest tightness, cough, and shortness of breath - pulmonary oedema (may be delayed in onset) <u>Cardiovascular:</u> - hypotension, reduction in cardiac output - tachycardia, irregular heart beat, or - cardiac arrest. <u>Gastrointestinal:</u> - nausea, vomiting, abdominal pain, and diarrhoea.	<u>Chronic Exposure</u> Chronic exposure to very low concentrations may result in anaemia, bronchitis, gastrointestinal disturbances, and visual, speech, and motor disturbances.

Type of Pesticide / Fumigant	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
		Hepatic: - jaundice, enlarged liver, elevated serum transaminases, and increased bilirubin in the blood. - Usually presents 48 to 72 hours later Renal: - Blood and protein in the urine - acute kidney failure Electrolytes: - combined respiratory and metabolic acidosis. - reports of significant hypomagnesaemia and hypermagnesaemia associated with massive focal myocardial damage	

+ Some pesticides may be categorised across multiple chemical classes.

Notes:

1. Each of the above compounds may have multiple isomers and trade names. Do check the SDS for more details on the chemical composition and health effects.
2. The highest exposure and incidences of poisoning occur with individuals involved in agricultural and horticultural pest control operations. Exposure can occur during the mixing of the compounds with water and spraying of the pesticides. Workers involved in the manufacture of pesticides may also be exposed to the hazard.
3. The majority of poisonings are based on a clinical diagnosis. Toxicology tests could be considered if the actual agents could not be identified or is required for confirmation.
4. Doctors should contact the laboratory for the availability of the toxicological tests if the tests are deemed necessary.
5. Workers exposed to organophosphate compounds (OPs) are required to undergo statutory medical examinations under the WSH (Medical Examinations) Regulations. The examinations include:
    a. The estimation of red blood count (RBC) cholinesterase. However, as this test is not available, estimation of the serum butyrylcholinesterase levels may be used.
    b. Serum butyrylcholinesterase (also known as serum cholinesterase) levels can be compared with baseline levels (if available). Exposure to high levels of organophosphates would depress the activity of serum cholinesterase, thus levels below the lower limit of normal would be deemed abnormal.
6. Workers who are exposed to carbamates would experience similar symptoms as those exposed to organophospate compounds. Carbamates also depress the activity of serum cholinesterase, however the effect is short acting and the worker would usually recover the following day.

2.11.8 Management

I. Advice to workers

- Follow the suspension period for organophosphates (OP) to avoid further exposure. For those exposed to OP, attend the doctor's follow up to monitor the serum cholinesterase level as outlined in the "WSH Guidelines on Statutory Medical Examinations".
- Advise worker on good personal hygiene and proper use of PPE, such as respirators, impervious gloves, PVC or rubber boots, face shields and overcoats/aprons.
- Workers with signs and symptoms of pesticide poisoning should be sent immediately to the Emergency Department of a public hospital for treatment and management.
- Advise the worker and/or supervisors to bring the SDS of the pesticide for the doctor's reference.

II. Advice to companies

- Send workers with signs and symptoms of pesticide poisoning immediately to the Emergency Department of a public hospital for treatment and management.
- Remove worker from further exposure to the pesticide and/or reassign him to pesticide-free area for the period specified by the DWD.
- In acute toxic exposures, activate emergency medical services immediately and evacuate exposed workers to safe zones if feasible. Institute first-aid measures and early decontamination as recommended in the SDS.
- Review the workplace risk assessment and put in place control measures to reduce exposure. Suitable PPE should be provided and worn. This could include a full-face mask with gas canister and protective suits and gloves. More information on PPE can be found in Appendix B.
- Advice on the requirements for medical examinations under the WSH (Medical Examinations) Regulations.
- Look for evidence of poisoning in other workers.
- Notify the Ministry of Manpower (poisoning by carbamates or organophosphates is a notifiable and compensable occupational disease).

2.11.9 Further Reading

1. Workplace Safety and Health Act
2. Workplace Safety and Health (Medical Examinations) Regulations
3. Environmental Protection and Management Act (EPMA)
4. Environmental Protection and Management (Hazardous Substances) Regulations
5. Control of Plants (Registration of Pesticides) Rules
6. National Environment Agency (NEA)
7. Singapore Food Agency (SFA)
8. Hydrogen Cyanide (Fumigation) Act 1947 and Hydrogen Cyanide (Fumigation) Regulations
9. International Labour Organisation: Diagnostic and exposure criteria for occupational diseases. Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010)
10. Workplace Safety and Health Guidelines: Statutory Medical Examinations
11. Agency for Toxic Substances and Disease Registry. Toxicological profile for Pyrethrins and pyrethroids (2003).
12. Agency for Toxic Substances and Disease Registry. Medical Management Guidelines for Phosphine
13. Agency for Toxic Substances and Disease Registry. Medical Management Guidelines for methyl bromide (Bromomethane)
14. US Environmental Protection Agency. Hazard Summary for Phosphine

2.12 Poisoning: Solvents

2.12.1 Introduction

The term solvent means "material used to dissolve another material". They are broadly classified as aqueous (water-based) and non-aqueous solvents (organic solvents). Most industrial solvents are used for cleaning, degreasing, thinning and extraction. Solvents are also used as chemical intermediates in the manufacture of other chemical products.

Commonly used organic solvents are usually volatile. These include aromatics such as benzene and toluene; ketones such as methyl ethyl ketone (MEK) and methyl isobutyl ketone (MIBK); and halogenated hydrocarbons such as trichloroethylene, perchloroethylene and carbon tetrachloride. Other commonly used solvents are alcohols, xylene and ether.

2.12.2 Clinical Presentation

Solvents are easily absorbed through the skin and inhaled in the workplace.

Acute high exposures through inhalation may cause central nervous system depression, respiratory arrest, unconsciousness and death.

Prolonged exposure may lead to neurological impairment such as peripheral neuropathy, irritability and memory loss. In severe cases, toxic encephalopathy, manifested by diminished concentration, memory, and learning ability may occur. Hepato-renal injuries are more common with halogenated hydrocarbons.

Dermal exposures can cause dermatitis. Carcinogenic risk is increased with certain solvents, such as benzene (known to cause leukaemia).

When a worker is exposed to more than one solvent, their effects on target organs may be additive.

2.12.3 Differential Diagnosis

Exclude non-occupational causes due to:

- Solvent abusers, e.g. glue sniffers
- Excessive alcohol intake
- Hobbies using glue
- Drugs such as phenobarbital and chloral hydrate

2.12.4 Diagnostic Criteria of Work-Relatedness

A thorough occupational history of exposure is critical. In addition, supporting documents of results of personal or workplace exposure monitoring will aid in determining the diagnosis of work-related solvent poisoning.

2.12.5 Investigation to Establish Work-Relatedness

I. Establish the exposure history to the solvent and correlate it with the signs and symptoms presented. The main exposure situations that would raise suspicion that the worker's clinical presentation is work-related are listed in the table. Please refer to Appendix A for further information on common work processes that may put the worker at risk of exposure to solvents.

