



# **ASEAN Diagnostic Criteria for Occupational Diseases**

**June 2017 edition**

**ASEAN Diagnostic Criteria for Occupational Diseases Committee**

**Secretary office at Nopparat Rajathanee Hospital**

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# Preface

Delegates from ten ASEAN countries (Brunei, Cambodia, Indonesia, Laos, Malaysia, Myanmar, Philippines, Singapore, Thailand and Vietnam) were participated in the first meeting in Bangkok on 8 July 2014 as a committee to set up the 'ASEAN Diagnostic Criteria on Occupational Diseases (ADCOD)'. In the meeting all delegates committed to formulate the ADCOD. The participants recognized the importance on ASEAN policy to unit the ten countries into one community by 31 December 2015 and agree to have ADCOD as a standard criteria for occupational diseases in ASEAN countries. With these criteria the statistics of occupational diseases will be registered with accuracy and referable and the in the first meeting on 8th of July, the representatives of each country agreed to accept the ADCOD principle. The participants at the meeting were occupational health physician and were the top persons in policy deployment on occupational health in each country. The result of the meeting were the format of the diseases criteria, the presentation of the most common occupational diseases occurring in each country to prioritized the criteria

making and the occupational diseases that each country promised to work on and send back to their country for correction and acceptance.

The first five occupational diseases selected were occupational hearing loss, occupational dermatitis, heat stress, asthma and silicosis. In this meeting the International committee for ADCOD was also formed. The meeting was organised by Nopparat Rajathanee Hospital, Department of medical services, in the Ministry of Public Health Thailand and the Association of Occupational and Environmental Disease of Thailand, while delegates from China and Japan joined to observe the meeting.

This Final Draft of ASEAN Diagnostic Criteria was prepared on the second meeting of the committee in Pattaya on 2-4 March 2015.

We look forwards to the 3<sup>rd</sup> meeting on 28-30 January 2016, where the committee which consist of delegates from all countries in ASEAN will make further success of more ADCOD and the new things that will be done together in the future.

Dr. Adul Bandhukul  
ADCOD Secretariat

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## **1. Introduction:**

The Association of South East Asian Nations (ASEAN) was formed in Aug 1967 as a regional grouping to promote economic growth and socio-cultural progress. The founder members were Indonesia, Malaysia, Philippines, Singapore, and Thailand. It expanded to a group of ten Asian countries, with the further inclusion of Brunei, Cambodia, Laos, Myanmar, and Vietnam. By the end of 2015, it was intended that a closer ASEAN community linkage would be fully established through economic integration, cooperation, and harmonization which we can foresee the migration of workforces between ten ASEAN countries.

With the formation of ASEAN community, there was also a recognition that there should be harmonization of occupational health services, especially the standards and procedures including diagnostic criteria for occupational diseases. We also learned that the EU has its diagnostic criteria for occupational diseases since 1994.

ASEAN occupational health clinicians have expressed a concern that existing criteria developed in western countries may not be wholly applicable to ASEAN countries. This is because they may include conditions that are rare or not seen in tropical regions, or they often do not refer to conditions that are encountered in Asia, such as cercarial dermatitis in farmers, rice millers' asthma, etc. There are also conditions with clinical manifestations that differ between workers affected in hot tropical climates compared to cold temperate countries, e.g. hand-arm vibration syndrome (HAVS) where the neurological and musculoskeletal features are more common than the vascular effects.

As the result of this, Thailand was the host to establish a committee in 2014 to develop ASEAN diagnostic criteria for occupational diseases. Where appropriate, existing criteria developed by other countries would be discussed and accepted in toto, or modified as necessary to take into account exposures and effects as encountered in ASEAN workplaces

## 2. The name list of ADCOD committee (order by alphabet)

### Chairman

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### Committee

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	3. Dr. Khamla Phoummany	Technical staff responsible of Occupation Health and Safety, Department of Hygiene and Health Promotion, MOH	
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### **3. Process for developing diagnostic criteria:**

The definition and scope of the key words and activities were considered. The committee agree to follow the WHO definition of “occupational diseases”. An occupational disease is any disease contracted primarily as a result of an exposure to risk factors arising from work activity.

ILO list of occupational diseases were considered as a guide for developing the criteria for occupational diseases for it includes a range of internationally recognized occupational diseases, from illness caused by chemical, physical and biological agents to respiratory and skin diseases, musculoskeletal disorders, mental and behavioral disorder and occupational cancer. (ILO 2010)

Occupational health representatives from the ASEAN countries were asked to provide information on common occupational diseases in their own countries to form a priority list. From this list, five occupational diseases were initially chosen as the first group to standardized the diagnostic criteria.

A search of the existing literature was conducted to determine what is currently available. Separate working groups reviewed each of the diseases to produce a summary based on the following headings. The working groups had the assistance of consultants from outside ASEAN with experience and expertise in occupational medicine and diagnostic criteria.

#### **Scope of each criterias for occupational disease.**

Name of each occupational disease must be focus clearly in the list by the scope as follow.

1. Definition of diseases
2. Definition of occupational hazard/s
3. Main occupational uses and exposure
4. Diagnostic criteria (Symptoms and signs)
5. Diagnostic tests, where available
6. Exposure criteria
  - 6.1 Minimum intensity of exposure
  - 6.2 Minimum duration of exposure
  - 6.3 Maximum latent period
7. Differential diagnosis

Treatment and prevention were outside the limit of the working group.

## **Users**

The format used in the EU diagnostic criteria was modified for compiling an ASEAN guide, which is intended as an aid for clinicians in regards to diagnosis. Compensation entitlement and criteria are outside the scope of this document, as these would depend on the regulations for compensation and benefits existing in each country. The systems for compensation are often depended on factors beyond purely medical considerations.

Where the document refers to documentation of exposure, it is acknowledged that systems for quantitative determination of workplace exposures are neither well developed nor readily available in many ASEAN countries. Industrial hygiene laboratories and trained personnel for occupational hygiene are at an early stage of provision. Occupational exposure standards where available are not consistent between countries. ASEAN countries may therefore need to rely on occupational histories, workplace records of incidents or accidents resulting in over-exposure to workplace agents, or other workplace records. Where reference is made to exposure standards, it is necessary to stress that a single excursion marginally above any current standard may be insufficient to support a diagnosis of an occupational disease. Where exposure standards are substantially exceeded and occurring on several occasions, this suggests that control of exposures at the workplace is inadequate. Under such circumstances this would provide better support for attributing a disease to occupational exposure.

## **Conclusion:**

The diseases included in this guide are the beginning towards a more comprehensive agreed list of ASEAN occupational diseases. The committee intends to add other conditions that are common in ASEAN countries. The ILO (International Labour Organization) list of occupational diseases could form the basis for including further occupational entities.

The purpose of this document is to help clinicians recognize occupational diseases as country statistics show that very few cases of occupational diseases are reported despite regulations requiring identification and notification of such diseases. A prime purpose of recognition is that it should lead to implementation of preventive measures at the workplace, as a single case of occupational disease would be a sentinel event indicating the possibility of other workers being exposed to adverse workplace exposures. Emphasis is placed on the preventive aspects of

management and control of workplace hazards. The clinical management of individuals with occupational disease is outside the scope of this document

**Further reading.**

1. ILO list of occupational diseases (revised 2010) from [http://www.ilo.org/wcmsp5/groups/public/---ed\\_protect/---protrav/---safework/documents/publication/wcms\\_125137.pdf](http://www.ilo.org/wcmsp5/groups/public/---ed_protect/---protrav/---safework/documents/publication/wcms_125137.pdf) accessed 5 June 2015.

2. European Commission (2009) Information notices on occupational diseases: a guide to diagnosis. EC (DG for Employment, Social Affairs and Equal Opportunities. Luxembourg: Office for Official Publications of the European Communities.

## 4. Criteria for diagnostic of occupational diseases

### 4.1 Diseases caused by arsenic or its toxic compounds

#### 1. Definition

Arsenic poisoning is caused by exposure to arsenic and arsenic compounds, and can be caused by exposures during work, as well as through non-work exposures. The diseases characterized by skin lesions (hyperkeratosis) and cancer of the skin, lungs and urinary bladder. In addition, long term effects can manifest as neurotoxicity, and cardiovascular effects.

