MCQS
ON
OCCUPATIONAL HEALTH

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MCQs on

Occupational Health

First Edition · Part 1

Part 1 — Pneumoconioses, Metals & Physical Agents

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MBBS, AFIH (Distinction)

Suitable for

Associate Fellow of Industrial Health (AFIH) aspirants

Preface

As AFIH students, we often struggled to find a structured resource for practising multiple-choice questions across the breadth of occupational health and industrial medicine. This book was created to bridge that gap.

Every question here is newly authored, tied to a specific concept, and paired with a worked explanation, so that each one doubles as a quick revision note while you practise.

This first edition launches as **Part 1**, covering three high-yield areas of the syllabus — Pneumoconioses, Metals, and Physical Agents — with 1187 questions in all. The remaining units are being prepared to the same standard and will follow as **Part 2**, so treat this as a living, growing resource rather than a finished textbook.

Best regards,

Dr Ravikiran Jadhav
Author

Acknowledgment

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To my wife, daughter, and parents — thank you for your unwavering love and support, which made this endeavour possible. Finally, I extend my sincere gratitude to my mentor, Dr. Ujjwal Vaid, whose guidance and inspiration have been instrumental in shaping my journey.

Warm regards,

Dr Ravikiran Jadhav

Disclaimer

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UNIT I — PNEUMOCONIOSES

Asbestos & Asbestos-Related Diseases

TOPICS IN THIS CHAPTER

Nature & Fibre Types

Q1. Asbestos minerals are divided into two mineralogical groups, namely:
A) Silicate and carbonate
B) Serpentine and amphibole
C) Crystalline and amorphous
D) Fibrous and granular

Answer: B) Serpentine and amphibole

Explanation: Asbestos comprises the serpentine group (chrysotile) and the amphibole group (crocidolite, amosite and others); the other pairings are unrelated mineral classifications.

Q2. The only serpentine asbestos, accounting for most commercial use, is:
A) Crocidolite
B) Amosite
C) Tremolite
D) Chrysotile

Answer: D) Chrysotile

Explanation: Chrysotile (white asbestos) is the sole serpentine fibre and about 95% of commercial asbestos; the distractors are all amphiboles.

Q3. Chrysotile fibres are physically distinguished by being:
A) Curly and serpentine
B) Straight and needle-like
C) Spherical
D) Branched and dendritic

Answer: A) Curly and serpentine

Explanation: Serpentine chrysotile is curly, whereas amphiboles are straight rigid rods; this shape influences deposition and clearance.

Q4. The amphibole asbestos most strongly associated with mesothelioma is:
A) Chrysotile
B) Anthophyllite
C) Crocidolite
D) Actinolite

Answer: C) Crocidolite

Explanation: Crocidolite (blue asbestos) has the highest mesothelioma potency owing to its long, thin, biopersistent fibres; chrysotile is far less potent.

Q5. 'Brown asbestos' refers to which amphibole fibre?
A) Crocidolite
B) Amosite
C) Chrysotile
D) Tremolite

Answer: B) Amosite

Explanation: Amosite is brown asbestos; crocidolite is blue and chrysotile is white. Colour names are a common examination point.

Q6. Amphibole fibres are more pathogenic than chrysotile mainly because they are:
A) More water-soluble
B) Larger than 100 micrometres
C) Non-fibrous
D) Straight and highly biopersistent in the lung

Answer: D) Straight and highly biopersistent in the lung

Explanation: Straight, durable amphiboles resist clearance and persist for decades, whereas chrysotile is partly cleared and dissolved; size and solubility distractors are wrong.

Q7. Compared with amphiboles, chrysotile is cleared from the lung:
A) More readily, being less biopersistent
B) Not at all
C) Only by the kidneys
D) More slowly than any amphibole

Answer: A) More readily, being less biopersistent

Explanation: Chrysotile is partially dissolved and cleared, lowering its biopersistence and potency relative to the durable amphiboles.

Q8. The Stanton hypothesis holds that the most carcinogenic asbestos fibres are:
A) Short and thick
B) Spherical and large
C) Long (over 8 micrometres) and thin (under 0.25 micrometres)
D) Soluble and short

Answer: C) Long (over 8 micrometres) and thin (under 0.25 micrometres)

Explanation: Stanton linked carcinogenicity to long thin durable fibres that penetrate deeply and resist clearance; short or thick fibres are less potent.

Q9. Which fibre is blue asbestos?
A) Amosite
B) Crocidolite
C) Chrysotile
D) Tremolite

Answer: B) Crocidolite

Explanation: Crocidolite is blue, amosite brown and chrysotile white; the colour-to-mineral pairing is a frequently tested point in asbestos questions.

Q10. Tremolite is important chiefly because it:
A) Is the main commercial asbestos
B) Is water-soluble and harmless
C) Is a serpentine fibre
D) Contaminates chrysotile and some talc deposits

Answer: D) Contaminates chrysotile and some talc deposits

Explanation: Tremolite is a non-commercial amphibole that contaminates chrysotile and talc, contributing to disease attributed to those materials.

Q11. Chemically, asbestos minerals are:
A) Hydrated silicates of magnesium and iron
B) Pure carbon allotropes
C) Calcium carbonates
D) Aluminium oxides

Answer: A) Hydrated silicates of magnesium and iron

Explanation: Asbestos minerals are fibrous hydrated silicates containing magnesium and/or iron; the distractors are unrelated compounds.

Q12. Approximately what proportion of historically used commercial asbestos was chrysotile?
A) About 5%
B) About 50%
C) About 95%
D) Essentially none

Answer: C) About 95%

Explanation: Chrysotile made up roughly 95% of commercial asbestos use, which is why most exposure involved it despite amphiboles' higher potency.

Q13. Which property of asbestos made it commercially valuable?
A) High water solubility
B) Heat resistance, tensile strength and insulation
C) Electrical conductivity
D) Radioactivity

Answer: B) Heat resistance, tensile strength and insulation

Explanation: Asbestos was prized for fire/heat resistance, strength and insulating capacity; these same durable fibres resist biological clearance.

Q14. Anthophyllite, tremolite and actinolite are classified as:
A) Serpentine asbestos
B) Non-asbestos zeolites
C) Man-made vitreous fibres
D) Amphibole asbestos

Answer: D) Amphibole asbestos

Explanation: These three are amphiboles (like crocidolite and amosite); chrysotile alone is serpentine, and zeolites/MMVF are different fibres.

Sources & Occupations

Q15. High-risk asbestos trades classically include all of the following EXCEPT:
A) Aluminium pot-room smelting
B) Shipbuilding and ship-breaking
C) Insulation and lagging work
D) Brake-lining manufacture and repair

Answer: A) Aluminium pot-room smelting

Explanation: Pot-room work exposes to fluoride/alumina, not asbestos; shipbuilding, insulation and brake work are classic asbestos exposures.

Q16. 'Para-occupational' (domestic) asbestos exposure classically occurs in:
A) Workers wearing respirators
B) People living in fibre-free cities
C) Family members exposed via the worker's dusty clothing
D) Office staff with no contact

Answer: C) Family members exposed via the worker's dusty clothing

Explanation: Household contacts inhaled fibres carried home on work clothes, a recognised cause of mesothelioma in spouses and children.

Q17. The Australian crocidolite mining town associated with an epidemic of mesothelioma is:
A) Bhopal
B) Wittenoom
C) Minamata
D) Yokkaichi

Answer: B) Wittenoom

Explanation: Wittenoom (Western Australia) crocidolite mining caused a notorious mesothelioma epidemic; the others are unrelated environmental disasters.

Q18. Asbestos was widely used in construction for all of the following EXCEPT:
A) Roofing and cement sheets
B) Pipe and boiler lagging
C) Fireproofing and insulation board
D) Pharmaceutical tablet coating

Answer: D) Pharmaceutical tablet coating

Explanation: Asbestos went into cement, lagging and fireproofing; it was not used to coat tablets, making that the correct exception.

Q19. Demolition and renovation of old buildings pose asbestos risk mainly because they:
A) Disturb and release previously bound fibres into the air
B) Create new asbestos chemically
C) Only involve chrysotile
D) Eliminate all fibres safely

Answer: A) Disturb and release previously bound fibres into the air

Explanation: Disturbing aged asbestos-containing materials releases respirable fibres; this is why controlled removal and clearance air-monitoring are required.

Q20. Environmental (non-occupational) mesothelioma has been linked in Turkey to:
A) Pure chrysotile cement
B) Tobacco smoke alone
C) Erionite-rich volcanic soil and tremolite
D) Silica dust

Answer: C) Erionite-rich volcanic soil and tremolite

Explanation: Anatolian villages with erionite (a fibrous zeolite) and tremolite soil show very high mesothelioma rates from environmental exposure.

Q21. Which occupation has classically high asbestos exposure?
A) Bank clerks
B) Asbestos-cement and insulation workers
C) Software developers
D) Retail cashiers

Answer: B) Asbestos-cement and insulation workers

Explanation: Asbestos-cement and insulation/lagging trades are classic high-exposure jobs; the office distractors carry no meaningful exposure.

Q22. Brake and clutch mechanics were historically exposed to asbestos from:
A) Welding fumes
B) Battery acid
C) Refrigerant gases
D) Friction linings

Answer: D) Friction linings

Explanation: Friction materials in vehicle brakes and clutches contained asbestos, and dust released while machining or servicing these linings exposed mechanics.

Q23. The latency between first asbestos exposure and disease is generally:
A) Long, typically one to several decades
B) A few days
C) A few weeks
D) Always under one year

Answer: A) Long, typically one to several decades

Explanation: Asbestos diseases have long latencies (asbestosis ~15-20 years, mesothelioma 20-40 years), reflecting slow fibre-driven processes.

Q24. Asbestos textile workers were at risk because the process generated:
A) Only large non-respirable clumps
B) Toxic gases but no fibres
C) Airborne respirable fibres during carding and spinning
D) No dust at all

Answer: C) Airborne respirable fibres during carding and spinning

Explanation: Carding, spinning and weaving of asbestos textiles released high concentrations of respirable fibres into the air, causing heavy exposure among textile workers.

Q25. Ship-breaking (vessel dismantling) is a major asbestos hazard in South Asia because older ships contain:
A) No asbestos at all
B) Extensive asbestos lagging and insulation
C) Only chrysotile cement floors
D) Radioactive material only

Answer: B) Extensive asbestos lagging and insulation

Explanation: Old ships were heavily insulated with asbestos; dismantling them releases large amounts of fibre, a significant regional occupational hazard.

Q26. Which statement about asbestos exposure dose and mesothelioma is correct?
A) A large dose is always required
B) Only continuous lifelong exposure causes it
C) Exposure must exceed the silica PEL
D) Even low or brief exposure can cause mesothelioma

Answer: D) Even low or brief exposure can cause mesothelioma

Explanation: Mesothelioma can follow relatively low or short exposures, and no clear threshold has been established, unlike dose-dependent asbestosis.

Q27. Asbestos cement (AC) sheets remain a common exposure source in India chiefly during:
A) Cutting, drilling and demolition of the sheets
B) Normal undisturbed use
C) Storage in sealed packaging
D) Transport by sealed container

Answer: A) Cutting, drilling and demolition of the sheets

Explanation: Intact AC sheets release little fibre; cutting, drilling and demolition liberate respirable dust, the main exposure route.

Deposition & Biopersistence

Q28. The fibre dimension that best predicts carcinogenic potential is:
A) Total mass only
B) Colour
C) Length and thinness (long, thin fibres)
D) Chemical solubility alone

Answer: C) Length and thinness (long, thin fibres)

Explanation: Long, thin, durable fibres penetrate deeply and resist clearance, the key determinants of carcinogenicity per the Stanton hypothesis.

Q29. 'Biopersistence' of an asbestos fibre refers to:
A) Its colour under the microscope
B) How long it resists dissolution and clearance in the lung
C) Its commercial durability in products
D) Its electrical resistance

Answer: B) How long it resists dissolution and clearance in the lung

Explanation: Biopersistence is the fibre's durability within lung tissue; high biopersistence (amphiboles) means prolonged injury and higher disease risk.

Q30. Asbestos fibres deposit deep in the lung partly because their aerodynamic behaviour is governed by:
A) Length alone, excluding thin fibres
B) Colour
C) Their iron content only
D) Diameter rather than length, allowing long thin fibres to reach alveoli

Answer: D) Diameter rather than length, allowing long thin fibres to reach alveoli

Explanation: Aerodynamic diameter depends mainly on fibre width, so long but thin fibres can still penetrate to the alveoli and pleura.

Q31. Fibres reaching the pleura to cause mesothelioma are typically:
A) Long, thin amphiboles that migrate to and persist at the pleura
B) Short soluble chrysotile only
C) Large non-respirable bundles
D) Spherical particles

Answer: A) Long, thin amphiboles that migrate to and persist at the pleura

Explanation: Durable long thin amphibole fibres translocate to the pleura and resist clearance, driving mesothelial transformation over decades.

Q32. Compared with amphiboles, chrysotile's lower biopersistence is due to:
A) Being larger than amphiboles
B) Containing no silicate
C) Partial dissolution and fragmentation, aiding clearance
D) Being water-repellent

Answer: C) Partial dissolution and fragmentation, aiding clearance

Explanation: Chrysotile breaks up and partly dissolves, so it is cleared more readily, reducing (though not eliminating) its pathogenic potential.

Q33. The main physiological clearance route for inhaled fibres deposited above the alveoli is the:
A) Renal tubules
B) Mucociliary escalator
C) Hepatic portal system
D) Lymphatic drainage of the gut

Answer: B) Mucociliary escalator

Explanation: The mucociliary escalator removes fibres deposited in conducting airways; alveolar fibres rely on slower macrophage clearance.

Q34. 'Frustrated phagocytosis' of long asbestos fibres means the macrophage:
A) Digests the fibre completely
B) Ignores the fibre
C) Converts the fibre to silica
D) Cannot fully engulf the fibre and releases mediators while remaining viable or dying

Answer: D) Cannot fully engulf the fibre and releases mediators while remaining viable or dying

Explanation: A macrophage unable to engulf a long fibre leaks reactive oxygen species and cytokines, perpetuating injury and fibrosis.

Q35. Why is no firm safe threshold accepted for asbestos-induced mesothelioma?
A) Cases occur after low and brief exposures with no clear dose threshold
B) Because exposure cannot be measured
C) Because all exposure is identical
D) Because it requires lifelong exposure

Answer: A) Cases occur after low and brief exposures with no clear dose threshold

Explanation: Mesothelioma has been documented after low or short exposures without a demonstrable threshold, so regulators assume no safe level.

Pathogenesis

Q36. A key mechanism of asbestos-induced injury is generation of reactive oxygen species catalysed by fibre-associated:
A) Calcium
B) Sodium
C) Iron
D) Potassium

Answer: C) Iron

Explanation: Iron on and within asbestos fibres catalyses Fenton-type reactions producing reactive oxygen species, damaging DNA, lipids and proteins.

Q37. Asbestos fibres contribute to carcinogenesis partly by mechanical interference with:
A) Renal filtration
B) Chromosome segregation during mitosis
C) Bile secretion
D) Gastric acid production

Answer: B) Chromosome segregation during mitosis

Explanation: Fibres can physically disrupt the mitotic spindle and chromosome segregation, causing aneuploidy and genetic instability in mesothelial cells.

Q38. Chronic asbestos exposure drives fibrosis through persistent release of profibrotic mediators, chiefly:
A) Interferon-gamma
B) Immunoglobulin E
C) Surfactant protein A
D) Transforming growth factor-beta

Answer: D) Transforming growth factor-beta

Explanation: TGF-beta from activated macrophages stimulates fibroblast collagen production; the distractors are not the principal fibrogenic mediator.

Q39. Asbestos bodies form when a fibre becomes coated with:
A) Iron and protein (ferritin/haemosiderin)
B) Calcium oxalate
C) Cholesterol
D) Melanin

Answer: A) Iron and protein (ferritin/haemosiderin)

Explanation: Macrophages coat a retained fibre with an iron-and-protein layer, producing the golden-brown, beaded or dumbbell-shaped asbestos (ferruginous) body seen on microscopy.

Q40. Asbestosis fibrosis characteristically begins in the:
A) Upper zones
B) Hilar lymph nodes
C) Lower zones and subpleural regions
D) Pleural plaques

Answer: C) Lower zones and subpleural regions

Explanation: Asbestosis is a basal, subpleural interstitial fibrosis, contrasting with the upper-zone nodular disease of silicosis.

Q41. The pleural changes of asbestos exposure are thought to result from fibres that:
A) Are injected intravenously
B) Migrate to and accumulate at the pleural surface
C) Form only in the trachea
D) Dissolve before reaching the lung

Answer: B) Migrate to and accumulate at the pleural surface

Explanation: Long thin fibres translocate to the pleura, where they provoke plaques, effusions, thickening and eventually mesothelioma.

Q42. Asbestos and tobacco smoke interact in lung carcinogenesis in a manner best described as:
A) Purely additive
B) Antagonistic
C) Independent with no interaction
D) Multiplicative (synergistic)

Answer: D) Multiplicative (synergistic)

Explanation: The combined lung-cancer risk greatly exceeds the sum of each, an approximately multiplicative synergy, which underlines smoking cessation.

Q43. Mesothelial transformation by asbestos involves all of the following EXCEPT:
A) IgE-mediated immediate hypersensitivity
B) Chronic inflammation and reactive oxygen species
C) Chromosomal damage and aneuploidy
D) Growth-factor-driven proliferation

Answer: A) IgE-mediated immediate hypersensitivity

Explanation: Mesothelioma arises through chronic inflammation, oxidative and chromosomal damage and growth signalling, not an IgE allergic mechanism.

Q44. Compared with silicosis, the lung fibrosis of asbestosis is:
A) Identical nodular fibrosis
B) Confined to lymph nodes
C) Diffuse and interstitial rather than discrete nodular
D) Purely pleural with no parenchymal disease

Answer: C) Diffuse and interstitial rather than discrete nodular

Explanation: Asbestosis produces diffuse interstitial fibrosis, whereas silicosis forms discrete collagenous nodules; the distinction is histological and radiological.

Q45. Fibre durability matters because longer biopersistence means:
A) Faster clearance
B) More prolonged tissue injury and higher disease risk
C) Lower cancer risk
D) No biological effect

Answer: B) More prolonged tissue injury and higher disease risk

Explanation: Durable fibres remain in tissue for years, sustaining oxidative and inflammatory injury and increasing the likelihood of fibrosis and cancer.

Q46. The role of the SV40 virus in mesothelioma is best described as:
A) Established as the sole cause
B) Protective against mesothelioma
C) Identical to asbestos in mechanism
D) Proposed but unproven and controversial

Answer: D) Proposed but unproven and controversial

Explanation: An association between SV40 and mesothelioma has been proposed but remains controversial and unconfirmed; asbestos is the established cause.

Q47. Why does asbestosis continue to progress after exposure stops?
A) Retained durable fibres keep driving inflammation and fibrosis
B) New fibres are produced endogenously
C) The lung regenerates abnormally without fibres
D) It actually always regresses

Answer: A) Retained durable fibres keep driving inflammation and fibrosis

Explanation: Biopersistent fibres remain in the lung and perpetuate fibrogenesis, so disease can advance years after exposure has ended.

Pathology

Q48. The asbestos (ferruginous) body appears microscopically as:
A) A whorled collagen nodule
B) A non-caseating granuloma
C) A golden-brown, beaded or dumbbell-shaped iron-coated fibre
D) A foamy lipid macrophage

Answer: C) A golden-brown, beaded or dumbbell-shaped iron-coated fibre

Explanation: The iron-protein coat gives a golden-brown beaded body; the distractors describe silicosis, beryllium/sarcoid and lipoid pneumonia respectively.

Q49. Asbestos bodies are best demonstrated with which stain?
A) Ziehl-Neelsen
B) Perls' Prussian blue (for iron)
C) Congo red
D) Periodic acid-Schiff

Answer: B) Perls' Prussian blue (for iron)

Explanation: The iron coat stains with Perls' Prussian blue; ZN detects mycobacteria, Congo red amyloid and PAS glycogen/mucin.

Q50. The presence of asbestos bodies in lung tissue indicates:
A) Active tuberculosis
B) Definite mesothelioma
C) Silica exposure
D) Past asbestos exposure (not necessarily disease)

Answer: D) Past asbestos exposure (not necessarily disease)

Explanation: Asbestos bodies are markers of exposure and help quantify fibre burden, but they do not by themselves establish a specific disease.

Q51. Pleural plaques are most often located on the:
A) Parietal pleura and diaphragmatic surface
B) Visceral pleura over the fissures
C) Pericardium
D) Apical pleura only

Answer: A) Parietal pleura and diaphragmatic surface

Explanation: Benign asbestos plaques arise on the parietal pleura and diaphragm and characteristically spare the apices and costophrenic angles.

Q52. Histologically, asbestosis is defined as:
A) Discrete whorled silicotic nodules
B) Non-caseating granulomas
C) Diffuse interstitial pulmonary fibrosis with asbestos bodies
D) Caseating necrosis

Answer: C) Diffuse interstitial pulmonary fibrosis with asbestos bodies

Explanation: Asbestosis is diffuse interstitial fibrosis with demonstrable asbestos bodies/fibres; the distractors are other pneumoconioses or infections.

Q53. Pleural plaques characteristically:
A) Encase the entire lung
B) Spare the costophrenic angles and lung apices
C) Always cavitate
D) Replace the lung parenchyma

Answer: B) Spare the costophrenic angles and lung apices

Explanation: Plaques are discrete parietal lesions that spare the angles and apices, distinguishing them from diffuse pleural thickening or tumour.

Q54. The gross appearance of malignant pleural mesothelioma is typically:
A) A solitary peripheral coin lesion
B) Multiple bilateral small nodules only
C) A purely endobronchial mass
D) A thick rind encasing and contracting the lung

Answer: D) A thick rind encasing and contracting the lung

Explanation: Mesothelioma forms a confluent pleural rind that encases and contracts the lung, often with effusion, rather than a discrete coin lesion.

Q55. Diffuse pleural thickening from asbestos differs from plaques in that it:
A) Involves the visceral pleura and can restrict lung expansion
B) Is confined to the parietal pleura
C) Is never associated with effusion
D) Always calcifies symmetrically

Answer: A) Involves the visceral pleura and can restrict lung expansion

Explanation: Diffuse pleural thickening involves the visceral pleura, may follow a benign effusion and can cause restriction, unlike discrete parietal plaques.

Q56. Immunohistochemistry of epithelioid mesothelioma is characteristically POSITIVE for:
A) CEA and TTF-1
B) Napsin A
C) Calretinin and WT-1
D) Chromogranin

Answer: C) Calretinin and WT-1

Explanation: Mesothelioma expresses calretinin, WT-1, CK5/6 and D2-40; CEA, TTF-1 and napsin A mark adenocarcinoma, aiding differentiation.

Q57. Markers that are typically NEGATIVE in mesothelioma but positive in pulmonary adenocarcinoma include:
A) Calretinin and WT-1
B) CEA and TTF-1
C) CK5/6
D) D2-40

Answer: B) CEA and TTF-1

Explanation: Adenocarcinoma expresses CEA and TTF-1, which mesothelioma lacks; the mesothelial markers (calretinin, WT-1, CK5/6, D2-40) are the converse.

Asbestosis

Q58. Asbestosis is defined as:
A) A benign pleural plaque
B) A malignant pleural tumour
C) An acute allergic alveolitis
D) Diffuse interstitial pulmonary fibrosis caused by asbestos

Answer: D) Diffuse interstitial pulmonary fibrosis caused by asbestos

Explanation: Asbestosis is the dose-dependent diffuse interstitial fibrosis of the lung parenchyma due to asbestos; plaques and mesothelioma are separate entities.

Q59. The zonal distribution of fibrosis in asbestosis is predominantly:
A) Lower zones and subpleural
B) Upper zones
C) Perihilar
D) Apical

Answer: A) Lower zones and subpleural

Explanation: Asbestosis favours the lung bases and subpleural regions, the opposite of the upper-zone predilection of silicosis.

Q60. The characteristic auscultatory finding in asbestosis is:
A) Loud expiratory wheeze
B) Pleural friction rub only
C) Fine, late inspiratory 'velcro' crackles at the bases
D) Coarse pan-inspiratory crackles at the apices

Answer: C) Fine, late inspiratory 'velcro' crackles at the bases

Explanation: Bibasal fine end-inspiratory crackles are typical of asbestosis and other interstitial fibroses; wheeze and apical signs are not features.

Q61. Unlike uncomplicated silicosis, finger clubbing in asbestosis is:
A) Never seen
B) Relatively common
C) Diagnostic of mesothelioma
D) A sign of cure

Answer: B) Relatively common

Explanation: Clubbing occurs in asbestosis (an interstitial fibrosis), whereas it is characteristically absent in uncomplicated silicosis.

Q62. The latency from first significant exposure to clinical asbestosis is usually:
A) A few weeks
B) 1 to 2 years
C) Over 60 years
D) 15 to 20 years or more

Answer: D) 15 to 20 years or more

Explanation: Asbestosis typically appears after 15-20+ years and is dose-dependent, so heavier exposure shortens the latency.

Q63. Asbestosis is a dose-dependent disease, meaning that:
A) Risk and severity rise with cumulative fibre exposure
B) A single low exposure reliably causes it
C) Dose is irrelevant
D) Only brief exposures cause it

Answer: A) Risk and severity rise with cumulative fibre exposure

Explanation: Asbestosis follows substantial cumulative exposure in a dose-response manner, in contrast to mesothelioma which lacks a clear threshold.

Q64. Pulmonary function testing in asbestosis characteristically shows:
A) An obstructive defect with raised DLCO
B) Entirely normal physiology
C) A restrictive defect with reduced DLCO
D) Isolated air-trapping

Answer: C) A restrictive defect with reduced DLCO

Explanation: Asbestosis, being a fibrosis, gives restriction (low FVC and TLC) with a preserved or raised FEV1/FVC ratio and reduced gas transfer.

Q65. The FEV1/FVC ratio in asbestosis is typically:
A) Reduced below 0.7
B) Normal or increased
C) Exactly 0.5
D) Unmeasurable

Answer: B) Normal or increased

Explanation: As a restrictive process, asbestosis lowers FVC proportionally more than FEV1, leaving the ratio normal or raised; a low ratio implies obstruction.

Q66. The earliest functional abnormality in asbestosis is often a fall in:
A) FEV1/FVC ratio
B) Peak expiratory flow
C) Residual volume
D) Diffusing capacity (DLCO)

Answer: D) Diffusing capacity (DLCO)

Explanation: Impaired gas transfer (low DLCO) frequently precedes measurable volume loss in early interstitial fibrosis such as asbestosis.

Q67. On HRCT, asbestosis typically shows:
A) Subpleural reticulation, traction bronchiectasis and honeycombing at the bases
B) Upper-zone rounded nodules
C) Eggshell nodal calcification
D) Lobar consolidation

Answer: A) Subpleural reticulation, traction bronchiectasis and honeycombing at the bases

Explanation: Asbestosis shows basal subpleural reticulation progressing to honeycombing with traction bronchiectasis; the distractors point to other diseases.

Q68. In the ILO classification, the opacities of asbestosis are typically:
A) Small rounded opacities (p, q, r)
B) Large opacities (A, B, C) only
C) Small irregular opacities (s, t, u)
D) Eggshell calcifications

Answer: C) Small irregular opacities (s, t, u)

Explanation: Asbestosis produces small irregular (linear/reticular) opacities graded s/t/u, unlike the rounded opacities of silicosis and CWP.

Q69. A radiographic feature that helps distinguish asbestosis from silicosis is:
A) Upper-zone rounded nodules
B) Lower-zone irregular opacities, often with pleural plaques
C) Eggshell hilar calcification
D) A miliary pattern

Answer: B) Lower-zone irregular opacities, often with pleural plaques

Explanation: Asbestosis is basal and irregular and is frequently accompanied by pleural plaques, whereas silicosis is upper-zone, nodular and may show eggshell calcification.

Q70. Asbestosis predisposes to which malignancy, especially in smokers?
A) Mesothelioma only
B) Renal cell carcinoma
C) Thyroid carcinoma
D) Bronchogenic carcinoma

Answer: D) Bronchogenic carcinoma

Explanation: Asbestosis (and asbestos exposure) increases lung-cancer risk, multiplicatively with smoking; mesothelioma is a separate, smoking-independent tumour.

Q71. Management of established asbestosis is:
A) Supportive, with removal from exposure and smoking cessation
B) Curative with corticosteroids
C) Reversed by chelation
D) Cured by a course of antibiotics

Answer: A) Supportive, with removal from exposure and smoking cessation

Explanation: No treatment reverses asbestosis; care is supportive, with exposure cessation, vaccination, smoking cessation, oxygen and rehabilitation as needed.

Q72. The natural history of asbestosis after exposure ceases is that it:
A) Always resolves completely
B) Resolves within weeks
C) May continue to progress slowly
D) Converts to mesothelioma

Answer: C) May continue to progress slowly

Explanation: Retained fibres keep driving fibrosis, so asbestosis can progress even after exposure ends, though usually more slowly than during exposure.

Q73. Clubbing and bibasal crackles with a restrictive defect and pleural plaques in an ex-insulation worker suggest:
A) Simple silicosis
B) Asbestosis
C) Occupational asthma
D) Byssinosis

Answer: B) Asbestosis

Explanation: The basal fibrosis signs plus pleural plaques and the exposure history point to asbestosis; the distractors have different patterns and exposures.

Q74. Cor pulmonale in advanced asbestosis arises from:
A) Direct fibre toxicity to the myocardium
B) Pericardial plaques
C) Left-sided valve disease
D) Chronic hypoxaemia and pulmonary fibrosis causing pulmonary hypertension

Answer: D) Chronic hypoxaemia and pulmonary fibrosis causing pulmonary hypertension

Explanation: Progressive fibrosis and hypoxaemia raise pulmonary vascular resistance, leading to pulmonary hypertension and right heart failure.

Q75. Which statement contrasting asbestosis and mesothelioma is correct?
A) Asbestosis is dose-dependent fibrosis; mesothelioma can follow low-dose exposure
B) Both require very heavy exposure equally
C) Both are caused by smoking
D) Asbestosis is malignant and mesothelioma benign

Answer: A) Asbestosis is dose-dependent fibrosis; mesothelioma can follow low-dose exposure

Explanation: Asbestosis needs substantial cumulative exposure, whereas mesothelioma can arise after low or brief exposure and is unrelated to smoking.

Pleural Disease

Q76. The most common manifestation of asbestos exposure is:
A) Mesothelioma
B) Asbestosis
C) Pleural plaques
D) Lung cancer

Answer: C) Pleural plaques

Explanation: Pleural plaques are the commonest and usually asymptomatic marker of asbestos exposure; the others are less frequent and more serious.

Q77. Pleural plaques are best regarded as:
A) A premalignant lesion that becomes mesothelioma
B) A benign marker of exposure that does not become malignant
C) An infection
D) A form of lung cancer

Answer: B) A benign marker of exposure that does not become malignant

Explanation: Plaques indicate past exposure but do not transform into mesothelioma; their presence does, however, flag elevated risk of other asbestos diseases.

Q78. The earliest pleural manifestation of asbestos exposure, sometimes within ten years, is:
A) Calcified plaques
B) Mesothelioma
C) Diffuse pleural thickening
D) Benign asbestos pleural effusion

Answer: D) Benign asbestos pleural effusion

Explanation: A benign, often blood-stained exudative effusion can appear relatively early; plaques and mesothelioma have much longer latencies.

Q79. Benign asbestos pleural effusion is characteristically:
A) An exudate, often blood-stained, and a diagnosis of exclusion
B) A transudate
C) Always purulent
D) Pathognomonic on its own

Answer: A) An exudate, often blood-stained, and a diagnosis of exclusion

Explanation: It is an exudative, frequently haemorrhagic effusion diagnosed only after excluding infection and malignancy, and it may resolve or lead to thickening.

Q80. Diffuse pleural thickening differs from plaques in that it:
A) Is confined to the diaphragm
B) Never affects lung function
C) Involves the visceral pleura and can cause restrictive impairment
D) Always calcifies

Answer: C) Involves the visceral pleura and can cause restrictive impairment

Explanation: Diffuse visceral pleural thickening, often after a benign effusion, can encase the lung and restrict expansion, unlike discrete parietal plaques.

Q81. 'Rounded atelectasis' (folded lung) is:
A) A malignant pleural tumour
B) A benign mass-like lesion of collapsed lung adjacent to pleural thickening
C) An infective cavity
D) A congenital anomaly

Answer: B) A benign mass-like lesion of collapsed lung adjacent to pleural thickening

Explanation: Rounded atelectasis is benign folded lung beneath thickened pleura with a 'comet-tail' of vessels; it mimics tumour and must be recognised.

Q82. The radiological 'comet-tail sign' is associated with:
A) Mesothelioma
B) Pleural plaque calcification
C) Honeycomb lung
D) Rounded atelectasis

Answer: D) Rounded atelectasis

Explanation: Curving vessels and bronchi sweeping into the folded lung produce the comet-tail sign, helping distinguish rounded atelectasis from malignancy.

Q83. Calcified pleural plaques on a chest radiograph indicate:
A) Previous asbestos exposure
B) Active tuberculosis
C) Current heart failure
D) Acute pneumonia

Answer: A) Previous asbestos exposure

Explanation: Bilateral calcified pleural plaques are a reliable radiographic marker of past asbestos exposure, typically after a 20-30 year latency.

Q84. Pleural plaques typically become visible radiographically after a latency of about:
A) 6 months
B) 2 years
C) 20 to 30 years
D) Over 60 years

Answer: C) 20 to 30 years

Explanation: Plaques generally appear two to three decades after first exposure, reflecting the slow pleural response to translocated fibres.

Q85. A unilateral, blood-stained pleural effusion with pleural thickening in a former asbestos worker should raise concern for:
A) Simple pleural plaque
B) Malignant mesothelioma
C) Transudative heart failure
D) Asthma

Answer: B) Malignant mesothelioma

Explanation: A unilateral haemorrhagic effusion with pleural rind in an asbestos-exposed person is suspicious for mesothelioma and warrants pleural biopsy.

Q86. Which asbestos-related pleural condition is malignant?
A) Pleural plaques
B) Benign pleural effusion
C) Rounded atelectasis
D) Malignant mesothelioma

Answer: D) Malignant mesothelioma

Explanation: Of the pleural manifestations, only mesothelioma is malignant; plaques, benign effusion and rounded atelectasis are non-malignant.

Q87. Pleural plaques characteristically spare which regions?
A) The costophrenic angles and lung apices
B) The diaphragm
C) The parietal pleura
D) The posterolateral chest wall

Answer: A) The costophrenic angles and lung apices

Explanation: Plaques favour the parietal pleura, diaphragm and posterolateral wall but spare the costophrenic angles and apices, a useful identifying feature.

Q88. Diffuse pleural thickening most often follows:
A) A pleural plaque
B) Pneumonia unrelated to asbestos
C) A benign asbestos pleural effusion
D) A pneumothorax

Answer: C) A benign asbestos pleural effusion
Explanation: Diffuse visceral pleural thickening commonly develops after a benign asbestos effusion, sometimes obliterating the costophrenic angle.

Q89. The main clinical importance of recognising pleural plaques is that they:
A) Require urgent surgical removal
B) Confirm asbestos exposure and flag the need for surveillance
C) Indicate active infection
D) Cause severe disability themselves

Answer: B) Confirm asbestos exposure and flag the need for surveillance
Explanation: Plaques are usually asymptomatic but mark exposure, prompting counselling, smoking cessation and surveillance for more serious asbestos diseases.

Q90. Which feature favours benign pleural disease over mesothelioma?
A) Circumferential nodular pleural rind
B) Mediastinal pleural involvement
C) A large unilateral bloody effusion
D) Discrete, often calcified plaques sparing the angles

Answer: D) Discrete, often calcified plaques sparing the angles
Explanation: Discrete calcified plaques that spare the angles are benign, whereas a nodular circumferential rind, mediastinal involvement and bloody effusion suggest mesothelioma.

Lung Cancer & Synergy

Q91. Crystalline asbestos is classified by IARC as a:
A) Group 1 (definite) human carcinogen
B) Group 2A
C) Group 2B
D) Group 3

Answer: A) Group 1 (definite) human carcinogen
Explanation: All asbestos types are IARC Group 1 carcinogens, causing lung cancer, mesothelioma, and laryngeal and ovarian cancer.

Q92. Asbestos-related lung cancer:
A) Is only ever squamous
B) Is only ever small-cell
C) Can be of any histological type
D) Is only adenocarcinoma

Answer: C) Can be of any histological type
Explanation: Asbestos increases the risk of all major histological types of bronchogenic carcinoma rather than a single subtype.

Q93. The combined effect of asbestos and smoking on lung-cancer risk is approximately:
A) Subtractive
B) Multiplicative
C) Protective
D) Unrelated

Answer: B) Multiplicative
Explanation: The risks multiply rather than merely add, so an asbestos-exposed smoker has a dramatically higher risk, underscoring smoking cessation.

Q94. Relative to a non-smoking unexposed person, an asbestos-exposed smoker's lung-cancer risk is roughly:
A) Unchanged
B) Halved
C) Twice
D) Around fifty-fold (illustratively)

Answer: D) Around fifty-fold (illustratively)
Explanation: Illustratively, asbestos raises risk ~5-fold and smoking ~10-fold, and together approximately 50-fold, reflecting multiplicative interaction.

Q95. Asbestos-related lung cancer typically requires:
A) Substantial cumulative exposure, often with asbestosis
B) No exposure at all
C) Only a single fibre
D) Exposure only to chrysotile

Answer: A) Substantial cumulative exposure, often with asbestosis
Explanation: Lung cancer risk rises with cumulative dose and is strongly linked to the presence of asbestosis, unlike mesothelioma which can follow low exposure.

Q96. The latency for asbestos-related lung cancer is generally:
A) A few months
B) About 2 years
C) 15 to 20 years or more
D) Over 60 years

Answer: C) 15 to 20 years or more

Explanation: Lung cancer typically appears 15-20+ years after exposure begins, shorter than the often longer latency of mesothelioma.

Q97. Beyond lung and pleura, asbestos is also causally linked to cancer of the:
A) Prostate and testis
B) Larynx and ovary
C) Brain and bone
D) Skin and bladder

Answer: B) Larynx and ovary

Explanation: IARC recognises asbestos as a cause of laryngeal and ovarian cancer in addition to lung cancer and mesothelioma.

Q98. The single most effective measure to reduce lung-cancer risk in an asbestos-exposed worker is:
A) Increasing fibre exposure
B) Annual antibiotics
C) High-dose vitamins
D) Smoking cessation

Answer: D) Smoking cessation

Explanation: Because the asbestos-smoking interaction is multiplicative, stopping smoking yields the greatest reduction in lung-cancer risk.

Q99. Compared with mesothelioma, asbestos-related lung cancer is:
A) Strongly influenced by smoking and dose-dependent
B) Independent of smoking
C) Always of longer latency
D) Unrelated to asbestos

Answer: A) Strongly influenced by smoking and dose-dependent

Explanation: Lung cancer is dose-dependent and powerfully modified by smoking, whereas mesothelioma is smoking-independent and lacks a clear threshold.

Q100. A peripheral lung adenocarcinoma in a former asbestos worker who smoked reflects:
A) A purely genetic cause
B) An effect of pleural plaques alone
C) Multiplicative asbestos-tobacco carcinogenesis
D) A benign process

Answer: C) Multiplicative asbestos-tobacco carcinogenesis

Explanation: The case illustrates the synergy of two carcinogens; pleural plaques themselves are benign markers, not the cause of the cancer.

Mesothelioma

Q101. Malignant mesothelioma most commonly arises from the:
A) Peritoneum
B) Pleura
C) Pericardium
D) Tunica vaginalis

Answer: B) Pleura

Explanation: About 80% of mesotheliomas are pleural; peritoneal is next, with pericardial and tunica vaginalis sites rare.

Q102. The asbestos fibre most strongly linked to mesothelioma is:
A) Chrysotile
B) Amosite (least of amphiboles)
C) None of the fibres
D) Crocidolite

Answer: D) Crocidolite

Explanation: Crocidolite carries the highest mesothelioma risk; chrysotile is far weaker (and contested), with amosite intermediate.

Q103. The latency between asbestos exposure and mesothelioma is typically:
A) 20 to 40 years or longer
B) Under 1 year
C) 2 to 5 years
D) Always over 60 years

Answer: A) 20 to 40 years or longer

Explanation: Mesothelioma has one of the longest latencies in medicine, commonly 20-40+ years, so cases continue decades after exposure.