Table 13: **Clinical presentations of poisoning and at-risk exposures to solvents**

Common solvents	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Benzene*#	- Petrochemical industries - Petroleum refineries - Bulk storage terminals involved in loading and unloading of chemicals containing benzene - Manufacture of plastics, synthetic fibres, detergents, synthetic resins - Work involved with handling of fuels containing benzene (e.g. service stations, airport terminals, vehicle workshops) - Work involving use of commercial solvents such as toluene and xylene (where benzene may be present as a contaminant)	- Neurological (dizzy, headache, nausea, vomiting, slurred speech, disorientation, loss of consciousness) - Cardiovascular (palpitations) - Respiratory (cough, sore throat, running nose) - Skin irritation	- Bone marrow depression (leucopaenia, thrombocytopaenia, aplastic anaemia, pancytopaenia) - Skin (dry, scaly dermatitis, erythema and/or blistering) - Neurological (motor and neural damage, impaired vision, fatigue, sleep disturbances) - Carcinogenic (leukaemia)

Common solvents	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
Carbon disulphide# (also known as carbon bisulphide)	- Manufacture of viscose rayon, resins, plywood adhesives, cellophane, varnishes - Refining of petroleum and paraffin - Extraction of oil and rubber - Dry cleaning industries - Degreasing in semiconductor industry	- Neurological (central nervous system depression, delirium, peripheral neuropathy, coma, seizures) - Mucosa (irritation to the skin, the eyes, respiratory tract, blistering with secondary burns may occur with direct skin contact) - Gastrointestinal tract (GIT) (nausea, vomiting) - Renal (failure) - Cardiovascular collapse - Respiratory (pulmonary oedema, respiratory failure)	- Neurological (diminished mental and motor ability with staggering gait and loss of coordination, impaired vision, fatigue, sleep disturbances and defective memory) - Psychological (irritability, suicidal tendencies, manic depressive psychosis) - Cardiovascular (atherosclerosis, heart disease) - Genitourinary (microhaematuria, albuminuria, hypertensive nephrosclerosis) - Others (chronic gastritis, anaemia)
Perchloroethylene*# (PCE) (also known as tetrachloroethylene)	- Dry cleaning and textile processing	- Mucosa membranes (irritation to eyes, nose, and respiratory tract)	- Central nervous system (non-specific complaints like headache, dizziness, fatigue and incoordination)

Common solvents	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
	- Degreasing of metal parts and equipment in metal fabrication, automotive, shipyards, aircraft and aerospace industries - Cleaning of lenses in the optical industry - Manufacture and use of printing ink, varnishes, adhesives, polishes, rubber coatings and silicones	- Central nervous system (massive exposure can cause dizziness, headache, nausea, incoordination, coma and death)	- Skin (irritation and even burns) - Liver (cirrhosis has been observed in workers exposed to high levels)
Trichloroethylene*# (TCE)	- Degreasing of metal parts in metal, electronics, automotive, shipyards, aircraft and aerospace industries - Spot removers in dry cleaning - Cleaning of lenses in the optical industry - Use as solvent for extraction of waxes, fats, resins and oils	- Mucosa membranes (irritation to eyes, nose, and respiratory tract) - Central nervous system (massive exposure can cause excitation, dizziness and euphoria initially. This is followed by a depressive phase of headache, nausea, sleepiness and coma)	- Central nervous system (non-specific complaints like headache, irritability, fatigue and insomnia) - Skin (irritation and dermatitis) - Liver (few cases of hepatitis-like syndromes and steatosis have been reported) - Renal (proteinuria and raised blood urea)

Common solvents	Exposure situations/At-risk exposures	Acute Presentation	Chronic Presentation
	- Manufacture and use of printing inks, varnishes, adhesives, paints, lacquers, rug cleaners and disinfectants	- Respiratory system (chemical pneumonitis and death from respiratory failure may occur) - Cardiovascular (high exposure can sensitise the myocardium leading to arrhythmia and cardiac arrest)	- Others (severe potentially fatal systemic allergic reaction presenting as Stevens Johnson Syndrome or toxic epidermal necrolysis. This may occur even with minimal exposure and usually presents as fever, rash and jaundice within two to three weeks of exposure).
Toluene (a homologue of benzene)#	- Manufacture of rubber, dye, glue, printing inks, paints - Removal of paints from contaminated parts - Wood and furniture industry	- Mucosa membranes (irritation to eyes, nose, and respiratory tract) - Neurological (headaches, confusion, and memory loss, impaired judgment, unbalanced gait, blurred vision, loss of consciousness, coma, and death) - Respiratory (pneumonitis, respiratory arrest)	- Central nervous system (non-specific complaints like headache, irritability, fatigue and insomnia) - Renal (tubular damage) - Skin (dermatitis) - Gastrointestinal tract (GIT) (liver dysfunction)

**** Workers exposed to these chemicals are required to undergo mandatory medical examinations under the WSH (Medical Examinations) Regulations.***
 # Poisoning by these solvents is a notifiable OD under the Workplace Safety and Health Act.

II. Ask company for results of exposure monitoring and the SDSes of the chemicals used. Review if the chemicals handled contain the above solvents. If the exposure levels exceed the PELs, a work-related condition should be suspected. Please note that solvents can be easily absorbed through the skin even in the absence of high exposure monitoring results.

III. Carry out specific investigations to document worker's absorption of certain solvents and its effects on health as listed in the Workplace Safety and Health Guidelines: Statutory Medical Examinations. For example, workers exposed to benzene are monitored with either urinary tt-muconic acid (ttma) or s-phenylmercapturic acid (spma), while workers exposed to TCE and PCE are monitored with urinary trichloroacetic acid (U-TCAA[4]).

Workers with biological indicators exceeding the threshold limits specified in the Guidelines should be suspended from further exposure and reviewed closely. Test the blood toluene levels for workers exposed to toluene and who are suspected to have toluene poisoning.

IV. Exclude non occupational causes, such as exposure to solvents from hobbies or solvent abuse.

Note:
Liver function tests, urine analysis, nerve conduction and electromyographic studies may be performed in cases of suspected solvent poisoning, especially for workers with chronic exposure.

2.12.6 Management

Most workers improve once they are no longer exposed to the solvent (especially if the exposure is of short duration). For those with high and prolonged exposures, recovery may be incomplete.

I. Advice to workers

- Stop further exposure to the solvent for up to one month or as instructed by the doctor.
- Attend the medical follow up consultations to monitor the repeat tests at end of the suspension period. If results improve, continue monitoring until the results return to normal before advising on return to previous work. If not, attend the referral to an Occupational Medicine Clinic for further investigation.
- Workers with symptoms should be referred to an Occupational Medicine clinic. In cases of severe symptoms, the worker should be referred to the Emergency Department of a public hospital as soon as possible.
- Practise good personal hygiene to reduce absorption (e.g. avoid smoking and eating with hands at the workplace).
- Instruct workers on the proper use and maintenance of appropriate PPE, such as goggles, respirators and gloves (see Appendix B).

[4] Urine trichloroacetic acid was previously known as U-TCA.

II. Advice to companies

- Review the workplace risk assessment and put in place control measures to reduce exposure to hazardous chemicals.
- Conduct regular toxic substances monitoring to assess the workers' level of exposure to hazards. The frequency of monitoring required is dependent on the level of toxic substance measured as a percentage of the PEL stated in the First Schedule of the WSH (General Provisions) regulations.
- Identify and educate high-risk workers (those with liver diseases, solvent abuse history or alcoholism) on the health effects of toxic substance exposure, and advise that they should not work in areas with significant solvent exposure.
- Educate workers on proper use of PPE to minimise hazardous chemical exposure (see Appendix B).
- Find out more on requirements for medical examinations under the WSH (Medical Examinations) Regulations.
- Notify the Ministry of Manpower.

2.12.7 Further Reading

1. Workplace Safety and Health (Medical Examinations) Regulations
2. Workplace Safety and Health (General Provisions) Regulations
3. Workplace Safety and Health Guidelines: Statutory Medical Examinations
4. Ministry of Manpower Webpage - Enhanced workplace health surveillance (WHS+)
5. Ministry of Manpower Webpage – Requirements for hygiene monitoring
6. Casarett & Doull's Essentials of Toxicology, Fourth Edition
7. Agency for Toxic Substances and Disease Registry – Toxic Substances Portal
8. Diagnostic and exposure criteria for occupational diseases - Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010)
9. List of Occupational Medicine Specialist clinics on the MOM website

2.13 Toxic Anaemia

2.13.1 Introduction

Toxic anaemia is a notifiable disease. Toxic anaemia occurs when the erythrocytes are reduced in number or volume or are deficient in haemoglobin as a result of damage caused by medication, chemicals and circulating metabolites. The anaemia may be followed by leucopaenia, thrombocytopaenia and pancytopaenia.