#### 2. Occupational hazards

Inorganic arsenic compounds are highly toxic while organic arsenic compounds are less harmful to health. Arsenic is a natural component of the earth's crust and is widely distributed throughout the environment in the air, water and land.

#### 3. Main occupational use and exposure

1. Pesticides manufacturing
2. Smelting plant
3. Alloys manufacturing
4. Tanning
5. Wood preservation manufacturing
6. Fabric printing factory
7. Semiconductor manufacturing
8. Color pigment manufacturing
9. Metal plating factory
10. Pottery factory
11. Ore mining and color metallurgy
12. Optical technology
13. Glass processing

#### 4. Diagnostic criteria

##### 4.1 Symptoms and Signs

##### **Acute poisoning**

Irritation of the mouth, throat, larynx and in severe cases lower airway irritation and pneumonitis. There are neurological effects such as headache, dizziness, delirium, seizure, and coma. Nephrotoxic effects include decreased urinary excretion, tubular necrosis and acute cortical necrosis. Digestive effects are severe abdominal pain, nausea, vomiting and diarrhea and in severe cases

hypovolemic shock from loss of blood from upper GI Hemorrhage. Hematologic effects include anemia and disseminated intravascular coagulation in severe cases..

## **Chronic poisoning**

### **Skin**

There will be hyperpigmentation in the skin with a characteristic appearance (like raindrops on a dusty road). In some cases there may be keratotic papules, which are also known as corn-like papules, or punctate keratosis, which can coalesce into verrucous plaques. Skin malignancies can result e.g. Bowen's disease, squamous cell carcinoma, and basal cell carcinoma.

### **Nervous system**

There may be peripheral neuropathy - usually bilateral, and presenting as weakness with pain and edema in both legs.

### **Cancer**

Cancer of skin, lungs and bladder.

### **Other system**

Anemia, rhinitis and perforation of nasal septum. Some affected individuals have portal hypertension without cirrhosis. Mees' line, (transverse white lines on the nails of the digits) may be observed, but this also occurs in other diseases e.g. renal failure and thallium poisoning.

#### **4.2 Diagnostic test**

In acute poisoning the total urine arsenic concentration in the first 2 to 3 days is typically in excess of 1000 µg/L

Analysis of arsenic in urine: Arsenic in an early morning urine sample or in a 24-hr. sample of > 50 µg/gram creatinine indicates excessive exposure to arsenic. In the ACGIH BEIs, arsenic as elemental and soluble inorganic compounds can be measured as 'Inorganic arsenic plus methylated metabolites in urine' collected at the end of the workweek and it is considered abnormal if > 35µg/L.

## **5. Exposure limit**

### **5.1 Minimum exposure of arsenic**

The American Conference of Governmental Industrial Hygienists (ACGIH) has set the threshold limit value (TLV) for inorganic arsenic at 0.01 mg/m<sup>3</sup>

## **6. Differential diagnosis**

- Skin cancer from UV light
- Peripheral neuropathy
- Other skin diseases
- Anemia from other causes

Ca lung and ca bladder from other cause  
Aetiology should be distinguished from occupational arsenic poisoning and that not caused by occupation.

#### 7. Further reading

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and Toxic Exposures, Sullivan, JB, Krieger, GR (Eds), Lippincott Williams  
& Wilkins, Philadelphia 2001. P.858-66.

## 4.2 Asbestosis

### 1. Definition

Asbestosis – refers to the diffuse interstitial pulmonary fibrosis caused by inhalation of asbestos fibers

### 2. Occupational hazards :

Serpentines – chrysotile

Amphiboles – crocidolite, amosite, actinolite, tremolite, and anthophyllite

### 3. Main occupational uses and sources of exposure

3.1 Asbestos has been in use for commercial products, such as insulation and fireproofing materials, automotive brakes and textile products, and cement and wallboard materials.

3.2 The source is primarily occupational in nature and it constitutes hazard to workers engaged in activities involving:

1. Mining and milling of asbestos
2. Production of asbestos-containing materials
3. Construction activities like installation of products; cutting, removal, and demolition of structures and buildings made of asbestos
4. Use and disposal of asbestos-containing products

### 4. Diagnostic criteria

The following criteria, together with a history of asbestos fiber exposure, suggest a diagnosis of asbestosis and provide a basis for assessing its severity:

#### 4.1. Symptoms and signs

1. Dry cough which may distressing
2. Increased breathlessness associated with transient chest pains
3. Signs of respiratory insufficiency such as cyanosis
4. Clubbing of fingers
5. Increase sputum

Physical examination findings:

Basal and inspiratory rhonchi & crepitations on chest auscultation

#### 4.2. Diagnostic tests

1. Chest x-ray – small irregular opacities (usually reticular or reticulonodular) mainly in the lower lung fields, correspond to 1/0 or more on chest x-ray according to ILO radiographs reading for pneumoconiosis.

2. Chest CT ( High resolution CT scan; HRCT) may help in limited cases , e.g., borderline cases or some discrepancy between CXR findings and other conditions

3. Pulmonary function test

1. Spirometry showing restriction

2. Carbon monoxide diffusion showing reduction in gas transfer

4. Lung biopsy – it is not indicated for diagnosis

5. Exposure criteria

5.1. Minimum intensity of exposure – Confirmed occupational exposure assessed by history and study of working conditions providing evidence of prolonged or repeated exposure to asbestos fibers

5.2. Minimum duration of exposure

At least 5 years of exposure at work

5.3. Minimum induction period

10 years

6. Differential diagnoses

6.1. Idiopathic pulmonary fibrosis

6.2. Hypersensitivity pneumonitis

6.3. Other forms of pneumoconiosis

7. Further readings

1. European Commission. 2009. Asbestos (Annex I, No. 301.21), *Information notices on occupational diseases: a guide to diagnosis* (pp 170 - 172). Luxembourg: Office for Official Publications of the European Communities. Retrieved from [ec.europa.eu/social/BlobServlet?docId=3155&langId=en](http://ec.europa.eu/social/BlobServlet?docId=3155&langId=en).

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## 4.3 Disease caused by lead and its toxic compounds

### Inorganic lead poisoning

#### 1. Definition:

A Disease caused by excessive absorption of inorganic lead and its toxic compounds

#### 2. Occupational hazards

Inorganic : Lead oxides, metallic lead, and lead salts

#### 3. Main occupational uses and exposures

3.1 Manufacture and recycling of lead-acid storage battery

3.2 Smelting and refining

3.3 Manufacture and use of lead-based paints (eg. Car painting)

3.4 Manufacture and use of lead-based glazes for ceramics, pottery and glass

3.5 Manufacture and use of ammunition (eg. firing range instructor)

3.6 Manufacture and use of stabilizers (eg. PVC compounding)

3.7 Burning/welding/cutting/ demolition of lead-coated structures in occupational activities such shipbuilding, ship repair, welding, construction, radiation repair etc.

3.8 Lead soldering in semiconductor sector

3.9 Lead mining

3.10 Other industries and occupations that have significant inorganic lead exposure

#### 4. Diagnostic criteria:

##### 4.1 Symptoms and signs

Acute lead poisoning

1. Abdominal pain (colicky)

2. Severe anemia

3. Acute renal failure

4. Neurological (encephalopathy, convulsions, coma, paresthesia, pain and muscle weakness)

Chronic

Fatigue, asthenia

1. Arthralgias and myalgias

2. Anemia

3. Numbness, wrist drop, foot drop (from peripheral neuropathy)

4. Infertility

5. Spontaneous abortions

6. Chronic renal failure

7. Lead Line ( A blue-purplish line on the gums)

5. Exposure criteria:

Male/female non-pregnant: 60 micrograms/dl plus sign and symptoms

6. Differential Diagnosis:

Consideration of other disease entities that would produce Symptoms and signs similar to lead poisoning

7. Further reading :

1. European Commission. 2009. Lead (Annex I, No. 112), Information notices on occupational diseases: a guide to diagnosis (pp 54-57). Luxembourg: Office for Official Publications of the European Communities. Retrieved from [ec.europa.eu/social/BlobServlet?docId=3155&langId=en](http://ec.europa.eu/social/BlobServlet?docId=3155&langId=en).