Q104. With respect to smoking, mesothelioma risk is:
A) Doubled by smoking
B) Halved by smoking
C) Not increased by smoking
D) Caused mainly by smoking

Answer: C) Not increased by smoking

Explanation: Unlike asbestos-related lung cancer, mesothelioma shows no consistent association with smoking; asbestos is the dominant cause.

Q105. The typical clinical presentation of pleural mesothelioma is:
A) Acute haemoptysis and fever
B) Chest pain, dyspnoea and a unilateral pleural effusion
C) Painless jaundice
D) Sudden monoarthritis

Answer: B) Chest pain, dyspnoea and a unilateral pleural effusion

Explanation: Persistent chest wall pain, breathlessness and a unilateral (often bloody) effusion in an asbestos-exposed person are classic for mesothelioma.

Q106. The investigation of choice to confirm mesothelioma is:
A) Sputum cytology alone
B) A tuberculin skin test
C) Serum tumour markers alone
D) Thoracoscopic pleural biopsy with immunohistochemistry

Answer: D) Thoracoscopic pleural biopsy with immunohistochemistry

Explanation: Adequate tissue from thoracoscopy with immunostaining is required; pleural fluid cytology alone is often insufficient for diagnosis.

Q107. Immunohistochemistry of mesothelioma is typically positive for:
A) Calretinin, WT-1 and CK5/6
B) CEA and TTF-1
C) Napsin A and TTF-1
D) Chromogranin and synaptophysin

Answer: A) Calretinin, WT-1 and CK5/6

Explanation: Mesothelial markers (calretinin, WT-1, CK5/6, D2-40) are positive, while adenocarcinoma markers (CEA, TTF-1, napsin A) are negative.

Q108. The chief differential diagnosis of pleural mesothelioma on biopsy is:
A) Tuberculous pleuritis only
B) Simple pleural plaque
C) Metastatic pleural adenocarcinoma
D) Rounded atelectasis

Answer: C) Metastatic pleural adenocarcinoma

Explanation: Distinguishing mesothelioma from metastatic adenocarcinoma to the pleura is the key diagnostic challenge, resolved by immunohistochemistry.

Q109. The prognosis of malignant pleural mesothelioma is:
A) Excellent with cure in most
B) Poor, with median survival around a year
C) Unchanged life expectancy
D) Benign

Answer: B) Poor, with median survival around a year

Explanation: Mesothelioma is generally incurable with a median survival of roughly 8-14 months, reflecting late presentation and limited treatment options.

Q110. First-line systemic chemotherapy for mesothelioma has classically been:
A) Isoniazid and rifampicin
B) Penicillin
C) Methotrexate alone
D) Pemetrexed plus a platinum agent (cisplatin)

Answer: D) Pemetrexed plus a platinum agent (cisplatin)

Explanation: Pemetrexed with cisplatin is the established chemotherapy backbone; the distractors are unrelated antimicrobial or single-agent regimens.

Q111. Regarding an exposure threshold for mesothelioma:
A) No safe threshold is recognised
B) A high threshold must be exceeded
C) It needs lifelong daily exposure
D) It requires exposure above the silica limit

Answer: A) No safe threshold is recognised

Explanation: Because cases follow low and brief exposures without a demonstrable threshold, regulators treat asbestos as having no safe level for mesothelioma.

Q112. Peritoneal mesothelioma typically presents with:
A) Acute chest pain
B) Haematuria
C) Abdominal distension, ascites and pain
D) Jaundice

Answer: C) Abdominal distension, ascites and pain

Explanation: Peritoneal mesothelioma causes ascites, abdominal swelling and pain; it is associated with heavier asbestos exposure than pleural disease.

Q113. The histological subtype of mesothelioma with the best (relatively) prognosis is:
A) Sarcomatoid
B) Epithelioid
C) Biphasic
D) Desmoplastic

Answer: B) Epithelioid

Explanation: Epithelioid mesothelioma carries a comparatively better outlook than the sarcomatoid or biphasic subtypes, which behave more aggressively.

Q114. Mesothelioma classically encases the lung as a:
A) Solitary peripheral nodule
B) Cavitating apical lesion
C) Endobronchial polyp
D) Confluent pleural rind that contracts the hemithorax

Answer: D) Confluent pleural rind that contracts the hemithorax

Explanation: The tumour forms a thick circumferential rind that traps and shrinks the lung, often with effusion, rather than a discrete nodule.

Q115. Which statement about chrysotile and mesothelioma is most accurate?
A) It carries lower risk than amphiboles, partly due to lower biopersistence
B) It is the most potent cause
C) It never causes mesothelioma
D) It is a non-asbestos mineral

Answer: A) It carries lower risk than amphiboles, partly due to lower biopersistence

Explanation: Chrysotile is a weaker cause of mesothelioma than amphiboles, reflecting its lower biopersistence, though contaminating tremolite contributes.

Q116. Pleural mesothelioma on imaging characteristically shows:
A) Bilateral upper-zone nodules
B) Eggshell hilar calcification
C) Nodular, circumferential pleural thickening with effusion
D) A miliary pattern

Answer: C) Nodular, circumferential pleural thickening with effusion

Explanation: Nodular circumferential pleural thickening, often with effusion and volume loss, is typical; the distractors point to other diseases.

Q117. Compared with pleural plaques, mesothelioma:
A) Is benign and self-limiting
B) Is malignant and progressive, not a benign exposure marker
C) Always calcifies
D) Never causes effusion

Answer: B) Is malignant and progressive, not a benign exposure marker

Explanation: Mesothelioma is an aggressive malignancy, whereas plaques are benign exposure markers; the two must not be confused clinically.

Q118. Even brief or low-level asbestos exposure is relevant to mesothelioma because:
A) It only matters at high dose
B) Brief exposure is protective
C) Latency is only weeks
D) Risk exists without a clear threshold and latency is very long

Answer: D) Risk exists without a clear threshold and latency is very long

Explanation: Mesothelioma can follow low or short exposures, manifesting decades later, which is why exposure history must be sought carefully.

Q119. A medico-legal point about mesothelioma is that it is:
A) A compensable occupational disease requiring exposure documentation
B) Never compensable
C) Compensable only if the worker smoked
D) Unrelated to occupation

Answer: A) A compensable occupational disease requiring exposure documentation

Explanation: Mesothelioma is a recognised compensable occupational cancer; documenting the asbestos exposure history is central to claims and notification.

Q120. The fibre characteristic that makes crocidolite especially mesotheliomagenic is its:
A) Short soluble curly fibres
B) Spherical shape
C) Long, thin, highly biopersistent straight fibres
D) High water content

Answer: C) Long, thin, highly biopersistent straight fibres

Explanation: Crocidolite's long, thin, durable straight fibres reach and persist at the pleura, maximising mesothelial injury and transformation.

Diagnosis & Differential

Q121. The cornerstone of diagnosing asbestos-related disease is:
A) A single serum biomarker
B) A careful occupational and exposure history with imaging
C) Sputum culture
D) Skin patch testing

Answer: B) A careful occupational and exposure history with imaging

Explanation: A detailed exposure history combined with imaging (and, for malignancy, biopsy) underpins diagnosis; no single blood test is diagnostic.

Q122. Asbestosis must be distinguished from idiopathic pulmonary fibrosis chiefly by:
A) The pattern of crackles alone
B) Blood eosinophilia
C) Response to antibiotics
D) A history of asbestos exposure and presence of pleural plaques/asbestos bodies

Answer: D) A history of asbestos exposure and presence of pleural plaques/asbestos bodies

Explanation: Exposure history plus markers such as pleural plaques and asbestos bodies separate asbestosis from idiopathic pulmonary fibrosis, which they otherwise resemble.

Q123. Counting asbestos bodies or fibres in tissue or lavage is used to:
A) Estimate past fibre burden and support attribution
B) Diagnose tuberculosis
C) Measure current smoking
D) Grade silicosis

Answer: A) Estimate past fibre burden and support attribution

Explanation: Fibre/asbestos-body counts quantify retained burden and help attribute disease to asbestos; they do not assess infection or smoking.

Q124. On HRCT, asbestosis is distinguished from silicosis by:
A) Eggshell nodal calcification
B) A miliary pattern
C) Basal subpleural reticulation rather than upper-zone nodules
D) Lobar consolidation

Answer: C) Basal subpleural reticulation rather than upper-zone nodules

Explanation: Asbestosis shows basal, subpleural reticulation and honeycombing, contrasting with the upper-zone rounded nodules and eggshell calcification of silicosis.

Q125. To differentiate mesothelioma from metastatic adenocarcinoma, the key tool is:
A) Plain chest radiograph
B) Immunohistochemistry on biopsy tissue
C) Serum CEA alone
D) Tuberculin testing

Answer: B) Immunohistochemistry on biopsy tissue

Explanation: A panel of mesothelial versus epithelial markers on adequate tissue distinguishes the two; imaging and single serum markers cannot.

Q126. Pleural plaques on imaging in an asymptomatic worker should prompt:
A) Immediate pleurectomy
B) Antitubercular therapy
C) Reassurance with no follow-up
D) Documentation of exposure and counselling/surveillance

Answer: D) Documentation of exposure and counselling/surveillance

Explanation: Plaques are benign but mark exposure, so the appropriate response is exposure documentation, smoking-cessation advice and surveillance.

Q127. A pitfall in diagnosing rounded atelectasis is that it can be mistaken for:
A) A pleural or lung malignancy
B) Simple pneumonia
C) Pulmonary embolism
D) Asthma

Answer: A) A pleural or lung malignancy

Explanation: Rounded atelectasis forms a mass-like lesion that mimics tumour; recognising the comet-tail sign and adjacent pleural thickening avoids unnecessary surgery.

Q128. The presence of bilateral pleural plaques in a patient with new lung fibrosis strongly suggests the fibrosis is:
A) Sarcoidosis
B) Hypersensitivity pneumonitis
C) Asbestosis rather than idiopathic
D) Drug-induced

Answer: C) Asbestosis rather than idiopathic

Explanation: Bilateral plaques are a reliable exposure marker, so coexisting basal fibrosis is most likely asbestosis rather than an idiopathic interstitial pneumonia.

Management

Q129. There is no curative treatment for asbestosis, so management focuses on:
A) Chelation therapy
B) Supportive care, exposure cessation and smoking cessation
C) A short antibiotic course
D) Curative corticosteroids

Answer: B) Supportive care, exposure cessation and smoking cessation

Explanation: Asbestosis is irreversible; management is supportive with removal from exposure, smoking cessation, vaccination, oxygen and rehabilitation.

Q130. An essential preventive intervention for every asbestos-exposed patient is:
A) Increasing fibre intake
B) Routine lung resection
C) Lifelong antibiotics
D) Smoking cessation

Answer: D) Smoking cessation

Explanation: Because smoking multiplies lung-cancer risk in the asbestos-exposed, cessation is the single most valuable intervention offered.

Q131. Treatment of malignant mesothelioma is best described as:
A) Largely palliative, with multimodal options of limited benefit
B) Curative surgery in most cases
C) Cured by antibiotics
D) Reversed by removing asbestos

Answer: A) Largely palliative, with multimodal options of limited benefit

Explanation: Mesothelioma is rarely curable; chemotherapy (pemetrexed-platinum), selected surgery and radiotherapy offer modest, mainly palliative benefit.

Q132. Surveillance of asbestos-exposed workers should include:
A) Daily fibre counts in blood
B) Routine bronchoscopy for all
C) Periodic symptom review, chest imaging and lung function
D) Annual CT angiography

Answer: C) Periodic symptom review, chest imaging and lung function

Explanation: Periodic clinical, radiographic and spirometric review is standard; the distractors are inappropriate or non-existent surveillance methods.

Q133. For a worker found to have pleural plaques only, appropriate management is:
A) Urgent chemotherapy
B) Reassurance about the plaques, smoking cessation and ongoing surveillance
C) Pleurectomy
D) Isolation

Answer: B) Reassurance about the plaques, smoking cessation and ongoing surveillance

Explanation: Plaques themselves need no treatment, but the worker should stop smoking and remain under surveillance for other asbestos-related diseases.

Q134. Oxygen, pulmonary rehabilitation and vaccination in asbestosis are aimed at:
A) Reversing the fibrosis
B) Eliminating retained fibres
C) Curing mesothelioma
D) Symptom relief and preventing complications, not cure

Answer: D) Symptom relief and preventing complications, not cure

Explanation: These supportive measures improve symptoms and reduce complications but do not reverse the established fibrosis or remove fibres.

Prevention & Legislation

Q135. The most effective control for asbestos risk is:
A) Elimination/substitution (banning use and substituting safer materials)
B) Issuing dust masks only
C) Job rotation
D) Annual radiographs

Answer: A) Elimination/substitution (banning use and substituting safer materials)

Explanation: Removing asbestos from use, with safer substitutes, ranks highest in the hierarchy of control; PPE and surveillance are lower-order measures.

Q136. Many countries have responded to asbestos disease by:
A) Encouraging wider use
B) Removing all exposure limits
C) Banning or severely restricting asbestos use
D) Reclassifying it as non-hazardous

Answer: C) Banning or severely restricting asbestos use

Explanation: Numerous countries have banned or tightly restricted asbestos, though chrysotile use continues in some regions, sustaining future disease.

Q137. The OSHA permissible exposure limit for asbestos is expressed as:
A) 50 micrograms per cubic metre
B) 0.1 fibres per cubic centimetre (8-hour TWA)
C) 5 milligrams per cubic metre
D) 1 fibre per litre

Answer: B) 0.1 fibres per cubic centimetre (8-hour TWA)

Explanation: Asbestos limits are counted as fibres per cc of air; the OSHA PEL is 0.1 f/cc as an 8-hour average, reflecting the fibrous hazard.

Q138. Safe removal of asbestos-containing materials requires:
A) Dry demolition with no controls
B) Burning the material
C) Open-air crushing
D) Controlled work, wetting, enclosure and clearance air-monitoring

Answer: D) Controlled work, wetting, enclosure and clearance air-monitoring

Explanation: Licensed removal uses wetting, enclosure, negative pressure and clearance sampling to prevent fibre release; uncontrolled methods are hazardous.

Q139. In India, asbestosis and mesothelioma are:
A) Notifiable and compensable occupational diseases
B) Not recognised legally
C) Compensable only for smokers
D) Reportable only after death

Answer: A) Notifiable and compensable occupational diseases

Explanation: Asbestos-related diseases are listed among notifiable, compensable occupational diseases, with claims handled under the relevant labour statutes.

Q140. Why does asbestos disease continue to appear despite bans?
A) Because bans increase exposure
B) Because the diseases are newly emerging
C) Long latency means past exposures still manifest, and use persists in some countries
D) Because fibres regenerate

Answer: C) Long latency means past exposures still manifest, and use persists in some countries

Explanation: The decades-long latency means historic exposures keep producing cases, and ongoing use in some countries adds new exposures.

Q141. Medical surveillance of asbestos workers primarily aims to:
A) Cure existing disease
B) Detect disease early and document exposure for prevention and compensation
C) Replace engineering controls
D) Increase permissible exposure

Answer: B) Detect disease early and document exposure for prevention and compensation

Explanation: Surveillance detects early disease, informs prevention and supports compensation; it complements, not replaces, exposure control.

Q142. A pre-placement chest radiograph for an asbestos worker mainly serves to:
A) Treat early asbestosis
B) Replace the need for fibre controls
C) Diagnose mesothelioma early
D) Establish a baseline and detect pre-existing disease

Answer: D) Establish a baseline and detect pre-existing disease

Explanation: A baseline film allows future comparison and identifies pre-existing disease; it neither treats disease nor substitutes for exposure control.

Q143. Engineering control of asbestos dust during unavoidable work relies chiefly on:
A) Wetting and local exhaust ventilation/enclosure
B) Issuing respirators alone
C) Job rotation alone
D) Posting warning signs alone

Answer: A) Wetting and local exhaust ventilation/enclosure

Explanation: Wet methods and LEV/enclosure control fibre release at source; PPE, rotation and signage are lower-order or supplementary measures.

Q144. The principle that there is 'no safe level' is applied most strictly to:
A) Noise-induced hearing loss
B) Heat stress
C) Mesothelioma risk from asbestos
D) Vibration white finger

Answer: C) Mesothelioma risk from asbestos

Explanation: Because mesothelioma lacks a demonstrable threshold, asbestos policy assumes no safe level; the other hazards have recognised exposure limits.

Q145. Substitution of asbestos in products has used materials such as:
A) More crocidolite
B) Man-made mineral/vitreous fibres and other synthetic fibres
C) Tremolite-rich talc
D) Pure quartz

Answer: B) Man-made mineral/vitreous fibres and other synthetic fibres

Explanation: Synthetic fibres and other substitutes replaced asbestos; substituting another amphibole or silica would reintroduce hazard.

Q146. Clearance air-monitoring after asbestos removal is performed to:
A) Increase ventilation permanently
B) Detect silica
C) Measure noise
D) Confirm airborne fibre levels are acceptable before re-occupation

Answer: D) Confirm airborne fibre levels are acceptable before re-occupation

Explanation: Clearance sampling verifies that fibre concentrations have fallen to acceptable levels before the area is reoccupied.

Q147. Worker education for asbestos exposure should emphasise:
A) Smoking cessation and correct use of controls and PPE
B) That asbestos is harmless if curly
C) That masks alone eliminate all risk
D) Ignoring respiratory symptoms

Answer: A) Smoking cessation and correct use of controls and PPE

Explanation: Education stresses smoking cessation (because of synergy), proper engineering controls and PPE use, and prompt reporting of symptoms.

Imaging, Spirometry & Signature Findings

Q148. The characteristic chest X-ray pattern of asbestosis is:
A) Upper-zone rounded nodules
B) Lower-zone irregular (reticular) opacities, often with pleural plaques
C) Eggshell hilar calcification
D) A miliary pattern

Answer: B) Lower-zone irregular (reticular) opacities, often with pleural plaques

Explanation: Asbestosis shows basal small irregular opacities (ILO s/t/u) commonly with pleural plaques, the mirror image of upper-zone silicosis.

Q149. The spirometry pattern in asbestosis is typically:
A) Obstructive with raised DLCO
B) Normal in advanced disease
C) Pure air-trapping
D) Restrictive with a reduced DLCO

Answer: D) Restrictive with a reduced DLCO

Explanation: Asbestosis is an interstitial fibrosis causing restriction (low FVC/TLC, normal/raised ratio) with an early fall in diffusing capacity.

Q150. A signature microscopic finding indicating asbestos exposure is the:
A) Ferruginous (asbestos) body
B) Silicotic nodule
C) Coal macule
D) Caseating granuloma

Answer: A) Ferruginous (asbestos) body

Explanation: The iron-coated golden-brown ferruginous body marks asbestos exposure; the distractors are lesions of other dust diseases.

Q151. The most common radiographic marker of asbestos exposure overall is:
A) Eggshell calcification
B) Cavitation
C) Pleural plaques
D) A solitary nodule

Answer: C) Pleural plaques

Explanation: Pleural plaques (parietal, often calcified, sparing angles and apices) are the commonest asbestos-related radiographic finding.

Q152. A unilateral nodular pleural rind encasing the lung with a bloody effusion suggests:
A) A benign plaque
B) Malignant mesothelioma
C) Simple silicosis
D) Siderosis

Answer: B) Malignant mesothelioma

Explanation: A nodular circumferential pleural rind with haemorrhagic effusion in an asbestos-exposed person is the signature picture of mesothelioma.

Q153. Finger clubbing accompanying basal crackles in an asbestos worker points to:
A) Simple silicosis
B) Stannosis
C) Baritosis
D) Asbestosis

Answer: D) Asbestosis

Explanation: Clubbing with bibasal velcro crackles and restriction characterises asbestosis, unlike silicosis and the benign metal pneumoconioses.

2

UNIT I — PNEUMOCONIOSES

Coal Workers' Pneumoconiosis

TOPICS IN THIS CHAPTER

Coal Dust, Rank & Exposure

Q1. Coal workers' pneumoconiosis is caused by the inhalation and retention of:
A) Pure crystalline silica only
B) Asbestos fibres
C) Coal mine dust
D) Cotton dust

Answer: C) Coal mine dust

Explanation: CWP results from inhaled coal mine dust; silica may coexist but the disease is defined by coal dust, not asbestos or organic dusts.

Q2. The risk of coal workers' pneumoconiosis rises with higher coal 'rank' because higher-rank coal has:
A) Greater carbon content and is more fibrogenic
B) More water
C) Larger non-respirable particles
D) No carbon

Answer: A) Greater carbon content and is more fibrogenic

Explanation: Higher-rank coals such as anthracite have high carbon content and are more fibrogenic, so anthracite mining carries the greatest CWP risk.

Q3. The coal of highest rank, associated with the greatest pneumoconiosis risk, is:
A) Lignite
B) Peat
C) Bituminous (low rank)
D) Anthracite

Answer: D) Anthracite

Explanation: Anthracite is the highest-rank, highest-carbon coal and carries the greatest CWP risk; lignite and peat are low-rank with lower risk.

Q4. Within a coal mine, the highest dust exposures classically occur at the:
A) Surface offices
B) Coal face during cutting
C) Canteen
D) Lamp room

Answer: B) Coal face during cutting

Explanation: Mechanised cutting at the coal face generates the highest respirable dust concentrations; non-production areas carry minimal exposure.

Q5. The respirable fraction of coal dust relevant to CWP consists of particles approximately:
A) Above 50 micrometres
B) Above 100 micrometres
C) Below 5 micrometres
D) Exactly 1 millimetre

Answer: C) Below 5 micrometres

Explanation: Only particles in the respirable range (broadly under about 5 micrometres) reach the gas-exchanging regions; larger particles are filtered out proximally.

Q6. Quartz (silica) content of coal mine dust matters because it:
A) Increases fibrogenicity and the risk of rapidly progressive disease
B) Makes the dust harmless
C) Only affects skin
D) Prevents pneumoconiosis

Answer: A) Increases fibrogenicity and the risk of rapidly progressive disease

Explanation: Silica is more fibrogenic than carbon, so a high quartz content accelerates disease and raises the risk of PMF and silicosis-like change.

Q7. The latency between starting coal mining and developing simple CWP is generally:
A) A few days
B) A few weeks
C) Always under one year
D) Many years (often 10 or more)

Answer: D) Many years (often 10 or more)

Explanation: CWP develops after prolonged exposure, typically a decade or more of underground work; very short latencies are not typical of simple disease.

Q8. Compared with the coal face, drivage through rock (e.g. tunnelling) tends to expose miners to:
A) No dust at all
B) More silica (quartz)
C) Only organic dust
D) Asbestos fibres

Answer: B) More silica (quartz)

Explanation: Cutting through rock strata raises the quartz fraction of the dust, increasing the silica hazard relative to cutting coal alone.

Q9. 'Anthracosis' refers to:
A) Malignant lung disease
B) A bacterial infection
C) Asymptomatic black pigment deposition in the lung without significant fibrosis
D) An allergic alveolitis

Answer: C) Asymptomatic black pigment deposition in the lung without significant fibrosis

Explanation: Anthracosis is benign carbon-pigment accumulation (also seen in urban dwellers and smokers) without the fibrosis that characterises CWP.

Q10. The disease burden of CWP in a workforce depends chiefly on:
A) Cumulative dust exposure and coal rank
B) Worker height
C) Ambient temperature
D) Shift colour coding

Answer: A) Cumulative dust exposure and coal rank

Explanation: Cumulative respirable dust dose and the rank/silica content of the coal are the principal determinants of CWP prevalence and severity.

Q11. Anthracosis without fibrosis is commonly found at autopsy in:
A) Only coal miners
B) Only children
C) Only asbestos workers
D) Urban dwellers and smokers

Answer: D) Urban dwellers and smokers

Explanation: Carbon-pigment anthracosis is common in city dwellers and smokers and is benign; CWP requires the fibrotic tissue reaction to coal mine dust.

Q12. Coal rank, from lowest to highest carbon content, runs:
A) Anthracite, peat, lignite, bituminous
B) Peat, lignite, bituminous, anthracite
C) Lignite, anthracite, peat, bituminous
D) Bituminous, anthracite, peat, lignite

Answer: B) Peat, lignite, bituminous, anthracite

Explanation: Rank rises from peat through lignite and bituminous to anthracite; higher rank means higher carbon and greater fibrogenic potential.

Deposition & Clearance

Q13. Coal dust deposited in the alveoli is cleared principally by:
A) The renal tubules
B) Gastric acid
C) Alveolar macrophages
D) Hepatic metabolism

Answer: C) Alveolar macrophages

Explanation: Alveolar macrophages engulf deposited particles and clear them via the mucociliary escalator and lymphatics; overload leads to retention.

Q14. CWP develops when dust deposition:
A) Overwhelms the lung's clearance capacity
B) Is fully cleared each day
C) Never reaches the alveoli
D) Is confined to the nose

Answer: A) Overwhelms the lung's clearance capacity

Explanation: Disease follows when the dust burden exceeds clearance, so particles and dust-laden macrophages accumulate and trigger the tissue response.

Q15. Coal dust retained in the lung is relatively:
A) Rapidly dissolved
B) Excreted in urine
C) Converted to silica
D) Biopersistent, being poorly soluble

Answer: D) Biopersistent, being poorly soluble

Explanation: Carbonaceous coal dust is poorly soluble and persists in tissue, sustaining the macrophage response and pigment accumulation over years.

Q16. Dust-laden macrophages in CWP tend to accumulate around the:
A) Pleural plaques
B) Respiratory bronchioles
C) Hilar cartilage
D) Vocal cords

Answer: B) Respiratory bronchioles

Explanation: Macrophages laden with coal collect around respiratory bronchioles, forming the coal macule, the primary lesion of simple CWP.

Q17. Impaired clearance in CWP can be worsened by concurrent:
A) Regular exercise
B) Adequate hydration
C) Cigarette smoking
D) Vaccination

Answer: C) Cigarette smoking

Explanation: Smoking impairs mucociliary clearance and adds airway inflammation, compounding dust retention and obstructive impairment in coal miners.

Q18. Some retained coal dust reaches regional lymph nodes via:
A) Lymphatic drainage
B) The portal vein
C) Cerebrospinal fluid
D) The bile ducts

Answer: A) Lymphatic drainage

Explanation: Particles and dust-laden macrophages drain through pulmonary lymphatics to hilar nodes, which become pigmented and may calcify.

Q19. The mucociliary escalator clears coal dust deposited in the:
A) Renal pelvis
B) Pleural space
C) Hilar cartilage
D) Conducting airways above the alveoli

Answer: D) Conducting airways above the alveoli

Explanation: Particles landing on ciliated conducting airways are swept up the mucociliary escalator; alveolar dust relies on slower macrophage clearance.

Q20. Overload of alveolar macrophage clearance leads to:
A) Complete recovery
B) Dust accumulation and the tissue reaction of CWP
C) Immediate malignancy
D) Pleural plaque formation

Answer: B) Dust accumulation and the tissue reaction of CWP

Explanation: When the dust burden exceeds macrophage clearance capacity, particles accumulate and provoke the macule formation and fibrosis of CWP.

Pathogenesis

Q21. The primary cellular event in CWP is:
A) Direct viral infection of pneumocytes
B) IgE-mediated mast-cell degranulation
C) Ingestion of coal dust by alveolar macrophages with release of mediators
D) Autoantibody attack on basement membrane

Answer: C) Ingestion of coal dust by alveolar macrophages with release of mediators

Explanation: Macrophages ingest coal dust and, when overloaded, release oxidants and cytokines that recruit more cells and drive the tissue reaction.

Q22. Reactive oxygen species in CWP arise from both activated macrophages and:
A) Surface-derived radicals on the coal/quartz particles
B) Dietary antioxidants
C) Pulmonary surfactant
D) Inhaled oxygen alone

Answer: A) Surface-derived radicals on the coal/quartz particles

Explanation: Both cellular oxidative burst and particle-surface free radicals (especially from quartz) generate ROS that injure lung tissue.

Q23. Progression from simple CWP to fibrosis is promoted by a higher dust content of:
A) Water
B) Calcium carbonate
C) Iron oxide
D) Silica (quartz)

Answer: D) Silica (quartz)

Explanation: Quartz is far more fibrogenic than carbon, so a higher silica fraction drives collagenous fibrosis and progression toward complicated disease.

Q24. The fibrogenic cytokine central to the fibrosis of progressive massive fibrosis is:
A) Interleukin-2
B) Transforming growth factor-beta
C) Interferon-gamma
D) Immunoglobulin E

Answer: B) Transforming growth factor-beta

Explanation: TGF-beta from activated macrophages stimulates fibroblast collagen synthesis, central to the dense fibrosis of complicated CWP.

Q25. Compared with the silicotic nodule, the simple coal macule is:
A) Composed of dense hyalinised collagen
B) A caseating granuloma
C) Largely non-collagenous (reticulin, not dense collagen)
D) An amyloid deposit

Answer: C) Largely non-collagenous (reticulin, not dense collagen)

Explanation: The coal macule is a non-collagenous dust-and-macrophage lesion with reticulin; the silicotic nodule is dense collagen, a key contrast.

Q26. Focal emphysema in CWP develops:
A) Around coal macules at the respiratory bronchioles
B) Only in the upper airway
C) In the pleura
D) In the hilar nodes

Answer: A) Around coal macules at the respiratory bronchioles

Explanation: Focal (centrilobular-type) emphysema forms around macules at the respiratory bronchioles, contributing to airflow limitation in CWP.

Q27. The role of cigarette smoking in coal miners' lung disease is to:
A) Protect against pneumoconiosis
B) Reverse established fibrosis
C) Have no effect
D) Add airway obstruction and emphysema, worsening impairment

Answer: D) Add airway obstruction and emphysema, worsening impairment

Explanation: Smoking independently causes COPD and emphysema, compounding the dust-related obstruction and accelerating functional decline in miners.

Q28. The coal macule is described as non-collagenous because it consists chiefly of:
A) Hyalinised collagen
B) Dust-laden macrophages and reticulin rather than dense collagen
C) Caseous necrosis
D) Amyloid fibrils

Answer: B) Dust-laden macrophages and reticulin rather than dense collagen

Explanation: The macule is a soft, largely non-collagenous accumulation of dust-laden macrophages with reticulin; dense collagen marks the nodule and PMF.

Q29. Quartz contamination accelerates CWP because silica causes:
A) Less inflammation
B) Rapid clearance
C) More collagenous, fibrotic lesions than carbon alone
D) Pleural calcification only

Answer: C) More collagenous, fibrotic lesions than carbon alone

Explanation: Silica is markedly more fibrogenic than carbon, so dust with high quartz produces collagenous nodules and faster progression to PMF.

Pathology

Q30. The primary lesion of simple coal workers' pneumoconiosis is the:
A) Coal macule
B) Silicotic nodule
C) Ferruginous body
D) Caseating granuloma

Answer: A) Coal macule

Explanation: The coal macule, a focal collection of dust-laden macrophages around respiratory bronchioles, is the defining lesion of simple CWP.

Q31. The coal macule is typically located at the:
A) Pleural surface
B) Hilar cartilage
C) Main bronchus
D) Respiratory bronchiole

Answer: D) Respiratory bronchiole

Explanation: Macules form around the respiratory bronchioles, where deposited dust and macrophages accumulate, often with surrounding focal emphysema.

Q32. A coal 'nodule' differs from a macule in that it:
A) Is purely emphysematous
B) Contains collagen and is palpable/firmer
C) Lacks any dust
D) Is malignant

Answer: B) Contains collagen and is palpable/firmer

Explanation: The coal nodule contains collagen (reflecting some silica effect) and is firmer than the largely non-collagenous macule.

Q33. Progressive massive fibrosis in CWP is defined pathologically as a fibrotic mass exceeding:
A) 1 millimetre
B) 10 centimetres
C) 1 centimetre
D) 0.1 millimetre

Answer: C) 1 centimetre

Explanation: PMF is a confluent fibrotic mass greater than 1 cm, formed by coalescence of lesions, paralleling the definition used in silicosis.

Q34. The black fluid sometimes expectorated from a cavitating PMF mass is termed:
A) Melanoptysis
B) Haemoptysis
C) Bronchorrhoea
D) Chyloptysis

Answer: A) Melanoptysis

Explanation: Central necrosis of a PMF mass can liquefy and be coughed up as black fluid, called melanoptysis, characteristic of complicated CWP.

Q35. Hilar lymph nodes in CWP are characteristically:
A) Replaced by pus
B) Caseous
C) Filled with amyloid
D) Pigmented with coal dust, sometimes calcified

Answer: D) Pigmented with coal dust, sometimes calcified

Explanation: Dust draining via lymphatics blackens the hilar nodes, which may calcify; caseation or amyloid would indicate different processes.

Q36. Focal emphysema associated with coal macules is best described as:
A) Panlobular and basal
B) Centrilobular-type around respiratory bronchioles
C) Bullous and apical only
D) Paraseptal only

Answer: B) Centrilobular-type around respiratory bronchioles

Explanation: The emphysema of CWP is focal and centrilobular, centred on the macules at respiratory bronchioles, contributing to airflow obstruction.

Q37. Compared with silicotic PMF, coal PMF masses more often show:
A) Eggshell nodal calcification
B) Ferruginous bodies
C) Central necrosis with black (melanoptysis) fluid
D) Non-caseating granulomas

Answer: C) Central necrosis with black (melanoptysis) fluid

Explanation: Coal PMF can liquefy centrally and discharge black fluid (melanoptysis); eggshell calcification and ferruginous bodies belong to silica and asbestos.

Simple CWP

Q38. Simple coal workers' pneumoconiosis is usually:
A) Asymptomatic and detected on chest radiography
B) Rapidly fatal
C) Associated with severe dyspnoea from onset
D) An acute febrile illness

Answer: A) Asymptomatic and detected on chest radiography

Explanation: Simple CWP is typically asymptomatic and found on surveillance films; significant symptoms usually indicate complicating PMF or coexisting disease.

Q39. On the chest radiograph, simple CWP shows:
A) Bilateral pleural plaques
B) Lobar consolidation
C) A miliary cavitating pattern
D) Small rounded opacities, often upper-zone

Answer: D) Small rounded opacities, often upper-zone

Explanation: Simple CWP produces small rounded opacities (graded by the ILO p/q/r scale), commonly in the upper zones, without large masses.

Q40. In the ILO classification, the small opacities of simple CWP are graded by:
A) A/B/C large-opacity grades only
B) Profusion and size (rounded p/q/r)
C) s/t/u irregular grades only
D) NYHA class

Answer: B) Profusion and size (rounded p/q/r)

Explanation: Simple CWP is scored for profusion and the size of small rounded opacities (p/q/r); A/B/C grades apply to the large opacities of PMF.

Q41. The main clinical significance of simple CWP is that it:
A) Is invariably disabling at once
B) Causes immediate respiratory failure
C) Can progress to complicated disease (PMF)
D) Always regresses spontaneously

Answer: C) Can progress to complicated disease (PMF)

Explanation: Simple CWP itself causes little impairment but is important because it may progress to PMF, which carries disability and mortality.

Q42. Lung function in uncomplicated simple CWP is often:
A) Near-normal or only mildly affected
B) Severely restrictive always
C) Severely obstructive always
D) Unmeasurable

Answer: A) Near-normal or only mildly affected

Explanation: Simple CWP usually leaves lung function near-normal; marked impairment suggests PMF, coexisting COPD from smoking, or another disease.

Q43. Simple CWP is distinguished from complicated CWP chiefly by:
A) The presence of pleural plaques
B) A positive tuberculin test
C) Finger clubbing
D) Absence of opacities larger than 1 cm

Answer: D) Absence of opacities larger than 1 cm

Explanation: The presence of large opacities (>1 cm) defines complicated disease (PMF); simple CWP has only small opacities.

Q44. The defining radiographic difference between simple and complicated CWP is the:
A) Presence of pleural effusion
B) Presence of opacities larger than 1 cm in complicated disease
C) Loss of lung volume
D) Hilar lymphadenopathy

Answer: B) Presence of opacities larger than 1 cm in complicated disease

Explanation: Complicated CWP (PMF) is defined by large opacities exceeding 1 cm; simple CWP shows only small rounded opacities.

Q45. A coal miner with category 2 or 3 profusion of small opacities is at:
A) No increased risk
B) Reduced risk of any disease
C) Increased risk of progression to PMF
D) Risk only of asthma

Answer: C) Increased risk of progression to PMF

Explanation: Higher profusion of small opacities is a recognised risk factor for progression to progressive massive fibrosis.

Complicated CWP (PMF)

Q46. Complicated coal workers' pneumoconiosis is synonymous with:
A) Progressive massive fibrosis
B) Simple anthracosis
C) Acute silicoproteinosis
D) Benign pleural effusion

Answer: A) Progressive massive fibrosis

Explanation: Complicated CWP is progressive massive fibrosis, defined by large fibrotic opacities; the distractors are unrelated entities.

Q47. Progressive massive fibrosis in CWP is radiologically defined as an opacity larger than:
A) 1 millimetre
B) 5 millimetres
C) 10 centimetres
D) 1 centimetre

Answer: D) 1 centimetre

Explanation: PMF is a large opacity exceeding 1 cm, formed by coalescence of smaller lesions; this threshold mirrors that used in silicosis.

Q48. PMF masses in CWP characteristically arise in the:
A) Lower zones and bases
B) Upper zones, often migrating toward the hila
C) Costophrenic angles
D) Anterior mediastinum
Answer: B) Upper zones, often migrating toward the hila
Explanation: Like silicotic PMF, coal PMF forms in the upper zones and migrates centrally over time, leaving peripheral emphysema.

Q49. Unlike simple CWP, complicated CWP (PMF) typically causes:
A) No symptoms ever
B) Only skin changes
C) Progressive dyspnoea and significant disability
D) Acute fever and rash
Answer: C) Progressive dyspnoea and significant disability
Explanation: PMF produces breathlessness, cough and progressive functional loss, in contrast to the usually asymptomatic simple disease.

Q50. The pulmonary function pattern of established PMF in CWP is typically:
A) Mixed restrictive and obstructive
B) Pure restrictive with raised DLCO
C) Entirely normal
D) Pure obstructive with raised DLCO
Answer: A) Mixed restrictive and obstructive
Explanation: Conglomerate fibrosis reduces volumes while associated focal emphysema adds obstruction, giving a mixed defect with reduced gas transfer.

Q51. A PMF mass that develops central cavitation should prompt evaluation for:
A) Simple anthracosis
B) A benign cyst
C) Normal ageing
D) Tuberculosis
Answer: D) Tuberculosis
Explanation: New cavitation within a PMF mass raises concern for superimposed tuberculosis (and occasionally necrosis with melanoptysis), needing investigation.

Q52. Progressive massive fibrosis in CWP can:
A) Only progress during active exposure
B) Progress even after exposure has ceased
C) Always regress after retirement
D) Resolve within weeks
Answer: B) Progress even after exposure has ceased
Explanation: Retained dust continues to drive fibrosis, so PMF may advance after leaving the industry, an important counselling and compensation point.

Q53. The usual terminal complication of advanced PMF is:
A) Nephrotic syndrome
B) Portal hypertension
C) Respiratory failure and cor pulmonale
D) Constrictive pericarditis
Answer: C) Respiratory failure and cor pulmonale
Explanation: Progressive loss of functioning lung and chronic hypoxaemia cause pulmonary hypertension and right heart failure, the common terminal pathway.

Q54. Risk factors for progression of simple CWP to PMF include all EXCEPT:
A) Low cumulative dust burden
B) High profusion of small opacities
C) High silica content of dust
D) Coexisting tuberculosis
Answer: A) Low cumulative dust burden
Explanation: A higher profusion, greater silica content and superimposed TB promote PMF; a low dust burden is associated with lower, not higher, risk.

Q55. The fibrotic mass of coal PMF is composed of:
A) Pure caseous material
B) Amyloid
C) Mucinous fluid only
D) Dense collagen with abundant coal pigment
Answer: D) Dense collagen with abundant coal pigment
Explanation: Coal PMF masses are dense collagenous fibrosis heavily laden with coal pigment, sometimes with central necrosis producing black fluid.

Q56. Management of PMF in CWP is:
A) Curative corticosteroids
B) Supportive, since no treatment reverses it
C) Chelation of carbon
D) A short antibiotic course

Answer: B) Supportive, since no treatment reverses it

Explanation: PMF is irreversible; care is supportive with removal from exposure, smoking cessation, treatment of TB and complications, oxygen and rehabilitation.