2.13.2 Clinical Presentation

Abnormal blood count and/or peripheral blood film is probably the first indication of a blood disorder. Mild forms of toxic anaemia may not cause any noticeable symptoms. Regardless of the cause, all types of anaemia have similar signs and symptoms because of the blood's reduced capacity to carry oxygen. A physical examination may show the presence of anaemia and mild jaundice. Common symptoms include anorexia, general weakness, dizziness and exercise intolerance.

Certain exposures may give rise to more specific clinical signs, which suggest the underlying cause. For example, a blue line on the gingival margins (lead), peripheral neuropathy (arsenic, lead), plantar-palmar hyperkeratosis and transverse lines in the nail bed (arsenic). In addition, some chemicals can cause specific blood disorders (e.g. leukaemia from exposure to benzene, and ionising radiation and aplastic anaemia from exposure to trinitrotoluene). These are however, rarely found in today's occupational settings.

In some cases, toxic anaemia develops within hours or days of exposure to a toxin (e.g. arsine causing massive intravascular haemolysis). But in most cases, it takes months of exposure before symptoms of toxic anaemia appear. Often, the symptoms of toxic anaemia clear when exposure to the toxin stops. Prolonged exposure may result in chronic renal failure and/or neurologic impairment.

2.13.3 Differential Diagnosis

Exclude non-occupational causes such as those due to:

- Hereditary conditions - G6PD deficiency, Thalassemia, Haemophilia
- Drugs, e.g. sulphonamides, NSAIDs, chloramphenicol
- Nutritional deficiency - iron, folic acid and Vitamin B12 deficiency

2.13.4 Diagnostic Criteria of Work-Relatedness

A thorough occupational history of exposure is critical. In addition, supporting documents of results of personal or workplace exposure monitoring will aid in determining the diagnosis of work-related anaemia.

2.13.5 Investigation to Establish Work-Relatedness

I. Take a detailed occupational history to establish if there is exposure to the following toxic agents:

Table 14: **At-risk exposures and agents causing toxic anaemia**

Classification of Agents	Type of Agent	Exposure situations/ At-Risk Exposure
Metals	Arsenic*	- Manufacture of glass, paint, enamels, weed killers, pesticides
	Inorganic lead*	- Manufacture of lead acid batteries - Cutting of scrap metal containing lead - Production of polyvinyl chloride (PVC) stabilisers - Manufacture of paint pigments
	Organic lead*	- Manufacture of anti-knock additives for petroleum (not used in Singapore since 1980)
	Mercury*	- Petrochemical and chemical industry - Repair, maintenance and disposal of medical instruments, e.g. blood pressure sets containing mercury - Analytical laboratories
Solvents	Benzene*	- Petrochemical industry
Gases	Arsine	- Semiconductor industry where galvanising, etching, soldering and lead plating are carried out - Smelting and refining industry - Cleaning of acid storage tanks - Waste treatment plants (acid comes in contact with metals containing arsenic)
Other chemicals	Aniline	- Manufacture of rubber, dyes
	Trinitrotoluene	- Manufacture of explosives
Physical	Ionising radiation	- Radio-isotopes imaging in medical and industrial setting (particularly to evaluate the quality of welded joints in ship and aircraft maintenance)

**** Workers exposed to these chemicals are required to undergo prescribed medical examinations under the WSH (Medical Examinations) Regulations. Please refer to the Workplace Safety and Health Guidelines: Statutory Medical Examinations for further details.***

II. Correlate the exposure history with the individual biological result

III. Ask company for results of exposure monitoring
   a. Occupational hygiene monitoring
      - Obtain results of the occupational hygiene monitoring for chemicals at the workplace e.g. inorganic lead, benzene etc. The results can provide additional support for the diagnosis if the levels exceed the PELs.

   b. Medical monitoring
      - Carry out specific investigations including toxicological tests or biomarkers to document worker's absorption of agents (biomarkers of exposure) and the effects on health (biomarkers of effect) for some of the chemicals in the above table. Reference can be made to the Workplace Safety and Health Guidelines: Statutory Medical Examinations.
      - Workers with biomarkers of exposure and/or effect exceeding the threshold limits specified in the Guidelines should be suspended from further exposure and reviewed and followed up closely. Using lead exposure as an example, the patient's serial haemoglobin results should be reviewed and correlated with the blood lead and/or urine lead levels. Basophilic stippling of the red cells may be seen on a peripheral blood film and could be evidence of lead exposure. Do note that the degree of stippling does not correlate with the body burden of lead.

IV. Exclude non-occupational causes, pre-existing and predisposing factors (e.g. nutritional deficiencies, Thalassemia and other blood disorders).

2.13.6 Management

Investigate workers with abnormal blood test results. Depending on the cause and severity of the anaemia, treatment may involve advice on adequate nutrition, haematinics, and removal from exposure to toxic chemicals. For toxic anaemia, treatment is mainly supportive, but antidotes and chelation therapy may be available in specific poisoning, such as lead. Some patients improve quickly once they are no longer exposed to the toxin, especially if the disease is detected early. For others, recovery may take months.

I. Advice to workers

- Advice to stop further exposure by suspension from further exposure or reassign to another area without exposure to the toxic agent for a period of 3 months (especially if the worker is a susceptible worker with pre-existing or predisposing condition, or there are additional abnormal parameters and/or there are other predisposing factors).
- Repeat the blood tests at end of 3 months. If improving, continue monitoring every 3 months till results revert to normal before returning to previous work and exposure. If not, refer to an Occupational Medicine Clinic for further investigation.
- Workers with symptoms should be referred to an Occupational Medicine Clinic. In cases of severe symptoms, the worker should be referred to the Emergency Department of a public hospital as soon as possible.
- Advice on diet, good personal hygiene to reduce absorption (e.g. avoid smoking and eating with hands at the workplace) and the proper use and maintenance of appropriate personal protective equipment (PPE), such as respirators and gloves when going to workplaces with exposure.

II. Advice to companies

- Identify high risk workers to reduce their chance of toxic exposure.
- Review the workplace RA and put in place control measures to reduce exposure.
- Find out more on requirements for medical examinations under the WSH (Medical Examinations) Regulations.
- Notify the Ministry of Manpower (Toxic Anaemia is a notifiable occupational disease).

2.13.7 Further Reading

1. Workplace Safety and Health (Medical Examinations) Regulations
2. Workplace Safety and Health Guidelines: Statutory Medical Examinations
3. Casarett & Doull's Essentials of Toxicology, Fourth Edition
4. Agency for Toxic Substances and Disease Registry – Toxic Substances Portal
5. Diagnostic and exposure criteria for occupational diseases - Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010)
6. List of Occupational Medicine Specialist clinics on the MOM website

2.14 Toxic Hepatitis

2.14.1 Introduction

Toxic hepatitis is an inflammation of the liver which occurs when the liver is damaged by drugs, chemicals and circulating metabolites. Except for a few chemicals that cause specific lesions (e.g. occupational liver angiosarcoma is strongly associated with exposure to vinyl chloride monomer), liver damage due to occupational exposure does not differ clinically or morphologically from non-occupational causes, such as from certain drugs and excessive alcohol intake.

2.14.2 Clinical Presentation

An abnormal liver function test result is usually the first indication of liver damage. Mild forms of toxic hepatitis may not cause any noticeable symptoms. The clinical features are similar irrespective of their underlying cause. Common symptoms include anorexia and lethargy. Clinical presentations of anaemia, jaundice, palmar erythema, liver/spleen enlargement are rare in an occupational setting.

In some cases, toxic hepatitis develops within hours or days of exposure to a hepatoxic agent. But in most cases, it takes months of exposure before symptoms of toxic hepatitis appear. Often, the symptoms of toxic hepatitis clear when exposure to the hepatoxic agent stops. Prolonged exposure, however, can permanently damage the liver, leading to cirrhosis and liver failure.