2. Poisoning & Drug Overdose, 6th edition

3. Current Occupational & Environmental Medicine, 4th edition (Ladou)

## **4.4 Solvent poisoning, including Perchloroethylene (PCE) and Trichloroethylene (TCE)**

### **1. Definition**

The term solvent means "any 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.

Commonly used organic solvents are usually volatile. These include aromatics (such as benzene and toluene) and halogenated hydrocarbons (such as trichloroethylene, perchloroethylene and carbon tetrachloride). Other commonly used solvents are the alcohols, xylene and ether.

Solvents are easily absorbed through the skin and by inhalation 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 hydrocarbon.

Dermal exposures can cause dermatitis. Carcinogenic risk is increased with certain solvents, such as benzene (known to cause leukemia). When a worker is exposed to more than one solvent, their effects on target organs may be additive.

This section will focus on the solvents perchloroethylene (PCE) and trichloroethylene (TCE) as there are common solvents use in the industries ASEAN countries. The usage of solvents generally involves a mix of multiple solvents in a particular workplace. However with PCE and TCE, they are usually used as a single solvent. For e.g. PCE is generally the main solvent for dry cleaning of laundry and TCE for metal degreasing. These specific usage of PCE and TCE makes it easier evaluating exposure to these solvents at the workplaces.

## 2. Main occupational uses and exposures

### **PCE**

- Dry cleaning and textile processing
- Degreasing of metal parts in metal fabricating, 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

### **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,
- Extraction of waxes, fats, resins and oils
- Manufacture and use of printing inks, varnishes, adhesives, paints, lacquers, rug cleaners and disinfectants

## 3. Diagnostic criteria

### 3.1 Symptoms and Signs

#### 3.1.1 PCE

#### **Acute exposures:**

- Mucosa membranes (Irritation to eyes, nose, and respiratory tract)
- Central Nervous System (Massive exposure can cause dizziness, headache, nausea, incoordination, coma and death).

#### **Chronic exposures:**

- Central Nervous System (Chronic Toxic encephalopathy - Non-specific complaints such as headache, dizziness, fatigue and incoordination).
- Skin (irritation and even burns)
- Liver (cirrhosis has been observed in workers exposed to high levels)

### 3.1.2 TCE

#### **Acute exposures:**

- Mucosal 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).
- Respiratory System (Chemical pneumonitis and death from respiratory failure may occur)
- Cardiovascular (high exposure can sensitize the myocardium leading to arrhythmia and cardiac arrest)

#### **Chronic exposures:**

- Central Nervous System (Chronic Toxic encephalopathy - non-specific complaints like headache, irritability, fatigue and insomnia.)
- Skin (irritation and dermatitis)
- Liver (A few cases of hepatitis-like syndromes and steatosis have been reported)
- Renal (proteinuria and raised blood urea)
- 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 with fever, rash and jaundice within 2-3 weeks of exposure).

### 3.2 Diagnostic tests

These tests can be used as indicators of exposure and a diagnosis of poisoning should be based on presence of relevant clinical Symptoms and signs.

Urinary trichloroacetic acid (U-TCA) done mid-week end of shift

Exposure to PCE – BEI for U-TCA = 7 mg/l

Exposure to TCE – BEI for U-TCA = 100 mg/l

(when there is a mixed exposure to PCE and TCE, the BEI for U-TCA of 50 mg/l should be adopted if the air level for PCE is less than half the PEL.

Where the air level for PCE is more than half the PEL, a BEI of 7 mg/l should be adopted.

Other tests: 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. Specialized neuropsychometric tests (e.g. WHO Neurobehavioural core test battery) may be used to evaluate behavioural effects.

#### 4. Differential diagnosis

Consider non-occupational exposures:

- a) Solvent abusers e.g. Glue sniffers
- b) Hobbies using glue

#### 5. References

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## 4.5 Occupational Pesticide poisoning: Organophosphate and Carbamate poisoning

### 1. Definition :

Occupational pesticide poisoning is a poisoning disease caused by exposure to pesticide. This can be acute to chronic and exposures can occur through dermal ,inhalation and ingestion.

### 2. Occupational hazards

#### 2.1 Organophosphate:

- blockage of Acetylcholinesterase (AChE) and Butyrylcholinesterase or pseudocholinesterase(PChE)
- permanent inhibition of AChE (aging)

#### 2.2 Carbamate : reversible blockage of enzyme

- blockage of Acetylcholinesterase (AChE) and Butyrylcholinesterase or pseudocholinesterase(PChE)

### 3. Main occupational organophosphate/carbamate use and exposure

- Pest control
- Farming and gardening used
- Crop management
- Manufacture of pesticide
- Other industries

### 4. Diagnostic criteria

1. History of exposure
2. Physical examination
3. Laboratory diagnosis

#### 4.1 Symptoms and signs:

**Acute presentation:** occurring within 2-3 days

1. Muscarinic manifestation :
  - Bronchospasm

- Bradycardia
  - Vomiting
  - Diarrhea
  - Miosis
  - Excessive sweating
2. Nicotinic manifestation :
    - Skeletal muscle : weakness, tremors and/or fasciculation
  3. Central nervous system :
    - Agitation
    - Seizure
    - Coma

**Intermediate syndrome** :occurring 24-96 hr after exposure and last for 1-2 weeks)

- Bulbar and Proximal muscle weakness : Neck flexion
- Respiratory failure

**Chronic presentation:**(Occurs 1-5 weeks after exposure)

1. Neurological
  - Distal sensory, motor neuropathy (Occurs 1-5 weeks after exposure)
2. Neurobehavioural
  - reduced fine motor co-ordination
  - slowed reaction time
3. Neuropsychiatric
  - apathy
  - depressed memory function
  - irritability

#### 4.2 Diagnostic test :

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.

- Serum, urinary organophosphate/carbamate and metabolites
- RBC acetylcholinesterase and plasma cholinesterase levels  
Plasma cholinesterase is for screening, RBC acetylcholinesterase is for confirm diagnosis
  - Cholinesterase levels should be compared **with baseline levels** (if available) or **with the laboratory lower limit of normal** to determine if any decrease in levels is significant.
  - **If the RBC acetylcholinesterase is <50%** of the baseline, referral to hospital is require if symptomatic. Suspension is also required.

## 5. Exposure Criteria : Biological monitoring

- RBC acetylcholinesterase and plasma cholinesterase levels  
Plasma cholinesterase is for screening, RBC acetylcholinesterase is for confirm diagnosis
  - Cholinesterase levels should be compared **with baseline levels** (if available) or **with the laboratory lower limit of normal** to determine if any decrease in levels is significant.
  - **If the RBC acetylcholinesterase is <50%** of the baseline, referral to hospital is require if symptomatic. Suspension is also required.

## 6. Differential diagnosis

6.1 Other causes of neurological disorders, such as motor neuron disease, impending CVA, diabetic ketoacidosis.

6.2 Gastroenteritis – acute pesticide poisoning may present with diarrhea and vomiting.

6.3 Non occupational exposure to pesticides used in the domestic settings, accidental ingestion of pesticides and exposure to nerve agents used in chemical warfare, especially in a mass casualty situation.

## 7. Further Reading

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2. Poisoning & Drug Overdose, 6th edition

3. Current Occupational & Environmental Medicine, 4th edition (Ladou)

## 4.6 Decompression Illness

### 1. Definition

Decompression illness (synonyms: bends, Caisson disease) is a disorder in which nitrogen dissolved in the blood and tissues by high pressure forms bubbles as pressure decreases, either in the surrounding air (e.g. high altitude) or water (e.g. deep sea diving). Decompression illness encompasses two diseases, decompression illness (DCS) and arterial gas embolism (AGE).