Q57. Compared with silicotic PMF, the tuberculosis risk in coal PMF is:
A) Higher than in silicosis
B) Absent
C) Increased, though generally less than in silicosis
D) Identical to the general population

Answer: C) Increased, though generally less than in silicosis

Explanation: Coal PMF carries an increased TB risk, but the association is generally weaker than the very strong silica-tuberculosis link.

Caplan Syndrome

Q58. Caplan syndrome is the association of pneumoconiosis with:
A) Rheumatoid arthritis and large necrobiotic nodules
B) Systemic sclerosis
C) Ankylosing spondylitis
D) Gout

Answer: A) Rheumatoid arthritis and large necrobiotic nodules

Explanation: Caplan syndrome is rheumatoid pneumoconiosis: coal (or silica) dust exposure plus rheumatoid arthritis producing large necrobiotic lung nodules.

Q59. Caplan syndrome was originally described in:
A) Asbestos textile workers
B) Cotton mill workers
C) Foundry moulders
D) Welsh coal miners

Answer: D) Welsh coal miners

Explanation: Anthony Caplan described the rheumatoid pneumoconiotic nodules in Welsh coal miners who also had rheumatoid arthritis, giving the syndrome its name.

Q60. The pulmonary nodules of Caplan syndrome are characteristically:
A) Single and calcified
B) Multiple, well-defined and may cavitate
C) Confined to the pleura
D) Always under 1 mm

Answer: B) Multiple, well-defined and may cavitate

Explanation: Caplan nodules are multiple, well-defined, rounded lesions that can appear in crops and sometimes cavitate, distinct from ordinary CWP opacities.

Q61. Histologically, Caplan nodules resemble:
A) Caseating tuberculous granulomas
B) Silicotic onion-skin nodules
C) Rheumatoid (necrobiotic) nodules with dust
D) Asbestos bodies

Answer: C) Rheumatoid (necrobiotic) nodules with dust

Explanation: The nodules show rheumatoid necrobiotic features with central necrosis and surrounding palisaded cells, admixed with dust pigment.

Q62. The serology usually associated with Caplan syndrome is:
A) Positive rheumatoid factor
B) Positive anti-GBM antibody
C) Positive anti-dsDNA only
D) Negative for all autoantibodies

Answer: A) Positive rheumatoid factor

Explanation: Rheumatoid factor is typically positive, reflecting the underlying rheumatoid arthritis that drives the necrobiotic pulmonary nodules of the syndrome.

Q63. Caplan syndrome can occur in workers exposed to:
A) Only asbestos
B) Only cotton dust
C) Only welding fumes
D) Coal or silica dust

Answer: D) Coal or silica dust

Explanation: Caplan syndrome is recognised in coal and silica (and other) dust exposures combined with rheumatoid arthritis, not exclusively coal.

Q64. A coal miner with rheumatoid arthritis who develops multiple rounded cavitating lung nodules most likely has:
A) Metastatic carcinoma
B) Caplan syndrome
C) Miliary tuberculosis
D) Simple anthracosis

Answer: B) Caplan syndrome

Explanation: The combination of rheumatoid arthritis, dust exposure and multiple rounded (sometimes cavitating) nodules is characteristic of Caplan syndrome.

Q65. A distinguishing radiographic feature of Caplan nodules is that they:
A) Are always solitary and stable
B) Only appear in the pleura
C) Appear relatively rapidly, often in crops, and may cavitate
D) Never change over time

Answer: C) Appear relatively rapidly, often in crops, and may cavitate

Explanation: Caplan nodules can appear quickly in crops and may cavitate, unlike the slowly accumulating small opacities of ordinary CWP.

Q66. Caplan syndrome indicates an interaction between dust exposure and:
A) An abnormal (rheumatoid) immune response
B) An allergic IgE response
C) A bacterial infection
D) A clotting disorder

Answer: A) An abnormal (rheumatoid) immune response

Explanation: The syndrome reflects a modified tissue reaction to dust in the setting of rheumatoid immunological activity, producing necrobiotic nodules.

Radiology & ILO

Q67. The standardised system for classifying pneumoconiosis radiographs is the:
A) NYHA classification
B) TNM staging
C) Glasgow scale
D) ILO classification

Answer: D) ILO classification

Explanation: The International Labour Office (ILO) system grades pneumoconiosis films by profusion, size and zone of opacities for reproducible reporting.

Q68. Small rounded opacities in the ILO system are graded:
A) s, t, u
B) p, q, r by diameter
C) A, B, C
D) 0, I, II, III only

Answer: B) p, q, r by diameter

Explanation: Rounded opacities are graded p (up to 1.5 mm), q (>1.5-3 mm) and r (>3-10 mm); s/t/u are irregular and A/B/C are large opacities.

Q69. Large opacities (PMF) in the ILO system are graded:
A) p, q, r
B) s, t, u
C) A, B, C by size
D) I, II, III

Answer: C) A, B, C by size

Explanation: Large opacities are graded A, B or C by combined size; A is >1 cm up to 50 mm, with B and C progressively larger.

Q70. ILO 'profusion' refers to:
A) The concentration of small opacities per zone
B) The size of large masses
C) The degree of calcification
D) The number of pleural plaques

Answer: A) The concentration of small opacities per zone

Explanation: Profusion records how many small opacities are present per lung zone, graded on a 12-point scale refining the major 0-3 categories.

Q71. ILO classification is performed by comparing the film with:
A) The patient's old ECG
B) A CT angiogram
C) A ventilation scan
D) A set of standard reference radiographs

Answer: D) A set of standard reference radiographs

Explanation: Readers compare the chest film against standard ILO reference radiographs to assign profusion and opacity grades reproducibly.

Q72. HRCT in CWP is more sensitive than plain radiography for detecting:
A) Only rib fractures
B) Early nodules, masses and emphysema
C) Pleural fluid only
D) Cardiac size only

Answer: B) Early nodules, masses and emphysema
Explanation: HRCT detects small nodules, early coalescence into masses and associated emphysema before they are evident on plain films.

Q73. The small opacities of simple CWP predominate in which zones?
A) Lower zones only
B) Costophrenic angles
C) Upper zones
D) Cardiophrenic angles

Answer: C) Upper zones
Explanation: Like silicosis, the rounded opacities of CWP tend to predominate in the upper zones, helping distinguish them from basal asbestosis.

Q74. A progressive increase in opacity size from category A toward C indicates:
A) Worsening progressive massive fibrosis
B) Resolution of disease
C) A normal variant
D) An artefact only

Answer: A) Worsening progressive massive fibrosis
Explanation: Enlargement of large opacities through A, B and C reflects advancing PMF with greater conglomerate fibrosis and worse prognosis.

Q75. In the ILO system, an irregular small opacity is graded with the letters:
A) p, q, r
B) A, B, C
C) 0, 1, 2
D) s, t, u

Answer: D) s, t, u
Explanation: Irregular (linear/reticular) small opacities are graded s, t and u by width; p/q/r are rounded and A/B/C are large opacities.

Q76. ILO large-opacity category C denotes a mass:
A) Smaller than 1 cm
B) Larger than category B (extensive PMF)
C) Equal to a small rounded opacity
D) Confined to one rib space

Answer: B) Larger than category B (extensive PMF)
Explanation: Category C is the largest grade of large opacity, indicating extensive progressive massive fibrosis with the worst functional impact.

Function & Complications

Q77. Simple CWP typically produces:
A) Severe restriction always
B) Severe obstruction always
C) Little or no functional impairment
D) An unmeasurable defect

Answer: C) Little or no functional impairment
Explanation: Uncomplicated simple CWP usually leaves lung function near-normal; significant impairment points to PMF, coexisting COPD or other disease.

Q78. Coal mine dust exposure is now recognised as a cause of:
A) Chronic obstructive pulmonary disease, independent of smoking
B) Only restrictive disease
C) Asthma exclusively
D) No functional change

Answer: A) Chronic obstructive pulmonary disease, independent of smoking
Explanation: Coal dust independently causes COPD and emphysema, so airflow obstruction in miners is not solely attributable to smoking.

Q79. The functional defect of complicated CWP (PMF) is characteristically:
A) Pure restrictive with raised DLCO
B) Normal
C) Isolated raised DLCO
D) Mixed restrictive and obstructive with reduced DLCO

Answer: D) Mixed restrictive and obstructive with reduced DLCO
Explanation: PMF combines restriction from fibrosis with obstruction from focal emphysema, producing a mixed defect and impaired gas transfer.

Q80. Cor pulmonale in advanced CWP results from:
A) Direct coal toxicity to the heart muscle
B) Chronic hypoxaemia and fibrosis causing pulmonary hypertension
C) Pericardial coal deposits
D) Coronary embolism by dust

Answer: B) Chronic hypoxaemia and fibrosis causing pulmonary hypertension

Explanation: Progressive fibrosis and hypoxaemia raise pulmonary vascular resistance, leading to pulmonary hypertension and right ventricular failure.

Q81. Which is a recognised association/complication of CWP?
A) Mesothelioma
B) Byssinosis
C) Progressive massive fibrosis and Caplan syndrome
D) Asbestos bodies

Answer: C) Progressive massive fibrosis and Caplan syndrome

Explanation: PMF and Caplan syndrome are complications of CWP; mesothelioma and asbestos bodies relate to asbestos, and byssinosis to cotton dust.

Q82. Focal emphysema around coal macules contributes to:
A) Airflow obstruction
B) Improved gas exchange
C) Pleural calcification
D) Increased lung elasticity

Answer: A) Airflow obstruction

Explanation: Centrilobular focal emphysema at the macules destroys airspace architecture, adding an obstructive component and impairing gas exchange.

Q83. Compared with silicosis, the tuberculosis risk in coal workers' pneumoconiosis is:
A) Greater than in silicosis
B) Identical to the general population
C) Reduced below baseline
D) Increased but generally less marked than in silicosis

Answer: D) Increased but generally less marked than in silicosis

Explanation: CWP carries an increased TB risk, but the association is weaker than the strong silica-tuberculosis link seen in silicosis.

Q84. Smoking in a coal miner principally adds:
A) Protection from pneumoconiosis
B) Further airflow obstruction and emphysema
C) Reversal of fibrosis
D) A reduced lung cancer risk

Answer: B) Further airflow obstruction and emphysema

Explanation: Smoking compounds dust-related obstruction with its own emphysema and COPD, accelerating functional decline; it is never protective.

Q85. The contribution of coal dust to COPD in miners is now regarded as:
A) Entirely due to smoking
B) Protective against COPD
C) Independent of, and additive to, smoking
D) Negligible

Answer: C) Independent of, and additive to, smoking

Explanation: Coal mine dust independently causes airflow obstruction and emphysema, adding to any smoking-related COPD rather than being explained by it.

Q86. Progressive breathlessness with a large upper-zone opacity in an ex-miner suggests:
A) Complicated CWP (PMF), once TB and cancer are excluded
B) Simple anthracosis
C) Pleural plaque
D) Occupational asthma

Answer: A) Complicated CWP (PMF), once TB and cancer are excluded

Explanation: A large upper-zone mass with progressive dyspnoea in a coal miner indicates PMF, after excluding tuberculosis and carcinoma.

Diagnosis & Differential

Q87. The diagnosis of CWP rests chiefly on:
A) A single serum biomarker
B) Sputum culture
C) Skin patch testing
D) Occupational dust-exposure history with characteristic radiography

Answer: D) Occupational dust-exposure history with characteristic radiography

Explanation: A compatible coal-mining history plus typical ILO-graded radiographic changes establishes CWP; no specific blood test is diagnostic.

Q88. Simple CWP must be radiologically distinguished from:
A) Asbestos pleural plaques
B) Silicosis and other nodular pneumoconioses
C) Lobar pneumonia
D) Pulmonary embolism

Answer: B) Silicosis and other nodular pneumoconioses

Explanation: Upper-zone small rounded opacities of CWP resemble silicosis; exposure history and dust content help distinguish them from other patterns.

Q89. PMF in CWP must be differentiated from:
A) Simple anthracosis
B) Pleural plaques
C) Lung cancer and tuberculosis
D) Asthma

Answer: C) Lung cancer and tuberculosis

Explanation: A large opacity can mimic carcinoma or a tuberculous mass; biopsy or follow-up may be needed when features are atypical.

Q90. A feature favouring CWP over asbestosis on imaging is:
A) Upper-zone rounded opacities without pleural plaques
B) Basal reticulation with plaques
C) Honeycombing at the bases
D) Diffuse pleural thickening

Answer: A) Upper-zone rounded opacities without pleural plaques

Explanation: CWP gives upper-zone rounded opacities and lacks the basal reticulation and pleural plaques characteristic of asbestos-related disease.

Q91. Lung biopsy in CWP is:
A) Mandatory in every case
B) The first-line investigation
C) Needed before notification
D) Rarely required, diagnosis being clinico-radiological

Answer: D) Rarely required, diagnosis being clinico-radiological

Explanation: History and imaging usually suffice; biopsy is reserved for atypical cases or to exclude malignancy in a suspicious mass.

Q92. New asymmetry, cavitation or rapid change in a coal miner's film should prompt evaluation for:
A) Simple anthracosis
B) Tuberculosis or carcinoma
C) A normal variant
D) Improved health

Answer: B) Tuberculosis or carcinoma

Explanation: CWP is slowly progressive and symmetric; rapid change, asymmetry or cavitation suggests superimposed TB or malignancy and needs investigation.

Q93. Distinguishing coal PMF from silicotic PMF may be aided by:
A) A tuberculin test
B) Serum calcium
C) Occupational history and dust analysis (coal pigment vs silica)
D) Echocardiography

Answer: C) Occupational history and dust analysis (coal pigment vs silica)

Explanation: The exposure history and, if biopsied, the predominant pigment (coal vs silica-associated collagen) help attribute PMF to coal or silica.

Q94. Bilateral upper-zone masses with background small nodules in a coal miner are most consistent with:
A) Complicated CWP (PMF)
B) Metastatic cancer
C) Sarcoidosis
D) Heart failure

Answer: A) Complicated CWP (PMF)

Explanation: Conglomerate upper-zone masses on a background of small rounded opacities in a coal miner are typical of PMF; alternatives need exclusion.

Prevention & Legislation

Q95. The most effective way to prevent CWP is to:
A) Rely on dust masks alone
B) Rotate canteen menus
C) Increase shift length
D) Control respirable coal dust at source

Answer: D) Control respirable coal dust at source

Explanation: Reducing airborne respirable dust by engineering controls (ventilation, water sprays, dust suppression) is the most effective preventive measure.

Q96. Engineering control of coal dust underground includes:
A) Issuing respirators only
B) Water sprays, ventilation and dust suppression at the face
C) Job rotation only
D) Warning posters only

Answer: B) Water sprays, ventilation and dust suppression at the face

Explanation: Wetting, adequate ventilation and dust-suppression at the cutting face control dust at source; PPE and administrative measures are secondary.

Q97. Periodic medical surveillance of coal miners centres on:
A) Routine bronchoscopy
B) Annual CT angiography
C) Chest radiography with ILO classification and symptom/spirometry review
D) Daily blood dust counts

Answer: C) Chest radiography with ILO classification and symptom/spirometry review

Explanation: Surveillance uses periodic ILO-classified chest films plus symptom and lung-function review to detect disease early and inform prevention.

Q98. A pre-employment chest radiograph for a new miner mainly serves to:
A) Provide a baseline and detect pre-existing disease
B) Cure early CWP
C) Replace dust controls
D) Diagnose mesothelioma

Answer: A) Provide a baseline and detect pre-existing disease

Explanation: A baseline film allows future comparison and identifies pre-existing lung disease; it does not treat disease or replace exposure control.

Q99. In India, coal workers' pneumoconiosis is:
A) Not legally recognised
B) Compensable only after death
C) Reportable only for smokers
D) A notifiable, compensable occupational disease

Answer: D) A notifiable, compensable occupational disease

Explanation: CWP is among the notifiable, compensable occupational diseases, with statutory medical examination and benefit provisions under labour law.

Q100. Statutory periodic medical examination of workers in a hazardous dust process is the duty of the:
A) Safety Officer
B) Certifying Surgeon / Factory Medical Officer
C) Welfare Officer
D) Mine cook

Answer: B) Certifying Surgeon / Factory Medical Officer

Explanation: Statutory examinations are carried out by the Certifying Surgeon/Factory Medical Officer; the other roles have non-medical duties.

Q101. A key reason CWP continues to occur is:
A) The disease being newly emerging
B) Coal dust regenerating in the lung
C) Inadequate dust control and long disease latency
D) Vaccination failure

Answer: C) Inadequate dust control and long disease latency

Explanation: Persistent inadequate dust control, combined with the long latency of pneumoconiosis, means cases keep appearing despite known prevention.

Q102. Removing a miner from further dust exposure once CWP is detected aims to:
A) Slow progression, since the disease is not reversible
B) Cure the existing fibrosis
C) Reverse all radiographic change
D) Eliminate retained dust

Answer: A) Slow progression, since the disease is not reversible

Explanation: Cessation of exposure cannot reverse established disease but reduces further dust burden and the risk of progression to PMF.

Q103. Respirable dust sampling in mines is used to:
A) Diagnose individual disease
B) Measure noise
C) Assess heat stress
D) Monitor exposure against permissible limits

Answer: D) Monitor exposure against permissible limits

Explanation: Personal and area respirable-dust sampling checks exposures against limits, guiding controls; it assesses exposure, not individual disease.

Q104. Worker education for coal miners should emphasise:
A) That dust is harmless
B) Smoking cessation and correct use of dust controls and PPE
C) That masks alone remove all risk
D) Ignoring breathlessness

Answer: B) Smoking cessation and correct use of dust controls and PPE

Explanation: Education stresses smoking cessation (added COPD risk), proper engineering controls and PPE use, and prompt reporting of respiratory symptoms.

Q105. The hierarchy of control ranks which measure highest for coal dust?
A) Respirator issue
B) Job rotation
C) Elimination or engineering control of dust at source
D) Health education

Answer: C) Elimination or engineering control of dust at source

Explanation: Eliminating or engineering out the dust hazard ranks highest; administrative measures and PPE are lower-order, less reliable controls.

Q106. Return-to-work decisions after CWP diagnosis should consider:
A) Limiting further dust exposure to reduce progression risk
B) Increasing exposure to build tolerance
C) Ignoring the radiograph
D) Only the worker's age

Answer: A) Limiting further dust exposure to reduce progression risk

Explanation: Because progression depends on cumulative dust, limiting further exposure is central to managing a worker with established CWP.

Imaging, Spirometry & Signature Findings

Q107. The characteristic chest X-ray of simple coal workers' pneumoconiosis is:
A) Lower-zone irregular opacities with plaques
B) Small rounded opacities, upper-zone predominant
C) Eggshell calcification
D) Diffuse ground-glass

Answer: B) Small rounded opacities, upper-zone predominant

Explanation: Simple CWP shows upper-zone small rounded opacities like silicosis; irregular basal opacities and plaques indicate asbestos disease.

Q108. The spirometry in simple CWP is usually:
A) Severely restrictive
B) Severely obstructive
C) Mixed with low DLCO
D) Near-normal

Answer: D) Near-normal

Explanation: Simple CWP causes little functional impairment; marked change suggests PMF, coexisting COPD or another disease.

Q109. Established complicated CWP (PMF) characteristically gives which spirometry pattern?
A) Mixed restrictive-obstructive with reduced DLCO
B) Pure restriction with raised DLCO
C) Normal
D) Pure obstruction with raised DLCO

Answer: A) Mixed restrictive-obstructive with reduced DLCO

Explanation: PMF combines fibrotic restriction with focal emphysema obstruction, producing a mixed defect and impaired gas transfer.

Q110. A signature finding of complicated CWP, reflecting central mass necrosis, is:
A) Eggshell calcification
B) Ferruginous bodies
C) Melanoptysis (black sputum)
D) Non-caseating granuloma

Answer: C) Melanoptysis (black sputum)

Explanation: Liquefied necrotic PMF material coughed up as black fluid (melanoptysis) is characteristic of coal disease.

Q111. The signature lesion underlying the simple CWP X-ray is the:

A) Collagenous silicotic nodule
B) Non-collagenous coal macule at the respiratory bronchiole
C) Ferruginous body
D) Caseating granuloma

Answer: B) Non-collagenous coal macule at the respiratory bronchiole

Explanation: The non-collagenous coal macule is the defining lesion of simple CWP, contrasting with the collagenous silicotic nodule.

Q112. A coal miner with rheumatoid arthritis and multiple rounded, sometimes cavitating nodules has:

A) Mesothelioma
B) Silicotuberculosis
C) Baritosis
D) Caplan syndrome

Answer: D) Caplan syndrome

Explanation: Multiple rounded (sometimes cavitating) nodules with rheumatoid arthritis in a dust-exposed worker indicate Caplan syndrome.

3

UNIT I — PNEUMOCONIOSES

Silica & Silicosis

TOPICS IN THIS CHAPTER

Sources, Forms & Occupations

Q1. Chemically, crystalline silica is:
A) Silicon carbide
B) Silicon dioxide (SiO2)
C) Calcium silicate
D) Magnesium silicate

Answer: B) Silicon dioxide (SiO2)

Explanation: Silica is SiO2. Magnesium silicate is talc, silicon carbide is carborundum, calcium silicate is a different mineral — all plausible silicate traps.

Q2. The commonest and most important crystalline form responsible for silicosis is:
A) Cristobalite
B) Tridymite
C) Amorphous silica
D) Quartz

Answer: D) Quartz

Explanation: Quartz is by far the commonest occupational form. Cristobalite and tridymite are more fibrogenic but less commonly encountered; amorphous silica is much less hazardous.

Q3. Compared with quartz, which forms of silica are MORE fibrogenic:
A) Cristobalite and tridymite
B) Amorphous silica
C) Vitreous (fused) silica
D) Raw diatomaceous earth

Answer: A) Cristobalite and tridymite

Explanation: Heat-formed cristobalite and tridymite are more fibrogenic than quartz. Amorphous and vitreous silica are relatively inert.

Q4. Freshly fractured silica is more toxic than aged dust because its cleaved surface generates:
A) Asbestos (ferruginous) bodies
B) Bacterial endotoxin
C) Reactive oxygen species via surface radicals
D) Ferritin deposits

Answer: C) Reactive oxygen species via surface radicals

Explanation: Newly cleaved silanol surfaces produce free radicals and ROS, enhancing toxicity. The distractors belong to other dust mechanisms.

Q5. The respirable particle size that reaches alveoli to cause silicosis is about:
A) 10 to 15 micrometres
B) 1 to 5 micrometres
C) 20 to 30 micrometres
D) Over 50 micrometres

Answer: B) 1 to 5 micrometres

Explanation: Particles ~1–5 μm penetrate to the alveoli; larger particles are trapped in upper airways and cleared.

Q6. Abrasive (sand) blasting causing very heavy exposure is classically linked to which form of disease:
A) Simple chronic silicosis only
B) Siderosis
C) Byssinosis
D) Acute silicosis (silicoproteinosis)

Answer: D) Acute silicosis (silicoproteinosis)

Explanation: The extreme dust of sandblasting causes acute silicosis. Siderosis is iron, byssinosis is cotton dust.

Q7. In India, slate-pencil workers of Mandsaur and agate grinders of Khambhat are classically at risk of:
A) Silicosis
B) Asbestosis
C) Bagassosis
D) Berylliosis

Answer: A) Silicosis

Explanation: These trades involve high quartz dust. Bagassosis is mouldy sugarcane, berylliosis is beryllium, asbestosis is asbestos.

Q8. Which occupation carries the LEAST crystalline silica exposure:
A) Foundry sand moulding
B) Granite quarrying
C) Aluminium pot-room operation
D) Stone crushing

Answer: C) Aluminium pot-room operation

Explanation: Pot-room work exposes to fluoride and alumina, not silica. The other three are classic high-silica trades.

Q9. An historic outbreak of acute silicosis among tunnellers drilling quartz rock occurred at:
A) Bhopal
B) Hawk's Nest (Gauley Bridge) Tunnel
C) Minamata Bay
D) Yokkaichi

Answer: B) Hawk's Nest (Gauley Bridge) Tunnel
Explanation: The Hawk's Nest Tunnel disaster caused mass acute silicosis. Bhopal (MIC), Minamata (mercury) and Yokkaichi (air pollution) are different events.

Q10. Diatomaceous earth becomes a serious silicosis hazard mainly after:
A) Simple wetting
B) Freezing
C) Magnetisation
D) Calcination, which converts amorphous silica to cristobalite

Answer: D) Calcination, which converts amorphous silica to cristobalite
Explanation: Calcination converts inert amorphous diatomaceous earth into crystalline cristobalite, which is highly fibrogenic; this is why calcined diatomite, unlike the raw material, causes silicosis.

Pathogenesis

Q11. The first cell to phagocytose inhaled silica in the alveolus is the:
A) Alveolar macrophage
B) Type I pneumocyte
C) Circulating neutrophil
D) Mast cell

Answer: A) Alveolar macrophage
Explanation: The alveolar macrophage ingests deposited silica and, once damaged, releases the mediators that begin fibrosis; type I pneumocytes and neutrophils are not the initiating cells.

Q12. Silica kills macrophages chiefly by:
A) Intercalating into nuclear DNA
B) Competitive enzyme inhibition
C) Rupturing the phagolysosomal membrane
D) Depleting serum complement

Answer: C) Rupturing the phagolysosomal membrane
Explanation: Silica's silanol surface ruptures the phagolysosome, releasing lysosomal enzymes and the indigestible particle and killing the macrophage, so injury is perpetuated rather than cleared.

Q13. Mediators released by silica-laden macrophages that drive fibroblast collagen synthesis include:
A) Histamine and bradykinin only
B) TNF-alpha, IL-1 and fibronectin
C) Surfactant proteins A and D
D) Immunoglobulin E

Answer: B) TNF-alpha, IL-1 and fibronectin
Explanation: These macrophage-derived mediators recruit and activate fibroblasts to lay down collagen; histamine, surfactant proteins and immunoglobulin E play no part in the fibrogenic cascade.

Q14. The surface chemical groups on quartz implicated in membrane toxicity are:
A) Sulfhydryl groups
B) Carboxyl groups
C) Amino groups
D) Silanol groups

Answer: D) Silanol groups
Explanation: Surface silanol (Si-OH) groups hydrogen-bond to and disrupt cell and lysosomal membranes; sulfhydryl, carboxyl and amino groups are not the toxic moiety on quartz.

Q15. Silica-induced fibrosis persists because silica is:
A) Not biodegradable, so macrophages re-ingest it in a self-sustaining cycle
B) Rapidly dissolved and cleared
C) Neutralised by surfactant
D) Converted to harmless silicate enzymatically

Answer: A) Not biodegradable, so macrophages re-ingest it in a self-sustaining cycle
Explanation: Because silica cannot be digested, it is released when the macrophage dies and re-ingested by fresh macrophages, creating the self-sustaining cycle that makes silicosis progressive.

Q16. The characteristic zonal distribution of silicotic nodules is:
A) Lower-zone predominance
B) Peribronchovascular bases
C) Upper-zone predominance
D) Subpleural lower zones

Answer: C) Upper-zone predominance
Explanation: Silicotic nodules cluster in the upper zones, whereas asbestosis is basal and subpleural; this distribution difference is useful for distinguishing the two pneumoconioses radiologically.

Q17. Acute silicosis differs pathologically from chronic silicosis by showing:
A) Well-formed whorled collagen nodules
B) Alveolar lipoproteinaceous (proteinosis-like) exudate
C) Ferruginous bodies
D) Non-caseating granulomas

Answer: B) Alveolar lipoproteinaceous (proteinosis-like) exudate

Explanation: Acute silicosis fills alveoli with PAS-positive lipoproteinaceous material like alveolar proteinosis; whorled collagen nodules are the chronic lesion and granulomas suggest beryllium disease.

Q18. Silica predisposes to tuberculosis principally by:
A) Causing systemic CD4 depletion like HIV
B) Blocking all antibody production
C) Inducing severe neutropenia
D) Impairing macrophage killing of M. tuberculosis

Answer: D) Impairing macrophage killing of M. tuberculosis

Explanation: Silica disables the alveolar macrophages that normally contain tubercle bacilli, so latent infection reactivates and progresses; this is the basis of the strong silicotuberculosis link.

Q19. The usual latency of classic chronic simple silicosis is:
A) 10 to 30 years
B) Days to weeks
C) 1 to 2 years
D) Over 50 years

Answer: A) 10 to 30 years

Explanation: Chronic (classic) silicosis appears only after one to three decades of moderate exposure; much shorter latencies indicate the accelerated or acute forms instead.

Q20. Accelerated silicosis is characterised by:
A) Onset after a single brief exposure
B) Lower-zone fibrosis
C) Onset in 5 to 10 years with heavier exposure and more autoimmune features
D) Pleural plaques

Answer: C) Onset in 5 to 10 years with heavier exposure and more autoimmune features

Explanation: Accelerated silicosis follows heavier exposure over five to ten years, progressing faster than chronic disease and carrying a notably higher burden of autoimmune complications.

Q21. Silica is a potent activator of which inflammasome, amplifying interleukin-1-beta release?
A) The AIM2 inflammasome
B) The NLRP3 inflammasome
C) The NLRC4 inflammasome
D) The pyrin inflammasome

Answer: B) The NLRP3 inflammasome

Explanation: Crystalline silica activates NLRP3, driving caspase-1 and IL-1-beta. The others are real inflammasomes triggered by different signals (DNA, flagellin, etc.).

Q22. The master profibrotic cytokine driving fibroblast collagen synthesis in silicosis is:
A) Interleukin-2
B) Interferon-gamma
C) Interleukin-4
D) Transforming growth factor-beta (TGF-beta)

Answer: D) Transforming growth factor-beta (TGF-beta)

Explanation: TGF-beta is the principal fibrogenic cytokine. IFN-gamma is largely anti-fibrotic, and IL-2/IL-4 have other roles.

Q23. The increased cytotoxicity of freshly fractured silica is attributed to:
A) Silicon-based surface free radicals generated at cleaved surfaces
B) An adsorbed iron-oxide coat
C) Residual elemental carbon
D) Surface sulfhydryl groups

Answer: A) Silicon-based surface free radicals generated at cleaved surfaces

Explanation: Newly cleaved silica bears reactive Si and SiO radicals that generate ROS; the distractors describe other dusts' surface chemistry.

Q24. Silica-laden macrophages predominantly die by:
A) Caseous necrosis
B) Coagulative infarction
C) Pyroptosis/apoptosis following lysosomal membrane rupture
D) Programmed survival with no cell death

Answer: C) Pyroptosis/apoptosis following lysosomal membrane rupture

Explanation: Lysosomal rupture triggers inflammasome-mediated pyroptosis and apoptosis; caseation is a TB pattern, coagulative necrosis follows ischaemia.

Q25. Mixed-dust fibrosis develops when silica is inhaled together with appreciable amounts of:
A) Pure cotton fibre
B) Iron oxide (as in haematite mining and steel work)
C) Elemental mercury
D) Organic grain dust

Answer: B) Iron oxide (as in haematite mining and steel work)

Explanation: Co-exposure to iron and other minerals modifies the nodular pattern into mixed-dust fibrosis; the distractors cause unrelated diseases.

Q26. The proposed basis of silica-associated autoimmunity is:
A) Molecular mimicry between silica and type IV collagen
B) An IgE-mediated hypersensitivity
C) An inherited complement deficiency
D) An adjuvant-like, non-specific amplification of immune responses

Answer: D) An adjuvant-like, non-specific amplification of immune responses

Explanation: Silica behaves as an immune adjuvant, breaking tolerance; the distractors are mechanisms of other disorders.

Pathology & Histology

Q27. The silicotic nodule is composed of:
A) Central whorled hyalinised collagen with dust-laden macrophages at the periphery
B) Iron-coated dumbbell fibres
C) Foamy lipid-laden macrophages
D) Caseating necrotic centre

Answer: A) Central whorled hyalinised collagen with dust-laden macrophages at the periphery

Explanation: The onion-skin collagen nodule is the histological hallmark. The distractors describe asbestos bodies, lipoid pneumonia and TB respectively.

Q28. Under polarised light, silica particles in lung tissue appear as:
A) Strongly birefringent long fibres
B) Non-birefringent black pigment
C) Weakly birefringent particles
D) Refractile cholesterol clefts

Answer: C) Weakly birefringent particles

Explanation: Silica is weakly birefringent; strongly birefringent fibres suggest asbestos and black non-birefringent pigment suggests coal.

Q29. Eggshell calcification refers to calcification at the:
A) Visceral pleura
B) Rim of hilar and mediastinal lymph nodes
C) Pericardium
D) Bronchial cartilage

Answer: B) Rim of hilar and mediastinal lymph nodes

Explanation: Peripheral 'eggshell' calcification of hilar and mediastinal nodes is a classic, fairly specific sign of silicosis, occurring in roughly five per cent of cases.

Q30. Progressive massive fibrosis in silicosis typically forms in the:
A) Costophrenic angles
B) Lingula only
C) Right middle lobe only
D) Upper zones, often migrating toward the hilum

Answer: D) Upper zones, often migrating toward the hilum

Explanation: Conglomerate masses form in the upper zones and migrate centrally toward the hila over years, leaving emphysematous lung at the periphery; this is the hallmark of PMF.

Q31. A silicotic nodule differs from a coal macule in that it contains:
A) Dense collagen (the macule is essentially non-collagenous)
B) More anthracotic pigment
C) Ferruginous bodies
D) Epithelioid granulomas

Answer: A) Dense collagen (the macule is essentially non-collagenous)

Explanation: The key difference is collagen: the silicotic nodule has a dense hyalinised collagen core, whereas the coal macule is essentially non-collagenous dust-laden tissue.

Q32. Mineralogical confirmation of silica in tissue can use:
A) Ziehl-Neelsen staining
B) Congo red staining
C) Polarised-light microscopy and analytical mineralogy
D) Perls' Prussian blue staining

Answer: C) Polarised-light microscopy and analytical mineralogy

Explanation: Polarised microscopy/analytical methods identify silica; ZN is for AFB, Congo red for amyloid, Perls' for iron.

Q33. The pneumoconiosis that produces non-caseating granulomas mimicking sarcoidosis is:
A) Silicosis
B) Chronic beryllium disease
C) Asbestosis
D) Coal workers' pneumoconiosis

Answer: B) Chronic beryllium disease

Explanation: Chronic beryllium disease produces non-caseating granulomas resembling sarcoidosis, whereas silicosis forms collagenous nodules; the BeLPT confirms beryllium sensitisation when the two are confused.

Q34. On examination, fine basal end-inspiratory crackles in silicosis are:
A) More prominent than in any other pneumoconiosis
B) Pathognomonic of silicosis
C) Always completely absent
D) Less prominent than in asbestosis (silicosis is upper-zone and nodular)

Answer: D) Less prominent than in asbestosis (silicosis is upper-zone and nodular)

Explanation: Basal crackles are characteristic of basal fibrosis (asbestosis); silicosis, being upper-zone nodular, has less prominent crackles.

Q35. The acellular core of the mature silicotic nodule is composed chiefly of:
A) Hyalinised, concentrically arranged collagen
B) Caseous necrotic debris
C) Amyloid deposits
D) Fibrinous exudate

Answer: A) Hyalinised, concentrically arranged collagen

Explanation: The core is dense hyalinised collagen in an onion-skin pattern; caseation suggests TB and amyloid/fibrin are unrelated.

Q36. The peripheral zone of a silicotic nodule characteristically contains:
A) A rim of caseous necrosis
B) Neutrophil microabscesses
C) Dust-laden macrophages and fibroblasts
D) Langhans giant-cell granulomas

Answer: C) Dust-laden macrophages and fibroblasts

Explanation: The nodule's periphery is a rim of active dust-laden macrophages and fibroblasts around the collagen core; caseation, microabscesses or granulomas indicate other diseases.

Q37. The alveolar material of acute silicosis (silicoproteinosis) stains:
A) Congo-red positive
B) PAS-positive
C) Acid-fast positive
D) Mucicarmine positive

Answer: B) PAS-positive

Explanation: The lipoproteinaceous exudate is PAS-positive, as in alveolar proteinosis; the other stains identify amyloid, mycobacteria and mucin.

Clinical Types

Q38. Acute silicosis (silicoproteinosis) develops within:
A) 20 to 40 years
B) Minutes of exposure
C) Only after retirement
D) Months to a few years of intense exposure

Answer: D) Months to a few years of intense exposure

Explanation: Acute silicosis follows overwhelming exposure to fine silica, classically unprotected sandblasting, producing disease within months to a few years rather than the usual decades.

Q39. The clinical course of acute silicosis is:
A) Rapidly progressive and frequently fatal
B) Benign and self-limiting
C) Static for life
D) Fully reversible with bronchodilators

Answer: A) Rapidly progressive and frequently fatal

Explanation: Acute silicosis progresses quickly to respiratory failure and is frequently fatal, unlike the slow chronic form; whole-lung lavage and transplantation are the only options.

Q40. The radiographic appearance of acute silicosis resembles:
A) Upper-zone rounded nodules
B) Bilateral pleural plaques
C) Alveolar filling / ground-glass (proteinosis pattern)
D) Basal honeycombing

Answer: C) Alveolar filling / ground-glass (proteinosis pattern)

Explanation: Acute silicosis shows alveolar filling and ground-glass change, sometimes crazy-paving, rather than the discrete upper-zone rounded nodules that characterise chronic silicosis.

Q41. Simple chronic silicosis is usually:
A) Rapidly fatal
B) Asymptomatic or mildly symptomatic, found on chest radiograph
C) Marked by early finger clubbing
D) Pleuritic from the outset

Answer: B) Asymptomatic or mildly symptomatic, found on chest radiograph

Explanation: Simple silicosis is usually asymptomatic and detected on a surveillance chest radiograph; symptoms and lung-function decline appear only as the disease advances toward complicated forms.

Q42. Progressive massive fibrosis (complicated silicosis) is defined as an opacity exceeding:
A) 3 mm
B) 5 mm
C) 2 cm
D) 1 cm

Answer: D) 1 cm

Explanation: Progressive massive fibrosis is defined as a coalescent opacity exceeding one centimetre, an ILO large opacity; smaller figures are cut-offs for small rounded opacities.

Q43. A silicotic worker developing weight loss, haemoptysis and worsening dyspnoea should first be evaluated for:
A) Tuberculosis
B) Asthma
C) Pulmonary embolism
D) Spontaneous pneumothorax

Answer: A) Tuberculosis

Explanation: Weight loss, fever and haemoptysis in a previously stable silicotic point to superimposed tuberculosis, which must be excluded before attributing decline to silicosis itself.

Q44. Caplan syndrome comprises pneumoconiosis together with:
A) Pleural effusion and ascites
B) Bronchiectasis
C) Rheumatoid pulmonary nodules
D) Emphysematous bullae

Answer: C) Rheumatoid pulmonary nodules

Explanation: Caplan syndrome is pneumoconiosis (silica or coal) combined with rheumatoid arthritis and large necrobiotic pulmonary nodules; effusion, ascites and bronchiectasis are not features.

Q45. Erasmus syndrome describes silica exposure associated with:
A) Rheumatoid arthritis
B) Systemic sclerosis (scleroderma)
C) Ankylosing spondylitis
D) Gout

Answer: B) Systemic sclerosis (scleroderma)

Explanation: Erasmus syndrome denotes the association of silica exposure with systemic sclerosis; silica is also linked to rheumatoid arthritis, lupus and ANCA-associated vasculitis.

Q46. Finger clubbing in a silicotic patient should raise suspicion of:
A) Uncomplicated simple silicosis
B) A benign pleural plaque
C) Allergic rhinitis
D) Complicating lung cancer or tuberculosis

Answer: D) Complicating lung cancer or tuberculosis

Explanation: Clubbing is not a feature of uncomplicated silicosis and warrants a search for malignancy or TB.

Q47. Which feature is characteristically ABSENT in uncomplicated simple silicosis:
A) Finger clubbing
B) Upper-zone nodules
C) A relevant dust exposure history
D) Largely preserved early lung function

Answer: A) Finger clubbing

Explanation: Finger clubbing is not a feature of uncomplicated silicosis; its presence should prompt a search for complicating lung cancer or tuberculosis, the usual causes.

Q48. On HRCT, acute silicosis (silicoproteinosis) characteristically shows a pattern of:
A) Honeycombing
B) Tree-in-bud nodularity
C) Crazy-paving
D) Mosaic attenuation

Answer: C) Crazy-paving

Explanation: Ground-glass with superimposed septal thickening (crazy-paving) reflects alveolar filling; the distractors indicate fibrosis, small-airway disease and air-trapping.