2.14.3 Differential Diagnosis

Exclude non-occupational causes such as those due to:

- Infections
- Drugs, e.g. contraceptives, statins, aspirin, tetracycline, methotrexate
- Alcohol
- Glue sniffing

2.14.4 Diagnostic Criteria of Work-Relatedness

A thorough occupational history of exposure is critical. In addition, supporting documents of results of personal or workplace exposure monitoring will aid in determining the diagnosis of work-related hepatitis.

2.14.5 Investigation to Establish Work-Relatedness

I. Take a detailed occupational history to establish if there is exposure to the hepatotoxic agents.

Table 15: **At risk exposures and agents causing toxic hepatitis**

Classification of Agents	Type of Agent	Exposure situations/ At-Risk Exposure
Metals	Arsenic*	Manufacture and use of pesticides
	Beryllium	Manufacture of nuclear devices, satellite and radar systems, aircraft bushings and bearings in the defence and aeronautical industry
Solvents	Carbon tetrachloride	Dry cleaning
	Chloroform	Laboratories, especially in pathology labs
	Dimethylformamide	Solvent manufacturing
	2-nitropropane	Painting
	Perchloroethylene (PCE)*	Degreasing/cleaning with solvents, dry cleaning
	Trichloroethylene (TCE)*	Degreasing/cleaning with solvents
	Tetrachloroethane	Manufacture of paints and varnishes
Other chemicals, including nitro-derivatives or amino-derivatives of benzene	Trinitrotoluene	Manufacture of explosives
	Vinyl Chloride Monomer (VCM)*	Rubber, plastic manufacturing
Biological	Dengue haemorrhagic fever	Construction sites, agriculture/farm
	Hepatitis A,B,C,D	Sewage workers, healthcare workers
	Leptospirosis	Sewage and drainage workers, waste collectors, abattoir workers, tunnelling workers
	Malaria	Agriculture/farm workers, military personnel
	Melioidosis	Construction sites, agriculture/farm, military personnel

**** Workers exposed to these chemicals are required to undergo prescribed medical examinations under the WSH (Medical Examinations) Regulations.***

II. Ask company for results of exposure monitoring
   a. Occupational hygiene monitoring
      - Obtain results of occupational hygiene monitoring at the workplace for hepatotoxic chemicals e.g. arsenic, PCE, TCE etc. Results of the occupational hygiene monitoring can provide additional support for the diagnosis if the levels exceed the PELs.
   b. Medical monitoring
      - Carry out specific investigations including toxicological tests or biomarkers to document worker's absorption of the chemicals and the effects on health for some of the chemicals in the above table. Reference can be made to the Workplace Safety and Health Guidelines: Statutory Medical Examinations. Workers with biomarkers exceeding the threshold limits specified in the Guidelines should be suspended from further exposure and reviewed closely. For example, workers exposed to TCE and PCE are monitored with urinary trichloroacetic acid (U-TCAA[5]).

III. Exclude non-occupational causes, pre-existing and predisposing factors (e.g. history of liver disease such as alcoholic liver cirrhosis, infection-related hepatitis).

2.14.6 Management

The cause of abnormal liver function test results should be investigated. For toxic hepatitis, no specific treatment exists. Therefore, treatment is usually supportive and include removal from toxic chemicals. If indicated, antiviral drugs may be used. Some patients improve quickly once they are no longer exposed to the hepatotoxic agent, especially if the disease is detected in the early stage. For others, recovery may take months.

I. Advice to workers

- Suspend the worker from further exposure or reassign them to another area without exposure to the hepatotoxic agent for up to three months (especially if two or more parameters are abnormal and/or there is a predisposing factor which makes the worker more susceptible to liver damage from exposure to hepatotoxic agents).
- Repeat liver function tests at end of the suspension period. If results improve, continue monitoring till results revert to normal before advising on return to previous work and exposure. If not, refer to an Occupational Medicine Clinic for further investigation.
- Workers with symptoms should be referred to an Occupational Medicine Clinic. In cases of severe symptoms, the worker should be referred to an emergency department as soon as possible.
- Advice to reduce alcohol intake, practise good personal hygiene to reduce absorption and proper use and maintenance of personal protective equipment (PPE), such as respirators and gloves when working in an environment with exposure to hazardous chemicals (see Appendix B).

[5] Urine trichloroacetic acid was previously known as U-TCA.

II. Advice to companies

- Advice to strictly follow suspension period to avoid further exposing worker to the hepatotoxic agent.
- Review the workplace risk assessments and put in place control measures to reduce exposure to hazardous chemicals.
- Conduct regular toxic substances monitoring to assess the workers' level of exposure to hazards. The frequency of monitoring required is dependent on the level of toxic substance measured as a percentage of the PEL stated in the First Schedule of the WSH (General Provisions) regulations.
- Advice on requirements for medical examinations under the WSH (Medical Examinations) Regulations.
- Notify the Ministry of Manpower (MOM) via incident report (www.gov.sg/ireport). Toxic Hepatitis is a notifiable occupational disease.

2.14.7 Further Reading

1. Workplace Safety and Health (Medical Examinations) Regulations
2. Workplace Safety and Health Guidelines: Statutory Medical Examinations.
3. Casarett & Doull's Essentials of Toxicology, Fourth Edition
4. Agency for Toxic Substances and Disease Registry – Toxic Substances Portal
5. Diagnostic and exposure criteria for occupational diseases - Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010)
6. List of Occupational Medicine Specialist clinics on the MOM website

2.15 Work-Related Musculoskeletal Disorders

2.15.1 Introduction

Musculoskeletal disorders (MSDs) are common in the general population. The clinical presentation ranges from discomfort and pain to numbness and restriction of movement involving various regions of the musculoskeletal system. From existing epidemiological studies, exposures at work have been associated with the development of musculoskeletal disorders and such disorders have been termed as work-related musculoskeletal disorders (WRMSDs). The burden of WRMSDs is anticipated to rise with an ageing workforce. As workers grow older, age-related musculoskeletal changes increase the risk of injury. Other terms such as repetitive strain injury, occupational overuse syndrome and cumulative trauma disorders have also been used to describe WRMSDs.

2.15.2 Clinical Presentation

The most frequently reported symptoms include discomfort and pain in the neck, shoulders, lower back and limbs. If there is nerve compression, numbness and weakness of the affected regions can occur. Details of some common WRMSDs with their presenting signs and symptoms are listed in the table below.

2.15.3 Differential Diagnosis

Exclude non-occupational sources of exposures that may cause or aggravate the musculoskeletal problem.

- Domestic exposures e.g. housework – manual washing of clothes can lead to wrist pain (de Quervain's tenosynovitis).
- Caring for children or elderly at home
- Sports, hobbies and leisure activities
- Past or pre-existing medical conditions (e.g. inflammatory arthritis, Diabetes Mellitus, degenerative bone conditions, cancer, ankylosing spondylitis)
- History of injuries

2.15.4 Diagnostic Criteria of Work-Relatedness

A detailed occupational history to determine the worker's work process, materials handled, practices and habits is essential to determine the work-relatedness of the musculoskeletal condition. The onset or worsening of MSD should be correlated to the following:

- Duration of employment or work activity.
- Recent change in the work process or practice (an increase in the workload or change in work activities) that may precipitate or aggravate the symptoms.
- Temporal relationship of the aches and pains with work periods (i.e. improvement in symptoms when the patient is away from work or worsening with recent increase in workload or change in type of work activities).

The risk factors for WRMSDs include prolonged awkward/static postures, repetitive motions, handling of excessive loads, forceful movements, vibration and contact stress.