Decompression illness can be categorised into:

Type I Decompression Illness – this tends to be mild and affects primarily the joints, skin, and lymphatic vessels.

Type II Decompression Illness – this may be life-threatening and often affects vital organ systems, including the brain and spinal cord, respiratory system, and circulatory system.

### 2. Causal agent

Air is composed mainly of nitrogen and oxygen. When the surrounding pressure decreases, extra oxygen is inhaled and used continuously by the body. The excess nitrogen molecules start to accumulate in the blood and tissues (intravascular and extravascular) and form bubbles. On rapid decompression, these bubbles may expand and cause tissue injury or block blood vessels in organs.

### 3. Main occupational uses and exposure

- Commercial divers (e.g. salvage workers, underwater logging workers, recreational diving instructors)
- Compressed air workers
- Fishermen divers
- Research divers
- Military divers
- Marine divers
- Underwater photographers/ videographers
- Aviators
- Underwater archeologist
- Tunnel worker

## 4. Diagnostic Criteria

### 4.1 Symptoms and Signs

Symptoms and signs commonly begin gradually and take some time to reach their maximum effect, and in most cases occur after 6 hours of the incident. Initial symptoms and signs commonly experienced may be:

- Fatigue
- Loss of appetite
- Headache
- Vague feeling of illness
- Joint pain (arms, legs, back)
- Pruritus (itching)
- Skin mottling, rash
- Swollen lymph nodes
- Numbness, tingling, limb weakness, inability to urinate or control micturition, partial paralysis
- Double vision, difficulty speaking, confusion
- Vertigo, tinnitus, hearing loss
- Chest pain, difficulty breathing ('the chokes'), cough, haemoptysis
- Pain in abdomen and back
- Dysbaric osteonecrosis or Avascular bone necrosis (particularly in the shoulder and hip)

### 4.2 Diagnostic test

Diagnosis of DCI (decompression illness) is based primarily on clinical examination and presence of classic Symptoms and signs. Urgent treatment and management is needed when DCI is suspected.

## 5 Exposure limit

Minimum intensity and duration of exposure is dependent on the frequency of activity within 24 hours (single vs multiple), and activity profile (depth or altitude reached, rest stops for recompression).

Risk factors which may increase the risk of developing decompression illness are:

### Personal

- Smoking
- Alcohol intake
- Flying after diving
- Fatigue
- Obesity
- Older age
- Heart defects

### Environment

- Rapid ascent
- Increasing pressure
- Increased length of time spent in a pressurised environment

## 6 Differential diagnosis

- Inner ear barotrauma
- Middle ear or maxillary sinus inflammation
- Oxygen toxicity
- Musculoskeletal strains
- Immersion pulmonary oedema
- Neurological disorders (e.g. stroke)
- Thermal stress (due to cold exposure)

## 7 Further reading

- (1) Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. *Lancet* 2011; 377 (976D): 153-164.
- (2) Walrath B, Smith JE, Raghunandan A, Boni B, Latham E. Differential diagnosis considerations of sickness after rapid pressure changes at altitude. *Aviat Space Environ Med* 2013; 84:1–4.
- (3) Lemaitre F, Fahlman A, Gardette B, Kohshi K. Decompression sickness in breath-hold divers: a review. *J Sports Sci* 2009; 27 (14): 1519-1534.

## 4.7 Heat Stress

### 1. Definition

Diseases caused by excessive heat (heat disorders) can be seen as a continuum of illnesses relating to the body's inability to cope with an increase in heat load. This can result in a spectrum of diseases ranging from heat cramps to heat exhaustion and heat stroke.

### 2. Causal agent

Heat is generated by the body and exposure can also be from the external environment. Heat stress occurs when the accumulation of heat in the body exceeds the ability of the body to remove the excess heat.

### 3. Main occupational uses and sources of exposure

These may involve outdoor workers working directly under the hot sun e.g.:

- construction workers;
- shipyard workers;
- landscaping and agricultural workers;
- Military personnel
- Indoor workers can also be at risk of developing heat stress.

e.g.:

- foundry and steel workers;
- oven and furnace operators;

Predisposing factors include:

- lack of acclimatisation\* (e.g. workers coming to ASEAN countries from a colder country would need to get adapted to the hot and humid tropical environment)\*;
- poor hydration (eg workers may not be adequately hydrated because of socio-cultural / religious practices, such as fasting where there is abstinence from consuming food and water during daylight hours);
- illness (e.g., diabetes) or those on medication;
- older workers;
- obesity; and
- alcohol consumption.

\*[Newly assigned workers, especially those who come from a colder climate, must be acclimatised to the hot weather which would allow the body to adapt slowly to the hot environment. They would need at least one to two weeks to adjust to the hot weather conditions and workloads.]

#### 4. Diagnostic Criteria

##### 4.1 Symptoms and signs

###### Heat rash

- Itchy red papules with a prickly sensation to blistering.

###### Heat syncope

- Transient loss of consciousness, preceded by pallor, blurring of vision, dizziness and nausea

###### Heat cramps

- Painful muscle cramps (usually in the legs)
- Weakness, nausea, vomiting
- Heat cramps can follow heavy physical work and is due to fluid and electrolyte loss caused by heavy sweating

###### Heat exhaustion

- May be associated with mild central nervous system or other non-specific symptoms such as profuse sweating, nausea, vomiting, headache, dizziness, light-headedness, intestinal cramps, weakness, hyperventilation and cool and clammy skin
- The core temperature is usually in the range of 37.7°C to 40°C.  
Heat exhaustion, if untreated, has the potential to develop into life-threatening heat stroke.

###### Heat fatigue

- Heat fatigue is a set of behavioral responses to acute or chronic heat exposure include impairment in (a) the performance of skilled sensorimotor tasks (b) cognitive performance, and (c) alertness.

- The behavioral responses to chronic heat exposure include reductions in performance capacity, standards of performance and concentration.

#### Heat stroke

- 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, seizures and even coma
- Cardiovascular deterioration
- Multi-organ failure

As a differential diagnosis, heat stroke must be suspected as one of the causes if a worker collapses in the workplace without sign of external injury. Heat stroke can be fatal if not treated quickly.

#### 4.2 Diagnostic tests

A good occupational history of work in hot environment, especially in the unacclimatised worker. Reports on air temperature, humidity and air movement/ventilation are useful measures of exposure to heat and an indication of the likelihood of heat stress in the environment. There are various composite indicators available for assessing the thermal environment and examples of these would include the the Wet Bulb Globe Temperature (WBGT) and the Heat Stress Index. . Investigations conducted would be related to the management of the worker arising from the heat stress and related disorders.

#### 5. Exposure limit

Heat stress can be assessed by measuring one or more of environmental, work, or worker factors, and then utilising the appropriate environmental heat stress index. This should be used in conjunction with other factors to assess overall risk.

There are various indicators available for assessing thermal environment. The most widely used indicator is the Wet Bulb Globe Temperature (WBGT) which takes into consideration the natural wet bulb temperature, globe temperature and dry bulb temperature.

Other methods of assessing the environmental heat stress include the Heat Stress Index (HSI) which takes into consideration the environmental heat and metabolic heat.

## 6. Differential diagnosis

Exclude non-occupational causes with similar symptoms such as:

- other causes of unconsciousness or syncope such as stroke, hypoglycemia; or
- other causes of increased body temperature such as fever due to infections.

## 7. Further reading

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[http://www.moh.gov.sg/content/dam/moh\\_web/HPP/Doctors/cpg\\_medical/current/2010/cpg\\_Heat%20injuries%20-%20Nov%202010.pdf](http://www.moh.gov.sg/content/dam/moh_web/HPP/Doctors/cpg_medical/current/2010/cpg_Heat%20injuries%20-%20Nov%202010.pdf) (last accessed 18 July 2014)

3. Wagner GR ed. Preventing occupational diseases and injury. Amer Public Health Assn (December 1991).

## 4.8 Occupational Noise-induced Hearing Loss

### 1. Definition

Occupational noise-induced hearing loss (NIHL) is defined as irreversible sensorineural hearing loss or impairment resulting from exposure to excessive noise at the workplace.