Q49. The zone of the chest radiograph predominantly involved in acute silicosis is the:
A) Upper zones (rounded nodules)
B) Mid and lower zones (alveolar filling)
C) Apices only
D) Costophrenic angles only

Answer: B) Mid and lower zones (alveolar filling)

Explanation: Acute silicosis produces mid- and lower-zone alveolar filling, in contrast to the upper-zone rounded nodules of chronic disease; this is a useful distinguishing feature.

Q50. Of the three forms, the strongest association with autoimmune connective-tissue disease is seen in:
A) Simple chronic silicosis
B) Acute silicosis
C) Mixed-dust fibrosis
D) Accelerated silicosis

Answer: D) Accelerated silicosis

Explanation: Of the three forms, accelerated silicosis carries the heaviest autoimmune burden, reflecting its more intense exposure and faster, more immunologically active course.

Q51. Accelerated silicosis is best characterised by a latency of:
A) 5 to 10 years
B) Less than 1 year
C) 10 to 30 years
D) More than 40 years

Answer: A) 5 to 10 years

Explanation: Accelerated silicosis has a latency of roughly five to ten years, sitting between acute disease (months) and chronic disease (one to three decades).

Radiology & ILO Classification

Q52. Small rounded opacities of silicosis are graded in the ILO system by:
A) The Bhalla score
B) CURB-65
C) Profusion and size (p/q/r)
D) NYHA class

Answer: C) Profusion and size (p/q/r)

Explanation: The ILO system grades small opacities by profusion and size (p/q/r); the Bhalla score, CURB-65 and NYHA class belong to other clinical contexts entirely.

Q53. An ILO small rounded opacity graded 'p' has a diameter:
A) Over 1.5 up to 3 mm
B) Up to 1.5 mm
C) Over 3 up to 10 mm
D) Over 10 mm

Answer: B) Up to 1.5 mm

Explanation: p = up to 1.5 mm; q and r are the larger rounded grades; over 10 mm is a large opacity.

Q54. An ILO small rounded opacity graded 'r' has a diameter:
A) Up to 1.5 mm
B) Over 1.5 up to 3 mm
C) Over 10 mm
D) Over 3 up to 10 mm

Answer: D) Over 3 up to 10 mm

Explanation: Rounded opacity 'r' measures over three up to ten millimetres; p (up to 1.5) and q (over 1.5 to 3) are smaller. These cut-offs must be memorised.

Q55. In the ILO classification, small IRREGULAR opacities are denoted:
A) s, t, u
B) p, q, r
C) A, B, C
D) 0, 1, 2, 3

Answer: A) s, t, u

Explanation: Irregular small opacities are graded s, t and u by width; rounded opacities use p/q/r and large opacities (PMF) use A/B/C in the ILO scheme.

Q56. ILO large opacities (progressive massive fibrosis) are categorised as:
A) 1, 2, 3
B) p, q, r
C) A, B, C by size
D) Mild, moderate, severe

Answer: C) A, B, C by size

Explanation: Large opacities (progressive massive fibrosis) are graded A, B or C by size; small rounded and irregular opacities use the lower-case p/q/r and s/t/u scales.

Q57. 'Profusion' in the ILO classification refers to:
A) The diameter of each opacity
B) The concentration (number per zone) of small opacities
C) Pleural thickness
D) Degree of calcification

Answer: B) The concentration (number per zone) of small opacities

Explanation: Profusion records the concentration (number per zone) of small opacities, not their diameter; opacity size is graded separately by the p/q/r and s/t/u letters.

Q58. Eggshell calcification of hilar nodes occurs in approximately what proportion of silicosis cases:
A) About 90%
B) About 50%
C) 0% (never seen)
D) About 5%

Answer: D) About 5%

Explanation: Eggshell calcification of hilar nodes is a classic but uncommon sign, present in only about five per cent of silicosis cases; its absence does not exclude disease.

Q59. The chest zones first and most involved by silicotic nodules are the:
A) Upper zones
B) Lower zones
C) Perihilar region only
D) Costophrenic angles

Answer: A) Upper zones

Explanation: Silicotic nodules predominate in the upper lung zones; this contrasts with the basal, subpleural distribution of asbestosis and is a key radiological discriminator.

Q60. The most sensitive imaging for early silicotic nodules and PMF is:
A) Plain abdominal radiograph
B) Bronchography
C) High-resolution CT (HRCT)
D) Ventilation-perfusion scan

Answer: C) High-resolution CT (HRCT)

Explanation: High-resolution CT detects early nodules, conglomerate masses and emphysema before they appear on a plain radiograph, making it the most sensitive imaging modality.

Q61. A coalescent upper-zone mass that has migrated centrally leaving a rim of emphysema suggests:
A) Mesothelioma
B) Progressive massive fibrosis
C) Rounded atelectasis
D) A simple pleural plaque

Answer: B) Progressive massive fibrosis

Explanation: Central migration of a conglomerate mass leaving peripheral emphysema is characteristic of progressive massive fibrosis, not mesothelioma, rounded atelectasis or a benign pleural plaque.

Q62. ILO film reading requires comparison of the radiograph against:
A) The patient's prior ECG
B) A CT coronary calcium score
C) Spirometry tracings
D) Standard reference radiographs

Answer: D) Standard reference radiographs

Explanation: ILO classification is performed by comparing the film against standard reference radiographs, ensuring reproducible grading of profusion and opacity size between different readers.

Q63. Dominant pleural plaques on imaging point away from silicosis and toward:
A) Asbestos exposure
B) Coal dust exposure
C) Cotton dust exposure
D) Grain dust exposure

Answer: A) Asbestos exposure

Explanation: Dominant pleural plaques indicate asbestos exposure; silicosis is a parenchymal nodular disease that does not characteristically produce plaques, so their presence redirects the diagnosis.

Q64. An ILO small rounded opacity graded 'q' has a diameter of:
A) Up to 1.5 mm
B) Over 3 up to 10 mm
C) Over 1.5 up to 3 mm
D) Over 10 mm

Answer: C) Over 1.5 up to 3 mm

Explanation: q is the intermediate rounded grade (>1.5-3 mm); p and r flank it and >10 mm is a large opacity.

Q65. ILO profusion of small opacities is recorded on a:
A) Simple present/absent binary
B) 12-point scale (four categories 0-3, each with three subdivisions)
C) Three-point A-B-C scale
D) Five-point NYHA-style scale

Answer: B) 12-point scale (four categories 0-3, each with three subdivisions)

Explanation: Profusion uses a twelve-point scale that subdivides the four major categories (0 to 3), allowing fine grading of small-opacity concentration; A/B/C grades large opacities.

Q66. An ILO large opacity classified as category A has a combined diameter of:
A) Less than 1 cm
B) Greater than the right upper zone
C) Exactly 1 mm
D) Greater than 1 cm and up to 50 mm

Answer: D) Greater than 1 cm and up to 50 mm

Explanation: Large opacity category A spans a combined diameter greater than one centimetre up to fifty millimetres; categories B and C denote progressively larger conglomerate masses.

Q67. Rapidly changing or asymmetric opacities on a silicotic's film should prompt evaluation for:
A) Superimposed tuberculosis or malignancy
B) Simple uncomplicated silicosis
C) Eggshell calcification
D) Normal ageing change

Answer: A) Superimposed tuberculosis or malignancy

Explanation: Silicosis itself is slowly progressive and symmetric; rapid or asymmetric change signals TB or cancer.

Pulmonary Function

Q68. Lung function in advanced complicated silicosis is typically:
A) Pure obstructive with a raised DLCO
B) Entirely normal
C) Restrictive or mixed with a reduced DLCO
D) An isolated rise in TLC

Answer: C) Restrictive or mixed with a reduced DLCO

Explanation: Fibrotic silicosis gives a restrictive defect with reduced DLCO; complicated disease with PMF adds obstruction, producing a mixed pattern rather than a purely obstructive picture.

Q69. In a pure restrictive ventilatory defect the FEV1/FVC ratio is:
A) Reduced below 0.7
B) Normal or increased
C) Exactly 0.5
D) Unmeasurable

Answer: B) Normal or increased

Explanation: Both FEV1 and FVC fall proportionally (or FVC more), so the ratio is preserved or raised.

Q70. In a restrictive defect the total lung capacity (TLC) is:
A) Increased
B) Unchanged by definition
C) Always normal
D) Reduced

Answer: D) Reduced

Explanation: A reduced total lung capacity defines a restrictive defect; in obstructive disease TLC is normal or raised, so falling lung volumes point toward fibrotic restriction.

Q71. Early simple chronic silicosis lung function is usually:
A) Normal or only mildly impaired
B) Severely obstructive
C) Markedly restrictive from the outset
D) Marked by a very high DLCO

Answer: A) Normal or only mildly impaired

Explanation: Early simple silicosis often leaves spirometry near-normal or only mildly impaired; significant functional loss appears as nodules coalesce and complicated disease develops.

Q72. Progressive massive fibrosis tends to add which element to the restrictive picture:
A) A raised DLCO
B) Metabolic alkalosis
C) Airflow obstruction, producing a mixed pattern
D) A pleural effusion

Answer: C) Airflow obstruction, producing a mixed pattern

Explanation: PMF distorts airways and is accompanied by emphysema, adding airflow obstruction to the underlying restriction and producing the mixed defect typical of complicated silicosis.

Q73. In significant interstitial silicosis the diffusing capacity (DLCO) is:
A) Increased
B) Reduced
C) Always normal
D) Not measurable in restriction

Answer: B) Reduced

Explanation: Interstitial thickening impairs gas transfer, reducing the diffusing capacity (DLCO); this is among the earliest functional abnormalities in interstitial lung disease, including silicosis.

Q74. A 'mixed' ventilatory defect in complicated silicosis means:
A) Normal FEV1/FVC with normal TLC
B) An isolated fall in FVC only
C) An isolated fall in the FEV1/FVC ratio only
D) A reduced FEV1/FVC ratio together with a reduced TLC

Answer: D) A reduced FEV1/FVC ratio together with a reduced TLC

Explanation: A mixed defect shows both a reduced FEV1/FVC ratio (obstruction) and a reduced total lung capacity (restriction), the pattern produced when PMF combines fibrosis with emphysema.

Q75. The flow-volume loop in a pure restrictive defect is typically:
A) Tall and narrow with reduced volume
B) Scooped on the expiratory limb
C) Flattened on inspiration
D) Identical to normal

Answer: A) Tall and narrow with reduced volume

Explanation: Restriction narrows the loop and shifts it toward low volumes; a scooped expiratory limb indicates obstruction.

Complications

Q76. Crystalline silica is classified by IARC as a:
A) Group 2A (probable)
B) Group 2B (possible)
C) Group 1 (definite human) carcinogen
D) Group 3 (not classifiable)

Answer: C) Group 1 (definite human) carcinogen

Explanation: Inhaled crystalline silica is an IARC Group 1 (definite) human lung carcinogen; groups 2A, 2B and 3 denote weaker or unproven carcinogenic evidence.

Q77. The malignancy most associated with crystalline silica is:
A) Pleural mesothelioma
B) Lung cancer
C) Bladder cancer
D) Acute leukaemia

Answer: B) Lung cancer

Explanation: Silica causes lung cancer; mesothelioma is linked to asbestos, bladder cancer to aromatic amines and leukaemia to benzene, making each distractor a real but mismatched agent.

Q78. Silicotuberculosis denotes coexistence of silicosis with:
A) Sarcoidosis
B) Histoplasmosis only
C) Aspergilloma only
D) Tuberculosis

Answer: D) Tuberculosis

Explanation: Silicotuberculosis is the well-known coexistence of silicosis and tuberculosis; sarcoidosis, histoplasmosis and aspergilloma are not the classic association tested here.

Q79. Relative to the general population, a silicotic worker's tuberculosis risk is:
A) Markedly increased (several-fold)
B) Unchanged
C) Slightly reduced
D) Effectively abolished

Answer: A) Markedly increased (several-fold)

Explanation: Silica greatly raises tuberculosis susceptibility, increasing risk several-fold; the risk climbs further with the radiographic profusion and severity of the silicosis.

Q80. All silicotic patients should be routinely screened for:
A) Diabetes insipidus
B) Haemochromatosis
C) Tuberculosis
D) G6PD deficiency

Answer: C) Tuberculosis

Explanation: Tuberculosis screening is mandatory in silicosis given the markedly raised risk; the distractors are conditions with no special association with silica exposure.

Q81. Autoimmune conditions linked to silica include all EXCEPT:
A) Systemic sclerosis
B) Ankylosing spondylitis
C) Rheumatoid arthritis
D) ANCA-associated vasculitis

Answer: B) Ankylosing spondylitis

Explanation: Silica is associated with systemic sclerosis, rheumatoid arthritis, lupus and ANCA vasculitis, but not with ankylosing spondylitis, which is the correct exception here.

Q82. A late cardiopulmonary complication of advanced silicosis is:
A) Left ventricular aneurysm
B) Constrictive pericarditis
C) Rheumatic mitral stenosis
D) Cor pulmonale (right heart failure)

Answer: D) Cor pulmonale (right heart failure)

Explanation: Chronic hypoxaemia and fibrosis cause pulmonary hypertension and right ventricular failure (cor pulmonale); the cardiac distractors are unrelated to silicotic lung disease.

Q83. Spontaneous pneumothorax in silicosis usually results from rupture of:
A) Subpleural bullae/emphysema
B) A silicotic lymph node
C) The trachea
D) The oesophagus

Answer: A) Subpleural bullae/emphysema

Explanation: Emphysematous bullae around fibrotic masses can rupture into the pleural space, producing spontaneous pneumothorax; lymph nodes, trachea and oesophagus are not the source.

Q84. A silicotic worker with chronic productive cough and fixed airflow limitation most likely has co-existing:
A) Fully reversible asthma
B) Pure restriction only
C) Chronic bronchitis / COPD
D) Entirely normal airways

Answer: C) Chronic bronchitis / COPD

Explanation: Silica dust, often with smoking, produces chronic bronchitis and fixed airflow obstruction (COPD), adding an obstructive element to the otherwise restrictive silicotic picture.

Q85. Besides M. tuberculosis, silicotics are more prone to infection by:
A) Only respiratory viruses
B) Non-tuberculous (atypical) mycobacteria and fungi
C) No organisms (risk unchanged)
D) Only Gram-negative sepsis

Answer: B) Non-tuberculous (atypical) mycobacteria and fungi

Explanation: Impaired macrophage function raises the risk of non-tuberculous mycobacteria and fungal infection in addition to classic tuberculosis; immunity, not only M. tuberculosis, is affected.

Q86. The mechanism linking silica to autoimmunity is best described as:
A) Direct mutation of B-cell DNA
B) IgE cross-linking on mast cells
C) Inherited complement deficiency
D) Adjuvant-like chronic immune activation

Answer: D) Adjuvant-like chronic immune activation

Explanation: Persistent macrophage activation acts as an immune adjuvant, breaking self-tolerance; this explains the autoantibodies and connective-tissue diseases linked to silica, rather than direct mutation or IgE.

Q87. Progressive massive fibrosis worsens prognosis chiefly through:
A) Progressive respiratory failure and cor pulmonale
B) Distant metastatic spread
C) Isolated secondary polycythaemia
D) Renal failure

Answer: A) Progressive respiratory failure and cor pulmonale

Explanation: PMF causes relentless loss of functioning lung and right heart strain, leading to progressive respiratory failure and cor pulmonale rather than metastasis or renal failure.

Q88. Which is NOT a recognised complication of silicosis:
A) Tuberculosis
B) Lung cancer
C) Pleural mesothelioma
D) Progressive massive fibrosis

Answer: C) Pleural mesothelioma

Explanation: Mesothelioma is an asbestos-related malignancy, not a complication of silica; tuberculosis, lung cancer and progressive massive fibrosis are all genuine silicotic complications.

Q89. Whole-lung lavage is used therapeutically mainly in:
A) Simple chronic silicosis
B) Acute silicosis with an alveolar-proteinosis pattern
C) Pleural plaque disease
D) Byssinosis

Answer: B) Acute silicosis with an alveolar-proteinosis pattern

Explanation: Whole-lung lavage clears the lipoproteinaceous alveolar material of acute silicosis (silicoproteinosis); it has no role in simple chronic silicosis, plaques or byssinosis.

Q90. Silica-associated renal disease most often takes the form of:
A) Recurrent nephrolithiasis
B) Isolated renal tubular acidosis
C) Renal artery stenosis
D) Glomerulonephritis, sometimes ANCA-associated

Answer: D) Glomerulonephritis, sometimes ANCA-associated

Explanation: Silica is linked to glomerular and ANCA-associated renal disease; the distractors are unrelated renal conditions.

Q91. The autoantibody profile commonly raised in silica-associated autoimmunity includes:
A) Antinuclear antibody, rheumatoid factor and ANCA
B) Anti-cyclic-citrullinated peptide only
C) Anti-double-stranded DNA exclusively
D) Anti-Jo-1 only

Answer: A) Antinuclear antibody, rheumatoid factor and ANCA

Explanation: Non-specific elevations of antinuclear antibody, rheumatoid factor and ANCA are typical, reflecting the adjuvant immune mechanism rather than a single disease-specific autoantibody.

Q92. Caplan syndrome was originally described in dust-exposed workers who also had:
A) Ankylosing spondylitis
B) Gout
C) Rheumatoid arthritis
D) Psoriatic arthritis

Answer: C) Rheumatoid arthritis

Explanation: Caplan described rheumatoid pneumoconiotic nodules in Welsh coal miners with rheumatoid arthritis; the syndrome also occurs in silica-exposed workers with the disease.

Silicotuberculosis

Q93. Compared with the general population, the risk of active tuberculosis in silicosis is increased by a factor of about:
A) No increase
B) Three-fold or more
C) Reduced by half
D) One hundred-fold

Answer: B) Three-fold or more

Explanation: Silicosis raises tuberculosis risk several-fold, commonly cited around three-fold and rising with disease severity; it is never protective or unchanged.

Q94. Regarding silica-exposed workers without radiographic silicosis, the tuberculosis risk is:
A) Exactly the same as unexposed people
B) Lower than baseline
C) Increased only if they smoke
D) Still increased above baseline

Answer: D) Still increased above baseline

Explanation: Heavy silica exposure increases tuberculosis risk even before radiographic silicosis appears, so exposure alone, not just established disease, is a warning sign.

Q95. The single most useful microbiological test in suspected silicotuberculosis, given its paucibacillary nature, is:
A) Mycobacterial culture
B) Smear microscopy alone
C) The tuberculin skin test
D) Serology for anti-TB antibodies

Answer: A) Mycobacterial culture

Explanation: Culture has the best yield in often smear-negative disease; the tuberculin test is unreliable and serology is not recommended.

Q96. The tuberculin skin test in a patient with silicosis is:
A) Diagnostic of active disease
B) One hundred per cent sensitive
C) Unreliable for diagnosing active tuberculosis
D) Absolutely contraindicated

Answer: C) Unreliable for diagnosing active tuberculosis

Explanation: The tuberculin skin test cannot distinguish latent from active disease and is unreliable in silicosis; microbiological confirmation by culture or NAAT is required.

Q97. A single negative sputum smear in a silicotic suspected of tuberculosis:
A) Reliably excludes tuberculosis
B) Does not exclude it; culture and NAAT should follow
C) Confirms cure
D) Means treatment can be stopped

Answer: B) Does not exclude it; culture and NAAT should follow

Explanation: Silicotuberculosis is often paucibacillary, so smears are frequently negative; a single negative smear does not exclude it and culture or NAAT should follow.

Q98. The response of silicotuberculosis to standard anti-tuberculous therapy is characteristically:
A) Faster than in non-silicotic patients
B) Identical to ordinary tuberculosis
C) Absent, so treatment is futile
D) Slower, with higher rates of failure and relapse

Answer: D) Slower, with higher rates of failure and relapse

Explanation: Drug penetration into avascular fibrotic masses is poor, delaying sputum conversion and raising rates of treatment failure and relapse compared with ordinary tuberculosis.

Q99. The chief reason anti-tuberculous drugs work less well in silicotuberculosis is:
A) Poor penetration into avascular fibrotic (PMF) masses
B) Universal multidrug resistance
C) Inevitable malabsorption of all drugs
D) Rapid hepatic clearance of all agents

Answer: A) Poor penetration into avascular fibrotic (PMF) masses

Explanation: Dense, avascular fibrotic (PMF) masses limit delivery of anti-tuberculous drugs to the bacilli within them, explaining the slower and less complete therapeutic response.

Q100. Radiographic features suggesting tuberculosis superimposed on silicosis include:
A) Symmetric stable small nodules
B) Eggshell nodal calcification
C) Cavitation, asymmetry and rapid change
D) Bilateral pleural plaques

Answer: C) Cavitation, asymmetry and rapid change

Explanation: New cavitation, asymmetry and rapid change point to active TB against the stable silicotic background.

Q101. Routine practice for all silica-exposed workers should include:
A) Tuberculosis screening only if they smoke
B) Periodic tuberculosis screening
C) A single screen at recruitment only
D) No tuberculosis screening

Answer: B) Periodic tuberculosis screening

Explanation: Given the sustained raised risk, periodic tuberculosis screening throughout employment is recommended; one-off or symptom-only screening misses early or paucibacillary disease.

Q102. Besides Mycobacterium tuberculosis, silicotics also have an increased risk of infection by:
A) Mycobacterium leprae specifically
B) No mycobacteria other than M. tuberculosis
C) Only rapidly growing saprophytes
D) Non-tuberculous mycobacteria (e.g. M. avium, M. kansasii)

Answer: D) Non-tuberculous mycobacteria (e.g. M. avium, M. kansasii)

Explanation: Impaired pulmonary immunity raises the risk of NTM and fungal infection as well as classic TB.

Q103. Silicotuberculosis is clinically important above all because it is:
A) A leading cause of death in silicosis
B) A cause of eggshell calcification
C) Protective against PMF
D) A purely radiographic curiosity

Answer: A) A leading cause of death in silicosis

Explanation: The TB association is the chief driver of mortality in silicosis, especially in endemic regions.

Q104. The public-health response to a diagnosed case of silicotuberculosis includes:
A) Permanent isolation of the worker for life
B) Closure of the entire factory
C) Notification and contact tracing
D) Chemoprophylaxis of every co-worker regardless of status

Answer: C) Notification and contact tracing

Explanation: Notification and contact tracing are the standard public-health response; lifelong isolation, factory closure or blanket chemoprophylaxis of all co-workers are disproportionate or unindicated.

Progressive Massive Fibrosis (PMF)

Q105. An alternative name for progressive massive fibrosis is:
A) Simple silicosis
B) Complicated (conglomerate) silicosis
C) Acute silicosis
D) Siderosis

Answer: B) Complicated (conglomerate) silicosis
Explanation: PMF is synonymous with complicated or conglomerate silicosis; simple, acute silicosis and siderosis are distinct entities, not alternative names for the same process.

Q106. PMF masses characteristically arise in the:
A) Lower zones and bases
B) Perihilar regions only
C) Costophrenic angles
D) Upper and posterior zones of both lungs

Answer: D) Upper and posterior zones of both lungs
Explanation: Conglomerate masses form in the upper and posterior zones of both lungs, mirroring the distribution of the small nodules from which they coalesce.

Q107. Over time, PMF masses typically:
A) Migrate towards the hila, leaving peripheral emphysema
B) Migrate towards the bases
C) Calcify uniformly and shrink
D) Resolve spontaneously

Answer: A) Migrate towards the hila, leaving peripheral emphysema
Explanation: Over years the masses migrate centrally toward the hila, leaving hyperinflated emphysematous lung at the periphery; they do not move to the bases or resolve.

Q108. The lung-function pattern of established PMF is:
A) Pure obstructive with a raised DLCO
B) Entirely normal
C) Mixed restrictive and obstructive
D) Pure restrictive with a raised DLCO

Answer: C) Mixed restrictive and obstructive
Explanation: Conglomerate fibrosis reduces volumes while associated emphysema adds obstruction, so PMF produces a mixed restrictive-obstructive defect with a reduced diffusing capacity.

Q109. Central cavitation appearing within a PMF mass should raise suspicion of:
A) Benign calcification
B) Tuberculosis
C) Spontaneous resolution
D) A pleural plaque

Answer: B) Tuberculosis
Explanation: Central cavitation within a PMF mass most often signals superimposed tuberculosis, and occasionally carcinoma; it is not a sign of benign calcification or resolution.

Q110. PMF can continue to develop after exposure has ceased because:
A) Fresh exposure is always required
B) The masses invariably regress
C) Collagen spontaneously dissolves
D) Retained silica keeps driving fibrosis

Answer: D) Retained silica keeps driving fibrosis
Explanation: Silica retained in the lung continues to drive fibrogenesis, so PMF can develop and progress even after exposure has ceased, without any new exposure.

Q111. The combination of PMF with rheumatoid arthritis and large necrobiotic nodules is termed:
A) Caplan syndrome
B) Erasmus syndrome
C) Loffler syndrome
D) Hamman-Rich syndrome

Answer: A) Caplan syndrome
Explanation: Caplan syndrome is the rheumatoid-arthritis-with-pneumoconiosis association producing large nodules; Erasmus is silica-scleroderma, and Loffler and Hamman-Rich are unrelated entities.

Q112. The usual terminal complication of advanced PMF is:
A) Nephrotic syndrome
B) Portal hypertension
C) Cor pulmonale and respiratory failure
D) Constrictive pericarditis

Answer: C) Cor pulmonale and respiratory failure
Explanation: Progressive loss of lung and chronic hypoxaemia lead to pulmonary hypertension and right heart failure.

Q113. A distinctive feature of coal-workers' PMF compared with silicotic PMF is:
A) Eggshell nodal calcification
B) Melanoptysis (expectoration of black material from a softened mass)
C) Ferruginous bodies in sputum
D) Bilateral pleural plaques

Answer: B) Melanoptysis (expectoration of black material from a softened mass)

Explanation: Coal PMF masses may liquefy centrally and discharge black fluid (melanoptysis); silicosis classically shows eggshell calcification.

Q114. The background microscopic lesion underlying silicotic PMF is the:
A) Non-collagenous coal macule
B) Asbestos (ferruginous) body
C) Non-caseating granuloma
D) Collagenous silicotic nodule

Answer: D) Collagenous silicotic nodule

Explanation: Silicotic PMF is built from coalescing collagenous silicotic nodules, whereas coal PMF arises from non-collagenous coal macules; the background lesion distinguishes the two.

Q115. Definitive treatment for end-stage PMF with respiratory failure is:
A) Lung transplantation
B) A curative corticosteroid course
C) Chelation therapy
D) Curative lobectomy

Answer: A) Lung transplantation

Explanation: Lung transplantation is the only definitive option for end-stage PMF; corticosteroids, chelation and lobectomy do not cure the established fibrosis.

Q116. Development of PMF is accelerated by:
A) Early removal from exposure
B) Smoking cessation
C) Superimposed tuberculosis and continued heavy exposure
D) Low cumulative dust burden

Answer: C) Superimposed tuberculosis and continued heavy exposure

Explanation: Superimposed tuberculosis and continued heavy exposure hasten conglomeration into PMF, whereas early removal from exposure, low dust burden and smoking cessation slow it.

Diagnosis & Differential

Q117. The cornerstone of silicosis diagnosis is:
A) A specific serum anti-silica antibody
B) A compatible occupational exposure history plus chest imaging
C) Sputum bacterial culture
D) Skin patch testing

Answer: B) A compatible occupational exposure history plus chest imaging

Explanation: Diagnosis rests on a compatible occupational exposure history together with characteristic chest imaging; there is no specific serum antibody, culture or skin test for silicosis.

Q118. Because no specific biomarker exists, silicosis diagnosis relies on:
A) A serum silica level
B) A bronchoalveolar silica-antibody titre
C) Plasma D-dimer
D) Radiology and exposure history, with biopsy rarely needed

Answer: D) Radiology and exposure history, with biopsy rarely needed

Explanation: History plus imaging suffice in most cases; there is no specific biomarker, and biopsy is reserved for atypical presentations rather than routine confirmation.

Q119. Differential diagnoses for upper-zone nodular/fibrotic lung disease include:
A) Tuberculosis and sarcoidosis
B) Congestive cardiac failure
C) Lobar pneumonia
D) Acute pulmonary embolism

Answer: A) Tuberculosis and sarcoidosis

Explanation: Tuberculosis and sarcoidosis share an upper-zone predilection and enter the differential; heart failure, lobar pneumonia and embolism have quite different radiographic patterns.

Q120. The pneumoconiosis distinguished from silicosis by its lower-zone fibrosis and pleural plaques is:
A) Berylliosis
B) Byssinosis
C) Asbestosis
D) Siderosis

Answer: C) Asbestosis

Explanation: Asbestosis is basal with pleural plaques, contrasting with the upper-zone nodular pattern of silicosis; this distribution difference separates the two pneumoconioses.

Q121. Chronic beryllium disease is separated from silicosis by a positive:
A) Mantoux test
B) Beryllium lymphocyte proliferation test (BeLPT)
C) Methacholine challenge
D) Sweat chloride test

Answer: B) Beryllium lymphocyte proliferation test (BeLPT)

Explanation: The beryllium lymphocyte proliferation test confirms beryllium sensitisation and distinguishes chronic beryllium disease from silicosis; Mantoux, methacholine and sweat-chloride tests are irrelevant here.

Q122. The radiographic clue that specifically favours silicosis over coal workers' pneumoconiosis is:
A) Lower-zone reticulation
B) Pleural plaques
C) Ferruginous bodies on biopsy
D) Eggshell nodal calcification

Answer: D) Eggshell nodal calcification

Explanation: Eggshell nodal calcification specifically favours silicosis over coal workers' pneumoconiosis; pleural plaques and ferruginous bodies instead indicate asbestos exposure.

Q123. A silica-exposed worker with bilateral hilar lymphadenopathy and non-caseating granulomas on biopsy more likely has:
A) Sarcoidosis or chronic beryllium disease
B) Simple silicosis
C) Asbestosis
D) Byssinosis

Answer: A) Sarcoidosis or chronic beryllium disease

Explanation: Granulomas are not the lesion of silicosis (which is collagenous nodules); they point to sarcoid or beryllium disease.

Q124. Lung biopsy in suspected silicosis is:
A) Mandatory for every case
B) The first-line investigation
C) Rarely required, since diagnosis is clinico-radiological
D) Required before the disease can be notified

Answer: C) Rarely required, since diagnosis is clinico-radiological

Explanation: History plus imaging usually suffice, so biopsy is rarely required and is reserved for atypical cases; it is neither mandatory, first-line nor needed for notification.

Management, Prevention & Legislation

Q125. Specific curative treatment for established silicosis is:
A) D-penicillamine chelation
B) None — care is supportive with removal from exposure
C) A curative corticosteroid course
D) Long-term curative antibiotics

Answer: B) None — care is supportive with removal from exposure

Explanation: Silicosis is irreversible, so management is supportive with removal from exposure and treatment of complications; penicillamine, steroids and antibiotics are not curative.

Q126. The single most effective control for silica exposure is:
A) Issuing dust masks
B) Job rotation
C) Annual chest X-rays
D) Elimination or substitution of silica at source

Answer: D) Elimination or substitution of silica at source

Explanation: Removing the hazard by elimination or substitution (e.g. non-silica abrasives) ranks highest in the hierarchy; masks, rotation and surveillance are lower-order measures.

Q127. In abrasive blasting, silica sand should be replaced with:
A) Non-silica abrasives such as alumina or steel shot
B) A finer grade of sand
C) Wetted sand only
D) Crushed quartz

Answer: A) Non-silica abrasives such as alumina or steel shot

Explanation: Substituting a non-silica abrasive such as alumina or steel shot removes the hazard, whereas finer, wetted or crushed quartz all still contain silica.

Q128. Wet drilling and local exhaust ventilation are examples of:
A) Administrative controls
B) Personal protective equipment
C) Engineering controls
D) Substitution

Answer: C) Engineering controls

Explanation: Wet drilling and local exhaust ventilation control dust at source by engineering means; they are neither administrative measures, PPE nor true elimination of the hazard.

Q129. Respiratory protection against silica should be regarded as:
A) The first and best control
B) The last line, used after higher controls
C) Unnecessary once masks are worn
D) Equivalent to elimination

Answer: B) The last line, used after higher controls

Explanation: PPE is the lowest tier of the hierarchy, used only after higher controls; it relies on correct fit and use and supplements rather than replaces source control.

Q130. Medical surveillance of silica-exposed workers centres on:
A) Serum silica monitoring
B) Routine bronchoscopy
C) Annual CT pulmonary angiography
D) Periodic chest radiography with symptom and spirometry review

Answer: D) Periodic chest radiography with symptom and spirometry review

Explanation: Periodic chest radiography with symptom and spirometry review is the standard surveillance approach; serum silica, routine bronchoscopy and CT angiography are not used.

Q131. Because of the raised tuberculosis risk, silica-exposed workers require:
A) Periodic tuberculosis screening
B) Lifelong isoniazid for everyone regardless of status
C) Annual BCG re-vaccination
D) No tuberculosis follow-up

Answer: A) Periodic tuberculosis screening

Explanation: Regular tuberculosis screening is essential in exposed workers; blanket lifelong isoniazid for everyone or annual BCG re-vaccination is not standard practice.

Q132. In India, silicosis is:
A) Not a notifiable disease
B) Notifiable only in mines
C) A notifiable occupational disease under the Factories Act (Third Schedule)
D) Reportable only after death

Answer: C) A notifiable occupational disease under the Factories Act (Third Schedule)

Explanation: Silicosis is listed in the Third Schedule of the Factories Act as a notifiable occupational disease; notification is not limited to mines or to post-mortem reporting.

Q133. Compensation for an Indian factory worker disabled by silicosis is provided mainly under:
A) The Factories Act 1948 alone
B) The ESI Act / Employees' Compensation Act
C) The Payment of Wages Act 1936
D) The Maternity Benefit Act 1961

Answer: B) The ESI Act / Employees' Compensation Act

Explanation: Statutory compensation flows through the ESI Act or Employees' Compensation Act. The Factories Act mandates safety/notification but not cash compensation; the others are unrelated labour statutes.

Q134. The OSHA permissible exposure limit for respirable crystalline silica (8-hour TWA) is about:
A) 5 milligrams per cubic metre
B) 1 milligram per cubic metre
C) 500 micrograms per cubic metre
D) 50 micrograms per cubic metre

Answer: D) 50 micrograms per cubic metre

Explanation: The OSHA PEL is 50 µg/m3; the distractors are off by one to two orders of magnitude.

Q135. A pre-placement chest radiograph for a worker entering a silica job mainly serves to:
A) Establish a baseline and detect pre-existing disease
B) Treat early silicosis
C) Substitute for ongoing dust controls
D) Screen only for tuberculosis

Answer: A) Establish a baseline and detect pre-existing disease

Explanation: A baseline film allows future comparison and identifies prior disease; it neither treats nor replaces controls.

Q136. End-stage silicosis with respiratory failure may be considered for:
A) Curative pneumonectomy
B) A curative corticosteroid course
C) Lung transplantation
D) Chelation therapy

Answer: C) Lung transplantation

Explanation: Lung transplantation is the only option in end-stage disease; pneumonectomy, corticosteroids and chelation are not curative for established silicosis.

Q137. Controlling silica dust at source rather than relying on the worker reflects the principle of the:
A) Healthy worker effect
B) Hierarchy of control
C) Dose-response threshold
D) Precautionary recall

Answer: B) Hierarchy of control

Explanation: Controlling dust at source rather than relying on the worker is the essence of the hierarchy of control; the distractors describe unrelated epidemiological or dosing concepts.

Q138. Health education for silica workers should emphasise:
A) That silicosis is curable with medication
B) That respirators alone eliminate all risk
C) That nodules will regress over time
D) Smoking cessation and awareness of tuberculosis symptoms

Answer: D) Smoking cessation and awareness of tuberculosis symptoms

Explanation: Smoking compounds lung-cancer and tuberculosis risk, and early recognition of tuberculosis symptoms is vital; the distractors are dangerous misconceptions to be corrected.

Q139. The ACGIH threshold limit value for respirable crystalline silica is approximately:
A) 0.025 mg/m3
B) 0.1 mg/m3
C) 0.5 mg/m3
D) 5 mg/m3

Answer: A) 0.025 mg/m3

Explanation: The ACGIH TLV is 0.025 mg/m3 (25 micrograms/m3), stricter than the OSHA PEL of 50 micrograms/m3.

Q140. Wet drilling controls silica exposure mainly by:
A) Chemically neutralising the silica
B) Increasing particle size so it cannot be inhaled
C) Suppressing the generation of airborne dust at source
D) Accelerating mucociliary clearance

Answer: C) Suppressing the generation of airborne dust at source

Explanation: Water suppresses dust release at the point of generation; it does not alter silica chemistry or particle physiology.

Q141. The workplace practice most restricted or banned in many countries because of silicosis risk is:
A) Wet rock drilling
B) Abrasive blasting with silica sand
C) Local exhaust ventilation
D) Routine medical surveillance

Answer: B) Abrasive blasting with silica sand

Explanation: Silica-sand blasting produces extreme exposures and is banned/restricted in several jurisdictions; the others are protective measures.

Q142. Under the Indian Factories Act, the statutory medical examination of workers in a hazardous (silica) process is carried out by the:
A) Safety Officer
B) Welfare Officer
C) Factory Inspector
D) Certifying Surgeon / Factory Medical Officer

Answer: D) Certifying Surgeon / Factory Medical Officer

Explanation: Statutory medical examination is the role of the Certifying Surgeon/FMO; the others have non-medical statutory duties.

Q143. For a silicotic worker found to have latent tuberculosis infection, the recommended action is:
A) Offer tuberculosis preventive therapy (e.g. isoniazid)
B) Take no action unless active disease develops
C) Give BCG vaccination
D) Start lifelong rifampicin monotherapy

Answer: A) Offer tuberculosis preventive therapy (e.g. isoniazid)

Explanation: Silicotics are among the groups in whom treating latent TB is strongly recommended owing to the high reactivation risk.

Imaging, Spirometry & Signature Findings

Q144. The characteristic chest X-ray pattern of simple silicosis is:
A) Lower-zone irregular opacities
B) Small rounded opacities, upper-zone predominant
C) Bilateral pleural plaques
D) Diffuse ground-glass at the bases

Answer: B) Small rounded opacities, upper-zone predominant

Explanation: Simple silicosis shows upper-zone small rounded opacities (ILO p/q/r); irregular basal opacities and plaques point to asbestos instead.

Q145. The spirometry pattern in established silicosis is typically:
A) Pure obstructive with raised DLCO
B) Always entirely normal
C) Isolated raised TLC
D) Restrictive, becoming mixed when PMF and emphysema develop

Answer: D) Restrictive, becoming mixed when PMF and emphysema develop

Explanation: Silicotic fibrosis is restrictive; complicated disease (PMF) adds obstruction, giving a mixed pattern with reduced gas transfer.

Q146. A near-signature radiographic finding of silicosis is:
A) Eggshell calcification of hilar lymph nodes
B) Ferruginous bodies in sputum
C) Basal honeycombing
D) Melanoptysis

Answer: A) Eggshell calcification of hilar lymph nodes

Explanation: Peripheral eggshell nodal calcification is a classic, fairly specific sign of silicosis; the distractors belong to asbestos and coal disease.

Q147. Large opacities of complicated silicosis are graded by the ILO system as:
A) p, q, r
B) s, t, u
C) A, B, C
D) 0 to 3

Answer: C) A, B, C

Explanation: Progressive massive fibrosis produces large opacities graded A, B or C by size; the small rounded opacities use p/q/r.

Q148. A silicotic whose film shows new cavitation and whose disease accelerates should be evaluated for:
A) Mesothelioma
B) Tuberculosis
C) Siderosis
D) Baritosis

Answer: B) Tuberculosis

Explanation: Silicosis carries a strong TB association; new cavitation or rapid change signals silicotuberculosis, the signature complication to exclude.

Q149. The signature histological lesion underlying the silicosis X-ray is the:
A) Non-collagenous coal macule
B) Ferruginous body
C) Non-caseating granuloma
D) Collagenous whorled (onion-skin) silicotic nodule

Answer: D) Collagenous whorled (onion-skin) silicotic nodule

Explanation: The dense collagenous onion-skin nodule is the silicosis hallmark; the distractors are the lesions of coal, asbestos and beryllium disease.

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UNIT I — PNEUMOCONIOSES

Baritosis

TOPICS IN THIS CHAPTER

Baritosis

Q1. Baritosis is caused by inhalation of:
A) Barium (e.g. barite/barium sulphate) dust
B) Iron oxide
C) Tin oxide
D) Crystalline silica

Answer: A) Barium (e.g. barite/barium sulphate) dust
Explanation: Baritosis is the benign pneumoconiosis from barium dust, seen in barite mining and processing; it is non-fibrogenic.