2.15.5 Investigation to Establish Work-Relatedness

I. Obtain a detailed occupational history to establish if there is exposure to the different types of job activities in the workplace for the specific clinical presentation. Correlate the exposure history with the individual symptoms and investigation results.
   - Correlate symptoms with work periods – a WRMSD is likely to worsen during work and improve when away from work. There may be a period of increased workload or a change in work process which triggers the onset of symptoms.
   - The clinician should always be on the alert for clustering of cases with similar occupational groups or in specific workplaces. Ask if there are other workers in the workplace doing the same task who have similar complaints.

II. Exclude non-occupational causes, pre-existing and predisposing factors such as domestic-related exposures and hobbies (as listed above).

III. Ask company for details of work carried out to determine if the work exposures are sufficient to cause the WRMSD. Work involving prolonged static postures, heavy lifting, forceful or repetitive movements of a specific joint provides additional support for the diagnosis of WRMSD.

IV. Assess the risk level at work. The risk level should be sufficient to cause the specific disorder. Older, untrained or new workers on the job may be more prone to developing these disorders from work. Where applicable, objective assessments of the risk factors at work using appropriate ergonomic assessment* tools should be utilised, e.g. NIOSH Lifting Equation, Rapid Upper Limb Assessment, and the Strain Index.

Ergonomic assessment which involves assessing the level or intensity of force, repetitiveness, task duration, posture, rate of movement, vibration, psychosocial and organisational factors may be important in some cases. This may require the expertise of a professional trained in ergonomics.

2.15.6 Examples of Work-Related Musculoskeletal Disorders

Table 16: **Clinical presentation of MSDs and associated job activities**

MSD	Subjective Symptoms	Objective Signs	Typical Job Activities
Carpal tunnel syndrome (CTS)	- Pain, paraesthesia, numbness, tingling, reduced touch sensitivity in median nerve distribution of hand (first three fingers and base of thumb) - Weakness and clumsiness of hand - Aggravated by prolonged full active flexion of wrist - Worse in early hours of morning, waking from sleep and causing handshaking	- Positive* Phalen's test (complete flexion of wrist for one minute) - Positive* Tinel's sign (percuss at the CT for half a minute) - Wasting of thenar eminence - Nerve conduction tests (but can be normal in early CTS) **Reproduce symptoms in median nerve distribution*	- Buffing, grinding, polishing, sanding, assembly work, typing, keying, cashiering, hammering - Risk factors: sustained forceful wrist movements, frequent flexion and extension of the wrist, hand-arm vibration

MSD	Subjective Symptoms	Objective Signs	Typical Job Activities
Ulnar Nerve neuropathy at the elbow (Cubital tunnel syndrome)	- Numbness, tingling in the little and ring fingers - Weakness of hand grasp and thumb pinch - Symptoms may worsen with prolonged elbow flexion - Pain, if present, may be referred to the elbow	- Paraesthesia in the distribution of ulnar verve - Wasting of intrinsic muscles and weakness in pinch and grasp - Wasting of hypothenar muscles and clawing of ring and little fingers - Tinel's sign positive over ulnar groove - Positive Wartenberg's sign – weakness in adduction of the little finger	- Assembly work, manual work - Risk factors: resting forearm near elbow on a hard surface, excessive flexion/ extension of elbow
Tennis elbow (Lateral epicondylitis)	- Pain in lateral epicondyle during rest/active motion of wrist and fingers - Pain and/or weakness in gripping (e.g. shaking hands or holding heavy objects) - Pain may radiate to dorsum of wrist	- Tenderness over lateral epicondyle - Pain on lateral aspect of elbow on resisted extension of wrist and fingers with elbow fully extended - Pain/weakness on gripping	- Turning screws, small parts assembly, hammering, meat cutting - Risk factors: physical exertion with elbow movement, twisting of wrist and elbow

MSD	Subjective Symptoms	Objective Signs	Typical Job Activities
Rotator cuff tendinitis	- Pain in the shoulder exacerbated by abduction or elevation of the arm - Pain at night and at rest if more severe	- Pain on active or resisted abduction or internal/external rotation - Local tenderness on the supraspinatus tendon or rotator cuff - Possible limited abduction - Impingement test	- Overhead assembly, overhead welding, overhead auto repair, reaching, lifting, carrying load on shoulder - Risk factors: physical exertion and abduction of arms above shoulder level
De Quervain's tenosynovitis	- Pain on dorsal radial aspect of wrist which may radiate down thumb or forearm - Pain on thumb movement - Weak grip/pinch	- Finkelstein's test[6] (passive ulnar deviation of wrist with thumb held adducted in the palm). A positive test: aggravation of pain at the tip of the radial styloid process - Pain on resisted extension and abduction of thumb - Local tenderness on palpation at radial styloid	- Buffing, grinding, polishing, sanding, pushing, pressing, sawing, cutting, butchering, use of pliers, inserting screws in holes, forceful hand wringing - Risk factors: prolonged period of highly repetitive hand movements, bending/twisting of wrist in extreme postures

[6] StatPearls (Internet), StatPearls Publishing LLC. Finkelstein Sign

MSD	Subjective Symptoms	Objective Signs	Typical Job Activities
Trigger finger/ stenosing tenosynovitis	- Stiffness/ triggering/ clicking/catching of finger on extension - Finger locked in flexion or extension	- Palpable nodule at base of digit just proximal to the metacarpo-phalangeal joint	- Trigger finger: using and tools with sharp edges pressing into tissue or with handles too far apart for the user's hands - Tenosynovitis: buffing, grinding, polishing, sanding, pushing, pressing, sawing, cutting, butchering, use of pliers, inserting screws in holes, forceful hand wringing
Tension neck syndrome	- Tightness and pain in the neck, shoulders and interscapular region. - Numbness radiating to the arms and fingers	- Stiffness on movement - Localised tenderness	- Administrative work, assembly work - Risk factors: prolonged flexion, extension, twisting of the neck

MSD	Subjective Symptoms	Objective Signs	Typical Job Activities
- Low back disorders (e.g. prolapsed intervertebral disc, muscle sprain)	- Onset of symptoms back pain may be sudden with the symptoms related to a specific incident (e.g. lifting, excessive loads). Symptom onset may also be gradual with the most common complaint being low back pain. - Leg pain may occur with radiculopathy - Sciatica can occur with prolapsed intervertebral disc. Patients with severe disc problems may avoid sitting and prefer standing.	- Pain associated with motion and back movements can be restricted. Each level of the spine and sacroiliac joints may be palpated for localised tenderness - Straight leg raising test can be limited.	- Risk factors*: Heavy lifting, carrying or pushing/pulling of loads, whole body vibration. Sudden overload and/or repetitive loading are additional factors to consider.
Bursitis of the knee	Pain over kneecap	Tenderness and swelling at the kneecap	- Carpet and floor laying activities - Risk factors: prolonged kneeling or external pressure on the knee

The list of MSDs in the table above is not exhaustive. Assessment of the work-relatedness of other MSDs (e.g. osteoarthritis, meniscus disease of the knee) may be given consideration if the risk factors at work is deemed sufficient to have caused the specific MSD.

* A general consensus has been found on the association of low back disorders and heavy manual work. There is strong evidence for the association with work-related lifting and forceful movements. Bending and /or twisting (awkward postures) of the spine increases the risk of back disorders.

2.15.7 Reporting of Work-Related Musculoskeletal Disorders (WRMSD)

Cases of confirmed work-related musculoskeletal disorders due to ergonomic risk factors are deemed as Occupational Diseases and notification is required. Should the treating doctor be unsure whether the condition is work-related, a referral can be made to an Occupational Medicine Specialist or relevant clinical specialist for assistance in determining the diagnosis and confirmation of the work-relatedness of the condition. Confirmed cases of WRMSD must be notified by both the doctor and the employer. As such, doctors should inform the employer of the diagnosis so that the employer can file the notification and comply with their statutory duties. For musculoskeletal disorders which are due to work-related trauma e.g., hit by an object, slips, trips and falls, such cases should be notified as accidents and reporting is only required to be made by the employer. All notifications should be made electronically at *www.mom.gov.sg/ireport*.