It can result from an acute exposure (e.g. acoustic trauma from a blast injury) or chronic exposure.

### 2. Occupational Hazards

Noise or unwanted sound with intensity that exceeds the threshold limit value of 85 dBA.

### 3. Main occupational uses and exposure

#### 3.1 Main occupational uses

- aircraft workers
- construction workers
- agriculture workers
- steel workers
- metal cutters
- miners
- shooting range instructors/ trainers
- military personnel

#### 3.2 Sources of exposure

- machinery (power generator, turbine, compressor, print machine etc)
- pneumatic drills
- grinding
- sandblasting
- metal manufacturing
- saw mills
- textile mills

### 4. Diagnostic Criteria

#### 4.1 Symptoms and signs

Acute NIHL commonly presents as ear pain, tinnitus, bleeding or giddiness. The hearing loss may be unilateral or bilateral. It may be conductive, affecting all frequencies, or sensorineural.

Chronic NIHL develops insidiously over a long period of time, and can present as intermittent or continuous tinnitus in both ears. There may also be a gradual loss of clarity in perceived speech resulting in difficulty to understand and/or hold a conversation. In the later stage, the hearing loss affects all frequencies. In the early stage, the average hearing thresholds at the lower frequencies of 500, 1000 and 2000 Hertz are better than the average thresholds at 3000, 4000 and 6000 Hertz.

#### 4.2 Diagnostic tests

- positive Rinne test, or Weber test (lateralised in the better ear), or Schwabach test
- pure tone audiometry (sensorineural hearing loss typically affecting frequencies at 3000-6000 Hertz with a dip at 4000 Hertz)

### 5. Exposure limit

#### 5.1 Minimum intensity of exposure and minimum duration of exposure

The risk of occupational NIHL is considered to be low at exposures below 85 dBA (over an 8-hour time-weighted average), or adjusted values depending on duration of noise exposure in the work environment, but increases significantly as exposures rise above this level.

<b>Duration of noise exposure (hours)</b>	<b>Acceptable intensity of noise level (dBA)</b>
24	80
16	82
8	85
4	88
2	91
1	94
0.5	97
0.25	100

## 6. Differential Diagnosis

Hearing loss may also arise from non-occupational causes and therefore it is important that these are also considered when a diagnosis of occupational NIHL is made. Common non-occupational causes include:

- congenital hearing loss (may be associated with maternal rubella, flu, prenatal medication, or birth trauma)
- familial hearing loss
- childhood illnesses (e.g. measles which may result in bilateral hearing loss, mumps which may result in unilateral hearing loss, encephalitis, meningitis, cerebral abscesses etc)
- use of ototoxic drugs (e.g. streptomycin, gentamycin, neomycin etc)\*
- history of head injury that may result in sudden hearing loss
- history of deep x-ray therapy (DXT) to the head and/or neck region
- presbycusis (for those above 50 years old)
- conductive hearing loss (e.g. from otitis media)
- socio acusis
- non-occupational loud music activities (e.g. karaoke, night clubbing etc)

\*exposure to ototoxic chemicals (e.g. carbon monoxide and solvents such as carbon disulphide, xylene and toluene) at the workplace may also result in hearing loss.

## 7. Further reading

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2. Health and Safety Executive (UK). Noise Induced Hearing Loss. [www.hse.gov.uk](http://www.hse.gov.uk)
3. Occupational Safety and Health Administration. Occupational Noise Exposure. [www.osha.gov](http://www.osha.gov)
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## 4.9 Occupational Diseases due to Vibration

### 1. Definition

Occupational exposure to vibration can cause two distinct types of disorders, i.e. hand-arm vibration syndrome (HAVS) and disorders due to whole body vibration, depending on the source and characteristics of vibration energy.

Hand-arm vibration syndrome is a clinical syndrome comprising vascular, neurological, musculoskeletal disorders of the upper limbs due to prolonged impact from exposure to hand-transmitted vibration.

Whole body vibration exposure has been found to be associated mainly with a higher incidence of low back pain, mainly due to exposure when the body is supported on a surface which is vibrating

### 2. Occupational hazards

Hand-arm vibration syndrome (HAV) – Occupation that exposes to localized vibration to the hands from using powered tools such as chain saw, jack hammer, concrete breaker, impact drills, impact wrenches, grinders, sanders, concrete vibrator, etc.

Whole body vibration – Occupation that exposes to whole body vibration at a constant frequency, usually requiring seating on a vibrating seats.

### 3. Main occupational uses and exposure

Hand-arm vibration – tree fellers, construction workers, shipyard workers, manufacturing industries using vibratory tools etc.

Whole body vibration – long haul drivers, prime movers

### 4. Diagnosis criteria

Hand Arm Vibration Syndrome:

Symptoms and Signs:

Classically, the syndrome consists of secondary Raynaud's phenomenon, finger tingling, numbness and dullness, small muscles weakness and wasting, and bony degenerative changes. The health effects of HAV exposure can be categorised into:

Vibration-induced white fingers (Raynaud's phenomenon of occupational origin)  
- The disease is characterized by attacks of vasoconstriction of the digital arteries. Attacks can last for minutes to hours and are more likely to occur with exposure to the cold.

Peripheral sensorineural polyneuropathy - Symptoms include tingling and numbness in finger and hands. In later stages reduced sensation of touch, temperature and vibration and an impairment of manual dexterity.

Osteoarticular diseases - The conditions found to be associated with exposure to HAV are osteoarthritis of the elbow and wrist, carpal bone diseases, osteonecrosis of the semilunate bone (Kienböck's disease), and pseudoarthritis of the scaphoid bone. The osteoarticular diseases are confirmed by radiography.

There is moderate evidence that contracture of the palmar aponeurosis (Dupuytren's disease) may occur as an effect of HAV. The prevalence of musculoskeletal diseases of the upper limbs, shoulder or neck is increased in HAV-exposed but it has not been possible to separate the effects of HAV from the effects of other physical factors, e.g. force, repetition and posture.

In tropical environments (as in most ASEAN countries), the vibration-induced white finger may not occur due to the absence of cold provocation. Hence, the diagnosis of HAVS can be made based on the neurological component alone provided the exposure criteria are met and objective tests show impairment of the sensory components in the hands.

Diagnostic tests:

An objective test to evaluate impairment of the sensorineural component of HAVS involves the determination of vibrotactile perception using a vibrotactile perception meter; and thermotactile perception thresholds for hot and cold using a thermal aesthesiometer.

An objective test to evaluate impairment of the vascular component of HAVS involves the measurement of finger re-warming times after cold provocation using a temperature monitor; and the measurement of finger systolic blood pressures using a suitable sphygmomanometer.

Where the above tests are not available, the objective evaluation of the impairment of the neurological component of the HAVS can be performed using Semmes-Weinstein monofilaments.

The Stockholm Workshop (1986) scale classification system for cold-induced peripheral vascular symptoms and neurological symptoms is an internationally recognized grading system for staging the severity of HAVS.

Table : Stockholm Workshop Scale for the classification of cold-induced Raynaud's phenomenon in the hand-arm vibration syndrome

Stage	Grade	Description
0 <sub>v</sub>	-	No attacks
1 <sub>v</sub>	Mild	Occasional blanching attacks affecting only the tips of one or more fingers
2 <sub>v</sub>	Moderate	Occasional attacks affecting distal and middle (rarely also proximal) phalanges of one or more fingers
3 <sub>v</sub>	Severe	Frequent attacks affecting all phalanges of most fingers
4 <sub>v</sub>	Very Severe	As in Stage 3, with trophic skin changes in the finger tips

V = vascular

Stage	Description
0 <sub>SN</sub>	Exposed to vibration but no symptoms
1 <sub>SN</sub>	Intermittent numbness, with or without tingling
2 <sub>SN</sub>	Intermittent or persistent numbness, reduced sensory perception
3 <sub>SN</sub>	Intermittent or persistent numbness, reduced tactile discrimination and/or manipulative dexterity

SN = sensorineural

## Whole body vibration

Whole body vibration usually presented with discomfort at work; associated with the frequency of vibration. There were reports on the effect of whole body vibration to degenerative changes of the spine, reproductive disorders or changes in the physiological functions.