Q2. Baritosis is best classified as:
A) A collagenous fibrosis
B) A granulomatous immune disease
C) Benign and non-fibrogenic
D) A malignant disease

Answer: C) Benign and non-fibrogenic
Explanation: Barium compounds are biologically inert, so baritosis merely deposits radio-dense material in the lung without causing fibrosis or any functional impairment.

Q3. The chest radiograph in baritosis characteristically shows:
A) Eggshell calcification
B) Very dense discrete opacities (barium is highly radio-opaque)
C) Basal honeycombing
D) Pleural plaques

Answer: B) Very dense discrete opacities (barium is highly radio-opaque)
Explanation: Barium's high radio-density produces strikingly dense opacities, a benign appearance disproportionate to the negligible clinical effect.

Q4. A characteristic behaviour of the opacities in baritosis is that they:
A) Always progress to PMF
B) Cavitate
C) Become malignant
D) May regress after exposure ceases

Answer: D) May regress after exposure ceases
Explanation: Because the opacities are inert radio-dense deposits rather than fibrosis, they may regress once exposure stops, confirming the benign nature.

Q5. Lung function in pure baritosis is:
A) Normal
B) Restrictive
C) Obstructive
D) Mixed

Answer: A) Normal
Explanation: Pure baritosis does not impair lung function, being a benign non-fibrogenic deposition; functional loss should prompt a search for other causes.

Q6. The occupation classically associated with baritosis is:
A) Tin smelting
B) Arc welding
C) Barite mining and processing
D) Coal mining

Answer: C) Barite mining and processing
Explanation: Baritosis occurs in barium (barite) miners and processors; tin, iron and coal give stannosis, siderosis and CWP respectively.

Imaging, Spirometry & Signature Findings

Q7. The chest X-ray of baritosis characteristically shows:
A) Basal honeycombing
B) Very dense discrete opacities without fibrosis
C) Pleural plaques
D) Eggshell calcification

Answer: B) Very dense discrete opacities without fibrosis
Explanation: Barium is highly radio-opaque, producing strikingly dense opacities with no fibrosis, paralleling siderosis and stannosis.

Q8. The spirometry in pure baritosis is:
A) Restrictive
B) Obstructive
C) Mixed
D) Normal

Answer: D) Normal
Explanation: Baritosis is a benign non-fibrogenic deposition, so lung function is normal; impairment should prompt a search for other causes.

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UNIT I — PNEUMOCONIOSES

Berylliosis

TOPICS IN THIS CHAPTER

Beryllium & Exposure

Q1. Berylliosis is caused by exposure to:
A) Beryllium and its compounds
B) Iron oxide fume
C) Tin oxide
D) Barium sulphate

Answer: A) Beryllium and its compounds
Explanation: Berylliosis (chronic beryllium disease) results from beryllium exposure; the distractors cause benign non-fibrogenic pneumoconioses instead.

Q2. Classic high-risk industries for beryllium exposure include all EXCEPT:
A) Aerospace and defence alloys
B) Electronics and ceramics
C) Cotton spinning
D) Fluorescent lamp manufacture (historical)

Answer: C) Cotton spinning
Explanation: Beryllium is used in aerospace alloys, electronics/ceramics and historically in fluorescent lamps; cotton spinning is unrelated (byssinosis).

Q3. Beryllium is valued industrially because it is:
A) Radioactive
B) Light, strong and an excellent conductor
C) Highly water-soluble and inert
D) A cheap abundant filler

Answer: B) Light, strong and an excellent conductor
Explanation: Beryllium is light, stiff, strong and conductive, hence its aerospace and electronics use; these very properties drive demand despite toxicity.

Q4. Acute beryllium disease, now rare, is essentially a:
A) Slow granulomatous reaction
B) Malignant tumour
C) Benign dust deposition
D) Chemical pneumonitis from high exposure

Answer: D) Chemical pneumonitis from high exposure
Explanation: Acute beryllium disease is a dose-related chemical pneumonitis from heavy exposure; chronic disease is the immunological granulomatous form.

Pathogenesis & Immunology

Q5. Chronic beryllium disease is fundamentally a:
A) Cell-mediated (type IV) hypersensitivity
B) Type I IgE allergy
C) Type II cytotoxic reaction
D) Non-immune dust overload

Answer: A) Cell-mediated (type IV) hypersensitivity
Explanation: CBD is a delayed, cell-mediated (type IV) hypersensitivity to beryllium acting as a hapten, driving granuloma formation.

Q6. Beryllium acts immunologically as a:
A) Complete antigen causing IgE release
B) Direct cytotoxin only
C) Hapten presented to beryllium-specific T cells
D) Pure irritant gas

Answer: C) Hapten presented to beryllium-specific T cells
Explanation: Beryllium behaves as a hapten, processed and presented to sensitised CD4 T cells that proliferate and drive granulomatous inflammation.

Q7. Genetic susceptibility to chronic beryllium disease is linked to:
A) HLA-B27
B) HLA-DPB1 Glu69 marker
C) HLA-DQ2
D) Cystic fibrosis gene

Answer: B) HLA-DPB1 Glu69 marker
Explanation: A glutamate at position 69 of HLA-DPB1 increases beryllium-presentation and CBD risk; the others relate to unrelated conditions.

Q8. The hallmark histological lesion of chronic beryllium disease is:
A) Caseating granuloma
B) Collagenous whorled nodule
C) Ferruginous body
D) Non-caseating granuloma

Answer: D) Non-caseating granuloma
Explanation: CBD produces non-caseating granulomas indistinguishable from sarcoidosis on histology alone, hence the need for exposure history and testing.

Diagnosis

Q9. The disease that chronic beryllium disease most closely mimics, clinically and histologically, is:
A) Sarcoidosis
B) Silicosis
C) Asbestosis
D) Coal workers' pneumoconiosis

Answer: A) Sarcoidosis

Explanation: CBD is radiographically and histologically very like sarcoidosis (non-caseating granulomas, hilar nodes); exposure history and BeLPT distinguish them.

Q10. The confirmatory test that distinguishes chronic beryllium disease from sarcoidosis is the:
A) Tuberculin skin test
B) Kveim test
C) Beryllium lymphocyte proliferation test (BeLPT)
D) Serum ACE alone

Answer: C) Beryllium lymphocyte proliferation test (BeLPT)

Explanation: The BeLPT demonstrates beryllium-specific lymphocyte sensitisation, confirming CBD and separating it from sarcoidosis, which it otherwise resembles.

Q11. The BeLPT can be performed on:
A) Sputum only
B) Blood and/or bronchoalveolar lavage lymphocytes
C) Urine
D) Skin scrapings

Answer: B) Blood and/or bronchoalveolar lavage lymphocytes

Explanation: The lymphocyte proliferation test uses blood or BAL lymphocytes exposed to beryllium salts; lavage testing is more sensitive in established disease.

Q12. A positive BeLPT without lung granulomas indicates:
A) Definite chronic beryllium disease
B) Acute pneumonitis
C) Cured disease
D) Beryllium sensitisation (not yet disease)

Answer: D) Beryllium sensitisation (not yet disease)

Explanation: A positive BeLPT alone shows sensitisation; CBD requires both sensitisation and granulomatous lung pathology, so such workers need surveillance.

Clinical & Radiology

Q13. The radiographic picture of chronic beryllium disease typically shows:
A) Diffuse infiltrates with hilar lymphadenopathy
B) Upper-zone rounded nodules with eggshell calcification
C) Basal honeycombing with pleural plaques
D) A solitary cavitating mass

Answer: A) Diffuse infiltrates with hilar lymphadenopathy

Explanation: CBD shows diffuse reticulonodular infiltrates and hilar adenopathy resembling sarcoidosis, unlike the patterns of the mineral-dust pneumoconioses.

Q14. Clinical features of chronic beryllium disease include:
A) Acute high fever and rigors only
B) Painless haematuria
C) Progressive dyspnoea, cough and systemic features
D) Isolated finger pain

Answer: C) Progressive dyspnoea, cough and systemic features

Explanation: CBD presents insidiously with exertional dyspnoea, dry cough, fatigue and weight loss, reflecting a multisystem granulomatous disorder.

Q15. Unlike the inert-dust pneumoconioses, chronic beryllium disease can involve:
A) Only the pleura
B) Extrapulmonary organs (skin, liver, lymph nodes)
C) Only the upper airway
D) No organs at all

Answer: B) Extrapulmonary organs (skin, liver, lymph nodes)

Explanation: As a systemic granulomatous disease, CBD may affect skin, liver, spleen and lymph nodes, again echoing sarcoidosis.

Management & Prevention

Q16. First-line treatment of symptomatic chronic beryllium disease is:
A) Chelation therapy
B) Antitubercular drugs
C) No treatment is ever used
D) Corticosteroids

Answer: D) Corticosteroids

Explanation: Corticosteroids suppress the granulomatous inflammation of CBD; unlike fibrogenic pneumoconioses, it is an immune disease that responds to steroids.

Q17. The cornerstone of preventing chronic beryllium disease is:
A) Rigorous control of airborne beryllium exposure
B) Vaccination of workers
C) Routine antibiotics
D) Job rotation alone

Answer: A) Rigorous control of airborne beryllium exposure

Explanation: Because even low exposures can sensitise susceptible workers, strict engineering control and exposure limitation are the key preventive measures.

Q18. Medical surveillance of beryllium workers commonly uses:
A) Annual tuberculin testing
B) Serum iron studies
C) Periodic BeLPT and respiratory assessment
D) Audiometry

Answer: C) Periodic BeLPT and respiratory assessment

Explanation: Surveillance with periodic BeLPT detects sensitisation early, prompting closer follow-up and exposure reduction before disease develops.

Q19. Compared with silicosis, chronic beryllium disease differs in being:
A) A collagenous nodular fibrosis
B) Immunologically mediated and steroid-responsive
C) Caused by a benign inert dust
D) Unrelated to exposure

Answer: B) Immunologically mediated and steroid-responsive

Explanation: CBD is a hypersensitivity granulomatous disease that responds to steroids, contrasting with the irreversible collagenous fibrosis of silicosis.

Imaging, Spirometry & Signature Findings

Q20. The characteristic chest X-ray of chronic beryllium disease is:
A) Upper-zone rounded nodules with eggshell calcification
B) Diffuse reticulonodular infiltrates with hilar lymphadenopathy
C) Basal honeycombing with pleural plaques
D) A solitary cavitating mass

Answer: B) Diffuse reticulonodular infiltrates with hilar lymphadenopathy

Explanation: CBD shows diffuse reticulonodular change with hilar adenopathy, closely mimicking sarcoidosis rather than the mineral-dust patterns.

Q21. The spirometry pattern in chronic beryllium disease is usually:
A) Obstructive with raised DLCO
B) Always normal
C) Isolated air-trapping
D) Restrictive, often with a reduced DLCO

Answer: D) Restrictive, often with a reduced DLCO

Explanation: As a granulomatous interstitial disease, chronic beryllium disease typically restricts lung volumes and reduces the diffusing capacity for gas transfer.

Q22. The signature combination confirming chronic beryllium disease is:
A) Non-caseating granulomas PLUS a positive BeLPT
B) Ferruginous bodies plus plaques
C) Eggshell calcification plus melanoptysis
D) Coal macules plus rheumatoid factor

Answer: A) Non-caseating granulomas PLUS a positive BeLPT

Explanation: CBD requires non-caseating granulomas together with demonstrated beryllium sensitisation (positive BeLPT), distinguishing it from sarcoidosis.

Q23. Chronic beryllium disease is distinguished from sarcoidosis chiefly by:
A) The pattern of crackles
B) Serum calcium
C) Beryllium exposure history and a positive BeLPT
D) A tuberculin test

Answer: C) Beryllium exposure history and a positive BeLPT

Explanation: Exposure history plus the beryllium lymphocyte proliferation test separate CBD from the otherwise identical sarcoidosis.

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UNIT I — PNEUMOCONIOSES

ILO Classification of Radiographs

TOPICS IN THIS CHAPTER

Purpose & Method

Q1. The ILO classification of radiographs is designed to:
A) Standardise the description of pneumoconiosis films, not to diagnose
B) Establish a tissue diagnosis
C) Measure lung function
D) Replace occupational history

Answer: A) Standardise the description of pneumoconiosis films, not to diagnose

Explanation: The ILO system provides a reproducible, semi-quantitative description of radiographic appearances for epidemiology, surveillance and compensation; it does not itself diagnose disease.

Q2. ILO reading is performed by comparing the film against:
A) The patient's previous CT
B) A textbook photograph
C) A set of standard reference radiographs
D) Predicted spirometry values

Answer: C) A set of standard reference radiographs

Explanation: Classification requires side-by-side comparison with the ILO standard reference films, which anchors profusion and opacity grading and keeps reading reproducible.

Q3. The ILO classification was developed primarily for use in:
A) Acute emergency triage
B) Epidemiology, surveillance and compensation
C) Antibiotic selection
D) Pre-operative assessment

Answer: B) Epidemiology, surveillance and compensation

Explanation: Its purpose is population surveillance, research comparability and medicolegal/compensation decisions in dust-exposed workers, not acute clinical triage.

Q4. In many countries a film is formally read for ILO purposes by a:
A) Ward nurse
B) Safety officer
C) Radiographer alone
D) Certified 'B-reader'

Answer: D) Certified 'B-reader'

Explanation: Trained, certified readers (NIOSH 'B-readers') classify films to a recognised standard, improving consistency for surveillance and compensation.

Q5. The ILO classification can be applied to:
A) Pneumoconiosis changes from any mineral dust
B) Only silicosis
C) Only asbestos disease
D) Only tuberculosis

Answer: A) Pneumoconiosis changes from any mineral dust

Explanation: The scheme is generic to the pneumoconioses, coding rounded and irregular opacities, large opacities and pleural changes regardless of the specific dust.

Film Quality

Q6. Before classifying opacities, the reader first records the film's:
A) Patient's weight
B) Spirometry result
C) Technical quality
D) Smoking pack-years

Answer: C) Technical quality

Explanation: Technical quality (graded 1 to 4) is recorded first, because a poor film can mimic or obscure opacities and must be acknowledged.

Q7. ILO technical quality is graded on a scale of:
A) 0 to 3
B) 1 to 4 (1 = good)
C) A to C
D) p to r

Answer: B) 1 to 4 (1 = good)

Explanation: Quality is graded 1 (good) to 4 (unreadable), with a comment required for grades 2-4; grade 4 films are unacceptable for classification.

Q8. A film of ILO technical quality grade 4 is:
A) The best possible
B) Equivalent to grade 1
C) Required for compensation
D) Unacceptable for classification

Answer: D) Unacceptable for classification

Explanation: Grade 4 indicates an unreadable film that cannot be classified; only grades 1-3 (with comment for 2-3) are usable.

Profusion

Q9. 'Profusion' in the ILO system refers to the:
A) Concentration (number per zone) of small opacities
B) Size of large masses
C) Degree of pleural calcification
D) Number of bullae

Answer: A) Concentration (number per zone) of small opacities

Explanation: Profusion grades how densely small opacities populate the affected zones, judged against the standard films, and is the core measure of disease extent.

Q10. Profusion has four major categories, namely:
A) A, B and C
B) p, q and r
C) 0, 1, 2 and 3
D) I to IV

Answer: C) 0, 1, 2 and 3

Explanation: The four major profusion categories are 0, 1, 2 and 3, each subdivided to create the 12-point scale used for fine grading.

Q11. The 12-point profusion scale is created by:
A) Adding pleural scores
B) Subdividing each major category against the neighbouring ones
C) Counting bullae
D) Measuring FEV1

Answer: B) Subdividing each major category against the neighbouring ones

Explanation: Each major category is split (e.g. 0/-, 0/0, 0/1, 1/0, 1/1, 1/2 ...), giving 12 steps that record the reader's first and seriously-considered alternative choice.

Q12. A profusion recorded as '2/1' means the reader:
A) Saw two opacities
B) Measured 2.1 mm opacities
C) Found 21% involvement
D) Chose category 2 but seriously considered 1

Answer: D) Chose category 2 but seriously considered 1

Explanation: The notation gives the chosen category (2) and the alternative seriously considered (1); it does not denote counts, sizes or percentages.

Q13. Higher profusion of small opacities correlates with:
A) Heavier cumulative exposure and greater risk of progression
B) Lower exposure
C) Better prognosis
D) Pleural disease only

Answer: A) Heavier cumulative exposure and greater risk of progression

Explanation: Greater profusion reflects a heavier dust burden and predicts a higher likelihood of progression, including to complicated (large-opacity) disease.

Small Opacities

Q14. Small ROUNDED opacities are graded by diameter as:
A) s, t, u
B) A, B, C
C) p, q, r
D) 0 to 3

Answer: C) p, q, r

Explanation: Rounded opacities are graded p (up to 1.5 mm), q (>1.5-3 mm) and r (>3-10 mm); these are typical of silicosis and coal disease.

Q15. Small IRREGULAR opacities are graded by width as:
A) p, q, r
B) s, t, u
C) A, B, C
D) I to III

Answer: B) s, t, u

Explanation: Irregular (linear/reticular) opacities are graded s (up to 1.5 mm), t (>1.5-3 mm) and u (>3-10 mm) by width; they are typical of asbestosis.

Q16. A small rounded opacity graded 'q' measures:
A) Up to 1.5 mm
B) Over 3 up to 10 mm
C) Over 10 mm
D) Over 1.5 up to 3 mm

Answer: D) Over 1.5 up to 3 mm

Explanation: q is the intermediate rounded grade (>1.5-3 mm); p is smaller, r is larger, and anything over 10 mm is a large opacity.

Q17. Opacity shape and size are recorded using:
A) Two letters (predominant then any subsidiary type)
B) A single number
C) A percentage
D) A colour code

Answer: A) Two letters (predominant then any subsidiary type)

Explanation: Two letters are entered, the first for the predominant opacity and the second for any significant secondary type (e.g. q/t), capturing mixed patterns.

Q18. An opacity exceeding 10 mm is no longer 'small' but is classified as a:
A) Grade r
B) Grade u
C) Large opacity (A, B or C)
D) Profusion 3

Answer: C) Large opacity (A, B or C)

Explanation: Once an opacity exceeds 10 mm it is a large opacity, graded A, B or C, denoting complicated pneumoconiosis (progressive massive fibrosis).

Q19. The zones used to record the distribution of small opacities number:
A) Two
B) Six (upper, middle, lower; each lung)
C) Three
D) Twelve

Answer: B) Six (upper, middle, lower; each lung)

Explanation: Each lung is divided into upper, middle and lower zones, giving six zones in which the presence and profusion of opacities are recorded.

Large Opacities

Q20. ILO large opacity category A is defined as:
A) Any opacity under 1 cm
B) Larger than the right upper zone
C) Exactly 1 mm
D) One or more opacities >1 cm with combined diameter up to 50 mm

Answer: D) One or more opacities >1 cm with combined diameter up to 50 mm

Explanation: Category A is large opacities whose combined longest diameter is greater than 1 cm and up to 50 mm; B and C are progressively larger.

Q21. ILO large opacity category C is defined as opacities whose combined area:
A) Exceeds the equivalent of the right upper zone
B) Is under 1 cm
C) Equals a small rounded opacity
D) Is within one rib space

Answer: A) Exceeds the equivalent of the right upper zone

Explanation: Category C, the most extensive, denotes large opacities whose combined area exceeds the right upper zone, indicating advanced PMF.

Q22. Large opacities in the ILO system indicate:
A) Simple disease only
B) A normal variant
C) Complicated pneumoconiosis (progressive massive fibrosis)
D) Pleural disease

Answer: C) Complicated pneumoconiosis (progressive massive fibrosis)

Explanation: Large opacities (A/B/C) define complicated pneumoconiosis, i.e. PMF formed by coalescence of small opacities, carrying worse prognosis.

Pleural Coding

Q23. ILO pleural plaques are recorded by all of the following EXCEPT:
A) Site (chest wall, diaphragm, other)
B) Profusion category 0-3
C) Presence of calcification
D) Width and extent

Answer: B) Profusion category 0-3

Explanation: Plaques are coded by site, face-on or in-profile appearance, calcification, width and extent; the 0-3 profusion scale applies to small opacities, not plaques.

Q24. In the ILO system, pleural plaques are noted as being seen:
A) Only on CT
B) Only after biopsy
C) Only if calcified
D) Face-on and/or in profile

Answer: D) Face-on and/or in profile

Explanation: Plaques are recorded by whether they are seen in profile (along the chest wall) and/or face-on, plus their site, calcification, width and extent.

Q25. Obliteration of the costophrenic angle is recorded separately because it:
A) Indicates pleural disease (often diffuse thickening)
B) Is a normal finding
C) Means active infection
D) Reflects profusion

Answer: A) Indicates pleural disease (often diffuse thickening)

Explanation: Costophrenic-angle obliteration is coded separately as a marker of pleural disease such as diffuse thickening, distinct from discrete plaques.

Q26. Pleural calcification in the ILO scheme is recorded by:
A) Profusion 0-3
B) Opacity letters p-u
C) Site and extent
D) Quality grade

Answer: C) Site and extent

Explanation: Calcification is coded by its site (chest wall, diaphragm, other) and extent, helping document asbestos-related pleural disease objectively.

Symbols

Q27. ILO 'symbols' are used to record:
A) The patient's age
B) Additional radiographic features beyond opacities and pleura
C) Spirometry values
D) Exposure duration

Answer: B) Additional radiographic features beyond opacities and pleura

Explanation: A defined set of two-letter symbols flags additional features (e.g. cavitation, emphysema, cancer), enriching the description beyond opacity and pleural codes.

Q28. The ILO symbol 'es' denotes:
A) An effusion
B) Emphysema
C) Mesothelioma
D) Eggshell calcification of lymph nodes

Answer: D) Eggshell calcification of lymph nodes

Explanation: 'es' records eggshell calcification, a classic silicosis sign; 'ef' is effusion, 'em' emphysema and 'me' mesothelioma.

Q29. The ILO symbol 'ax' denotes:
A) Coalescence of small opacities
B) An axillary mass
C) A normal variant
D) Atelectasis

Answer: A) Coalescence of small opacities

Explanation: 'ax' marks coalescence of small rounded opacities, an early step toward the large opacities of progressive massive fibrosis.

Q30. Which symbol would flag a suspected complicating malignancy?
A) em
B) es
C) ca
D) ax

Answer: C) ca

Explanation: The symbol 'ca' denotes a suspected complicating malignancy; 'em' marks emphysema, 'es' eggshell calcification and 'ax' coalescence of opacities.

Q31. The symbol 'cv' on an ILO reading indicates:
A) Cardiac enlargement
B) A cavity
C) Calcified nodes
D) Coalescence

Answer: B) A cavity

Explanation: 'cv' records a cavity (relevant when tuberculosis complicates silicosis); 'co' relates to cardiac/cardiomediastinal abnormality and 'cn' to calcified small opacities.

Q32. The symbol 'tb' is available so the reader can flag features suggestive of:
A) Tin deposition
B) Total burden
C) Tumour stage
D) Tuberculosis

Answer: D) Tuberculosis

Explanation: 'tb' lets the reader note radiographic features suggestive of tuberculosis, an important complication especially of silicosis.

Application

Q33. A key strength of the ILO classification is that it:

A) Improves reproducibility and comparability between readers and over time
B) Provides a tissue diagnosis
C) Measures fibre burden
D) Replaces spirometry

Answer: A) Improves reproducibility and comparability between readers and over time

Explanation: By anchoring readings to standard films and codes, the system reduces inter-reader variation and allows valid comparison across surveys and time.

Q34. For occupational compensation, ILO classification provides:

A) A legal verdict
B) A cure
C) An objective, standardised record of radiographic change
D) An exposure measurement

Answer: C) An objective, standardised record of radiographic change

Explanation: It supplies a reproducible, documented description of radiographic abnormality that supports (but does not by itself decide) compensation claims.

Q35. A limitation of the ILO classification is that it:

A) Measures lung function
B) Describes appearances but does not establish the specific diagnosis
C) Is fully automated
D) Eliminates the need for exposure history

Answer: B) Describes appearances but does not establish the specific diagnosis

Explanation: The system standardises description only; diagnosis still requires the exposure history and clinical correlation, and inter-reader variation persists.

Q36. Serial ILO-classified films in surveillance are useful to:

A) Measure cumulative dust dose
B) Diagnose asthma
C) Replace the exposure history
D) Detect progression of profusion or new large opacities over time

Answer: D) Detect progression of profusion or new large opacities over time

Explanation: Comparing serial readings detects rising profusion or emerging large opacities, signalling progression and prompting intervention.

7

UNIT I — PNEUMOCONIOSES

Other Pneumoconioses

TOPICS IN THIS CHAPTER

Aluminosis

Q1. Aluminosis (aluminium lung) is associated with exposure to:
A) Fine aluminium dust/powder
B) Iron oxide only
C) Cotton dust
D) Barium sulphate

Answer: A) Fine aluminium dust/powder

Explanation: Aluminosis follows inhalation of fine aluminium powder (historically in abrasives and explosives manufacture) and can cause fibrosis, unlike the inert metals.

Q2. Unlike the benign inert-dust pneumoconioses, aluminosis can cause:
A) No reaction at all
B) Only pleural plaques
C) Pulmonary fibrosis
D) Mesothelioma

Answer: C) Pulmonary fibrosis

Explanation: Finely divided aluminium can provoke interstitial fibrosis, so aluminosis is not classed among the purely benign metal pneumoconioses.

Shaver's Disease

Q3. Shaver's disease arises from fume exposure during:
A) Cotton ginning
B) Bauxite smelting (alumina abrasives manufacture)
C) Coal cutting
D) Tin plating

Answer: B) Bauxite smelting (alumina abrasives manufacture)

Explanation: Shaver's disease is a fibrotic lung disease from fumes of alumina/silica during bauxite smelting for abrasives, historically severe.

Graphite Pneumoconiosis

Q4. Graphite pneumoconiosis resembles coal workers' disease because graphite is:
A) An iron compound
B) A silicate fibre
C) A barium salt
D) A form of carbon

Answer: D) A form of carbon

Explanation: Graphite is crystalline carbon, so its pneumoconiosis resembles CWP, with macules and, in heavy exposure, progressive massive fibrosis.

Q5. Heavy graphite exposure can lead to:
A) Progressive massive fibrosis
B) Mesothelioma
C) Eggshell calcification
D) Benign deposition only

Answer: A) Progressive massive fibrosis

Explanation: Like coal, graphite (carbon) dust in heavy exposure can produce PMF, distinguishing it from the truly inert metal dusts.

Kaolinosis

Q6. Kaolinosis is caused by inhalation of:
A) Tin oxide
B) Barium
C) China clay (kaolin) dust
D) Iron fume

Answer: C) China clay (kaolin) dust

Explanation: Kaolin (china clay, a hydrated aluminium silicate) causes kaolinosis in mining and pottery work, which can progress to fibrosis and PMF.

Q7. Kaolinosis is important because heavy exposure may cause:
A) Only benign deposition
B) Fibrosis and PMF
C) Mesothelioma
D) Acute asthma

Answer: B) Fibrosis and PMF

Explanation: Although less fibrogenic than silica, heavy kaolin exposure can produce significant fibrosis and even PMF, so it is not a benign pneumoconiosis.

Talcosis

Q8. Talcosis may produce fibrosis and pleural disease partly because talc is often contaminated with:
A) Iron only
B) Tin
C) Barium
D) Silica and asbestos (tremolite) fibres

Answer: D) Silica and asbestos (tremolite) fibres

Explanation: Commercial talc frequently contains silica and tremolite-type fibres, so talcosis can cause both parenchymal fibrosis and pleural changes.

Q9. Inhaled talc in occupational talcosis characteristically causes:
A) Mixed nodular and interstitial fibrosis (and pleural plaques)
B) Purely benign deposition
C) Mesothelioma only
D) No lung change

Answer: A) Mixed nodular and interstitial fibrosis (and pleural plaques)

Explanation: Talcosis produces a mixed fibrotic picture with possible pleural plaques, reflecting talc's silica and fibre contamination.

Hard-Metal Disease

Q10. Hard-metal (cobalt) lung disease is caused by exposure to:
A) Pure tin
B) Barium sulphate
C) Cobalt-tungsten carbide dust
D) Iron oxide

Answer: C) Cobalt-tungsten carbide dust

Explanation: Hard-metal disease results from cobalt in tungsten-carbide tools (grinding, sharpening); cobalt is the key sensitising/toxic agent.

Q11. The characteristic histological finding in hard-metal lung disease is:
A) Non-caseating granuloma
B) Giant-cell interstitial pneumonia (GIP)
C) Collagenous silicotic nodule
D) Ferruginous body

Answer: B) Giant-cell interstitial pneumonia (GIP)

Explanation: Hard-metal disease classically produces giant-cell interstitial pneumonia with bizarre multinucleate giant cells, a near-specific finding.

Q12. Hard-metal lung disease is best regarded as caused by:
A) Tungsten alone
B) Iron oxide
C) Barium
D) Cobalt acting as the toxic/sensitising agent

Answer: D) Cobalt acting as the toxic/sensitising agent

Explanation: Cobalt, not tungsten, drives the lung toxicity and hypersensitivity in hard-metal disease, which can cause asthma and interstitial fibrosis.

Mixed-Dust & General

Q13. Mixed-dust pneumoconiosis arises when silica is inhaled with:
A) Other less-fibrogenic dusts (e.g. iron, carbon, silicates)
B) Only organic dust
C) Only beryllium
D) Cotton dust

Answer: A) Other less-fibrogenic dusts (e.g. iron, carbon, silicates)

Explanation: Mixed-dust fibrosis occurs when silica is combined with other dusts, modifying the pattern from classic nodular silicosis to mixed lesions.

Q14. Among these less common pneumoconioses, those capable of causing true fibrosis include:
A) Only siderosis
B) Only stannosis and baritosis
C) Aluminosis, graphite, kaolin, talc and hard-metal disease
D) None of them

Answer: C) Aluminosis, graphite, kaolin, talc and hard-metal disease

Explanation: Aluminium, graphite, kaolin, talc and cobalt exposures can all cause fibrosis, unlike the purely benign iron, tin and barium pneumoconioses.

Q15. A useful organising principle for the metal pneumoconioses is that:
A) All metal dusts are benign
B) Iron, tin and barium are benign; beryllium and cobalt are immunological; aluminium, graphite, kaolin, talc can fibrose
C) All metal dusts cause granulomas
D) All metal dusts cause mesothelioma

Answer: B) Iron, tin and barium are benign; beryllium and cobalt are immunological; aluminium, graphite, kaolin, talc can fibrose

Explanation: Grouping by mechanism (inert vs immunological vs fibrogenic) is the clearest way to remember the diverse less-common pneumoconioses.

Imaging, Spirometry & Signature Findings

Q16. Graphite pneumoconiosis radiographically and pathologically resembles:
A) Asbestosis
B) Coal workers' pneumoconiosis
C) Berylliosis
D) Baritosis

Answer: B) Coal workers' pneumoconiosis

Explanation: Graphite is carbon, so its disease mimics CWP, with macules and possible progressive massive fibrosis in heavy exposure.

Q17. The near-characteristic histology of hard-metal (cobalt) lung disease is:
A) Non-caseating granuloma
B) Collagenous silicotic nodule
C) Ferruginous body
D) Giant-cell interstitial pneumonia (GIP)

Answer: D) Giant-cell interstitial pneumonia (GIP)

Explanation: Hard-metal disease classically shows giant-cell interstitial pneumonia with bizarre multinucleate giant cells, a near-specific finding.

Q18. Talcosis can produce both parenchymal fibrosis and pleural plaques because talc is often contaminated with:
A) Silica and tremolite-type fibres
B) Iron oxide
C) Pure carbon
D) Barium

Answer: A) Silica and tremolite-type fibres

Explanation: Commercial talc frequently contains silica and fibrous tremolite, so talcosis shows mixed fibrosis with pleural changes.

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UNIT I — PNEUMOCONIOSES

Pulmonary Function & Imaging — Foundations

TOPICS IN THIS CHAPTER

Spirometry Basics

Q1. Spirometry directly measures:
A) Air volumes and flow rates during forced breathing
B) Total lung capacity directly
C) Residual volume directly
D) Diffusing capacity

Answer: A) Air volumes and flow rates during forced breathing

Explanation: Spirometry records volumes and flows (FVC, FEV1, etc.); TLC and RV require body plethysmography or gas dilution, and DLCO is a separate test.

Q2. FVC (forced vital capacity) is:
A) The volume exhaled in the first second
B) The volume left after maximal expiration
C) The total volume forcibly exhaled after a full inspiration
D) The volume breathed at rest

Answer: C) The total volume forcibly exhaled after a full inspiration

Explanation: FVC is the entire volume forcibly exhaled from full inspiration to maximal expiration; FEV1 is the first-second portion of it.

Q3. FEV1 is:
A) The total exhaled volume
B) The volume forcibly exhaled in the first second
C) The maximal inspiratory volume
D) The tidal volume

Answer: B) The volume forcibly exhaled in the first second

Explanation: FEV1 is the volume expelled in the first second of a forced expiration and falls in both obstruction and restriction, so the ratio is key.

Q4. The FEV1/FVC ratio is most useful because it:
A) Measures diffusing capacity
B) Estimates residual volume
C) Detects anaemia
D) Distinguishes obstructive from restrictive defects

Answer: D) Distinguishes obstructive from restrictive defects

Explanation: A reduced ratio indicates obstruction, whereas a normal or raised ratio with low volumes indicates restriction; this is the central interpretive step.

Q5. For a spirometry effort to be acceptable, it should show:
A) A rapid start, no cough, and an adequate plateau/duration
B) A slow gentle exhalation
C) Breath-holding at the start
D) Maximal inspiration only

Answer: A) A rapid start, no cough, and an adequate plateau/duration

Explanation: Acceptable curves need a sharp start, freedom from cough or early termination, and a sufficient exhalation; repeatability across efforts is also required.

Q6. Spirometry results are interpreted against:
A) A single fixed cut-off for everyone
B) The patient's weight only
C) Predicted values for age, sex, height and ethnicity
D) Room temperature only

Answer: C) Predicted values for age, sex, height and ethnicity

Explanation: Predicted normals depend on age, sex, height and ethnicity, so interpretation is relative to the individual's predicted value, not an absolute number.

Obstructive vs Restrictive

Q7. An obstructive ventilatory defect is defined by:
A) A reduced FVC with normal ratio
B) A reduced FEV1/FVC ratio
C) A raised DLCO
D) A normal flow-volume loop

Answer: B) A reduced FEV1/FVC ratio

Explanation: Obstruction reduces FEV1 more than FVC, lowering the ratio (commonly <0.7); restriction lowers volumes with a preserved ratio.

Q8. A restrictive ventilatory defect is characterised by:
A) A reduced FEV1/FVC ratio
B) Air-trapping with raised RV
C) A scooped expiratory curve
D) Reduced FVC and TLC with a normal or raised FEV1/FVC

Answer: D) Reduced FVC and TLC with a normal or raised FEV1/FVC

Explanation: Restriction reduces lung volumes (FVC, TLC) while the ratio is preserved or increased; the pneumoconioses classically cause restriction.

Q9. A classic obstructive disease is:
A) Chronic obstructive pulmonary disease
B) Pulmonary fibrosis
C) Chest-wall deformity
D) Neuromuscular weakness

Answer: A) Chronic obstructive pulmonary disease
Explanation: COPD and asthma are obstructive; fibrosis, chest-wall disease and neuromuscular weakness are restrictive, with a preserved ratio.

Q10. A 'mixed' ventilatory defect shows:
A) Normal spirometry
B) Only a low FVC
C) Both a reduced FEV1/FVC ratio and a reduced TLC
D) Only a low ratio

Answer: C) Both a reduced FEV1/FVC ratio and a reduced TLC
Explanation: A mixed defect combines obstruction (low ratio) and restriction (low TLC), as seen in complicated pneumoconiosis with fibrosis plus emphysema.

Q11. To confirm restriction definitively, the key measurement is:
A) A reduced peak flow
B) A reduced total lung capacity (TLC)
C) A reduced FEV1 alone
D) A reduced oxygen saturation

Answer: B) A reduced total lung capacity (TLC)
Explanation: TLC is the defining measurement of restriction; spirometry can suggest it (low FVC, normal ratio) but TLC by plethysmography or dilution confirms it.

Q12. In obstruction, the total lung capacity is typically:
A) Markedly reduced
B) Always zero
C) Unmeasurable
D) Normal or increased (air-trapping)

Answer: D) Normal or increased (air-trapping)
Explanation: Air-trapping in obstruction raises RV and keeps TLC normal or high, unlike restriction where TLC falls.

Flow-Volume Loops

Q13. The flow-volume loop in airflow obstruction characteristically shows:
A) A scooped (concave) expiratory limb
B) A tall, narrow loop
C) A flattened inspiratory plateau
D) A normal shape

Answer: A) A scooped (concave) expiratory limb
Explanation: Obstruction produces a concave (scooped) expiratory limb from reduced flows at low volumes; restriction narrows the loop.

Q14. The flow-volume loop in a restrictive defect is typically:
A) Scooped on expiration
B) Flattened on both limbs
C) Tall and narrow, shifted to lower volumes
D) Identical to obstruction

Answer: C) Tall and narrow, shifted to lower volumes
Explanation: Restriction narrows the loop and shifts it toward low lung volumes while preserving the overall shape, unlike the scooping of obstruction.

Q15. Flattening of BOTH the inspiratory and expiratory limbs suggests:
A) Pulmonary fibrosis
B) A fixed upper-airway obstruction
C) Emphysema
D) Normal lungs

Answer: B) A fixed upper-airway obstruction
Explanation: A fixed large-airway obstruction (e.g. tracheal stenosis) plateaus both limbs; variable lesions flatten only one limb.

Q16. Flattening of only the inspiratory limb indicates:
A) Restriction
B) A fixed obstruction
C) Small-airway disease
D) A variable extrathoracic obstruction

Answer: D) A variable extrathoracic obstruction
Explanation: A variable extrathoracic lesion (e.g. vocal-cord dysfunction) limits inspiratory flow selectively; intrathoracic variable lesions affect expiration.

Diffusing Capacity (DLCO)

Q17. The DLCO measures:
A) The lung's ability to transfer gas across the alveolar membrane
B) Airway resistance
C) Residual volume
D) Respiratory muscle strength

Answer: A) The lung's ability to transfer gas across the alveolar membrane

Explanation: DLCO assesses gas transfer across the alveolar-capillary membrane, reflecting surface area, membrane thickness and capillary blood volume.

Q18. DLCO is characteristically REDUCED in:
A) Asthma between attacks
B) Obesity alone
C) Interstitial fibrosis and emphysema
D) Simple anxiety

Answer: C) Interstitial fibrosis and emphysema

Explanation: Both fibrosis (thick membrane, lost surface) and emphysema (lost surface) reduce DLCO; it helps separate emphysema from asthma in obstruction.

Q19. In the pneumoconioses with established fibrosis, DLCO is typically:
A) Increased
B) Reduced
C) Always normal
D) Supranormal

Answer: B) Reduced

Explanation: Fibrosis thickens the membrane and destroys surface area, lowering DLCO; a falling DLCO is often an early functional sign in asbestosis.

Q20. A reduced DLCO with an otherwise normal spirometry suggests:
A) Pure large-airway obstruction
B) A normal variant only
C) Poor effort only
D) Early interstitial or pulmonary vascular disease

Answer: D) Early interstitial or pulmonary vascular disease

Explanation: Isolated DLCO reduction can be the earliest sign of interstitial lung disease (e.g. early asbestosis) or pulmonary vascular disease.

Q21. DLCO may be spuriously LOW because of:
A) Anaemia
B) Polycythaemia
C) Recent exercise
D) Tall stature

Answer: A) Anaemia

Explanation: Anaemia reduces haemoglobin available for gas uptake, lowering measured DLCO; results are therefore corrected for haemoglobin.

Chest X-ray & ILO

Q22. The standardised system for reporting pneumoconiosis chest films is the:
A) TNM staging
B) NYHA classification
C) ILO classification
D) Bhalla score

Answer: C) ILO classification

Explanation: The ILO system grades profusion, size, shape and zone of opacities against standard reference films for reproducible occupational reporting.