2.15.8 Management

The objective of management is relieving pain and inflammation, restoring the patient's range of movement, and modifying the work environment and/or work task to prevent a recurrence or worsening of the condition. It is important to identify the factors leading to the MSD so that the appropriate advice may be given.

The worker may benefit from referral to physiotherapists or occupational therapists for guided strength and mobility exercises to enhance recovery. Additionally, assess the need for referral to an orthopaedic surgeon or a rehabilitation physician in cases of persistent pain or restriction of movement unresponsive to conservative management. Referral to an Occupational Medicine Specialist clinic may also be indicated to evaluate how workplace activities or conditions could be modified to prevent further injury and support a safe return to work.

I. Advice to workers

- Use manual material handling aids to handle loads where possible.
- Consider using wearable assistive devices to reduce strain during heavy lifting and improve posture.
- Adopt proper postures when handling loads, e.g. for lifting, keep the trunk straight and knees bent, carry loads close to the body and avoid twisting and jerking movements.
- Ask for assistance if heavy loads need to be carried.
- Take the required rest breaks.
- Incorporate regular strengthening exercise to improve core stability, which can help protect against strain during repetitive or heavy lifting tasks.
- Engage in flexibility and conditioning programmes, to reduce the risk of musculoskeletal injuries.

II. Advice to companies

- Re-organise the workplace and process to decrease weight of loads, reduce frequency of lifting, keeping heavy objects at knuckle height.
- Eliminate need for manual material handling, if possible, by providing mechanical lifting aids (e.g. lift tables, hoists, cranes).
- Provision of wearable devices such as exoskeletons, posture monitors, or smart lifting belts to support workers during heavy lifting tasks.
- Automating some of the repetitive tasks.
- Rescheduling of work processes to incorporate rest periods.
- Job rotation to different tasks so that the muscle groups can recover.
- Transfer of worker to area where there is less or no exposure to risk factor.
- Training of workers in manual handling techniques and ensuring that workers adopt safe techniques in manual handling.

2.15.8 Further Reading

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5. David Rodrick, Waldemar Karwowski and William S Marras: Work-related Upper Extremity Musculoskeletal Disorders in Handbook of Human Factors and Ergonomics, 4th ed 2012, ed by Gavriel Salvendy.
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3. Appendices

Appendix A – Common Work Processes

Confined Space Work

Entry into confined space

Work in confined space

Storage tank

Manhole work

Tunnelling

Confined space work includes work in underground vaults, tanks, storage bins, manholes, pits, silos, process vessels, pipelines and tunnels where there is a potential to contain or trap hazardous atmospheres.

Possible health hazards in confined spaces include:

- Toxic gases and vapours e.g. hydrogen sulphide, methane, carbon monoxide, carbon dioxide, solvents
- Low oxygen environment (O2 deficiency)
- Constrained or awkward postures

Workers may succumb to asphyxiation, poisoning by chemicals and develop musculoskeletal disorders.

Construction

Cutting of tiles

Pneumatic drilling

Plastering

Work in excavation site

Demolition

Manual handling of loads

Construction activities may expose the worker to:

- Asbestos fibres from removal of asbestos ceiling boards and roofsheets
- Chemicals such as cement, plaster and paints
- Compressed air environment in tunnelling works
- Ergonomic risks from manual handling
- Excessive noise and vibration from piling, cutting, hacking and drilling works
- Crystalline silica dust from hacking or cutting concrete
- Wet work

Workers may develop malignant mesothelioma from inhaling asbestos fibres, hearing loss from exposure to excessive noise, hand-arm vibration syndrome from exposure to vibration, compressed air-related illness such as decompression illness and barotrauma, musculoskeletal disorders from manual handling activities, contact dermatitis from wet work or contact with cement, and silicosis from inhaling crystalline silica dust.

Laundry and Dry-cleaning

Steam pressing

Hot pressing

Manual ironing

Loading of laundry

Workers in the laundry and dry-cleaning industry can be exposed to:

- Ergonomic hazards from handling of heavy laundry loads
- Excessive heat from dryer machines and dried clothing
- Solvents such as dry-cleaning chemicals and stain removers (e.g. hydrocarbons, perchloroethylene, isopropyl alcohol)

Workers may develop musculoskeletal disorders from manual handling tasks, heat rash from exposure to excessive heat and high humidity. Excessive absorption or poisoning due to solvents occurs when workers remove the clothes from the laundry machines.

Horticulture and Landscaping

Preparation of pesticides

Application of pesticides

Horticulture work – transplanting

Horticultural work – weeding

Tree pruning

Grass cutting

Workers in the horticulture and landscaping industry may be exposed to:

- Biological agents from contact with wild animals, insects and soil borne organisms
- Ergonomic risks from manual handling of plants, tools and equipment
- Excessive heat from working outdoors
- Pesticides (e.g. carbamates and organophosphates) from the preparation and application of pesticides
- Wet work from watering of plants and working in pond/waterways

Workers may succumb to poisoning from pesticides, infections such as melioidosis, musculoskeletal disorders and dermatitis from wet work.

Shipbuilding and Repair

Welding

Grit blasting

Non-destructive testing using radioactive isotopes

Solvent degreasing by dipping parts into basin containing solvent

Confined space work

Lagging/delagging

Workers in this industry share similar hazards to those found in confined space work and the metal working industry.

Workers in the ship building and ship repair industry may be exposed to hazards such as:

- Asbestos fibres (from lagging and delagging of asbestos containing insulated pipes)
- Ergonomic risk from awkward postures and manual handling
- Excessive noise from cutting and grinding works
- Heat and ultraviolet (UV) radiation from cutting and welding
- Ionising radiation from non-destructive testing
- Solvent exposure from degreasing, painting works and chemical cleaning

Malignant mesothelioma can develop in workers with past exposure to asbestos during lagging and delagging activities of insulated pipes. Workers may develop musculoskeletal disorders from awkward postures and manual handling, cataract and arc eye from exposure to heat and UV radiation, leukaemia from exposure to ionising radiation, noise-induced hearing loss from exposure to excessive noise and poisoning from solvent use.

Metalworking

Hammering

Grinding

Vapour degreasing

Solvent degreasing

Workers in the metal working industry may be exposed to hazards such as:

- Excessive noise from machining work, usage of hand tools such as air guns
- Foreign objects in eye (e.g. from flying particles), resulting in corneal ulcers, eye irritation
- Heat and UV radiation from cutting and welding
- Solvent exposure from degreasing, handling parts with coolants and spray painting

Workers may develop cataract and arc eye from exposure to heat and UV radiation, noise-induced hearing loss from exposure to excessive noise and poisoning from solvent use.

Woodworking

Sawing using machine saw

Sanding

Lamination with acrylate glue

Varnishing parquet flooring

Workers in the wood working industry may be exposed to hazards such as:

- Chemicals such as solvents, paints, epoxy glue, formaldehyde, isocyanates
- Excessive noise and vibration from sawing and sanding
- Ergonomic risks from manual handling, forceful exertions, repetitive motion
- Wood dust

Workers may develop noise-induced hearing loss from exposure to excessive noise, hand-arm vibration syndrome from the use of tools and musculoskeletal disorders from repetitive flexion or extension of the upper limbs. They may also be sensitised to the wood dust, epoxy glue formaldehyde and isocyanates.

Hospitality and Entertainment

Housekeeping – making of beds

Housekeeping – vacuuming

Cleaning

Cutting

Handling allergens such as seafood

Hospitality and Entertainment

Manual lifting

Carrying goods

Chef exposed to hot environment

Carrying chairs

Carrying food trays

Workers in the hospitality and entertainment industry may be exposed to:

- Contact with food proteins which may predispose to allergies
- Wet work during area cleaning, washing of dishes
- Ergonomic risks from forceful exertions, poor postures and repetitive flexion or extension of wrist and elbows)
- Hot work and hot environment

Workers may develop contact dermatitis from wet work and contact urticaria from sensitisation to raw food such as seafood. Adoption of poor postures and repetitive forceful work in manual handling can lead to musculoskeletal disorders.