However, the causal relationship between whole body vibration and the development of related health effects could not be established clearly, therefore is not discussed further in this section.

## 5. Exposure limit

### Hand Arm Vibration

#### i. Minimum intensity of exposure

Information about hand and arm vibration levels for specific tools used may be obtained from existing databases.

If exposed to a frequency weighted and time averaged acceleration  $\geq 3 \text{ m/s}^2$  (A(8)) for 10 years 10 % of exposed individuals will develop vibration induced white fingers and/or sensorineural polyneuropathy.

HAV with a frequency range below 100 Hz and of high magnitude appears to be associated with joint and bone pathology (pneumatic percussive tools).

The daily exposure limit value (A(8)) shall be  $5 \text{ m/s}^2$  and the action value  $2.5 \text{ m/s}^2$  (EU directive 2002/44/EC); to be used as control measure for prevention.

#### ii. **Minimum duration of exposure:**

Depending on acceleration level:

$3\text{-}10 \text{ m/s}^2$  (A(8)): 3-10 years

$>10 \text{ m/s}^2$  (A(8)): 1-3 years.

#### **Maximum latent period:**

Not known, probably months.

iv. **Induction period:**

Between 1-5 years.

**Whole Body Vibration**

The comfort zone for whole body vibration exposure ranges from  $<0.315 \text{ m/s}^2$  (comfortable) to  $>2.5 \text{ m/s}^2$  (extremely uncomfortable). No other definitive exposure limits are available.

**6. Further reading**

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## 4.10 Occupational Hepatitis B Virus infection

### 1. Definition

Hepatitis B Virus is a common cause of acute and chronic hepatitis in ASEAN countries.

### 2. Occupational hazard

Hepatitis B Virus (HBV) happened from work.

### 3. Main Occupational uses and exposure

Occupational exposure to viral hepatitis patients, infected blood and body fluids, contaminated instruments and needle stick injuries. Sex workers? Healthcare workers?.

### 4. Diagnostic Criteria

#### 4.1 Symptoms and Signs

Clinical presentation:

##### a) Typical hepatitis:

- A history of blood transfusions or blood products, injections (as in drug addicts), unprotected sex with multiple partners, between the past 4 weeks to 6 months.

- Clinical: may have symptoms and signs of anorexia, fatigue, jaundice, dark urine, liver pain, nausea, vomiting, and pale stools.

- Tests:

+ AST, ALT increased (often rise above 5 times the normal value).

+ Bilirubin increased, mainly direct bilirubin.

+ HBsAg (+) or (-) and anti-HBc IgM (+).

##### b) A number of other clinical presentation:

- *Hepatitis without jaundice:*

+ Clinical: may have fatigue, anorexia, muscle pain.

+ Tests: AST, ALT increased, anti-HBc IgM (+) and HBsAg (+/-).

- *Hepatitis with prolonged prolonged jaundice:*

+ Clinical: There are clinical symptoms similar to typical, accompanied by itching. Jaundice usually prolongs more than 6 weeks, sometimes 3-4 months.

+ Tests: AST, ALT increase, bilirubin increase, mainly direct bilirubin, HBsAg (+) or (-) and anti-HBc IgM (+).

- *acute hepatitis with liver failure:*

+ Clinical: Patients with acute liver failure manifested with symptoms of hepatic encephalopathy.

+ Tests: AST, ALT increase, bilirubin increase, mainly unconjugated bilirubin, HBsAg (+) or (-) and anti-HBc IgM (+), prolonged blood clotting time, thrombocytes decrease.

#### 4.2 Diagnostic tests for chronic hepatitis B virus

- HBsAg (+) > 6 months or HBsAg (+) and Anti-HBc IgG (+).

- AST, ALT increased intermittent or continuous over 6 months.

- There is evidence of progressive tissue lesions, cirrhosis (defined by liver biopsy or liver elastography or fibrotest or APRI index), not due to other causes.

#### 5. Exposure limit

5.1 Minimum intensity of exposure:

Single exposure incident sufficient.

5.2 Minimum duration of exposure:

6 months.

5.3 Maximum latent period:

- Hepatitis: 06 months.

- Cirrhosis, liver cancer: 10 years.

#### 6. Differential diagnosis:

Differential diagnosis for acute hepatitis B virus:

- Other types of hepatitis such as toxic hepatitis, other viral hepatitis (hepatitis A virus, hepatitis E virus, hepatitis C virus), autoimmune hepatitis, and alcohol hepatitis

- Other causes of jaundice:

+ Jaundice in some infectious diseases: e.g. Leptospirosis, malaria, and dengue fever

+ Jaundice due to mechanical biliary obstruction: e.g. Tumor of the head of pancreas, biliary duct tumors, and biliary stones.

7. Classification of disease extent/Complications:

+ Level 1: Full recovery, no sequelae.

+ Level 2: Chronic hepatitis due to HBV.

+ Level 3: HBV cirrhosis.

+ Level 4: Liver cancer or severe acute hepatitis causing death.

## 4.11 Chronic Silicosis

### 1. Definition

Silicosis is a chronic interstitial fibrotic disease of the lungs resulting from prolonged and intense exposure to free crystalline silica.

### 2. Occupational hazard

Dust contains free crystalline silicon dioxide

### 3. Main Occupational uses and sources of exposure

These are:

- Hard rock mining
- Tunneling
- Quarrying
- Foundry work
- Stone cutting & Dressing & Polishing
- Sandblasting & Abrasives
- Glass manufacturing
- Ceramics
- Shipbuilding
- Vitreous enameling

### 4. Diagnostic Criteria

#### 4.1 Symptoms and signs

Shortness of breath with exertion is typically the presenting symptom. The disease progresses to breathlessness at rest and increased cough with sputum production. Wheezing occurs when there is associated chronic obstructive bronchitis or asthma.

#### 4.2 Diagnostic tests

- Chest Radiographs

Abnormal chest radiograph, the severity of which can be classified according to the International Labour Office (ILO) System of Classification of Radiographs of Pneumoconiosis 2000 (Obvious pulmonary fibrosis with profusion graded from category 1/0 and above). Chest radiograph reveal

small round nodular lesion or and opacities in the upper lower lung fields. There may be associated hilar adenopathy, lymph node calcification.

- Pulmonary Function Tests

In simple silicosis, pulmonary function can be normal. As the disease progresses, a restrictive pattern develops, with reduction in forced vital capacity (FVC), Forced Expiratory Volume in 1 second (FEV1), total lung capacity (TLC) and lung compliance. Diffusion capacity also decreases.

- Chest Computerised Axial Tomography (CAT) scans

Chest CT (High Resolution CT Scan, HRCT) may reveal confluent lesions in what was previously graded simple Silicosis on a plain chest x-ray. Chest CT may also show abnormal lesions at an early stage of disease where no abnormalities are detected on a plain chest radiograph. Chest CT can help to evaluate the presence of nodules and the degree of emphysematous changes in complicated silicosis. A Chest CT is not essential for diagnosing chronic silicosis, but may help in borderline cases.

- Lung Biopsy

Lung biopsy is not indicated to make a diagnosis of chronic silicosis. On rare occasions, it is used primarily to rule out a potentially treatable disease rather than to diagnose Silicosis.

## 5. Exposure limit

### 5.1 Minimum intensity of exposure

Permissible limit for exposure to silica crystalline not exceed 50 mcg/cubicmeter.

### 5.2 Minimum duration of exposure

Exposure to crystalline silica for at least 5 years

### 5.3 Maximum latent period

No maximum latent period

## 6. Differential Diagnosis

### 6.1 Tuberculosis

### 6.2 Sarcoidosis

### 6.3 Hypersensitivity pneumonitis

### 6.4 Collagen diseases

- Scleroderma
- Rheumatoid Arthritis

## 6.5 Metastatic Lung cancer

## 6.6 Histoplasmosis

Other forms of Silicosis such as acute, accelerated, complicated silicosis or silicosis with associated diseases such as tuberculosis and cancer are not included in this section.