Q23. Small ROUNDED opacities are graded in the ILO system as:
A) s, t, u
B) p, q, r
C) A, B, C
D) 0, 1, 2, 3

Answer: B) p, q, r

Explanation: Rounded opacities (silicosis, CWP) are p/q/r by diameter; irregular opacities (asbestosis) are s/t/u; large opacities are A/B/C.

Q24. Small IRREGULAR opacities are graded as:
A) p, q, r
B) A, B, C
C) I, II, III
D) s, t, u

Answer: D) s, t, u

Explanation: Irregular (linear/reticular) opacities, typical of asbestosis, are graded s/t/u by width, distinguishing them from rounded p/q/r opacities.

Q25. 'Profusion' in the ILO system refers to:
A) The concentration of small opacities per zone
B) The size of large masses
C) The degree of calcification
D) Pleural plaque number

Answer: A) The concentration of small opacities per zone

Explanation: Profusion grades the concentration of small opacities on a 12-point scale, recorded separately from opacity size and shape.

Q26. Large opacities (progressive massive fibrosis) are graded:
A) p, q, r
B) s, t, u
C) A, B, C by size
D) 0 to 3

Answer: C) A, B, C by size

Explanation: Large opacities (>1 cm) are graded A, B or C by combined size, denoting complicated pneumoconiosis with conglomerate fibrosis.

Q27. HRCT is more sensitive than the plain chest film for detecting:
A) Anaemia
B) Early nodules, masses, emphysema and pleural disease
C) Airway resistance
D) Respiratory muscle strength

Answer: B) Early nodules, masses, emphysema and pleural disease

Explanation: HRCT detects subtle parenchymal and pleural change before the plain film, useful in early or equivocal occupational lung disease.

ABG & Oximetry

Q28. Arterial blood gas analysis primarily assesses:
A) Lung volumes
B) Airway calibre
C) Diffusing capacity
D) Oxygenation, ventilation (CO2) and acid-base status

Answer: D) Oxygenation, ventilation (CO2) and acid-base status

Explanation: ABG reports PaO2, PaCO2, pH and bicarbonate, characterising oxygenation, ventilation and acid-base balance in respiratory disease.

Q29. Type 1 respiratory failure is defined by:
A) Low PaO2 with normal or low PaCO2
B) Low PaO2 with high PaCO2
C) High PaO2 with low PaCO2
D) Normal gases

Answer: A) Low PaO2 with normal or low PaCO2

Explanation: Type 1 (hypoxaemic) failure shows hypoxaemia without hypercapnia, typical of parenchymal disease such as fibrosis; type 2 adds hypercapnia.

Q30. Type 2 respiratory failure is characterised by:
A) Hypoxaemia with low PaCO2
B) Normal PaO2 and PaCO2
C) Hypoxaemia WITH hypercapnia (raised PaCO2)
D) Isolated alkalosis

Answer: C) Hypoxaemia WITH hypercapnia (raised PaCO2)

Explanation: Type 2 (ventilatory) failure combines low PaO2 with raised PaCO2, seen in severe COPD and respiratory-muscle or chest-wall failure.

Q31. A raised alveolar-arterial (A-a) oxygen gradient indicates:
A) Pure hypoventilation only
B) A problem with gas exchange/diffusion or shunt
C) Normal lungs
D) High altitude only

Answer: B) A problem with gas exchange/diffusion or shunt

Explanation: A widened A-a gradient localises hypoxaemia to gas-exchange failure (diffusion defect, V/Q mismatch, shunt) rather than pure hypoventilation.

Q32. Pulse oximetry measures:
A) Arterial PaCO2
B) Blood pH
C) Diffusing capacity
D) Peripheral oxygen saturation (SpO2)

Answer: D) Peripheral oxygen saturation (SpO2)

Explanation: Oximetry estimates haemoglobin oxygen saturation non-invasively; it does not measure CO2 or pH, so it cannot exclude hypercapnia.

Q33. An important limitation of pulse oximetry is that it:

A) Is unreliable in carbon monoxide poisoning and poor perfusion
B) Measures PaCO2 accurately
C) Detects anaemia
D) Replaces ABG entirely

Answer: A) Is unreliable in carbon monoxide poisoning and poor perfusion

Explanation: Oximetry cannot distinguish carboxyhaemoglobin from oxyhaemoglobin and is unreliable with poor perfusion, so ABG is needed when these matter.

Occupational Application

Q34. In occupational lung surveillance, spirometry is mainly used to:

A) Diagnose the specific dust
B) Replace chest radiography
C) Detect and track functional decline over time
D) Measure cumulative dust dose

Answer: C) Detect and track functional decline over time

Explanation: Serial spirometry tracks functional change in exposed workers; it complements imaging but does not identify the dust or quantify exposure.

Q35. A pre-placement spirometry and chest film mainly serve to:

A) Cure early disease
B) Establish a baseline and detect pre-existing disease
C) Replace exposure controls
D) Quantify fibre burden

Answer: B) Establish a baseline and detect pre-existing disease

Explanation: Baseline tests allow future comparison and reveal pre-existing disease; they neither treat disease nor substitute for dust control.

Q36. The pneumoconioses characteristically produce which spirometric pattern?

A) Pure obstructive always
B) Always normal
C) Raised DLCO
D) Restrictive (with mixed pattern when emphysema/PMF coexist)

Answer: D) Restrictive (with mixed pattern when emphysema/PMF coexist)

Explanation: Fibrotic pneumoconioses cause restriction; coal dust and complicated disease add obstruction, giving a mixed pattern with reduced DLCO.

Q37. Bronchodilator reversibility testing is most useful for:

A) Identifying reversible obstruction (e.g. asthma)
B) Confirming fibrosis
C) Measuring TLC
D) Detecting pleural plaques

Answer: A) Identifying reversible obstruction (e.g. asthma)

Explanation: A significant FEV1 improvement after a bronchodilator indicates reversible airflow obstruction such as occupational asthma, not fibrosis.

Obstructive vs Restrictive — Synthesis

Q38. A patient has reduced FVC and TLC with an FEV1/FVC ratio of 0.85. The pattern is:

A) Obstructive
B) Mixed
C) Restrictive
D) Normal

Answer: C) Restrictive

Explanation: Low volumes with a preserved or raised ratio define restriction; a low ratio would indicate obstruction.

Q39. An FEV1/FVC ratio of 0.55 with a raised TLC indicates:

A) An obstructive defect with air-trapping
B) A restrictive defect
C) A normal study
D) A pure diffusion defect

Answer: A) An obstructive defect with air-trapping

Explanation: A low ratio with normal or raised TLC is obstruction; air-trapping raises lung volumes, unlike the reduced volumes of restriction.

Q40. A restrictive pattern with a REDUCED DLCO points to a cause that is:
A) Extrapulmonary (chest wall)
B) Neuromuscular
C) Obesity
D) Parenchymal (e.g. fibrosis or pneumoconiosis)

Answer: D) Parenchymal (e.g. fibrosis or pneumoconiosis)

Explanation: Reduced DLCO localises restriction to the lung parenchyma; chest-wall, neuromuscular and obesity-related restriction spare DLCO.

Q41. A restrictive pattern with a NORMAL DLCO suggests:
A) Pulmonary fibrosis
B) An extrapulmonary cause (chest wall or neuromuscular)
C) Asbestosis
D) Sarcoidosis

Answer: B) An extrapulmonary cause (chest wall or neuromuscular)

Explanation: Preserved DLCO in restriction indicates an extrapulmonary cause, since the lung tissue itself is normal; parenchymal diseases lower DLCO.

Q42. Within obstructive disease, a reduced DLCO best distinguishes:
A) Asthma from bronchiectasis
B) Fibrosis from sarcoid
C) Emphysema from asthma/chronic bronchitis
D) Plaques from effusion

Answer: C) Emphysema from asthma/chronic bronchitis

Explanation: Emphysema destroys alveolar surface and lowers DLCO, whereas asthma and chronic bronchitis leave DLCO normal or raised.

Q43. Hyperinflation with flattened diaphragms on the chest film is the radiographic hallmark of:
A) Obstructive disease
B) Restrictive parenchymal disease
C) Pleural plaques
D) A normal film

Answer: A) Obstructive disease

Explanation: Air-trapping in obstructive disease produces hyperinflation with low, flattened diaphragms, whereas a restrictive process instead gives small lung volumes.

Q44. A worker with upper-zone rounded opacities, a restrictive defect and reduced DLCO most likely has:
A) Asthma
B) Chest-wall restriction
C) Chronic bronchitis
D) A parenchymal pneumoconiosis such as silicosis

Answer: D) A parenchymal pneumoconiosis such as silicosis

Explanation: Rounded upper-zone opacities with restriction and low DLCO indicate a parenchymal nodular pneumoconiosis like silicosis or coal disease.

Q45. A normal FEV1/FVC ratio with an isolated reduction in DLCO and a normal X-ray may indicate:
A) Established emphysema
B) Early interstitial or pulmonary vascular disease
C) A fixed upper-airway obstruction
D) Chest-wall restriction

Answer: B) Early interstitial or pulmonary vascular disease

Explanation: An isolated low DLCO with normal spirometry and film can be the earliest sign of interstitial or pulmonary vascular disease.

Q46. A bronchodilator producing a large rise in FEV1 indicates:
A) Irreversible fibrosis
B) Chest-wall restriction
C) Reversible obstruction (asthma)
D) A diffusion defect

Answer: C) Reversible obstruction (asthma)

Explanation: Significant bronchodilator reversibility identifies reversible airflow obstruction such as asthma, not a restrictive or diffusion problem.

Q47. The single test that confirms restriction definitively is:
A) Total lung capacity (TLC)
B) Peak expiratory flow
C) FEV1 alone
D) Pulse oximetry

Answer: A) Total lung capacity (TLC)

Explanation: A reduced TLC confirms restriction; spirometry can only suggest it from a low FVC with a preserved ratio.

9

UNIT I — PNEUMOCONIOSES

Siderosis

TOPICS IN THIS CHAPTER

Siderosis

Q1. Siderosis is caused by the inhalation of:
A) Iron oxide dust or fume
B) Crystalline silica
C) Beryllium
D) Cotton dust

Answer: A) Iron oxide dust or fume

Explanation: Siderosis results from iron oxide inhalation, classically in arc welders and iron workers; it is a benign, non-fibrogenic pneumoconiosis.

Q2. The classic occupation associated with siderosis is:
A) Coal mining
B) Sandblasting
C) Electric arc welding
D) Cotton spinning

Answer: C) Electric arc welding

Explanation: Arc welders inhale iron oxide fume, the prototypical cause of siderosis (sometimes called welder's lung); the others cause different diseases.

Q3. Pure siderosis is best described as:
A) Rapidly fatal fibrosis
B) Benign and non-fibrogenic
C) A granulomatous immune disease
D) A malignant tumour

Answer: B) Benign and non-fibrogenic

Explanation: Iron oxide is relatively inert, so pure siderosis causes pigment deposition without significant fibrosis or functional impairment.

Q4. The chest radiograph in siderosis typically shows:
A) Eggshell calcification
B) Basal honeycombing
C) Bilateral pleural plaques
D) Small dense opacities (iron is radio-dense) with little fibrosis

Answer: D) Small dense opacities (iron is radio-dense) with little fibrosis

Explanation: Iron is radio-opaque, so siderosis can produce striking small dense opacities despite minimal fibrosis, a classic 'reassuringly benign' picture.

Q5. A characteristic feature of the radiographic opacities of pure siderosis is that they may:
A) Regress after exposure ceases
B) Always progress to PMF
C) Cavitate
D) Calcify in an eggshell pattern

Answer: A) Regress after exposure ceases

Explanation: Because the opacities reflect inert radio-dense iron rather than fibrosis, they may even regress once exposure stops, unlike true fibrotic disease.

Q6. Lung function in pure siderosis is typically:
A) Severely restrictive
B) Severely obstructive
C) Normal or near-normal
D) Unmeasurable

Answer: C) Normal or near-normal

Explanation: As a benign non-fibrogenic pneumoconiosis, pure siderosis leaves lung function essentially intact; impairment suggests mixed dust or other disease.

Q7. 'Siderosilicosis' refers to siderosis combined with exposure to:
A) Tin
B) Silica (producing genuine fibrosis)
C) Barium
D) Cotton dust

Answer: B) Silica (producing genuine fibrosis)

Explanation: When iron exposure is mixed with silica (e.g. some foundry/mining work), the silica produces real fibrosis, unlike pure benign siderosis.

Q8. The key teaching point about siderosis is that it is:
A) Always rapidly progressive
B) A common cause of lung cancer
C) Caused by an organic dust
D) Radiographically alarming but clinically benign

Answer: D) Radiographically alarming but clinically benign

Explanation: Siderosis classically produces impressive, dense radiographic opacities yet causes minimal clinical consequence, making it the benign mirror-image of silicosis.

Imaging, Spirometry & Signature Findings

Q9. The chest X-ray of pure siderosis characteristically shows:

A) Basal honeycombing
B) Numerous small dense opacities with little or no fibrosis
C) Pleural plaques
D) Eggshell calcification

Answer: B) Numerous small dense opacities with little or no fibrosis

Explanation: Iron is radio-dense, so siderosis produces striking dense opacities despite minimal fibrosis, a benign 'alarming film' picture.

Q10. The spirometry in pure siderosis is typically:

A) Severely restrictive
B) Severely obstructive
C) Mixed
D) Normal

Answer: D) Normal

Explanation: Because iron oxide is biologically inert and non-fibrogenic, pure siderosis leaves lung function essentially normal despite the dense radiographic opacities.

Q11. A signature behaviour of the radiographic opacities in pure siderosis is that they:

A) May regress after exposure ceases
B) Always progress to PMF
C) Cavitate
D) Calcify as eggshells

Answer: A) May regress after exposure ceases

Explanation: The opacities are inert radio-dense deposits, not fibrosis, so they may regress once exposure stops, confirming the benign nature.

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UNIT I — PNEUMOCONIOSES

Stannosis

TOPICS IN THIS CHAPTER

Stannosis

Q1. Stannosis is caused by inhalation of:
A) Tin oxide dust or fume
B) Iron oxide
C) Silica
D) Beryllium

Answer: A) Tin oxide dust or fume

Explanation: Stannosis is the benign pneumoconiosis caused by tin oxide, seen in tin smelting and processing; it is non-fibrogenic.

Q2. Stannosis is classified as a:
A) Fibrogenic collagenous disease
B) Granulomatous immune disease
C) Benign, non-fibrogenic pneumoconiosis
D) Malignant condition

Answer: C) Benign, non-fibrogenic pneumoconiosis

Explanation: Tin oxide is inert, so stannosis causes radio-dense deposition without fibrosis or functional loss, like siderosis and baritosis.

Q3. The chest radiograph in stannosis shows:
A) Eggshell calcification
B) Dense nodular opacities (tin is very radio-dense) without fibrosis
C) Honeycombing
D) Pleural plaques

Answer: B) Dense nodular opacities (tin is very radio-dense) without fibrosis

Explanation: Tin has a high atomic number and is very radio-opaque, producing striking dense opacities despite the absence of fibrosis.

Q4. Lung function in pure stannosis is:
A) Severely restrictive
B) Severely obstructive
C) Always mixed
D) Normal

Answer: D) Normal

Explanation: Because tin oxide is inert and non-fibrogenic, pure stannosis does not impair lung function; the radiograph overstates the clinical impact.

Q5. The occupation classically associated with stannosis is:
A) Tin smelting and processing
B) Coal mining
C) Cotton spinning
D) Sandblasting

Answer: A) Tin smelting and processing

Explanation: Workers in tin smelting and oxide handling inhale tin oxide; the other trades are linked to coal, cotton and silica diseases respectively.

Q6. The main clinical significance of stannosis is that it:
A) Frequently causes PMF
B) Causes mesothelioma
C) Is benign and mainly a radiographic finding
D) Requires steroids

Answer: C) Is benign and mainly a radiographic finding

Explanation: Stannosis matters chiefly as a benign radiographic finding not to be mistaken for serious fibrotic disease; it needs no specific treatment.

Imaging, Spirometry & Signature Findings

Q7. The chest X-ray of stannosis characteristically shows:
A) Basal reticulation
B) Very dense nodular opacities without fibrosis
C) Pleural plaques
D) Cavitation

Answer: B) Very dense nodular opacities without fibrosis

Explanation: Tin is highly radio-opaque, so stannosis produces very dense opacities despite the absence of fibrosis or functional loss.

Q8. The spirometry in pure stannosis is:
A) Restrictive
B) Obstructive
C) Mixed
D) Normal

Answer: D) Normal

Explanation: Tin oxide is inert and non-fibrogenic, so pure stannosis does not impair lung function; the film overstates the clinical impact.

11

UNIT II — METALS

Inorganic Lead

TOPICS IN THIS CHAPTER

Sources & Occupations

Q1. Which occupation carries the HIGHEST risk of inorganic lead exposure?
A) Lead-acid battery manufacture and recycling
B) Commercial fishing
C) Software development
D) Dairy farming

Answer: A) Lead-acid battery manufacture and recycling

Explanation: Battery manufacture and recycling involve melting, pasting and oxide handling, generating heavy lead fume and dust, the archetypal high-exposure lead trade.

Q2. Lead exposure during building renovation arises chiefly from:
A) Mixing fresh Portland cement
B) Installing glass-fibre insulation
C) Burning, sanding or abrading old lead-based paint
D) Laying ceramic floor tiles

Answer: C) Burning, sanding or abrading old lead-based paint

Explanation: Disturbing old lead paint by burning, dry-sanding or demolition aerosolises lead, a major exposure in renovation, repainting and ship-breaking.

Q3. Among inorganic lead compounds, the one generally regarded as MOST toxic is:
A) Lead chromate
B) Lead oxide
C) Metallic lead sheet
D) Lead glass

Answer: B) Lead oxide

Explanation: Lead oxide (litharge/red lead) is highly bioavailable and regarded as the most toxic inorganic compound; lead chromate is comparatively the least toxic.

Q4. The least toxic of the following inorganic lead compounds is:
A) Lead oxide
B) Lead acetate
C) Lead carbonate
D) Lead chromate

Answer: D) Lead chromate

Explanation: Lead chromate is poorly soluble and the least toxic of the listed compounds, whereas lead oxide and the soluble salts are far more hazardous.

Q5. Lead glazing of pottery and lead-crystal manufacture cause exposure principally to:
A) Lead oxide dust and fume
B) Crystalline silica only
C) Organic solvents
D) Iron oxide fume

Answer: A) Lead oxide dust and fume

Explanation: Pottery glaze and lead crystal use lead oxide, releasing lead dust and fume during weighing, dipping, spraying and firing.

Q6. Which of these is NOT a recognised inorganic lead exposure?
A) Lead smelting and refining
B) Soldering and cable sheathing
C) Cotton textile spinning
D) Type-foundry and printing work

Answer: C) Cotton textile spinning

Explanation: Smelting, soldering and printing are classic lead exposures; cotton spinning relates to byssinosis, an organic-dust lung disease, not lead.

Absorption & Routes

Q7. The two principal routes of inorganic lead absorption in industry are:
A) Direct intravenous and intramuscular injection
B) Inhalation of fume/dust and ingestion from contaminated hands
C) Transdermal absorption and ocular contact
D) Rectal absorption and direct inhalation

Answer: B) Inhalation of fume/dust and ingestion from contaminated hands

Explanation: Inorganic lead is absorbed mainly by inhaling fume and dust and by ingestion (hand-to-mouth), the latter important where hygiene is poor.

Q8. Approximately what fraction of INHALED respirable lead is absorbed?
A) About 1%
B) About 5%
C) Essentially 100%
D) About 30-40%

Answer: D) About 30-40%

Explanation: A substantial fraction (around 30-40%) of inhaled respirable lead is absorbed across the alveoli, making inhalation the most efficient occupational route.

Q9. Gastrointestinal absorption of ingested lead differs markedly between adults and children, being about:
A) 10% in adults but ~50% in children
B) Equal at 50% in both
C) Higher in adults
D) Negligible in both
Answer: A) 10% in adults but ~50% in children
Explanation: Adults absorb roughly a tenth of ingested lead, but children absorb about half, a key reason children are far more susceptible to lead toxicity.

Q10. Inorganic (as opposed to organic) lead is absorbed through intact skin:
A) Rapidly and completely
B) Only via sweat glands
C) Poorly
D) Better than by inhalation
Answer: C) Poorly
Explanation: Inorganic lead is poorly absorbed through intact skin; significant dermal absorption is a feature of lipid-soluble organic (alkyl) lead, not inorganic lead.

Q11. Hand-to-mouth ingestion of lead is best prevented by:
A) Displaying hazard warning posters in the area
B) Prohibiting eating and smoking in work areas and enforcing handwashing
C) Increasing the ambient lighting of the workshop
D) Rotating workers between tasks each week
Answer: B) Prohibiting eating and smoking in work areas and enforcing handwashing
Explanation: Because ingestion is a major route, banning eating/smoking in lead areas and enforcing handwashing before meals directly interrupts hand-to-mouth transfer.

Distribution

Q12. In the three-compartment model, the pool that mediates acute toxicity and reflects current exposure is the:
A) Slow skeletal pool
B) Compact-bone pool
C) Dentine pool
D) Rapidly exchangeable (blood) pool
Answer: D) Rapidly exchangeable (blood) pool
Explanation: The rapidly exchangeable pool, chiefly blood, mediates toxicity and indicates current exposure; the skeletal pools are long-term stores.

Q13. What proportion of the adult lead body burden is held in the skeleton?
A) About 95%
B) About 5%
C) About 25%
D) About 50%
Answer: A) About 95%
Explanation: Roughly 95% of the adult body burden resides in bone, which acts as a long-term store with a half-life measured in years to decades.

Q14. The intermediate lead pool is located mainly in the:
A) Compact bone
B) Dentine
C) Soft tissues
D) Plasma water
Answer: C) Soft tissues
Explanation: Between the rapidly exchangeable blood pool and the slow skeletal pool lies the intermediate pool, found chiefly in the soft tissues.

Q15. The slow skeletal lead pool corresponds to:
A) Trabecular bone and marrow
B) Compact (cortical) bone and dentine
C) Blood and plasma
D) Liver and kidney
Answer: B) Compact (cortical) bone and dentine
Explanation: The slow skeletal pool is compact cortical bone and dentine; trabecular bone and marrow form the more readily exchangeable bone component.

Q16. In blood, the great majority of lead is carried:
A) Free in plasma water
B) On plasma albumin only
C) Within leucocytes
D) Bound within red blood cells
Answer: D) Bound within red blood cells
Explanation: Over 95% of blood lead is bound within erythrocytes, which is why whole blood (not serum) is assayed for the blood lead level.

Q17. Skeletal lead can be remobilised into blood during:
A) Pregnancy, lactation, immobilisation and intercurrent illness
B) Routine light exercise
C) Adequate dietary calcium intake
D) Cold weather alone

Answer: A) Pregnancy, lactation, immobilisation and intercurrent illness

Explanation: Increased bone turnover (pregnancy, lactation, immobilisation, illness) releases stored lead back into blood, re-exposing the person and any fetus.

Q18. Besides urine, minor routes of lead elimination include:
A) Exhaled breath as a gas
B) Tears exclusively
C) Sweat, saliva and breast milk
D) Shed hair only

Answer: C) Sweat, saliva and breast milk

Explanation: Lead is excreted chiefly in urine, with small amounts in sweat, saliva and breast milk; excretion in milk raises concern for the breastfed infant.

Mechanism

Q19. Lead exerts much of its enzymatic toxicity through a high affinity for:
A) Carbohydrate residues
B) Sulphydryl (SH) groups on enzymes
C) Membrane phospholipids only
D) Ribosomal RNA only

Answer: B) Sulphydryl (SH) groups on enzymes

Explanation: Lead binds avidly to enzyme sulphydryl groups, inhibiting SH-dependent enzymes, most importantly several in the haem-synthesis pathway.

Q20. Lead disrupts the handling of which cation, competing with it at synapses and in mitochondria?
A) Sodium
B) Chloride
C) Bicarbonate
D) Calcium

Answer: D) Calcium

Explanation: Lead mimics and competes with calcium at synapses and in mitochondrial respiration; therapeutic calcium can displace lead, enhancing its urinary loss.

Q21. Lead causes anaemia primarily by:
A) Inhibiting enzymes of haem synthesis
B) Causing acute haemolysis alone
C) Suppressing erythropoietin
D) Destroying the bone marrow stroma

Answer: A) Inhibiting enzymes of haem synthesis

Explanation: Lead blocks several haem-synthesis enzymes, reducing haemoglobin production; a mild reduction in red-cell survival also contributes to the anaemia.

Q22. The two haem-pathway enzymes classically inhibited by lead are:
A) Hexokinase and pyruvate kinase
B) Catalase and superoxide dismutase
C) ALA dehydratase and ferrochelatase
D) Amylase and lipase

Answer: C) ALA dehydratase and ferrochelatase

Explanation: Lead inhibits delta-aminolaevulinic-acid dehydratase and ferrochelatase, causing ALA and protoporphyrin to accumulate and impairing haem formation.

Q23. Inhibition of ferrochelatase by lead raises the level of:
A) Serum sodium
B) Zinc protoporphyrin / free erythrocyte protoporphyrin
C) Blood glucose
D) Plasma albumin

Answer: B) Zinc protoporphyrin / free erythrocyte protoporphyrin

Explanation: Ferrochelatase normally inserts iron into protoporphyrin; when blocked, zinc protoporphyrin (and FEP) accumulates, providing an effect biomarker.

Q24. Inhibition of ALA dehydratase by lead leads to accumulation of:
A) Glucose
B) Bilirubin
C) Urea
D) Delta-aminolaevulinic acid (ALA)

Answer: D) Delta-aminolaevulinic acid (ALA)

Explanation: Blocking ALA dehydratase causes ALA to accumulate and spill into urine, an early biochemical marker of lead's effect on haem synthesis.

Q25. Basophilic stippling of red cells in lead poisoning results from inhibition of:
A) Pyrimidine-5'-nucleotidase
B) Glucose-6-phosphate dehydrogenase
C) Ferrochelatase alone
D) Carbonic anhydrase

Answer: A) Pyrimidine-5'-nucleotidase

Explanation: Lead inhibits pyrimidine-5'-nucleotidase, leaving ribosomal RNA aggregates that appear as basophilic stippling on the blood film.

Haematology

Q26. The anaemia of inorganic lead poisoning is typically:
A) Macrocytic with hypersegmented neutrophils
B) Purely haemolytic with spherocytes
C) Normocytic or microcytic with basophilic stippling
D) Aplastic with pancytopenia

Answer: C) Normocytic or microcytic with basophilic stippling

Explanation: Lead causes a normo- or microcytic anaemia with basophilic stippling; in adults it is usually mild to moderate, more pronounced in children.

Q27. Unlike iron-deficiency anaemia, the anaemia of lead poisoning characteristically shows:
A) Very low serum iron
B) Normal or raised serum iron / iron stores
C) Marked microcytosis with low ferritin
D) Target cells

Answer: B) Normal or raised serum iron / iron stores

Explanation: Lead blocks haem synthesis rather than iron supply, so iron stores are normal or raised, distinguishing it biochemically from iron-deficiency anaemia.

Q28. Basophilic stippling is characteristic of chronic lead poisoning but is NOT specific, also occurring in:
A) Only lead poisoning
B) Healthy athletes
C) Iron overload exclusively
D) Some anaemias, malignancy and malaria

Answer: D) Some anaemias, malignancy and malaria

Explanation: Basophilic stippling, though typical of chronic inorganic lead, also appears in certain anaemias, malignancy and malaria, so it is suggestive not diagnostic.

Q29. The characteristic pallor of lead poisoning is partly attributable to:
A) Cutaneous vasoconstriction, not anaemia alone
B) Jaundice
C) Central cyanosis
D) Polycythaemia

Answer: A) Cutaneous vasoconstriction, not anaemia alone

Explanation: Lead causes anaemia, but the striking pallor also reflects cutaneous vasoconstriction, a classic descriptive teaching point.

Gastrointestinal

Q30. A characteristic feature distinguishing lead colic from other abdominal pain is that it is:
A) Relieved promptly by antispasmodics
B) Worsened by firm pressure
C) Relieved by firm abdominal pressure, not by antispasmodics
D) Associated with profuse watery diarrhoea

Answer: C) Relieved by firm abdominal pressure, not by antispasmodics

Explanation: Lead colic is severe abdominal pain eased by firm pressure (and by chelation) rather than by antispasmodics, with constipation rather than diarrhoea.

Q31. Typical gastrointestinal features of inorganic lead poisoning include:
A) Profuse bloody diarrhoea with tenesmus
B) Constipation, colicky pain, anorexia and a metallic taste
C) Painless obstructive jaundice and pale stools
D) Acute haemorrhagic pancreatitis with shock

Answer: B) Constipation, colicky pain, anorexia and a metallic taste

Explanation: Lead classically causes constipation with colicky abdominal pain, anorexia, dyspepsia and a metallic taste; diarrhoea is comparatively uncommon.

Q32. The blue-grey 'Burton line' at the gum margin is formed by:
A) Lead binding to enamel calcium
B) Lead reacting with salivary amylase
C) Lead displacing gingival melanin
D) Deposition of lead sulphide from reaction with hydrogen sulphide

Answer: D) Deposition of lead sulphide from reaction with hydrogen sulphide

Explanation: With poor oral hygiene, sulphide generated by oral bacteria reacts with lead to deposit lead sulphide at the gingival margin, forming the Burton line.

Q33. The Burton line is best interpreted as a sign of:
A) Lead exposure, not necessarily lead poisoning
B) Definite encephalopathy
C) Established renal failure
D) Acute haemolysis

Answer: A) Lead exposure, not necessarily lead poisoning

Explanation: The Burton line confirms exposure to lead but is not by itself proof of clinical poisoning, which requires symptoms or biochemical disturbance.

Neurological

Q34. The peripheral neuropathy of adult inorganic lead poisoning is characteristically:
A) Purely sensory
B) Predominantly autonomic
C) Motor, affecting the long extensor muscles
D) Confined to cranial nerves

Answer: C) Motor, affecting the long extensor muscles

Explanation: Lead causes a predominantly motor neuropathy weakening the long extensors, producing wrist drop and foot drop, with little sensory involvement.

Q35. Wrist drop in lead poisoning reflects weakness of muscles supplied by the:
A) Median nerve
B) Radial nerve (extensors of wrist and fingers)
C) Ulnar nerve
D) Common peroneal nerve

Answer: B) Radial nerve (extensors of wrist and fingers)

Explanation: The classic wrist drop reflects extensor weakness in the radial-nerve distribution; foot drop (peroneal) is a less common lower-limb counterpart.

Q36. Lead encephalopathy is most frequent and most dangerous in:
A) Healthy adult men
B) The elderly only
C) No age group
D) Young children

Answer: D) Young children

Explanation: Children are far more susceptible to lead encephalopathy, which presents with irritability, ataxia, raised intracranial pressure, seizures and coma.

Q37. Features of acute lead encephalopathy include:
A) Headache, vomiting, ataxia, seizures and impaired consciousness
B) Isolated painless visual loss
C) A single peripheral nerve palsy only
D) Pure cerebellar tremor

Answer: A) Headache, vomiting, ataxia, seizures and impaired consciousness

Explanation: Lead encephalopathy, with cerebral oedema and raised intracranial pressure, causes headache, vomiting, ataxia, seizures and depressed consciousness.

Q38. Childhood lead exposure, even at low blood levels, is associated with:
A) No measurable effect on the developing brain
B) Improved cognition and school performance
C) Reduced IQ and neurodevelopmental impairment, with no clear safe threshold
D) Effects appearing only above 100 micrograms/dL

Answer: C) Reduced IQ and neurodevelopmental impairment, with no clear safe threshold

Explanation: Lead lowers IQ and impairs neurodevelopment in children with no demonstrated safe threshold, underpinning very low public-health reference values.

Renal & Systemic

Q39. Chronic lead nephropathy is characteristically a:
A) Acute proliferative glomerulonephritis
B) Chronic interstitial nephritis with secondary hypertension
C) Minimal-change nephrotic syndrome
D) Recurrent stone disease only

Answer: B) Chronic interstitial nephritis with secondary hypertension

Explanation: Chronic lead exposure causes an interstitial nephropathy with progressive renal impairment and secondary hypertension, sometimes with gout.

Q40. In children, lead may produce a proximal tubular defect resembling:
A) Bartter syndrome
B) Nephrotic syndrome
C) Renal tubular acidosis type 4
D) Fanconi syndrome (aminoaciduria, glycosuria, phosphaturia)

Answer: D) Fanconi syndrome (aminoaciduria, glycosuria, phosphaturia)

Explanation: Lead can cause a reversible Fanconi-like proximal tubular dysfunction in children, with aminoaciduria, glycosuria and phosphaturia.

Q41. 'Saturnine gout' arises because lead:
A) Reduces renal urate excretion, raising serum urate
B) Increases dietary purine load
C) Causes vitamin C excess
D) Dissolves articular cartilage directly

Answer: A) Reduces renal urate excretion, raising serum urate

Explanation: Lead impairs renal urate handling, raising serum urate and precipitating gout, often in association with chronic lead nephropathy.

Q42. In children, dense transverse radiographic 'lead lines' appear at the:
A) Skull sutures
B) Vertebral end-plates only
C) Metaphyses of long bones (growth-arrest bands)
D) Distal phalangeal tufts only

Answer: C) Metaphyses of long bones (growth-arrest bands)

Explanation: Lead disturbs metaphyseal remodelling, producing dense growth-arrest bands at the growing ends of long bones in heavily exposed children.

Q43. Regarding reproduction, lead is recognised to:
A) Have no reproductive effect
B) Impair male fertility and increase miscarriage and stillbirth
C) Improve sperm quality
D) Affect only postmenopausal women

Answer: B) Impair male fertility and increase miscarriage and stillbirth

Explanation: Lead impairs spermatogenesis and raises the risk of miscarriage and stillbirth, and crosses the placenta, prompting protective limits around pregnancy.

Q44. Lead readily crosses the placenta, so the fetus is considered at risk once maternal blood lead exceeds roughly:
A) 300 micrograms/dL
B) 3 micrograms/dL
C) No level is relevant
D) 30 micrograms/dL

Answer: D) 30 micrograms/dL

Explanation: Maternal blood lead above about 30 micrograms/dL is taken to threaten the fetus, reflecting placental transfer and developmental neurotoxicity.

Diagnosis & Monitoring

Q45. The single standard biomarker guiding management of lead exposure is the:
A) Blood lead concentration
B) Serum calcium
C) Urinary glucose
D) Hair zinc level

Answer: A) Blood lead concentration

Explanation: Blood lead, measured on whole blood, is the primary index of exposure severity and guides decisions on follow-up, removal and chelation.

Q46. A worker with a raised blood lead but no symptoms should be regarded as:
A) Definitely and clinically poisoned by lead
B) Entirely normal and needing no further action
C) Significantly exposed and at an action level, not necessarily 'poisoned'
D) Beyond any benefit from medical intervention

Answer: C) Significantly exposed and at an action level, not necessarily 'poisoned'

Explanation: A raised blood lead without symptoms denotes significant exposure warranting action (removal and retest) rather than a diagnosis of clinical poisoning.

Q47. For very recent exposure (less than about two weeks), the most informative test is:
A) Zinc protoporphyrin
B) Blood ALA-dehydratase (ALAD) activity
C) Bone X-ray fluorescence
D) Hair lead

Answer: B) Blood ALA-dehydratase (ALAD) activity
Explanation: ALAD activity falls early and reflects very recent exposure; urinary ALA and then ZPP reflect progressively longer exposure windows.

Q48. For exposure of intermediate duration (about two weeks to two months), a useful index is:
A) Blood ALAD only
B) ZPP only
C) Serum sodium
D) Urinary delta-aminolaevulinic acid (ALA)

Answer: D) Urinary delta-aminolaevulinic acid (ALA)
Explanation: Urinary ALA rises over an intermediate window as ALAD inhibition causes ALA to accumulate and be excreted in urine.

Q49. For longer exposure (more than about two months), the recommended index is:
A) Zinc protoporphyrin (ZPP)
B) Blood ALAD
C) Urinary ALA
D) Random serum lead

Answer: A) Zinc protoporphyrin (ZPP)
Explanation: ZPP reflects the marrow effect of lead over the preceding months and is the recommended index for longer-term exposure and large-scale screening.

Q50. A limitation of ZPP as a screening test is that it:
A) Detects new exposure within a few minutes
B) Measures the skeletal bone-lead burden directly
C) Responds slowly and only to higher exposures, missing recent low-level exposure
D) Reliably diagnoses coexisting iron overload

Answer: C) Responds slowly and only to higher exposures, missing recent low-level exposure
Explanation: ZPP rises slowly and mainly at higher exposures, so it misses recent low-level exposure that blood lead would reveal; the two are complementary.

Q51. The long-term skeletal lead burden can be measured non-invasively by:
A) Spirometry
B) K-shell X-ray fluorescence (XRF)
C) Plain abdominal ultrasound
D) Skin-prick testing

Answer: B) K-shell X-ray fluorescence (XRF)
Explanation: K-shell X-ray fluorescence estimates cortical bone lead non-invasively, providing a measure of cumulative exposure used mainly in research.

Management

Q52. The first and most important step on identifying significant lead toxicity is to:
A) Begin lifelong prophylactic chelation
B) Transfuse packed red cells
C) Start high-dose corticosteroids
D) Remove the worker from further exposure

Answer: D) Remove the worker from further exposure
Explanation: Removing the worker from exposure halts further uptake and is the essential first measure; chelation is reserved for high levels or symptoms.

Q53. Oral chelation of moderate lead poisoning is commonly achieved with:
A) DMSA (succimer)
B) Deferoxamine
C) Atropine
D) Penicillin

Answer: A) DMSA (succimer)
Explanation: Oral DMSA (succimer), a water-soluble dimercaprol analogue, is used for moderate lead poisoning; penicillamine is an older oral alternative.

Q54. Calcium-disodium EDTA in lead poisoning is notable for:
A) Provoking the colic
B) Having no symptomatic effect
C) Relieving the abdominal colic within a few hours
D) Being effective only orally

Answer: C) Relieving the abdominal colic within a few hours
Explanation: Calcium-disodium EDTA, given by slow intravenous infusion, chelates lead and characteristically relieves lead colic within hours.

Q55. Lead encephalopathy is treated with chelation using:
A) Oral iron alone
B) Dimercaprol (BAL) together with calcium-disodium EDTA
C) A single dose of DMSA
D) No chelation at all

Answer: B) Dimercaprol (BAL) together with calcium-disodium EDTA

Explanation: Severe poisoning or encephalopathy is treated with BAL plus calcium-disodium EDTA, BAL being started first to limit redistribution of lead into the brain.

Q56. BAL is administered before EDTA in lead encephalopathy in order to:
A) Prevent an allergic reaction to EDTA
B) Avoid renal stone formation
C) Prevent hypoglycaemia
D) Avoid EDTA mobilising lead into the brain

Answer: D) Avoid EDTA mobilising lead into the brain

Explanation: Starting EDTA alone can mobilise lead and transiently raise brain levels; giving BAL first reduces the risk of redistribution into the central nervous system.

Q57. After a course of chelation, the blood lead may 'rebound' because:
A) Skeletal stores re-equilibrate lead back into blood
B) The laboratory assay is unreliable
C) Lead is endogenously regenerated
D) Iron stores rise sharply

Answer: A) Skeletal stores re-equilibrate lead back into blood

Explanation: Chelation lowers blood lead acutely, but bone stores re-equilibrate afterwards, so levels can rebound and must be rechecked before further decisions.

Prevention & Legislation

Q58. The primary (most effective) control of inorganic lead exposure is:
A) Respirators issued to all workers
B) Job rotation between tasks
C) Engineering control: enclosure and local exhaust ventilation
D) Health-education posters

Answer: C) Engineering control: enclosure and local exhaust ventilation

Explanation: Controlling fume and dust at source by enclosure and local exhaust ventilation is the primary control; respiratory protection is a lower-order supplement.

Q59. 'Medical removal protection' in a lead programme means:
A) Permanent dismissal of the over-exposed worker
B) Withdrawing an over-exposed worker, with income protection, until blood lead falls
C) Taking no action until symptoms finally appear
D) Routine prophylactic chelation of all workers

Answer: B) Withdrawing an over-exposed worker, with income protection, until blood lead falls

Explanation: Medical removal protection removes an over-exposed worker from lead work, with job and earnings protection, until the blood lead falls to a safe level.