Electronics

Work in cold room

Silkscreen printing

Inspection of printed circuit board assembly

Inspection of printed circuit board assembly

Wave soldering system

Spot soldering

Workers in the electronics may be exposed to:

- Ergonomic risks from manual material handling and prolonged awkward postures
- Respiratory allergens and irritants from soldering fluxes
- Lead from soldering and silkcreen prinitng
- Eye strain from intense near/microscpic work

Workers may develop musculoskeletal disorders from prolonged awkward postures, occupational asthma from sensitisation to soldering fluxes and poisoning from lead used in silkscreen printing. Workers can also be exposed to the hazards during maintenance of these machines.

Healthcare

Handling patients

Patient transfer

Handling sharps

Disposal of used linen

Dental work

Workers in the healthcare sector may be exposed to:

- Biological hazards from infected body fluids and close contact with infected patients
- Ergonomic risks from lifting, carrying and other manual handling task
- Excessive noise from usage of mechanical tools in surgery and dentistry work
- Respiratory allergens, irritants and sensitisers from sterilising and disinfecting chemicals and natural rubber latex gloves use

Workers may develop infections from contact with infected body fluids, musculoskeletal disorders from manual handling tasks, noise-induced hearing loss from noisy processes, occupational asthma from sensitisation to latex gloves and glutaraldehyde and other cold disinfectants.

Logistics

Lifting loads

Reaching for objects on high shelves

Bending back when handling loads

Overstretching or twisting back when retrieving items from shelves

Workers in the logistics sector may be exposed to ergonomic risks from awkward postures, lifting, carrying and handling of loads, and may develop musculoskeletal disorders from such work.

For more information on industry processes and hazards, please refer to Workplace Safety and Health Council's website at https://www.tal.sg/wshc/resources.

Appendix B – Personal Protective Equipment

Hearing Protectors

Hearing protectors serve to protect users from exposure in the interim period before the noise is successfully reduced through engineering control measures, or when engineering or administrative measures are not feasible.

Proper selection of suitable hearing protectors and consistent usage when working in high noise work environment are critical for protection of employees' hearing.

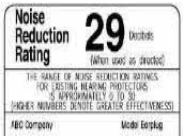
Selection of Suitable Hearing Protectors

Select hearing protectors that are:

- Certified with a noise rating, e.g. noise reduction rating (NRR) or single number rating (SNR). Attenuation derating[7] should be applied to account for real-world performance.

Example to determine if hearing protection is sufficient

A worker carrying out grinding was monitored to have a noise exposure of 88 dB(A) over an 8-hour work shift. He was given foam earplugs with NRR 29. Is the hearing protector sufficient to protect the worker's hearing?



Calculation:

Derating for foam earplugs = Subtract 50% of NRR

Adjusted NRR = [NRR] – 50% [NRR]

= [29] – 0.5 [29] = 14.5dB

Adjusted NRR in dB(A) = 14.5dB – 7 = 7.5 dB(A) (Rule-of-thumb conversion dB to dB(A))

The worker's noise exposure level with earplugs

= Noise exposure level – adjusted NRR in dB(A)

= 88dB – 7.5dB = 80.5 dB (sufficient protection since < 85 dB)

- Comfortable to wear and are compatible with other personal protective equipment used by worker, e.g. wearing earmuffs with safety spectacles which may affect the fit of the hearing protector.

[7] Refer to Section 5.2.2 of the Workplace Safety and Health Guidelines for Hearing Conservation Programme for guidance on derating of NRR.

It is important to avoid overprotection to environmental noise below 75 dBA as this may lead to lower situational awareness and increased safety risks. Refer to the Singapore Standard SS 549:2009 Code of practice and the WSH Council's WSH Guidelines for Hearing Conservation Programme for more information on selection, use, care and maintenance of hearing protectors.

Noise exposure levels	Type of hearing protector to select
85dBA to 100dBA	Ear plugs or ear muffs
More than 100 dBA or presence of impulse noise	Double protection of both earplugs with ear muffs

Type of hearing protectors	Application
Disposable foam earplugs (corded or uncorded) 	Usually made from compressible materials that the wearer rolls down into a tube shape before inserting them into the ear canals. These earplugs will expand to form a seal within the ear canals. Good hand hygiene should be practiced before rolling down. Discard earplugs after use.
Pre-moulded earplugs (corded or uncorded) 	Pre-moulded earplugs are usually made of soft forms of silicone, rubber or plastics, and are readily inserted into the ear canal without need for prior shaping. They are available in different sizes. Reusable but should be discarded when they become damaged, hardened or deformed.
Banded earplugs 	Easy to wear or remove. The flexible band can be used to loop around the neck when not in use. They are most commonly used for intermittent noise exposures. *Note: some noise can be transmitted from the band itself.*

Noise exposure levels	Type of hearing protector to select
Over-the-head earmuff 	Earmuffs are usually used where ear plugs cannot be fitted properly or when dual protection is required when the noise levels exceed 100 dBA. They are easily worn or taken off and generally give better acoustic seal than ear plugs thus providing better hearing protection. Available in different sizes. If the cushions have become hardened or worn out, they should be replaced before use.
Helmet-mounted earmuff 	Some may have built-in microphones and speakers that make it easier to talk to other workers in a noisy environment.

Reusable hearing protectors should be properly maintained and cleaned. Workers who are required to use hearing protectors should receive adequate training on its proper use, care and maintenance.

Tips on Proper Donning of Hearing Protectors

Proper donning of earplugs
Pull ear lobes backwards and upwards with the opposite hand to ensure proper fit and to allow the inserting hand for a better line of sight.

Use the right hand to insert the right earplug, and the left hand to insert the left earplug.

For foam ear plugs, place the thumb over the ear canal for about 10 seconds after inserting the ear plugs to ensure the foam slowly expands within ear canal.

Proper donning of earmuffs
Hair should be brushed away and earmuff should cover the entire ear with the cushion making a proper seal. Adjust any respirator straps and handles of the eyewear where appropriate.

Poor and improper donning

Proper donning

Check the fit after donning
Visual check: at least 75% of the plug should be inserted into the ear canal when using pre-moulded or disposable earplugs to be effective.

Seal check: when in a noisy environment, cup your hands over your ears. If the seal is good, covering your ears shouldn't significantly change the noise level.

Skin and Body Protection

Personal protective equipment (PPE) for skin and body protection such as gloves, sleeve coverings, aprons and coveralls should be used as the last line of defence where there is a possibility of exposure to chemical (such as acid or alkali solutions and organic solvents) and physical hazards (such as heat and vibration). Proper selection of suitable skin and body PPE is critical to protect employees against exposure to chemical and physical hazards.

Selection of PPE for skin and body protection

Select the PPE for skin or body protection that are:

- Able to provide adequate protection against the specific hazard such as chemical resistance, heat resistance, attenuation of vibration etc. For protection against chemical exposure, refer to the Safety Data Sheet on exposure controls/personal protection to determine the suitable PPE to be used.
- Physically suitable to withstand the wear and tear expected from the work activities and duration of usage.

Types of PPE for skin and body protection

Protection for arms	
Glove properties	**Application**
Length of gloves	Gloves are available in a range of materials and lengths, from wrist-length to shoulder-length, offering varying degrees of protection against different chemical and physical hazards. Selecting an optimum length of gloves suitable for the tasks is crucial to maximise protection for workers. PPE can increase the risk of skin exposure when used incorrectly. For example, when poor-fitting gloves are used, contaminant can be trapped within and result in direct exposure to the contaminants.