## 7. Further reading

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## 4.12 Occupational Asthma

### 1) Definition

Occupational asthma is a disease characterized by reversible airways obstruction and/or airway hyper-responsiveness caused by agents encountered in the work environment.

### 2) Occupational hazards

Occupational asthma may be caused by a variety of agents at the workplace. These include high molecular weight or low molecular weight agents, with specific clinical and laboratory characteristics for each group. Common examples are:

High-molecular-weight agents:

Laboratory animals, crab/seafood, mites, insects, flour and grain dusts, natural rubber latex gloves, bacterial enzymes, castor bean dust, vegetable gums, etc.

Low molecular weight agents:

Isocyanates, acid anhydrides, amines, platinum salts, cobalt

Exercised induced asthma

### 3) Main occupational uses and exposure

The listed agents are found in a variety of workplaces. Cases of occupational asthma have been identified from the following work activities. The specific agents and the extent of exposure differ depending on the work process. A risk assessment that takes into account the systems of work, and susceptibility of individuals is necessary in evaluating possible cases of occupational asthma.

- Animal Handlers
- Farming
- Food Processing
- Bakeries
- Health Care Workers
- Detergent Making
- Food Processing
- Auto Spray Painting
- Varnishing
- Woodworking
- Metal Grinding
- Sawmill Work
- Carpentry
- Pharmaceutical Manufacturing and Packaging
- Janitorial Work
- Meat Packing

\* Rice millers syndrome had been reported in Malaysia and other parts of Asia; comprising of acute and chronic irritant allergic responses such as nasal catarrh, chest tightness, asthma and eosinophilia.

#### 4) Diagnostic criteria

Occupational asthma is diagnosed by a history confirming an association between the occurrence of symptoms, and its relationship to workplace exposure

##### 4.1 Symptoms and signs

Symptoms of occupational asthma are no different from non-occupational asthma. They include coughing, wheezing, chest tightness and shortness of breath. Auscultation may reveal the presence of rhonchi. Asthmatic individuals can be relatively symptom-free in between attacks of asthma.

##### 4.2 Relevant history.

- No previous medical and clinical history of asthma before workplace exposure. (Caution: A previous history of childhood asthma does not necessarily rule out a diagnosis of occupational asthma in adults)
- The presence of a known asthma-causing agent in the workplace (although new agents may be recognized as capable of causing occupational asthma)
- Improvement of symptoms during periods of non-exposure to workplace agents (weekends or vacation) and recurrence of symptoms on returning to work (recognizing that asthmatic attacks may be immediate, delayed, dual, or episodic. Where symptoms occur hours after exposure to the causal agent, it may seem not to improve when away from the workplace).

##### 4.3 Relevant investigations:

- Serial Peak Expiratory Flow Rate (PEFR), demonstrating wide variation in readings at and away from work. Software systems such as OASYS are available for facilitating a more objective evaluation of serial peak flow readings
- Pre and post beta-agonist bronchodilator spirometry to confirm reversible airflow obstruction

- Bronchial provocation tests can help confirm a case of occupational asthma, but this should only be performed in clinical settings with experience and expertise in conducting such tests.

#### 5) Exposure limit

Minimum intensity and duration of exposure varies according to the susceptibility of the individuals to the agents at the workplace.

(i) For irritants; acute large dose exposure may result in the effect manifesting immediately.

(ii) For allergens; manifestation may appear within weeks to many years later. There is no specifically defined maximum latent period.

#### 6) Differential diagnosis

- Chronic Obstructive Pulmonary Diseases
- Infectious diseases of the lungs
- Pre-existing asthma, not of occupational origin

#### 7) Further reading

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4. R. Musa, L Naing, Z. Ahmad, Y. Kamarul. Respiratory health of rice millers in Kelantan, Malaysia. Southeast Asian Journal of Tropical Medicine and Public Health. 2000 Sep; 31(3): 575-578

## 4.13 Occupational Contact Dermatitis

### OCCUPATIONAL ALLERGIC CONTACT DERMATITIS

#### 1. Definition

Allergic contact dermatitis manifests as a skin rash following skin contact with allergens (in the workplace in the case of occupational allergic contact dermatitis). It is a cell-mediated reaction involving sensitized T cells in a delayed type IV immune response.

\* cercarial dermatitis, or ‘sawah’ itch is a dermatitis reported among paddy farmers. The condition is due to exposure to the schistosome *S. spindale*; reported in some states Malaysia, and likely exist in other ASEAN countries.

#### 2. Occupational hazards

- Potassium dichromate
- Nickel sulfate,
- Cobalt chloride
- Antioxidants and accelerators in natural rubber
- Paraphenylenediamine in organic dyes
- Glyceryl thioglycolate
- Ammonium persulfate
- Epoxy resins
- Formaldehyde resins
- Acrylic resins
  
- Rosin (Colophony)
  
- - Plant and animal products. Include schistomes from agricultural activities

The list is not exhaustive.

#### 3. Main occupational uses and exposure

- Construction workers
- Hairdressers
- Health care workers
- Cooks and caterers
- Mechanics
- Electronics workers

- Electroplating workers
- Machine operators
- Agriculture: paddy farmers especially during the field preparation and transplanting stages of paddy.

#### 4. Diagnostic Criteria

##### 4.1 Symptoms and Signs

- Signs of Allergic Contact Dermatitis include patches of erythema and edema on exposed areas of skin, which can progress to vesicle formation, rupture and oozing dermatitis.
- The first signs appear weeks after exposure to the allergen for the first time.
- The dermal response may continue to increase in severity even without further contact with the allergen.
- A subsequent exposure to even a small amount of the causative agent can trigger another bout of allergic skin reaction.

‘Sawah’ itch may present initially as itching, followed by burning sensation followed by macular, popular eruptions that may become severe dermatitis. The reaction is associated with skin sensitizing antibodies, with immediate and delayed hypersensitivity reaction playing an important role in the manifestation of the dermatitis.

##### 4.2 Diagnostic tests

- Patch test with a standard patch test battery

In some instances, a battery of allergens specific to the patients work or to test materials brought by the patient may be required

#### 5. Exposure limit

##### 5.1 Minimum intensity of exposure

None, although usually following considerable initial contact with the causal agent.

##### 5.2 Minimum duration of exposure

The duration of skin contact with the causal agent is at least 2 weeks, but can be much shorter for skin rashes to appear in an individual who is already sensitized.

### 5.3 Maximum latent period

No Maximum latent period

## 6. Differential diagnosis

- Seborrhoeic dermatitis
- Atopic eczema
- Urticaria
- Psoriasis
- Lichen planus
- Chickenpox (varicella)
- Scabies

## 7. Further reading

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5. Krishnasamy M, Cercarial Dermatitis Among Rice-field Workers in Labu, Negeri Sembilan, Peninsular Malaysia Trop Biomed 1995; 12: 109-113.

## **OCCUPATIONAL IRRITANT CONTACT DERMATITIS**

### 1. Definition

Irritant contact Dermatitis is caused by a direct toxic effect on the skin due to irritant chemicals and is characterized by erythema, scaling , fissuring and pruritus.

## 2. Occupational hazards

- Soaps, detergent , cleaning agents
- Solvents
- Acids & Alkalis
- Cement
- Soldering fluxes
- Cutting oils and coolants
- Food products
- Fiberglass
- Petroleum products
- The list is not exhaustive.

## 3. Main occupational uses and exposure

- Cleaners
- Hairdressers
- Chemical workers
- Health care workers
- Construction workers
- Gardeners
- Mechanics
- Vehicle assemblers
- Degreasers
- Metal workers

## 4. Diagnostic Criteria

### 4.1 Symptoms and signs

Irritant contact Dermatitis is characterized by erythema, scaling, fissuring, pruritus and a burning sensation over the affected area of skin. It commonly affects the hands and fingers.