Q60. Periodic biological monitoring of lead workers is essential because air monitoring alone:
A) Measures the internal absorbed dose
B) Is sufficient on its own
C) Detects anaemia directly
D) Misses ingestion and take-home contributions to dose

Answer: D) Misses ingestion and take-home contributions to dose

Explanation: Air sampling captures only inhaled lead; periodic blood lead monitoring reveals the additional ingested and take-home contributions to the internal dose.

Q61. Take-home (para-occupational) lead exposure of families is prevented by:

A) On-site changing, showering and separate laundering of work clothes
B) Laundering work clothing with family washing
C) Wearing work boots home
D) Sharing locker space with street clothes

Answer: A) On-site changing, showering and separate laundering of work clothes

Explanation: Lead carried on clothing, skin and hair can poison family members; on-site changing, showering and separate laundering interrupt this take-home pathway.

Q62. A pre-placement examination for a prospective lead worker should include:

A) Audiometry only
B) Chest computed tomography only
C) Baseline blood lead and a full blood count
D) Patch testing only

Answer: C) Baseline blood lead and a full blood count

Explanation: Baseline blood lead and haematology establish a reference for future comparison and detect any pre-existing burden or anaemia before lead work begins.

Q63. In occupational-health law, lead poisoning in a factory worker is:

A) Not legally recognised
B) A notifiable and compensable occupational disease
C) Compensable only after death
D) Reportable only for managers

Answer: B) A notifiable and compensable occupational disease

Explanation: Lead poisoning is a listed notifiable and compensable occupational disease, with statutory provisions for medical examination of exposed workers.

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UNIT II — METALS

Organic Lead

TOPICS IN THIS CHAPTER

Sources & Compounds

Q1. The principal organic lead compounds of occupational importance are:
A) Lead oxide and lead sulphide
B) Tetraethyl lead (TEL) and tetramethyl lead (TML)
C) Lead chromate and lead acetate
D) Lead carbonate and lead nitrate

Answer: B) Tetraethyl lead (TEL) and tetramethyl lead (TML)

Explanation: Organic lead is encountered chiefly as the petrol anti-knock additives tetraethyl lead and tetramethyl lead; the others listed are inorganic lead compounds.

Q2. Of the alkyl-lead compounds, the MOST toxic is:
A) Tetramethyl lead (TML)
B) Lead oxide
C) Lead chromate
D) Tetraethyl lead (TEL)

Answer: D) Tetraethyl lead (TEL)

Explanation: Tetraethyl lead is the most toxic organic lead form; tetramethyl lead is the least toxic of the two anti-knock additives.

Q3. Organic lead was used historically and industrially mainly as:
A) An anti-knock additive in petrol (automotive/aviation fuel)
B) A pottery glaze
C) A solder flux
D) A paint pigment

Answer: A) An anti-knock additive in petrol (automotive/aviation fuel)

Explanation: Tetraethyl lead served as an anti-knock additive in petrol, so the automotive and aviation-fuel sectors are the classic organic-lead exposures.

Q4. The classic high-exposure scenario for acute organic lead poisoning is:
A) Welding stainless steel
B) Spray-painting cars
C) Cleaning sludge from leaded-petrol storage tanks
D) Grinding castings

Answer: C) Cleaning sludge from leaded-petrol storage tanks

Explanation: Workers cleaning residual sludge from the inside of leaded-petrol storage tanks have classically suffered severe acute tetraethyl-lead poisoning.

Q5. Compared with inorganic lead, organic (alkyl) lead is distinctive in being:
A) Water-soluble and bone-seeking
B) Lipid-soluble, with toxicity dominated by the central nervous system
C) Non-absorbable by any route
D) Only a local skin irritant

Answer: B) Lipid-soluble, with toxicity dominated by the central nervous system

Explanation: Alkyl-lead compounds are lipophilic and readily enter the central nervous system, so their toxicity is predominantly neuropsychiatric, unlike inorganic lead.

Toxicokinetics & Metabolism

Q6. Because it is lipid-soluble, organic lead is well absorbed through:
A) Bone only
B) The renal tubule only
C) Tooth enamel
D) Skin as well as the lungs

Answer: D) Skin as well as the lungs

Explanation: Being lipophilic, alkyl lead is absorbed through intact skin as well as by inhalation, a route of little importance for inorganic lead.

Q7. Tetraethyl lead is first metabolised in the liver to:
A) Triethyl lead
B) Diethyl lead directly
C) Lead oxide
D) Elemental lead vapour

Answer: A) Triethyl lead

Explanation: TEL is de-alkylated in the liver to triethyl lead, the proximate toxic metabolite, which then de-alkylates further to diethyl lead.

Q8. The proximate TOXIC metabolite of tetraethyl lead is considered to be:
A) Diethyl lead
B) Tetramethyl lead
C) Triethyl lead
D) Lead chromate

Answer: C) Triethyl lead

Explanation: Triethyl lead, the first de-alkylation product, is regarded as the proximate toxic species responsible for organic lead's neurotoxicity.

Q9. The triethyl-lead metabolite de-alkylates further to:
A) Tetraethyl lead again
B) Diethyl lead, which is excreted in bile
C) Lead sulphide
D) Insoluble lead oxide

Answer: B) Diethyl lead, which is excreted in bile

Explanation: Triethyl lead spontaneously de-alkylates to diethyl lead, which is excreted in bile and further de-alkylated in the gut to ionic lead.

Q10. The only metabolite of tetraethyl lead that characteristically appears in the URINE is:
A) Triethyl lead
B) Tetramethyl lead
C) Lead chromate
D) Diethyl lead

Answer: D) Diethyl lead

Explanation: Diethyl lead is the urinary metabolite of TEL, which is why urinary lead measurement is central to monitoring organic-lead exposure.

Q11. Like inorganic lead, organic lead also:
A) Crosses the blood-brain barrier and the placenta
B) Is confined to red cells
C) Cannot enter the nervous system
D) Is excreted only in sweat

Answer: A) Crosses the blood-brain barrier and the placenta

Explanation: Lipophilic organic lead readily crosses the blood-brain barrier (accounting for its CNS toxicity) and the placenta, threatening the fetus.

Mechanism & Contrast

Q12. The dominant toxic effect of organic lead is on the:
A) Bone marrow
B) Renal tubule
C) Central nervous system
D) Gastrointestinal tract

Answer: C) Central nervous system

Explanation: Organic lead is principally a central neurotoxin, in contrast to inorganic lead, whose hallmark effects are on haem synthesis and the peripheral nerves.

Q13. Which feature of INORGANIC lead poisoning is typically ABSENT or minor in organic lead poisoning?
A) Central nervous system disturbance
B) Basophilic stippling and prominent anaemia
C) Insomnia and psychiatric change
D) Raised urinary lead

Answer: B) Basophilic stippling and prominent anaemia

Explanation: The haematological hallmarks of inorganic lead (anaemia, basophilic stippling) are not prominent in organic lead, whose picture is neuropsychiatric.

Q14. Unlike inorganic lead, organic lead poisoning is NOT characterised by:
A) Insomnia and vivid dreams
B) Anxiety and hallucinations
C) Tremor and hyperexcitability
D) A peripheral motor neuropathy with wrist drop

Answer: D) A peripheral motor neuropathy with wrist drop

Explanation: Wrist drop from a peripheral motor neuropathy is a feature of inorganic lead; organic lead instead produces a central, neuropsychiatric syndrome.

Q15. The Burton (blue) gum line is:
A) A feature of inorganic lead, not typical of organic lead
B) Pathognomonic of organic lead
C) Seen only in tetraethyl lead poisoning
D) Caused by triethyl lead in the gums

Answer: A) A feature of inorganic lead, not typical of organic lead

Explanation: The Burton line reflects lead sulphide deposition seen with inorganic lead exposure; it is not a characteristic feature of organic lead poisoning.

Clinical Features

Q16. Organic lead poisoning characteristically presents with a syndrome that is predominantly:
A) Haematological
B) Renal
C) Neuropsychiatric
D) Skeletal

Answer: C) Neuropsychiatric

Explanation: Organic lead produces a predominantly neuropsychiatric illness, reflecting its lipid solubility and ready entry into the central nervous system.

Q17. Early symptoms of organic lead intoxication often include:
A) Acute wrist drop from a motor neuropathy
B) Abdominal discomfort, anorexia and numbness of the head and hands
C) Painless obstructive jaundice with pale stools
D) Bony swelling and necrosis of the jaw

Answer: B) Abdominal discomfort, anorexia and numbness of the head and hands

Explanation: Organic lead often begins with abdominal discomfort and anorexia together with numbness of the head and hands, before frank neuropsychiatric features.

Q18. Sleep and perceptual disturbances in organic lead poisoning include:
A) Excessive somnolence only
B) Complete deafness
C) Painful red eyes only
D) Insomnia, vivid dreams and hearing strange sounds

Answer: D) Insomnia, vivid dreams and hearing strange sounds

Explanation: Organic lead disturbs sleep with insomnia and vivid dreams and may cause abnormal auditory experiences, part of its neuropsychiatric picture.

Q19. A visual symptom reported in organic lead poisoning is:
A) Blurred vision, especially in dim light
B) Sudden painless permanent blindness
C) Bitemporal hemianopia
D) Central scotoma with pain

Answer: A) Blurred vision, especially in dim light

Explanation: Blurred vision, particularly in dim light, is among the reported neuropsychiatric features of organic lead intoxication.

Q20. As organic lead poisoning advances, the patient may develop:
A) Constipation and a blue gum line only
B) Wrist drop and foot drop only
C) Anxiety, tremor, hyperexcitability, delusions and hallucinations
D) A microcytic anaemia only

Answer: C) Anxiety, tremor, hyperexcitability, delusions and hallucinations

Explanation: Progressive organic lead poisoning causes anxiety, tremor, hyperexcitability and an organic psychosis with delusions and hallucinations, sometimes mania.

Q21. Severe acute tetraethyl lead poisoning may culminate in:
A) A self-limiting skin rash
B) Convulsions, coma and death
C) Isolated hearing loss
D) Reversible wrist drop only

Answer: B) Convulsions, coma and death

Explanation: Severe organic lead poisoning can progress through psychosis and hyperexcitability to convulsions, coma and death, a potentially fatal encephalopathy.

Q22. A recognised autonomic feature of severe tetraethyl lead poisoning is a triad of:
A) Fever, hypertension and tachycardia
B) Hyperthermia and flushing
C) Hypertension with bradycardia only
D) Hypothermia, hypotension and bradycardia

Answer: D) Hypothermia, hypotension and bradycardia

Explanation: Severe tetraethyl lead poisoning is classically described with low body temperature, low blood pressure and a slow pulse, alongside the CNS disturbance.

Q23. The onset of organic lead symptoms relative to any rise in blood lead is that symptoms may:
A) Develop earlier than any rise in blood level
B) Appear only after blood lead is grossly raised
C) Never precede laboratory change
D) Correlate exactly with blood lead

Answer: A) Develop earlier than any rise in blood level

Explanation: In organic lead poisoning, clinical symptoms may develop before the blood lead rises, so reliance on blood lead can dangerously underestimate toxicity.

Diagnosis & Monitoring

Q24. In organic lead poisoning, the blood lead level is:

A) The single most reliable diagnostic test available
B) Always grossly elevated well above normal
C) Often unhelpful, as it may rise little (rarely above ~50 micrograms/dL) or not at all
D) Identical in behaviour to inorganic lead in blood

Answer: C) Often unhelpful, as it may rise little (rarely above ~50 micrograms/dL) or not at all

Explanation: Blood lead frequently fails to rise much in organic lead poisoning (often staying below ~50 micrograms/dL), so it is an unreliable index of toxicity.

Q25. The most helpful confirmatory laboratory test in organic lead poisoning is:

A) Blood lead level
B) Urinary lead concentration
C) Serum calcium
D) Hair calcium

Answer: B) Urinary lead concentration

Explanation: Urinary lead is the most useful confirmatory test for organic lead, since blood lead is unreliable; the urine reflects the diethyl-lead metabolite.

Q26. A commonly quoted normal range for urinary lead is approximately:

A) 1-2 micrograms/litre
B) 5-10 milligrams/litre
C) No measurable level
D) 150-350 micrograms/litre

Answer: D) 150-350 micrograms/litre

Explanation: A normal urinary lead of roughly 150-350 micrograms/litre is quoted; values above this support significant organic-lead exposure when blood lead is unhelpful.

Q27. Besides urinary lead, which additional indices may be measured in organic lead exposure?

A) Zinc protoporphyrin and porphyrin levels
B) Serum sodium and potassium
C) Blood glucose and HbA1c
D) Serum amylase and lipase

Answer: A) Zinc protoporphyrin and porphyrin levels

Explanation: ZPP and porphyrin levels may be assessed in organic-lead exposure, complementing the key measurement of urinary lead.

Q28. Why is biological monitoring of organic lead based on urine rather than blood?

A) Venous blood cannot be sampled safely in these workers
B) Urine is simply cheaper and easier to collect
C) The urinary diethyl-lead metabolite reflects exposure, whereas blood lead rises little
D) Blood lead consistently overestimates the true exposure

Answer: C) The urinary diethyl-lead metabolite reflects exposure, whereas blood lead rises little

Explanation: Because diethyl lead, the urinary metabolite, reflects organic-lead exposure while blood lead rises little, urine is the preferred monitoring medium.

Treatment

Q29. A fundamental difference in treating organic lead poisoning is that:

A) Calcium-disodium EDTA is curative
B) Chelating agents are NOT useful
C) Dimercaprol (BAL) is first-line
D) Penicillamine is mandatory

Answer: B) Chelating agents are NOT useful

Explanation: Chelation is ineffective in organic lead poisoning, in sharp contrast to inorganic lead, so management is essentially supportive.

Q30. Why is chelation ineffective in organic lead poisoning?

A) Chelating agents markedly worsen the associated anaemia
B) Organic lead is not absorbed into the body at all
C) There is effectively no lead present to be removed
D) The toxic alkyl-lead metabolites are not bound or removed effectively by chelators

Answer: D) The toxic alkyl-lead metabolites are not bound or removed effectively by chelators

Explanation: The toxic organic (alkyl) lead metabolites are not effectively bound and excreted by the standard chelators, so chelation does not help.

Q31. Symptomatic management of the psychiatric features of organic lead poisoning may use:
A) Diazepam
B) High-dose calcium-disodium EDTA
C) Oral iron
D) Atropine

Answer: A) Diazepam

Explanation: As chelation is unhelpful, organic lead is managed supportively, with diazepam (a benzodiazepine) used for agitation, psychosis and seizures.

Q32. The first and essential step in managing organic lead exposure is:
A) Lifelong chelation
B) Immediate exchange transfusion
C) Removal from exposure and decontamination (including skin)
D) Thyroidectomy

Answer: C) Removal from exposure and decontamination (including skin)

Explanation: As with all metals, stopping exposure is the essential first step; because organic lead is absorbed through skin, decontamination of skin and clothing matters.

Q33. Overall, management of organic lead poisoning is best described as:
A) Aggressive chelation as for inorganic lead
B) Largely supportive, with sedation and removal from exposure
C) A single curative antidote
D) Surgical

Answer: B) Largely supportive, with sedation and removal from exposure

Explanation: Organic lead poisoning is managed supportively — removal from exposure, decontamination and sedation (diazepam) — because no effective chelation exists.

Prevention & Legislation

Q34. A key reason organic lead is now a much smaller public-health problem is:
A) The banning of lead-acid batteries
B) The end of all smelting
C) The disappearance of soldering
D) The phase-out of leaded petrol

Answer: D) The phase-out of leaded petrol

Explanation: The worldwide phase-out of tetraethyl-lead petrol has greatly reduced environmental and occupational organic-lead exposure, though niche uses persist.

Q35. Because organic lead is absorbed through skin, a particularly important control is:
A) Preventing skin contact with the liquid and contaminated surfaces
B) Pure-tone audiometry repeated at intervals
C) Dietary calcium and vitamin supplementation
D) Routine prophylactic chelation of workers

Answer: A) Preventing skin contact with the liquid and contaminated surfaces

Explanation: Skin absorption makes prevention of dermal contact (impervious PPE, decontamination, no skin exposure to liquid or sludge) a particularly important control.

Q36. Residual niche exposures to tetraethyl lead may still occur in:
A) Modern unleaded-petrol filling stations
B) Electric-vehicle battery plants
C) Aviation gasoline (avgas) handling and tank cleaning
D) Solar-panel manufacture

Answer: C) Aviation gasoline (avgas) handling and tank cleaning

Explanation: Leaded aviation gasoline and the cleaning of old leaded-petrol tanks remain residual sources of organic-lead exposure despite the petrol phase-out.

Q37. Like inorganic lead, organic lead exposure warrants:

A) No monitoring at all
B) Biological monitoring (urinary lead) and health surveillance of exposed workers
C) Monitoring only after symptoms
D) Air monitoring as the sole measure

Answer: B) Biological monitoring (urinary lead) and health surveillance of exposed workers

Explanation: Exposed workers warrant biological monitoring (urinary lead, given blood lead's unreliability) and health surveillance, alongside exposure control.

13

UNIT II — METALS

Arsenic

TOPICS IN THIS CHAPTER

Sources

Q1. Occupational and environmental arsenic exposure occurs in all EXCEPT:
A) Smelting of metal ores
B) Cotton ginning
C) Pesticide/wood-preservative manufacture
D) Contaminated groundwater (drinking)

Answer: B) Cotton ginning

Explanation: Smelting, pesticides/preservatives and contaminated water are key arsenic sources; cotton ginning relates to byssinosis, not arsenic.

Q2. Large-scale environmental arsenic poisoning from groundwater has notably affected:
A) The Arctic
B) High-altitude deserts
C) Polar oceans
D) West Bengal and Bangladesh

Answer: D) West Bengal and Bangladesh

Explanation: Naturally arsenic-rich aquifers in West Bengal and Bangladesh have caused mass chronic arsenicosis from drinking water.

Q3. Gallium arsenide exposure is a modern hazard in the:
A) Semiconductor industry
B) Bakery industry
C) Textile industry
D) Dairy industry

Answer: A) Semiconductor industry

Explanation: Gallium arsenide is used in semiconductors and optoelectronics, an emerging source of occupational arsenic exposure.

Q4. A historical medicinal and homicidal arsenic compound was:
A) Sodium chloride
B) Calcium carbonate
C) Arsenic trioxide ('inheritance powder')
D) Magnesium sulfate

Answer: C) Arsenic trioxide ('inheritance powder')

Explanation: Arsenic trioxide, tasteless and once widely available, was a notorious poison ('inheritance powder') and features in toxicology history.

Historical Note

Q5. Arsenic trioxide was historically the ideal homicidal poison ('inheritance powder') because it was:
A) Bright blue and bitter-tasting
B) Tasteless, odourless and colourless, with cholera-like effects
C) Immediately and visibly corrosive
D) Detectable by simple smell

Answer: B) Tasteless, odourless and colourless, with cholera-like effects

Explanation: White arsenic was tasteless, odourless and colourless, and its vomiting and diarrhoea mimicked natural illnesses such as cholera, so poisonings went unnoticed.

Q6. The forensic test that first allowed arsenic to be reliably detected in the body, ending its era as the 'perfect poison', was the:
A) Schick test
B) Mantoux test
C) Benedict test
D) Marsh test (James Marsh, 1836)

Answer: D) Marsh test (James Marsh, 1836)

Explanation: The Marsh test, devised by James Marsh in 1836, detected minute amounts of arsenic in tissue, enabling convictions and ending arsenic's reign as an undetectable poison.

Acute Toxicity

Q7. Acute arsenic poisoning characteristically causes:
A) Severe vomiting and 'rice-water' diarrhoea with shock
B) Painless jaundice
C) Isolated wrist drop
D) Acute arthritis

Answer: A) Severe vomiting and 'rice-water' diarrhoea with shock

Explanation: Acute arsenic causes violent gastroenteritis with rice-water stools, dehydration and shock, sometimes with cardiac arrhythmia and encephalopathy.

Q8. A classic breath odour described in arsenic poisoning is:
A) A purely neurological syndrome without haemolysis
B) Only localised skin changes at contact sites
C) Garlic-like
D) No measurable toxic effect at any dose

Answer: C) Garlic-like

Explanation: A garlic-like breath/odour is described with arsenic; bitter almonds suggests cyanide and rotten eggs hydrogen sulfide.

Q9. Arsine gas exposure differs from other arsenic exposure in causing:
A) A purely neurological syndrome
B) Massive intravascular haemolysis and haemoglobinuria
C) Only skin changes
D) No toxicity

Answer: B) Massive intravascular haemolysis and haemoglobinuria

Explanation: Arsine (AsH_3) is a potent haemolytic agent, causing acute intravascular haemolysis, haemoglobinuria and renal failure, distinct from other arsenic forms.

Q10. Acute arsenic poisoning may cause a cardiac effect of:
A) Bradycardia only
B) No cardiac effect
C) Pericarditis only
D) QT prolongation and arrhythmia

Answer: D) QT prolongation and arrhythmia

Explanation: Acute arsenic can prolong the QT interval and provoke ventricular arrhythmia, contributing to cardiovascular collapse in severe poisoning.

Q11. The mechanism underlying acute arsenic gastroenteritis is:
A) Dilatation and increased permeability of small gut blood vessels
B) Direct corrosive mucosal burns of the gut only
C) An IgE-mediated allergic reaction in the bowel
D) A secondary bacterial infection of the gut

Answer: A) Dilatation and increased permeability of small gut blood vessels

Explanation: Acute inorganic arsenic dilates and increases the permeability of small gut (and other) vessels, producing the cholera-like rice-water diarrhoea.

Chronic - Skin

Q12. Chronic arsenicosis classically affects the skin with:
A) Bullous pemphigoid
B) Vitiligo only
C) Hyperpigmentation and palmoplantar hyperkeratosis
D) Acne only

Answer: C) Hyperpigmentation and palmoplantar hyperkeratosis

Explanation: Chronic arsenic produces diffuse hyperpigmentation with raindrop depigmentation and palmar and plantar hyperkeratoses (arsenical keratoses), a classic dermatological signature.

Q13. Transverse white lines across the nails in chronic arsenic poisoning are:
A) Burton's lines
B) Mees' lines
C) Beau-Reil only
D) Muehrcke only specifically

Answer: B) Mees' lines

Explanation: Mees' lines are transverse white bands crossing the nails after arsenic (and some other) exposures, reflecting a growth disturbance.

Q14. Arsenical skin cancer characteristically includes:
A) Malignant melanoma of sun-exposed skin only
B) Kaposi sarcoma of the skin and mucosa
C) No increase in any form of skin cancer
D) Bowen disease (squamous carcinoma in situ) and basal/squamous carcinomas

Answer: D) Bowen disease (squamous carcinoma in situ) and basal/squamous carcinomas

Explanation: Chronic arsenic causes multiple Bowen lesions and basal/squamous cell carcinomas, often on non-sun-exposed skin, decades after exposure.

Q15. Bowen disease, one of arsenic's skin cancers, is a form of:
A) Squamous cell carcinoma in situ
B) Basal cell carcinoma
C) Melanoma
D) Sarcoma

Answer: A) Squamous cell carcinoma in situ

Explanation: Bowen disease is squamous carcinoma in situ; arsenic causes it along with invasive squamous and basal cell carcinomas of the skin.

Chronic - Cancer

Q16. Arsenic is an IARC Group 1 carcinogen causing cancer of the:
A) Brain and bone
B) Thyroid and testis
C) Skin, lung and urinary bladder
D) Pancreas and stomach only

Answer: C) Skin, lung and urinary bladder

Explanation: Arsenic causes skin (including Bowen disease), lung and bladder cancers; it is a definite (Group 1) human carcinogen.

Chronic - Vascular & Neuro

Q17. Chronic arsenic exposure causes a peripheral neuropathy that is:
A) Purely motor with wrist drop
B) Predominantly sensorimotor (often sensory first)
C) Purely cranial
D) Absent

Answer: B) Predominantly sensorimotor (often sensory first)

Explanation: Arsenic causes a length-dependent sensorimotor polyneuropathy (typically sensory-predominant early), contrasting with the motor neuropathy of lead.

Q18. 'Blackfoot disease', a peripheral vascular gangrene, is linked to chronic:
A) Lead exposure
B) Mercury exposure
C) Cadmium exposure
D) Arsenic exposure

Answer: D) Arsenic exposure

Explanation: Blackfoot disease, an obliterative peripheral arteriopathy with gangrene, was described in arsenic-endemic areas of Taiwan.

Q19. Chronic arsenic ingestion is also linked to systemic effects including:
A) Peripheral vascular disease, hypertension and diabetes
B) Improved circulation
C) Lowered cancer risk
D) Bone overgrowth

Answer: A) Peripheral vascular disease, hypertension and diabetes

Explanation: Beyond skin and nerve, chronic arsenic is associated with peripheral vascular disease (blackfoot), hypertension and an increased diabetes risk.

Biological Monitoring

Q20. The best biomarker of recent inorganic arsenic exposure is:
A) Blood lead
B) Serum calcium
C) Speciated urinary arsenic
D) Hair zinc

Answer: C) Speciated urinary arsenic

Explanation: Speciated urinary arsenic (inorganic plus methylated metabolites) reflects recent exposure; total urinary arsenic is confounded by dietary seafood arsenic.

Q21. To avoid false elevation of urinary arsenic, workers should avoid before testing:
A) Drinking water
B) Seafood (organic arsenobetaine)
C) Green vegetables
D) Dairy products

Answer: B) Seafood (organic arsenobetaine)

Explanation: Seafood contains non-toxic organoarsenicals that inflate total urinary arsenic, so seafood is avoided before testing or speciation is used.

Q22. Why are hair and nails useful for chronic arsenic assessment?
A) They filter blood
B) They store inorganic mercury
C) They reflect today's air level
D) Arsenic binds keratin, recording past exposure along the growth

Answer: D) Arsenic binds keratin, recording past exposure along the growth

Explanation: Arsenic binds sulfhydryl groups in keratin, so hair and nails preserve a temporal record of past exposure, complementing urinary arsenic.

Q23. The airborne arsenic TWA is recommended not to exceed about:
A) 50 micrograms/m3
B) 500 micrograms/m3
C) 5 mg/m3
D) No limit

Answer: A) 50 micrograms/m3

Explanation: Airborne arsenic (TWA) should not exceed roughly 50 micrograms/m3, and urinary arsenic is monitored against a similar reference for exposure control.

Management

Q24. Chelation of significant inorganic arsenic poisoning uses:
A) Deferoxamine
B) Atropine
C) Dimercaprol (BAL), DMSA or DMPS
D) Calcium gluconate

Answer: C) Dimercaprol (BAL), DMSA or DMPS

Explanation: Arsenic is chelated with dimercaprol or its water-soluble analogues DMSA/DMPS; deferoxamine is specific for iron.

Q25. Arsine-induced haemolysis is managed primarily by:
A) Routine dimercaprol chelation used alone
B) Supportive care, transfusion and renal support (not standard chelation)
C) Oral iron replacement to correct the anaemia
D) Calcium-disodium EDTA chelation used alone

Answer: B) Supportive care, transfusion and renal support (not standard chelation)

Explanation: Arsine haemolysis is treated supportively with transfusion and management of renal failure; conventional chelation is of limited value for the haemolysis.

Q26. A key supportive priority in acute arsenic gastroenteritis is:
A) Withholding all fluids
B) Immediate chelation alone
C) Antibiotics alone
D) Aggressive fluid resuscitation

Answer: D) Aggressive fluid resuscitation

Explanation: Massive fluid loss from rice-water diarrhoea demands vigorous rehydration alongside chelation to prevent hypovolaemic shock and renal injury.

Forms & Toxicokinetics

Q27. The biological half-life of arsenic in blood is approximately:
A) 60 hours
B) 6 hours
C) 60 days
D) 6 minutes

Answer: A) 60 hours

Explanation: Arsenic has a blood half-life of about 60 hours and is rapidly eliminated in urine, unchanged or after biotransformation.

Chronic - Neuro

Q28. The arsenic peripheral neuropathy is attributed to interference with:
A) Myelin synthesis only
B) Sodium channels only
C) Glycolysis within neurons
D) Acetylcholine release

Answer: C) Glycolysis within neurons

Explanation: Arsenic impairs neuronal glycolysis, producing a mixed sensorimotor neuropathy with paraesthesiae, vibration loss and calf pain followed by weakness.

Chronic - Liver

Q29. Hepatic effects of chronic arsenic (enlargement, cirrhosis) are due to:
A) An autoimmune attack
B) Inhibition of enzymes of intracellular respiration
C) Fatty infiltration only
D) Viral co-infection

Answer: B) Inhibition of enzymes of intracellular respiration

Explanation: Chronic arsenic inhibits enzymes of intracellular respiration, causing liver enlargement and cirrhosis among its systemic effects.

Chronic - Blood

Q30. The anaemia of chronic arsenic poisoning is characteristically:
A) Microcytic with stippling
B) Haemolytic only
C) Aplastic only
D) Megaloblastic (interference with SH-enzymes of haem synthesis)

Answer: D) Megaloblastic (interference with SH-enzymes of haem synthesis)

Explanation: Chronic arsenic interferes with sulphydryl-group enzymes of haem synthesis, producing a megaloblastic anaemia, distinct from the picture of lead.

Chronic - Respiratory

Q31. A painless upper-airway lesion that may occur soon after arsenic exposure is:

A) Perforation of the nasal cartilaginous septum
B) Pleural plaque
C) Lobar consolidation
D) Eggshell calcification

Answer: A) Perforation of the nasal cartilaginous septum

Explanation: Erosive lesions of the nasal mucosa can painlessly perforate the nasal cartilaginous septum within a short time, alongside bronchitis and lung cancer risk.

Arsine Gas

Q32. Arsine gas is formed whenever arsenic contacts:

A) Carbon dioxide
B) Water vapour
C) Nascent (freshly generated) hydrogen
D) Nitrogen

Answer: C) Nascent (freshly generated) hydrogen

Explanation: Arsine (AsH_3) forms when arsenic meets nascent hydrogen ($As + 3H \rightarrow AsH_3$), as in smelting, galvanising and semiconductor processes.

Q33. The commonest cause of occupational jaundice, occurring 24-48 hours after exposure, is:

A) Lead colic
B) Arsine gas (haemolytic jaundice)
C) Cadmium fume
D) Manganese dust

Answer: B) Arsine gas (haemolytic jaundice)

Explanation: Arsine causes haemolytic jaundice within 24-48 hours, regarded as the commonest cause of occupational jaundice, with haemoglobinuria and renal failure.

Q34. Treatment of significant arsine-induced haemolysis centres on:

A) Routine dimercaprol chelation used on its own
B) Oral iron supplementation to replace losses
C) Calcium-disodium EDTA chelation used alone
D) Exchange blood transfusion (forced diuresis in mild cases)

Answer: D) Exchange blood transfusion (forced diuresis in mild cases)

Explanation: Arsine haemolysis is treated by exchange blood transfusion, with forced diuresis in mild haemoglobinuria; conventional chelation does little for the haemolysis.

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UNIT II — METALS

Cadmium

TOPICS IN THIS CHAPTER

Sources

Q1. Occupational cadmium exposure occurs in all EXCEPT:
A) Cotton weaving
B) Nickel-cadmium battery manufacture
C) Smelting and electroplating
D) Pigment and welding/soldering work

Answer: A) Cotton weaving

Explanation: Battery, smelting, plating and pigment work expose to cadmium; cotton weaving relates to byssinosis, not cadmium.

Q2. A major non-occupational source of cadmium intake in the general population is:
A) Drinking water fluoride
B) Iodised salt
C) Cigarette smoking
D) Sunlight

Answer: C) Cigarette smoking

Explanation: Tobacco accumulates cadmium, so smoking is a major source of body cadmium in the general population, adding to any occupational exposure.

Acute Toxicity

Q3. Acute inhalation of cadmium fume causes:
A) Acute glomerular injury with heavy albuminuria
B) Chemical pneumonitis and pulmonary oedema
C) An acute, fully reversible tubular insult
D) A primarily nephrotic pattern of disease

Answer: B) Chemical pneumonitis and pulmonary oedema

Explanation: Heavy cadmium-fume inhalation causes a delayed chemical pneumonitis and pulmonary oedema, potentially fatal, after an initial metal-fume-fever-like illness.

Q4. Acute cadmium-oxide fume inhalation typically causes symptoms after a latent period of about:
A) 10 minutes
B) 10 days
C) No delay
D) 10 hours

Answer: D) 10 hours

Explanation: Acute cadmium-fume inhalation characteristically causes delayed (about 10 hours later) retrosternal pain, dyspnoea, cough and a metal-fume-fever-like illness.

Chronic - Renal

Q5. The hallmark of chronic cadmium toxicity is damage to the:
A) Renal proximal tubule
B) Cerebellum
C) Bone marrow
D) Optic nerve

Answer: A) Renal proximal tubule

Explanation: Cadmium accumulates in the renal cortex and damages the proximal tubule, producing low-molecular-weight proteinuria and progressive nephropathy.

Q6. Cadmium proximal tubular damage is detected by:
A) Heavy albuminuria only
B) Glycosuria alone
C) Low-molecular-weight proteinuria (beta-2-microglobulin)
D) Haematuria

Answer: C) Low-molecular-weight proteinuria (beta-2-microglobulin)

Explanation: Tubular injury leaks low-molecular-weight proteins such as beta-2-microglobulin into urine, the standard effect biomarker for cadmium.

Q7. Cadmium nephropathy is typically:
A) Fully reversible on chelation
B) Largely irreversible once established
C) Acute and self-limiting
D) A glomerular nephrotic picture

Answer: B) Largely irreversible once established

Explanation: Cadmium-induced tubular damage is generally irreversible and may progress even after exposure stops, underscoring prevention over treatment.

Q8. The earliest sign of chronic cadmium nephropathy is:
A) Anuria
B) Haematuria
C) Glycosuria alone
D) Proteinuria (low-molecular-weight)

Answer: D) Proteinuria (low-molecular-weight)

Explanation: Proteinuria is the earliest renal sign, with low-molecular-weight proteins such as beta-2-microglobulin and retinol-binding protein appearing in urine.

Q9. A urinary enzyme marker of cadmium tubular injury is:
A) N-acetyl-beta-D-glucosaminidase (NAG)
B) Amylase
C) Alkaline phosphatase only
D) Creatine kinase

Answer: A) N-acetyl-beta-D-glucosaminidase (NAG)

Explanation: A raised urinary N-acetyl-beta-D-glucosaminidase, together with beta-2-microglobulin and retinol-binding protein, marks the proximal tubular damage caused by chronic cadmium exposure.

Q10. Cadmium nephropathy also produces urinary changes including:
A) Heavy albuminuria only
B) Pure haematuria
C) Aminoaciduria, glycosuria, phosphaturia and calcium stones
D) No urinary change

Answer: C) Aminoaciduria, glycosuria, phosphaturia and calcium stones

Explanation: Beyond low-molecular-weight proteinuria, cadmium proximal tubular damage causes aminoaciduria, glycosuria, phosphaturia, increased urinary calcium and the formation of calcium stones.

Chronic - Bone

Q11. Chronic cadmium toxicity affects bone, causing:
A) Osteopetrosis
B) Osteomalacia and osteoporosis with fractures
C) Avascular necrosis only
D) Paget disease

Answer: B) Osteomalacia and osteoporosis with fractures

Explanation: Cadmium (partly via renal phosphate/vitamin-D handling) causes osteomalacia and osteoporosis, with bone pain and fractures.

Q12. 'Itai-itai' disease, marked by severe bone pain and fractures, is caused by:
A) Lead exposure
B) Mercury exposure
C) Manganese exposure
D) Cadmium exposure

Answer: D) Cadmium exposure

Explanation: Itai-itai ('ouch-ouch') disease, from cadmium-contaminated rice in Japan, combined painful osteomalacia with renal tubular dysfunction.

Q13. Bone disease in chronic cadmium toxicity is partly secondary to:
A) Renal loss of calcium and phosphate and disturbed vitamin D
B) An excess of activated vitamin D in the circulation
C) A high dietary calcium intake overwhelming the gut
D) Iron overload depositing in the renal tubules

Answer: A) Renal loss of calcium and phosphate and disturbed vitamin D

Explanation: Tubular damage causes urinary calcium/phosphate loss and impaired vitamin-D activation, contributing to the osteomalacia of cadmium toxicity.

Chronic - Lung

Q14. Chronic cadmium inhalation is associated with:
A) Asthma cure
B) Pleural plaques
C) Emphysema/COPD and lung cancer
D) Mesothelioma

Answer: C) Emphysema/COPD and lung cancer

Explanation: Long-term cadmium-fume inhalation causes emphysema and is a recognised (IARC Group 1) lung carcinogen; it does not cause plaques or mesothelioma.

Biological Monitoring

Q15. Urinary cadmium chiefly reflects:
A) Today's air level only
B) Body burden / long-term accumulation
C) Iron stores
D) Renal blood flow

Answer: B) Body burden / long-term accumulation

Explanation: Urinary cadmium correlates with the accumulated body burden, whereas blood cadmium better reflects recent exposure.

Q16. Blood cadmium is a better index of:
A) Lifetime bone stores
B) Iron status
C) Lung function
D) Recent exposure

Answer: D) Recent exposure

Explanation: Blood cadmium reflects relatively recent exposure, complementing urinary cadmium (body burden) and beta-2-microglobulin (tubular effect).

Q17. Once beta-2-microglobulinuria from cadmium is established, it is:
A) Generally irreversible and may progress
B) Quickly reversible
C) A normal finding
D) Due to glomerular disease

Answer: A) Generally irreversible and may progress

Explanation: Established cadmium tubular proteinuria tends to persist or progress even after exposure ceases, marking permanent tubular injury.

Management

Q18. Chelation in cadmium poisoning is:
A) First-line with EDTA
B) Curative with DMSA
C) Generally avoided, as it can worsen nephrotoxicity
D) Always with BAL

Answer: C) Generally avoided, as it can worsen nephrotoxicity

Explanation: Cadmium chelation can redistribute the metal to the kidney and aggravate renal damage, so management is supportive with removal from exposure.

Q19. The mainstay of managing chronic cadmium toxicity is:
A) Aggressive chelation to remove stored cadmium
B) Removal from exposure and supportive renal/bone care
C) Long-term systemic corticosteroid therapy
D) Thyroidectomy to remove the affected gland

Answer: B) Removal from exposure and supportive renal/bone care

Explanation: Because effective safe chelation is lacking, management centres on stopping exposure and supporting renal and skeletal complications.

Sources & Metabolism

Q20. Cadmium occurs naturally with, and is obtained as a by-product of smelting, which metal's ores?
A) Iron
B) Aluminium
C) Tin
D) Zinc

Answer: D) Zinc

Explanation: Cadmium occurs with zinc and is recovered as a by-product of zinc (and lead) ore smelting, a key occupational source.

Q21. Gastrointestinal absorption of ingested cadmium is approximately:
A) Only about 6%
B) About 90%
C) Nearly 100%
D) Zero

Answer: A) Only about 6%

Explanation: Only about 6% of ingested cadmium is absorbed; the lung is the main route of significant absorption, especially of cadmium-oxide fume.

Q22. About 50% of the cadmium body burden is held in the:
A) Brain
B) Bone only
C) Liver and kidneys
D) Lungs only

Answer: C) Liver and kidneys

Explanation: Roughly half the cadmium body burden resides in the liver and kidneys; in blood about 70% is bound to red cells.

Q23. The biological half-life of cadmium in the body is approximately:
A) About 10 hours
B) About 10 years
C) About 10 days
D) About 100 years

Answer: B) About 10 years

Explanation: Cadmium has a very long biological half-life of around 10 years, reflecting tight binding to metallothionein and slow excretion.

Q24. Cadmium is stored and excreted largely bound to:
A) Albumin only
B) Haemoglobin only
C) Transferrin
D) Metallothionein

Answer: D) Metallothionein

Explanation: Cadmium binds metallothionein (a metal-storage protein) in tissues and is excreted in urine as the cadmium-metallothionein complex, slowly.

Chronic - Other

Q25. Besides renal and bone effects, chronic cadmium can cause anaemia by interfering with:

A) Copper metabolism
B) Folate absorption
C) Vitamin B12 only
D) Iron storage only

Answer: A) Copper metabolism

Explanation: Chronic cadmium can cause anaemia partly by interfering with copper metabolism, in addition to its renal, pulmonary and skeletal effects.

Itai-itai

Q26. The osteomalacia of itai-itai disease is explained by mechanisms including:

A) An excess of activated vitamin D in the blood
B) Greatly increased calcium absorption from the gut
C) A Fanconi-like tubular defect and disturbed vitamin-D activation
D) Iron overload deposited within the bones

Answer: C) A Fanconi-like tubular defect and disturbed vitamin-D activation

Explanation: Itai-itai osteomalacia arises from a Fanconi-like phosphate-wasting tubular defect and impaired renal activation of vitamin D (calcitriol).