For chemical protection

POLYVINYL CHLORIDE (Vinyl)

SUPPORTED POLYVINYL ALCOHOL

PVA™

VITON/BUTYL UNSUPPORTED

Gloves used for chemical protection are often made from various material(s) to provide protection against specific chemical(s) which the workers will be exposed to.

Latex gloves are usually used as protection against wet work, plastering, contact with mild chemicals, such as, detergents and cement.

Nitrile and neoprene gloves are used as protection against chemical hazards such as acids, alkalis, solvents, fats, oils and organic acids.

Refer to ISO 374-1: *Protective gloves against dangerous chemicals and micro-organisms* for more information on chemical glove selection.

Other common gloves materials and their general characteristics:

Polyvinyl Chloride (PVC): good for Acids, bases, oils, fats. Not recommended for Ketones, aldehydes, and many organic solvents

Polyvinyl Alcohol (PVA): good for protection against aromatic and chlorinated solvents, ketones, esters. Not recommended for water-based solvent.

Butyl: good for aldehydes, ketones, esters, glycol ethers, strong acids and bases. Not recommended for aliphatic and aromatic hydrocarbons, halogenated solvents.

Fluoroelastomer: good for aromatic and chlorinated solvents, aliphatic hydrocarbons, oils. Not recommended for ketones, esters, amines.

For physical protection

- **Cotton gloves**

Cotton gloves are breathable, typically used for light duty work, general assembly and as liners for coated gloves.

- **Leather gloves**

Leather gloves may be used as protection against heat, cold and mechanical hazards such as abrasions, cuts and punctures.

- **Thermal resistant gloves**

Thermal resistant gloves (e.g. heat or cryogenic resistant) are used in work involving high temperature or very low temperatures such as hot mechanical work, forging, vulcanising applications, plastic extruding, handling hot castings, smelting works or handling cryogenic substances such as dry ice.

- **Anti-vibration gloves**

Anti-vibration gloves are used to reduce vibration transmitted to the hands and arms when operating machines or equipment.

Sleeve Covers	Application
	Chemical resistant sleeve covers provide protection to part of or the entire arm that come into contact with hazardous chemical(s) or physical hazard such as heat. Sleeve covers are typically made of the same materials as the gloves selected to be used for protection against the physical or chemical hazard exposed by the worker. Refer to ISO 16602: *Protective clothing for protection against chemicals — Classification, labelling and performance requirements* for details on the minimum performance classification and labelling requirements for protective clothing designed to provide protection against chemicals.

Protection for body

Aprons	Application
	Aprons prevent workers' body from coming into contact with hazardous chemical(s) or physical hazard such as heat. Aprons are typically made of the same materials as the gloves and sleeve covers selected for protection against the physical or chemical hazard exposed by the worker. For details on the minimum performance classification and labelling requirements for protective clothing designed to provide protection against chemicals, refer to ISO 16602: *Protective clothing for protection against chemicals — Classification, labelling and performance requirements*.

Coveralls	Application
• **Reusable Coveralls** 	Coveralls (reusable or disposable) provide the entire body, including arms, legs, and torso against physical or chemical hazards by acting as a physical barrier above the skin to prevent direct contact. Construction method and material of the coveralls can alter its protection against physical or chemical hazards.
• **Disposable coveralls** Type 5 disposable coveralls  Type 6 disposable coveralls 	Disposable coveralls are used to prevent contamination from exposure to hazardous chemical(s). Type 5 (BS EN ISO 13982-121) disposable coveralls are designed to protect against airborne solid particulates and are commonly used in work involving exposure to asbestos fibres, fibreglass, powder and in some pharmaceutical manufacturing. Type 6 (BS EN 13034:2005+A1:2009) disposable coveralls are designed to protect against light liquid splashes or sprays and are commonly used in work such as light-duty cleaning, general industrial work and paint spraying. For details on the minimum performance classification and labelling requirements for protective clothing designed to provide protection against chemicals, refer to ISO 16602: *Protective clothing for protection against chemicals — Classification, labelling and performance requirements.*

Respiratory Protective Devices

Respirators are used to protect the user from inhalation of harmful dusts, fumes, vapours, gases and against oxygen-deficient atmospheres. They can be broadly classified into air-purifying (non-powered and powered) and supplied-air respirators (airline and self-contained breathing apparatus).

Selection of respiratory protective devices

To determine the suitable respiratory protection for workers exposed to hazardous chemical(s), companies should refer to:

- Section 8 of the SDS on exposure controls/personal protection.
- SS 548:2022 Code of practice for the selection, use and maintenance of respiratory protective devices.

Respirators should be selected based on the type of hazards in the workplace and be properly maintained and cleaned. Fit-testing must be conducted to ensure adequate protection when in use.

Types of respirators	Application
Non-powered Air-purifying respirators • **Disposable respirator**  	Air-purifying respirators may be powered and non-powered. They may be disposable or equipped with a filter or cartridge and are designed for specific contaminants. They are not suitable for use in oxygen-deficient environments. A common disposable respirator is the N95 respirator (designed with at least 95% filter efficiency) used to protect against respirable particulates such as mists, welding fumes and some biological agents. Presence of contaminant such as oil aerosol in the work environment may reduce or degrade the particulate filter efficiency.

- **Respirator with filter or cartridge**

Respirators fitted with the appropriate filters or cartridges can be used to remove mist, particulates, and certain gases and vapours. Exposure to such hazard or combination of hazards can be found in some common applications such as welding, degreasing or spray painting works.

- **Full face-piece respirator**

A full face-piece respirator gives a higher assigned protection factor than a half face-piece. A full face-piece also provides eye and face protection.

- **Half face-piece respirator**
  Powered air-purifying respirators

For powered air-purifying respirators, the contaminated air is drawn through a filter or cartridge by a fan and delivered to user.

Supplied-air respirators

- **Airline Respirators**

Supplied-air respirators, also known as breathing apparatus, are devices where breathing air is provided from a source independent of the working environment. They are used in situations with unknown air contaminants, high contamination levels and in oxygen-deficient environment, such as in confined spaces.

For airline respirators, breathing air is supplied through a hose from a compressor drawing air from an uncontaminated source or compressed air cylinder. User movement may be restricted by the length of the hose.

- **Self-Contained Breathing Apparatus**

For self-contained breathing apparatus, a full face-piece is commonly used, and the air source is carried by the user. This allows the user to move around without restriction.

Appendix C – List of Reportable Occupational Diseases in Singapore under the Workplace Safety and Health Act

1. Barotrauma
2. Cataract due to infra-red, ultraviolet or ionising radiation
3. Compressed air illness or its sequelae, including dysbaric osteonecrosis
4. Diseases caused by excessive heat
5. Diseases caused by ionising radiation
6. Noise-induced hearing loss
7. Poisoning by aniline
8. Poisoning by arsenic
9. Poisoning by benzene or a homologue of benzene
10. Poisoning by beryllium
11. Poisoning by cadmium
12. Poisoning by carbamates
13. Poisoning by carbon disulphide
14. Poisoning by carbon monoxide gas
15. Poisoning by cyanide
16. Poisoning by halogen derivatives of hydrocarbon compounds
17. Poisoning by hydrogen sulphide
18. Poisoning by lead
19. Poisoning by manganese
20. Poisoning by mercury
21. Poisoning by organophosphates
22. Poisoning by oxides of nitrogen
23. Poisoning by phosphorus
24. Toxic anaemia
25. Toxic hepatitis
26. Anthrax
27. Glanders
28. Leptospirosis or its sequelae
29. Occupational infectious disease
30. Work-related musculoskeletal disorder
31. Asbestosis
32. Byssinosis
33. Occupational asthma
34. Silicosis
35. Occupational skin disease
36. Malignant mesothelioma
37. Occupational liver angiosarcoma
38. Occupational skin cancer

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