### 4.2 Diagnostic tests

Patch tests (Not for irritant contact dermatitis, but can help to distinguish allergic from irritant dermatitis)

## 5. Exposure limit

### 5.1 Minimum intensity of exposure

Effects can be almost immediate or within minutes of contact depending on the concentration and pH of the causal agent

### 5.2 Minimum duration of exposure

Effects can be almost immediate or within minutes of contact depending on the concentration and pH of the causal agent

### 5.3 Maximum latent period

No maximum latent period

## 6. Differential diagnosis

- Urticaria
- Seborrhoeic dermatitis
- Psoriasis
- Fixed drug eruption
- Lichen planus
- Scabies
- Chickenpox

## 7. Further reading

1. Fisher AA. Occupational dermatitis, in Contact Dermatitis. 5th ed. Philadelphia : Lippincott Willcans & Wilkins. 2001; 419-449.

2. Frosch PJ. Cutaneous irritation, in Textbook of Contact Dermatitis. Berlin : Springer –Verlag; 1995, 28-61.

3. Mathias CG. Contact dermatitis and workers' compensation: criteria for establishing occupational causation and aggravation. J Am Acad Dermatol 1989;20: 842-8.

## 4.14 Carpal tunnel syndrome (CTS)

### 1. Definition

CTS is a constellation of symptoms and signs resulting from mononeuropathy of the median nerve due to the compression in the Carpal Tunnel.

### 2. Occupational hazards

It occurs in workers who repetitively move their hands in work and/or forceful movement at wrist. The worker who uses their hand in lifting heavy object or holds the equipment with vibration frequently. Working with over extension more than 45 degree of wrist or ulnar deviation of wrist or work with increase squeezing of hand can cause carpal tunnel syndrome.

### 3. Main occupational uses and exposure

Activities involving repetitive ,forceful and awkward position of wrist and hand use e.g. meat cutting, sorting of parcels, manual assembling, carpenters, masonry, bricklayer, plasterer, shoemaker, tailor etc.

Working with hand-held tools with pressure against the carpus, e.g. a chisel, hammer or repeated impacts against the carpus (e.g. using the hand as a hammer)

### 4. Diagnostic criteria

#### 4.1 Symptoms and signs

Pain, paraesthesia, numbness, tingling, reduced touch sensitivity in median nerve distribution of hand (in the radial palm and palmar aspect of the thumb, index, middle and ring finger). Weakness and clumsiness of hand Aggravated by prolonged full active flexion of wrist

#### 4.2 Diagnosis Test

1. Phalen test : By fully flexing both hand if positive there will be pain, paresthesia or numbness at tip of middle finger in 30 seconds. If there is symptoms in less than 30 seconds it means the patient has severe diseases.

2. Positive Tinel's sign (percussion over the Carpal Tunnel) produce discomfort to the distribution of the median nerve

3. Nerve conduction velocity test: The latency of Median nerve is slower by 25-30% when compare with the normal hand.

## 5. Exposure criteria

Measurements of repetition at the work place (e.g. number of items handled, no. of hand repetitions) and assessment of force exerted (e.g. handled weights) may add valuable information although threshold limits for exposure are not established.

### Minimum intensity of exposure (guiding)

- Highly repetitive procedures (guiding) : >10 items handled/minute or >20 hand repetitions/minute.
- High force: > 1 kg load handled.
- Awkward wrist posture: posturing in more than 45 degree of wrist flexion or extension without excessive radial or ulnar deviation.

### Minimum duration of exposure (guiding)

- Repetitive and forceful wrist and hand, direct pressure: Months

### Induction period

- As for minimum duration of exposure

## 6. Differential diagnosis

1. Cervical radiculopathy
2. Flexor carpi radialis tenosynovitis
3. Median nerve compression at elbow
4. Rheumatoid arthritis
5. Diabetes Mellitus
6. Hypothyroidism.

## 7. Further reading:

1. Hou WH, Hsu JH, Lin CH, Liang HW. Carpal tunnel syndrome in male visual displayterminal (VDT) workers. *Am J Indust med* 2006;50(1):1-7.

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3. Keith T. Palmer, E. Clare Harris, and David Coggon. Carpal tunnel syndrome and itsrelation to occupation: a systematic literature review. *Occup Med* 2007;57:57-66.

4.AMA Guides® to the Evaluation of Disease and Injury Causation. Edited by J. Mark Melhorn, William E. Ackerman III, James B. Talmage, Mark H. Hyman. 2<sup>nd</sup> edition, 2014.

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6.Carpal tunnel syndrome and its relationship to occupation: a meta-analysis. Annica Barcenilla, Lyn M. March, Jian Sheng Chen and Phillip N. Sambrook. Rheumatology 2012;51:25-261

7.ACOEM's Occupational Medicine Practice Guidelines. 3<sup>rd</sup> edition, 2011

## 4.15 Rotator Cuff Tendinopathies

### 1. Definition

- Repetitive microtrauma and wear-and-tear due to overstraining, vigorous and repetitive movements of the upper limb causing inflammation of the muscle or tendon around the shoulder. This may lead to partial tear or rupture of the tendon.
- This includes rotator cuff tendinitis, rotator cuff tears (partial or total), supraspinatus tendinitis, calcific tendinitis, impingement syndrome, bicipital tendinitis, and subacromial bursitis.
- The most disabling condition is the total rupture of the rotator cuff tendon.

### 2. Occupational hazards

Rotator cuff tendinopathies may result acutely from trauma to the shoulder joint from accidents at work. In a chronic presentation; may be due to

- Forceful and repetitive flexion, abduction and rotation of the shoulder joint (e.g. repetitive throwing and swinging motions)
- Awkward postures: sustained shoulder posture of more than 60 degrees of flexion or abduction

### 3. Main occupational uses and exposure

#### a. Work tasks:

Activities involving repetitive upper limb movement with elevated arm or repetitive throwing and swinging motions. Examples: overhead assembly, overhead welding, overhead auto repair, reaching, lifting and carrying loads on the shoulders.

#### b. Main occupations:

Mechanics, welders, textile workers and professional sportsmen and women.

#### 4. Diagnostic criteria

Symptoms and signs:

Shoulder pain exacerbated by movement in one or more of the following: abduction, flexion, external rotation and internal rotation .

Specific examination may show:

- Local tenderness on the supraspinatus tendon, rotator cuff, subacromial bursa, coracoacromial ligament, acromium, deltoid or the greater tuberosity of the humerus
- muscle atrophy may be noticeable if the condition has been present for several weeks or longer;
- with significant disruption of the rotator cuff, a patient may have no active elevation past mid-range
- Impingement sign and test may be positive

Relevant Investigations

- X-Rays – shoulder xrays are usually normal or may show a small bone spur, however is useful to exclude other pathologies.
- MRI or Ultrasound – may localize any tear or lesions on the rotator cuff tendons.

#### 5. Exposure limit

The assessment of exposures relevant to rotator cuff tendinopathies is complex. This is because the relevant factors include not just the number of repeated movements at the joint, but is also dependent on the size, shape and weight of any loads that are carried, and on the postures maintained by the individual worker when performing such tasks at work.

i. Minimum intensity of exposure

- Indication of frequent, forceful, repetitive joint movements, often at the extremes of the range of movement during work activities. (e.g. number of items handled per unit time, no. of arm repetitive

movements) and assessment of force exerted (e.g. handled weights), percent of the work time with the arms elevated may add valuable information although threshold limits for exposure are not established.

- Highly repetitive procedures : As a rough guide, e.g. More than 10 items handled/minute or more than 20 repetitions/minute.
- High force : More than 1 kg load handled.
- Awkward arm elevation : As a rough guide, arms elevated more than 60 degrees more than 50% of the work time.

ii. Minimum duration of exposure

Days

iii. Latent period

Days

**6. Further reading**

- 1) [Internet]. 2009 [cited 2 June 2017]. Available from: <http://1>) Information notices on occupational diseases: a guide to diagnosis. European Commission 2009.
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- 4) Bodin J, Ha C, Petit Le Manac'h A, Sérazin C, Descatha A, Leclerc A et al. Risk factors for incidence of rotator cuff syndrome in a large working population. *Scandinavian Journal of Work, Environment & Health*. 2012;38(5):436-446.
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