Q27. Itai-itai disease is dominated clinically by:

A) Acute intravascular haemolysis with jaundice
B) Bone pain in the back and legs from osteomalacia
C) A pure motor neuropathy causing wrist drop
D) A garlic-like odour on the patient's breath

Answer: B) Bone pain in the back and legs from osteomalacia

Explanation: Itai-itai disease presents with severe bone pain in the back and legs from osteomalacia, on a background of cadmium-induced renal tubular dysfunction.

Treatment

Q28. Chelation with BAL or EDTA in cadmium poisoning is limited because it:

A) Reliably cures established chronic cadmium disease
B) Is entirely free of any adverse side effects
C) Is the definitive curative treatment of choice
D) Removes only non-metallothionein-bound cadmium, so helps little in chronic poisoning

Answer: D) Removes only non-metallothionein-bound cadmium, so helps little in chronic poisoning

Explanation: Chelators remove only the small non-metallothionein-bound fraction, so they are of little value in chronic cadmium poisoning; management is supportive.

Other Effects

Q29. Chronic cadmium can cause discolouration of the:

A) Teeth
B) Nails only
C) Sclera
D) Hair

Answer: A) Teeth

Explanation: Chronic cadmium exposure can cause a characteristic discolouration of the teeth, among its varied chronic effects.

Q30. Chronic cadmium exposure has been associated with cancer of the:

A) Bladder only
B) Liver only
C) Prostate (and lung)
D) Thyroid

Answer: C) Prostate (and lung)

Explanation: Chronic cadmium is associated with prostate cancer and is a recognised lung carcinogen, besides its renal, bone and pulmonary effects.

Liver

Q31. Liver damage in chronic cadmium poisoning is:

A) The principal lesion
B) Not significant (the liver has some protective device)
C) Always cirrhotic
D) The cause of death

Answer: B) Not significant (the liver has some protective device)

Explanation: Unlike the kidney, the liver is relatively protected in chronic cadmium poisoning, so hepatic damage is not a significant feature.

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UNIT II — METALS

Chromium

TOPICS IN THIS CHAPTER

Properties & Valency

Q1. Chromium is toxicologically important mainly in its:
A) Trivalent state
B) Hexavalent state
C) Metallic state
D) Divalent state
Answer: B) Hexavalent state
Explanation: Hexavalent chromium is the irritant, corrosive and carcinogenic form; trivalent chromium (e.g. chromium oxide, sulphate) is essentially non-toxic.

Q2. Trivalent chromium is much less toxic than hexavalent chromium mainly because it is:
A) More volatile
B) More corrosive
C) Radioactive
D) Very poorly absorbed
Answer: D) Very poorly absorbed
Explanation: Trivalent chromium is poorly absorbed, so it is largely non-toxic, whereas hexavalent chromium is absorbed and is irritant, corrosive and carcinogenic.

Q3. Examples of relatively non-toxic trivalent chromium compounds are:
A) Chromium oxide and chromium sulphate
B) Chromic acid
C) Chromate salts
D) Dichromates
Answer: A) Chromium oxide and chromium sulphate
Explanation: Chromium oxide and chromium sulphate are trivalent and non-toxic; chromates, dichromates and chromic acid are hexavalent and hazardous.

Absorption

Q4. Hexavalent chromium is absorbed by all routes EXCEPT it is poorly absorbed through the:
A) Lungs
B) Gut
C) Skin
D) Respiratory mucosa
Answer: C) Skin
Explanation: Hexavalent chromium is absorbed through lungs and gut but only poorly through skin; it is rapidly excreted in urine (half-life ~15-41 hours).

Skin Effects

Q5. 'Chrome ulcers' (chrome holes) are characteristically:
A) Diffuse blistering across exposed skin
B) Circular, well-demarcated, punched-out lesions
C) Pigmented flat macules on the forearms
D) Fine linear scratch-like skin markings
Answer: B) Circular, well-demarcated, punched-out lesions
Explanation: Chrome ulcers are circular, well-demarcated, punched-out skin lesions, only slightly painful, that heal spontaneously unless secondarily infected.

Q6. Chrome ulcers are notable in occupational health because they are:
A) Always malignant
B) Untreatable
C) Caused by trivalent chromium
D) A notifiable occupational disease
Answer: D) A notifiable occupational disease
Explanation: Chrome ulcers are a notifiable occupational disease; they can be treated with a 10% calcium-EDTA solution and tend to heal spontaneously.

Q7. Chromium can cause contact dermatitis because it acts as a:
A) Sensitiser
B) Simple lubricant
C) Vasodilator
D) Nutrient
Answer: A) Sensitiser
Explanation: Hexavalent chromium is a skin sensitiser, causing allergic contact dermatitis, in addition to its irritant chrome-ulcer and respiratory effects.

Respiratory & Other

Q8. A classic upper-airway lesion of chromium exposure is:
A) Pleural plaque
B) Eggshell calcification
C) Perforation of the nasal cartilaginous septum
D) Honeycombing
Answer: C) Perforation of the nasal cartilaginous septum
Explanation: Hexavalent chromium erodes the nasal mucosa, perforating the nasal cartilaginous septum, and also causes chronic rhinitis and bronchitis.

Q9. Inhalation of a large concentration of hexavalent chromium may cause:
A) Only a mild rash
B) Cough, wheeze, fever, weight loss and pulmonary oedema
C) No symptoms
D) Isolated deafness

Answer: B) Cough, wheeze, fever, weight loss and pulmonary oedema

Explanation: Heavy hexavalent chromium inhalation causes cough, wheeze, inspiratory pain, fever, weight loss and pulmonary oedema, besides renal and lung effects.

Carcinogenicity

Q10. Chromium is recognised as causing:
A) Bladder cancer only
B) Skin melanoma only
C) No cancer
D) Lung carcinoma

Answer: D) Lung carcinoma

Explanation: Hexavalent chromium is a recognised cause of lung carcinoma, alongside renal damage and its irritant/corrosive mucosal effects.

Eyes

Q11. Ocular effects of chromium exposure include:
A) Conjunctivitis, keratitis and eyelid ulceration
B) Cataract only
C) Retinal detachment
D) Glaucoma

Answer: A) Conjunctivitis, keratitis and eyelid ulceration

Explanation: Hexavalent chromium causes conjunctivitis, keratitis and ulceration of the eyelids, reflecting its irritant and corrosive nature.

Treatment

Q12. Chrome ulcers can be treated adequately with:
A) Oral antibiotics alone
B) Topical steroids only
C) A 10% calcium-EDTA solution
D) Surgical amputation

Answer: C) A 10% calcium-EDTA solution

Explanation: Chrome ulcers respond to a 10% solution of calcium EDTA, with attention to secondary infection; prevention rests on skin protection and hygiene.

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UNIT II — METALS

Cobalt

TOPICS IN THIS CHAPTER

Sources & Uses

Q1. Cobalt is recovered mainly as a by-product of mining for:
A) Iron and coal
B) Copper and silver
C) Tin and zinc
D) Gold and lead

Answer: B) Copper and silver

Explanation: Cobalt is a silvery-grey magnetic metal recovered as a by-product of copper and silver mining.

Q2. A principal industrial use of cobalt is in:
A) Cotton dyeing
B) Glass etching
C) Food preservation
D) High-temperature 'super alloys' for jet engines

Answer: D) High-temperature 'super alloys' for jet engines

Explanation: Cobalt makes high-temperature-resistant 'super alloys' used in jet engines, plus steel/rock cutting tools, magnets, paints and battery catalysts.

Q3. Radioactive cobalt-60 is used as a source of:
A) High-energy gamma radiation (radiotherapy, industrial radiography)
B) Alpha particles
C) Ultraviolet light
D) Infrared heat

Answer: A) High-energy gamma radiation (radiotherapy, industrial radiography)

Explanation: Cobalt-60 is a high-energy gamma source used in radiotherapy and industrial radiography, a distinct radiation-related use of the metal.

Absorption

Q4. Absorption of cobalt occurs by the gut (~5-40%) and lungs (~30%), with main storage in:
A) Bone only
B) Brain only
C) Liver, spleen, kidneys, muscle and fat
D) Skin only

Answer: C) Liver, spleen, kidneys, muscle and fat

Explanation: Cobalt is absorbed via gut and lungs and stored mainly in liver, spleen, kidneys, muscle and fat; about 80% is excreted in urine.

Lung Toxicity

Q5. Inhaled cobalt powder causes 'hard-metal' lung disease, which is an:
A) An obstructive defect with hyperinflation
B) Interstitial fibrosis with a restrictive defect and reduced DLCO
C) Benign deposition without functional change
D) A pleural-plaque pattern of disease

Answer: B) Interstitial fibrosis with a restrictive defect and reduced DLCO

Explanation: Hard-metal cobalt fibrosis is an interstitial lung fibrosis with wheeze, dyspnoea, chest tightness, a restrictive defect (low FVC) and reduced diffusion capacity.

Q6. Cobalt can also cause occupational asthma, characteristically a:
A) Type IV reaction only
B) Non-immune irritation only
C) Granulomatous reaction
D) Type I reaction with specific IgE

Answer: D) Type I reaction with specific IgE

Explanation: Cobalt causes occupational asthma as a type I hypersensitivity reaction, with detectable specific IgE, besides its fibrotic hard-metal lung effect.

Cardiac

Q7. Cobalt was historically added to beer as a foam stabiliser, causing a dangerous cardiac syndrome of:
A) Abrupt left ventricular failure, pericardial effusion and polycythaemia
B) Isolated symptomless bradycardia
C) Painless chronic heart-valve disease
D) No cardiac effect of any clinical importance

Answer: A) Abrupt left ventricular failure, pericardial effusion and polycythaemia

Explanation: Cobalt-contaminated beer caused abrupt left ventricular failure with pericardial effusion and polycythaemia, especially in malnourished heavy drinkers.

Q8. The cobalt-beer cardiomyopathy was severe because cobalt has, with alcohol, a:
A) Protective cardiac effect
B) Purely renal interaction
C) Synergistic adverse effect on myocardial function
D) No interaction

Answer: C) Synergistic adverse effect on myocardial function

Explanation: Cobalt acts synergistically with alcohol to impair myocardial function, so malnourished beer drinkers suffered the cardiomyopathy most severely.

Thyroid

Q9. Cobalt can cause thyroid hyperplasia because it:
A) Destruction of the anterior pituitary gland
B) Inhibits tyrosine and blocks thyroxine synthesis, raising TSH
C) Direct mimicry of circulating thyroxine
D) A block of renal iodine excretion

Answer: B) Inhibits tyrosine and blocks thyroxine synthesis, raising TSH

Explanation: Cobalt inhibits tyrosine and thus thyroxine synthesis, leading to compensatory over-secretion of TSH and thyroid hyperplasia.

Polycythaemia

Q10. Cobalt-induced polycythaemia arises from increased renal release of:
A) Renin
B) Insulin
C) Aldosterone
D) Erythropoietin (from tissue hypoxia)

Answer: D) Erythropoietin (from tissue hypoxia)

Explanation: Cobalt causes tissue hypoxia (impaired oxygen release via 2,3-DPG inactivation and cobalt-haemoglobin), increasing renal erythropoietin and producing polycythaemia.

Q11. Cobalt impairs oxygen release from red cells partly by inactivating:
A) 2,3-diphosphoglycerate (2,3-DPG)
B) Haemoglobin A only
C) Carbonic anhydrase
D) Methaemoglobin reductase

Answer: A) 2,3-diphosphoglycerate (2,3-DPG)

Explanation: Cobalt inactivates 2,3-DPG and forms cobalt-haemoglobin, shifting the oxygen-dissociation curve left so less oxygen is released, causing tissue hypoxia.

Monitoring

Q12. Medical surveillance of cobalt workers focuses on the lung and skin, using:
A) Pure-tone audiometry of workers only
B) Routine liver biopsy of exposed workers
C) Periodic examination, pulmonary function tests and chest X-ray
D) Bone densitometry scanning only

Answer: C) Periodic examination, pulmonary function tests and chest X-ray

Explanation: Cobalt surveillance includes periodic health examination (lung and skin), pulmonary function tests and chest radiography; urinary cobalt is the biological index.

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UNIT II — METALS

Combined Review — Mechanisms & Comparison

TOPICS IN THIS CHAPTER

Enzymes & Molecular Targets

Q1. The single most common molecular mechanism shared by lead, mercury, arsenic and cadmium is binding to:
A) DNA promoter regions only
B) Sulphydryl (–SH) groups of enzymes
C) Cell-membrane phospholipids only
D) Ribosomal RNA only

Answer: B) Sulphydryl (–SH) groups of enzymes

Explanation: Most toxic heavy metals bind avidly to enzyme sulphydryl (thiol) groups, inactivating them; this shared chemistry underlies dithiol-chelator antidotes.

Q2. Dithiol chelators such as BAL, DMSA and DMPS work against several metals because they:
A) Acidify the urine to trap metals
B) Bind only calcium-mimicking metals
C) Oxidise the metal to an inert form
D) Provide alternative thiol (–SH) groups for the metal to bind

Answer: D) Provide alternative thiol (–SH) groups for the metal to bind

Explanation: BAL and its relatives carry two –SH groups, offering the metal an alternative thiol to bind, which is why one agent chelates arsenic, mercury and lead.

Q3. Which metal characteristically inhibits ALA dehydratase and ferrochelatase in the haem-synthesis pathway?
A) Lead
B) Manganese
C) Cobalt
D) Nickel

Answer: A) Lead

Explanation: Lead inhibits ALA dehydratase, ferrochelatase and coproporphyrinogen oxidase, raising ALA and zinc protoporphyrin and impairing haemoglobin synthesis.

Q4. Trivalent arsenic blocks cellular respiration mainly by inhibiting which enzyme complex via its lipoic-acid cofactor?
A) Carbonic anhydrase
B) Ferrochelatase
C) Pyruvate dehydrogenase
D) Glutamine synthetase

Answer: C) Pyruvate dehydrogenase

Explanation: Trivalent arsenic binds the dithiol of lipoic acid, inhibiting the pyruvate dehydrogenase complex and blocking conversion of pyruvate to acetyl-CoA.

Q5. Pentavalent arsenate is toxic because it:
A) Binds sulphydryl groups irreversibly
B) Substitutes for phosphate and uncouples oxidative phosphorylation
C) Chelates intracellular calcium
D) Is converted to arsine gas

Answer: B) Substitutes for phosphate and uncouples oxidative phosphorylation

Explanation: Arsenate mimics phosphate, forming unstable arsenate esters in place of ATP, thereby uncoupling oxidative phosphorylation (arsenolysis).

Q6. Basophilic stippling of red cells in lead poisoning results from inhibition of:
A) Cytochrome c oxidase
B) Glutamine synthetase
C) Lysyl oxidase
D) Pyrimidine-5'-nucleotidase

Answer: D) Pyrimidine-5'-nucleotidase

Explanation: Lead inhibits pyrimidine-5'-nucleotidase, leaving residual RNA aggregates that appear as basophilic stippling on the blood film.

Q7. Cadmium exerts much of its cellular toxicity by displacing which essential metal from metalloenzymes?
A) Zinc
B) Iodine
C) Sodium
D) Iron

Answer: A) Zinc

Explanation: Cadmium binds metallothionein and displaces zinc from zinc-dependent metalloenzymes, contributing to its tubular and oxidative toxicity.

Q8. Hexavalent chromium is carcinogenic chiefly because, once inside the cell, it is:
A) Oxidised to an inert trivalent salt
B) Bound harmlessly to metallothionein
C) Reduced to reactive intermediates that damage DNA
D) Excreted unchanged in bile

Answer: C) Reduced to reactive intermediates that damage DNA

Explanation: Hexavalent chromium is taken up and reduced intracellularly, generating reactive oxygen species and chromium-DNA adducts that drive carcinogenesis.

Q9. Which metal is an essential cofactor for glutamine synthetase and manganese superoxide dismutase, yet causes parkinsonism in excess?
A) Cadmium
B) Manganese
C) Lead
D) Arsenic

Answer: B) Manganese

Explanation: Manganese is an essential cofactor for glutamine synthetase and Mn-superoxide dismutase, but accumulation in the basal ganglia causes manganism.

Q10. Copper is a cofactor for cytochrome c oxidase and superoxide dismutase, but in excess causes haemolysis by inhibiting:
A) ALA dehydratase
B) Pyruvate dehydrogenase
C) Carbonic anhydrase
D) G6PD and glutathione reductase

Answer: D) G6PD and glutathione reductase

Explanation: Excess copper inhibits the protective enzymes G6PD and glutathione reductase, leaving red cells vulnerable to oxidative haemolysis.

Common Pathways & Mimicry

Q11. Lead disrupts neuronal and other functions partly by mimicking and competing with which physiological ion?
A) Calcium
B) Chloride
C) Bicarbonate
D) Iodide

Answer: A) Calcium

Explanation: Lead competes with calcium at synapses and in mitochondria; therapeutic calcium can displace lead and enhance its urinary excretion.

Q12. Grouping the metals by mechanism, which set primarily acts through oxidative stress and carcinogenesis?
A) Lead and organic lead
B) Manganese and cobalt only
C) Chromium(VI), nickel and arsenic
D) Phosphorus and copper only

Answer: C) Chromium(VI), nickel and arsenic

Explanation: Hexavalent chromium, nickel and arsenic act substantially through reactive oxygen species, DNA adducts and impaired DNA repair, driving carcinogenesis.

Q13. Which three metals are best described as 'essential elements that become toxic only on overload'?
A) Lead, mercury and arsenic
B) Manganese, copper and cobalt
C) Cadmium, chromium and nickel
D) Phosphorus, arsenic and mercury

Answer: B) Manganese, copper and cobalt

Explanation: Manganese, copper and cobalt are required trace cofactors at low levels but injurious in excess, unlike the purely xenobiotic toxic metals.

Q14. Cobalt produces polycythaemia largely because it:
A) Directly stimulates the bone marrow stroma
B) Blocks renal erythropoietin breakdown
C) Mimics testosterone
D) Causes tissue hypoxia, raising erythropoietin

Answer: D) Causes tissue hypoxia, raising erythropoietin

Explanation: Cobalt impairs oxygen delivery (inactivating 2,3-DPG and forming cobalt-haemoglobin), causing tissue hypoxia that raises renal erythropoietin and red-cell mass.

Q15. A worker has anaemia with normal iron stores, raised zinc protoporphyrin and basophilic stippling. The unifying mechanism is:
A) Inhibition of haem-synthesis enzymes by lead
B) Displacement of zinc by cadmium
C) Phosphate mimicry by arsenate
D) Oxidative DNA damage by chromium

Answer: A) Inhibition of haem-synthesis enzymes by lead

Explanation: Normal iron stores with raised ZPP and basophilic stippling point to lead's inhibition of haem-synthesis enzymes rather than iron deficiency.

Antidotes & Mechanism

Q16. Calcium-disodium EDTA is given AFTER dimercaprol in lead encephalopathy because, used alone, EDTA may:
A) Cause acute haemolysis
B) Precipitate in the renal tubule
C) Mobilise lead into the brain
D) Inactivate vitamin B12

Answer: C) Mobilise lead into the brain

Explanation: In encephalopathy, EDTA alone can mobilise lead and transiently raise brain levels; BAL is started first to limit redistribution into the CNS.

Q17. Penicillamine is the chelator of choice for copper overload, as in:
A) Itai-itai disease
B) Wilson disease
C) Minamata disease
D) Manganism

Answer: B) Wilson disease

Explanation: Penicillamine chelates copper and is used in Wilson disease and copper toxicity; it is also an oral option for lead and mercury.

Q18. Match the metal to its preferred biological monitoring index: the metal whose effect is tracked by zinc protoporphyrin is:
A) Cadmium
B) Manganese
C) Chromium
D) Lead

Answer: D) Lead

Explanation: Zinc protoporphyrin reflects lead's block of ferrochelatase over the preceding months and is used to monitor longer-term lead exposure.

Q19. Which metal's chronic poisoning is LEAST helped by chelation, because the metal is tightly bound to metallothionein?
A) Cadmium
B) Inorganic lead
C) Arsenic
D) Inorganic mercury

Answer: A) Cadmium

Explanation: Chelation is of little value in chronic cadmium poisoning, as chelators remove only the small fraction not bound to metallothionein.

Q20. Dimercaprol (BAL) is effective for inorganic mercury but is contraindicated in organic (methyl) mercury because it:
A) Causes immediate haemolysis
B) Is destroyed by gastric acid
C) Increases mercury transfer across the blood-brain barrier
D) Has no thiol groups

Answer: C) Increases mercury transfer across the blood-brain barrier

Explanation: In organomercury poisoning BAL can increase movement of mercury across the blood-brain barrier, worsening central injury, so it is contraindicated.

Signature Syndromes

Q21. Inhibition of haem synthesis with basophilic stippling is the signature of:
A) Mercury
B) Lead
C) Arsenic
D) Manganese

Answer: B) Lead

Explanation: Lead inhibits haem-synthesis enzymes, producing anaemia with basophilic stippling, raised ZPP and urinary ALA, a picture unique among these metals.

Q22. The Minamata Bay disaster in Japan resulted from industrial poisoning with which agent?
A) Inorganic lead
B) Arsenic
C) Cadmium
D) Organic (methyl) mercury

Answer: D) Organic (methyl) mercury

Explanation: Minamata disease results from methylmercury that bioaccumulated in fish, causing cerebellar and sensory neurological damage in the exposed coastal population.

Q23. Itai-itai disease (painful osteomalacia with renal tubular damage) is caused by:
A) Cadmium
B) Lead
C) Mercury
D) Manganese

Answer: A) Cadmium

Explanation: Itai-itai disease is the classic chronic cadmium syndrome, combining osteomalacia/fractures with proximal renal tubular dysfunction.

Q24. A parkinsonism-like extrapyramidal syndrome from metal exposure suggests:
A) Lead
B) Cadmium
C) Manganese
D) Arsenic

Answer: C) Manganese

Explanation: Manganism targets the basal ganglia, producing a levodopa-resistant parkinsonian syndrome distinctive among the occupational metals.

Q25. Palmoplantar hyperkeratosis, hyperpigmentation and Mees' lines indicate chronic poisoning by:
A) Lead
B) Arsenic
C) Mercury
D) Manganese

Answer: B) Arsenic

Explanation: These skin and nail changes are hallmarks of chronic arsenicosis, alongside its sensorimotor neuropathy and cancer risk.

Q26. Erethism and intention tremor point to exposure to:
A) Lead
B) Cadmium
C) Arsenic
D) Elemental mercury

Answer: D) Elemental mercury

Explanation: Erethism (neuropsychiatric change) with intention tremor is the classic syndrome of chronic elemental mercury vapour exposure.

Q27. Punched-out, well-demarcated skin ulcers ('chrome holes') are characteristic of:
A) Hexavalent chromium
B) Lead
C) Cobalt
D) Copper

Answer: A) Hexavalent chromium

Explanation: Chrome ulcers, circular punched-out lesions and a notifiable disease, are characteristic of hexavalent chromium exposure.

Q28. Hard-metal interstitial lung fibrosis and a beer-drinker's cardiomyopathy point to:
A) Nickel
B) Chromium
C) Cobalt
D) Copper

Answer: C) Cobalt

Explanation: Cobalt causes hard-metal lung fibrosis and the historic cobalt-beer cardiomyopathy (left ventricular failure, pericardial effusion, polycythaemia).

Q29. A brick-dust-smelling, phosgene-like gas formed in the Mond process is:
A) Arsine
B) Nickel carbonyl
C) Phosphine
D) Stibine

Answer: B) Nickel carbonyl

Explanation: Nickel carbonyl, Ni(CO)4, is a highly toxic phosgene-like gas with a brick-dust odour, formed during the Mond process of nickel purification.

Q30. Phossy jaw (necrosis of the mandible) is the classic disease of:
A) Cadmium
B) Manganese
C) Mercury
D) White phosphorus

Answer: D) White phosphorus

Explanation: Phossy jaw, necrosis of the jaw described by Donald Hunter, is the classic occupational disease of white (yellow) phosphorus.

Neuropathy Pattern

Q31. A predominantly MOTOR neuropathy (wrist drop) suggests, whereas a SENSORY neuropathy suggests:
A) Lead (motor) vs arsenic (sensory)
B) Arsenic (motor) vs lead (sensory)
C) Mercury vs cadmium
D) Manganese vs lead

Answer: A) Lead (motor) vs arsenic (sensory)

Explanation: Lead causes a motor neuropathy (wrist/foot drop); arsenic causes a sensory-predominant sensorimotor neuropathy, a classic distinguishing pair.

Target Organs

Q32. The metal whose chief chronic target is the renal proximal tubule and bone is:
A) Lead
B) Mercury
C) Cadmium
D) Manganese

Answer: C) Cadmium

Explanation: Cadmium's hallmark targets are the proximal tubule (beta-2-microglobulinuria) and bone (osteomalacia), as seen in itai-itai disease.

Q33. The metal that principally accumulates in and damages the basal ganglia is:
A) Lead
B) Manganese
C) Cadmium
D) Arsenic

Answer: B) Manganese

Explanation: Manganese deposits in the globus pallidus, producing the extrapyramidal features of manganism and basal-ganglia MRI changes.

Q34. Greenish discolouration of the tongue, hair and teeth with haemolytic anaemia suggests:
A) Lead
B) Arsenic
C) Nickel
D) Copper

Answer: D) Copper

Explanation: Very high copper exposure causes greenish discolouration of tongue, hair and teeth, and ingestion can cause delayed haemolytic anaemia.

Biomarkers

Q35. The standard biomarker for each metal pairs correctly as:
A) Lead = blood lead; cadmium = urinary cadmium/beta-2-microglobulin
B) Lead = urine arsenic
C) Cadmium = blood lead
D) Mercury = zinc protoporphyrin

Answer: A) Lead = blood lead; cadmium = urinary cadmium/beta-2-microglobulin

Explanation: Blood lead indexes lead; urinary cadmium (burden) and beta-2-microglobulin (effect) index cadmium, while mercury and arsenic use their own urine/blood markers.

Q36. Speciated URINARY arsenic and URINE mercury are used because:
A) They both simply measure blood lead levels
B) They both measure the cadmium body burden
C) They reflect inorganic arsenic and elemental/inorganic mercury exposure respectively
D) They are completely interchangeable indices

Answer: C) They reflect inorganic arsenic and elemental/inorganic mercury exposure respectively

Explanation: Speciated urinary arsenic captures toxic inorganic arsenic, and urine mercury reflects elemental/inorganic mercury; organic mercury is better shown by blood.

Q37. Zinc protoporphyrin (ZPP) is an effect biomarker specific to:
A) Cadmium
B) Lead
C) Manganese
D) Mercury

Answer: B) Lead

Explanation: ZPP rises when lead blocks ferrochelatase in haem synthesis; it is not a marker for the other metals.

Carcinogens

Q38. Among these metals, the recognised human carcinogens (IARC Group 1) include:
A) Manganese and lead
B) Mercury and manganese
C) None of them
D) Arsenic and cadmium

Answer: D) Arsenic and cadmium

Explanation: Arsenic (skin, lung, bladder) and cadmium (lung) are Group 1 carcinogens; mercury and manganese are not classified as such, and lead is a probable carcinogen.

Q39. Among these, the metals causing nasal-sinus and lung cancer (and the chrome lung cancer) include:
A) Nickel and hexavalent chromium
B) Cobalt and copper
C) Manganese and lead
D) Phosphorus and mercury

Answer: A) Nickel and hexavalent chromium

Explanation: Nickel causes nasal-sinus, lung and pharyngeal cancer, and hexavalent chromium causes lung cancer; both also perforate the nasal septum.

Chelation

Q40. Chelation matches correctly as:
A) Cadmium = calcium-disodium EDTA as first-line
B) Manganese = curative DMSA chelation
C) Lead = EDTA/DMSA; arsenic and inorganic mercury = BAL/DMSA
D) Methylmercury = dimercaprol (BAL) as first-line

Answer: C) Lead = EDTA/DMSA; arsenic and inorganic mercury = BAL/DMSA

Explanation: Lead uses EDTA/DMSA (BAL+EDTA if severe), arsenic and inorganic mercury use BAL/DMSA; cadmium chelation is avoided and BAL is contraindicated in methylmercury.

Q41. Chelation is generally AVOIDED or contraindicated for:
A) Lead encephalopathy
B) Cadmium (nephrotoxic redistribution) and methylmercury with BAL
C) Acute arsenic poisoning
D) Inorganic mercury

Answer: B) Cadmium (nephrotoxic redistribution) and methylmercury with BAL

Explanation: Cadmium chelation worsens nephrotoxicity and BAL is contraindicated in methylmercury (CNS redistribution); the others are standard chelation indications.

Classic Eponyms

Q42. Match the eponymous sign to its metal: Burton's line is to lead as garlic breath is to:
A) Mercury
B) Cadmium
C) Manganese
D) Arsenic

Answer: D) Arsenic

Explanation: Burton's blue gum line is the eponymous sign of lead; a garlic-like breath or odour is the classic clue to arsenic poisoning.

Q43. 'Mad hatter' / erethism is to mercury as 'cock walk' / extrapyramidal gait is to:
A) Manganese
B) Lead
C) Cadmium
D) Arsenic

Answer: A) Manganese

Explanation: The mad-hatter neuropsychiatric picture is mercury; the cock-walk extrapyramidal gait is the classic motor sign of manganism.

Acute Distinctive

Q44. An acute haemolytic crisis with haemoglobinuria after a metal-hydride gas exposure indicates:
A) Lead fume
B) Cadmium fume
C) Arsine (AsH_3)
D) Mercury vapour

Answer: C) Arsine (AsH_3)

Explanation: Arsine gas causes acute massive intravascular haemolysis and haemoglobinuria, unlike the pneumonitis of cadmium fume or CNS effects of mercury vapour.

Q45. Acute heavy inhalation causing delayed chemical pneumonitis/pulmonary oedema is characteristic of:
A) Lead dust
B) Cadmium fume
C) Arsenic ingestion
D) Manganese ore

Answer: B) Cadmium fume

Explanation: Cadmium-fume inhalation classically causes a delayed and potentially fatal chemical pneumonitis with pulmonary oedema, often after an initial metal-fume-fever-like illness.

Q46. A normal carboxyhaemoglobin helps distinguish, from carbon-monoxide poisoning, poisoning by:
A) Arsine
B) Cadmium fume
C) Phosphine
D) Nickel carbonyl (Mond process)

Answer: D) Nickel carbonyl (Mond process)

Explanation: Because the Mond process uses carbon monoxide, a normal COHb points to nickel-carbonyl rather than carbon-monoxide poisoning.

Prevention

Q47. A principle common to all these metals is that:

A) Removal from exposure precedes and underpins any chelation
B) Chelation prevents exposure
C) Air monitoring measures internal dose
D) Biomarkers are interchangeable

Answer: A) Removal from exposure precedes and underpins any chelation

Explanation: For every toxic metal, stopping exposure is the essential first step; chelation is selective, adjunctive and sometimes contraindicated.

Chelation & Antidotes

Q48. Copper toxicity (and Wilson disease) is treated with:

A) Deferoxamine
B) Calcium gluconate
C) Penicillamine
D) Atropine

Answer: C) Penicillamine

Explanation: Penicillamine chelates copper, as used in Wilson disease; nickel-carbonyl poisoning instead uses disulfiram and prednisolone.

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UNIT II — METALS

Copper

TOPICS IN THIS CHAPTER

Properties & Uses

Q1. Copper is widely used because it is, among other things:
A) Radioactive
B) An excellent electrical conductor, erosion-resistant and malleable
C) Volatile and gaseous
D) Biologically inert

Answer: B) An excellent electrical conductor, erosion-resistant and malleable

Explanation: Copper's excellent electrical conductivity, erosion resistance and malleability underlie its wide use in wiring and alloys.

Q2. Brass and bronze are copper alloys with, respectively:
A) Tin and lead
B) Nickel and iron
C) Lead and zinc
D) Zinc and tin

Answer: D) Zinc and tin

Explanation: Brass is copper with zinc and bronze is copper with tin; Monel is copper with nickel, and copper-beryllium is another important alloy.

Biochemistry

Q3. Copper serves as a co-factor for oxidative enzymes including:
A) Cytochrome c oxidase and ALA dehydratase
B) Amylase and lipase
C) Pepsin and trypsin
D) Renin and aldosterone

Answer: A) Cytochrome c oxidase and ALA dehydratase

Explanation: Copper is a co-factor for oxidative enzymes such as cytochrome c oxidase and ALA dehydratase, and influences specific gene expression.

Metabolism

Q4. Excess copper is excreted mainly in the:
A) The urine only, with none in bile
B) The sweat only, with none in bile
C) Bile (firmly bound to amino acid to prevent reabsorption)
D) The exhaled breath as a volatile compound

Answer: C) Bile (firmly bound to amino acid to prevent reabsorption)

Explanation: Excess copper is excreted in bile, firmly bound to amino acid so it is not reabsorbed; in tissue it binds metallothionein-like proteins.

Q5. Copper can inhibit sulphydryl-group enzymes that protect cells from free radicals, such as:
A) Cytochrome oxidase only
B) G6PD and glutathione reductase
C) Carbonic anhydrase
D) Hexokinase

Answer: B) G6PD and glutathione reductase

Explanation: Copper can inhibit SH-group enzymes like G6PD and glutathione reductase, which normally protect cells from oxygen free radicals, predisposing to haemolysis.

Acute Toxicity

Q6. Acute copper ingestion causes nausea, vomiting and haemorrhagic gastritis, followed after some hours by:
A) Wrist drop
B) Garlic breath
C) Bone pain
D) Haemolytic anaemia

Answer: D) Haemolytic anaemia

Explanation: Ingested copper causes nausea, vomiting, haemorrhagic gastritis and diarrhoea, and after a delay a haemolytic anaemia (from oxidative red-cell injury).

Chronic - Discolouration

Q7. Very high copper exposure can cause a characteristic greenish discolouration of the:
A) Tongue, hair and teeth
B) Discolouration confined to the fingernails
C) A bluish discolouration of the sclera
D) Greenish staining limited to the palms

Answer: A) Tongue, hair and teeth

Explanation: Very high copper exposure causes greenish discolouration of the tongue and hair and superficial green staining of the teeth.

Chronic - Lung & Liver

Q8. Copper sprayers may develop, from inhaling copper dust or mist, a:
A) Pure obstructive asthma
B) Pleural plaque disease
C) Granulomatous lung disorder with interstitial fibrosis
D) Mesothelioma

Answer: C) Granulomatous lung disorder with interstitial fibrosis

Explanation: Inhaled copper dust/mist in sprayers causes a granulomatous lung disorder followed by interstitial fibrosis ('vineyard sprayer's lung'), and liver granulomas.

Other Effects

Q9. Other recognised effects of copper exposure include:
A) Itai-itai disease
B) Metal fume fever and contact dermatitis
C) Manganism
D) Phossy jaw

Answer: B) Metal fume fever and contact dermatitis

Explanation: Copper exposure can cause metal fume fever and contact dermatitis on skin contact with copper dust, besides its systemic effects.

Wilson Disease

Q10. Copper is central to which inherited disorder of copper accumulation?
A) Haemochromatosis
B) Thalassaemia
C) Gaucher disease
D) Wilson disease

Answer: D) Wilson disease

Explanation: Wilson disease is a disorder of copper accumulation; a gene on the X chromosome and another locus control copper absorption and accumulation in the body.

Treatment

Q11. Copper toxicity (and Wilson disease) is treated with the chelator:
A) Penicillamine
B) Deferoxamine
C) Atropine
D) Calcium gluconate

Answer: A) Penicillamine

Explanation: Penicillamine is used to chelate copper, given as in Wilson disease, to enhance copper excretion and lower the body burden.

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UNIT II — METALS

Metal Toxicology — Foundations

TOPICS IN THIS CHAPTER

Routes & Toxicokinetics

Q1. The principal route of occupational exposure to most toxic metals is:
A) Dermal contact only
B) Inhalation of dust or fume
C) Ingestion of food
D) Injection

Answer: B) Inhalation of dust or fume

Explanation: Inhalation of metal dust or fume is the dominant occupational route; ingestion (often via contaminated hands) and skin contribute less for most metals.

Q2. Metal 'fume' is generated chiefly by:
A) Cold grinding only
B) Liquid spillage
C) Simple handling of solids
D) Heating/welding processes that vaporise then condense the metal

Answer: D) Heating/welding processes that vaporise then condense the metal

Explanation: Fume forms when high temperature vaporises a metal that condenses into ultrafine, highly respirable particles, as in welding and smelting.

Q3. Lead is stored in the body mainly in:
A) Bone
B) The brain
C) Subcutaneous fat
D) The thyroid

Answer: A) Bone

Explanation: About 95% of the adult lead body burden is in bone, from which it is released slowly, giving lead a long biological half-life.

Q4. The principal target organ for chronic cadmium accumulation is the:
A) Liver only
B) Skin
C) Kidney (renal cortex)
D) Thyroid

Answer: C) Kidney (renal cortex)

Explanation: Cadmium accumulates in the renal cortex bound to metallothionein, with a very long half-life, making the proximal tubule its key chronic target.

Q5. Elemental mercury vapour is especially toxic to the brain because it:
A) Cannot be inhaled
B) Is lipid-soluble and crosses the blood-brain barrier
C) Is excreted instantly
D) Binds only to bone

Answer: B) Is lipid-soluble and crosses the blood-brain barrier

Explanation: Inhaled elemental mercury vapour is lipophilic, crosses the blood-brain barrier and is then oxidised, accounting for its neurotoxicity.

Q6. Gastrointestinal absorption of ingested lead is greater in children than adults, approximately:
A) Equal in both
B) Higher in adults
C) Negligible in both
D) 50% in children vs 10% in adults

Answer: D) 50% in children vs 10% in adults

Explanation: Children absorb roughly half of ingested lead versus about a tenth in adults, one reason they are far more susceptible to lead toxicity.

Biological Monitoring

Q7. Biological monitoring of metal exposure means measuring the metal or its effect in:
A) Biological media such as blood, urine, hair or nails
B) Only the concentration of the metal in workroom air
C) Surface wipe samples taken from the workplace
D) Settled dust collected from working surfaces

Answer: A) Biological media such as blood, urine, hair or nails

Explanation: Biological monitoring assesses internal dose by measuring the metal (or a biological effect) in body fluids or tissues, complementing air monitoring.

Q8. An ACGIH 'BEI' (Biological Exposure Index) is:
A) A maximum permitted airborne concentration limit
B) A statutory noise-exposure limit for the workplace
C) A reference value for a determinant in biological media
D) A permissible skin-dose limit for the agent

Answer: C) A reference value for a determinant in biological media

Explanation: BEIs are guidance values for biological determinants (e.g. blood lead, urinary cadmium) corresponding to inhalation at the airborne limit.

Q9. The primary biomarker of lead exposure is:
A) Serum calcium
B) Blood lead level
C) Urine creatinine
D) Hair zinc

Answer: B) Blood lead level

Explanation: Blood lead is the standard index of recent and ongoing lead exposure and guides medical management and removal decisions.

Q10. A biomarker of the biological EFFECT of lead (not just exposure) is:
A) Blood lead alone
B) Urinary creatinine
C) Serum sodium
D) Zinc protoporphyrin / free erythrocyte protoporphyrin

Answer: D) Zinc protoporphyrin / free erythrocyte protoporphyrin

Explanation: ZPP/FEP rises when lead blocks haem synthesis, reflecting an effect on the marrow over preceding weeks, complementing the blood lead level.

Q11. Urinary beta-2-microglobulin is used to detect the effect of which metal?
A) Cadmium (proximal tubular damage)
B) Lead
C) Manganese
D) Nickel

Answer: A) Cadmium (proximal tubular damage)

Explanation: Low-molecular-weight proteinuria, detected as urinary beta-2-microglobulin, signals cadmium-induced proximal tubular dysfunction and is its key biological effect biomarker.

Q12. Speciated urinary arsenic is preferred over total urinary arsenic because:
A) Speciation is technically impossible in urine
B) Total urinary arsenic cannot be measured at all
C) Dietary (organic) arsenic from seafood can falsely raise the total
D) Urine cannot retain arsenic for measurement

Answer: C) Dietary (organic) arsenic from seafood can falsely raise the total

Explanation: Seafood organoarsenicals inflate total urinary arsenic; speciation measures the toxic inorganic and methylated forms relevant to occupational exposure.

Q13. Hair and nails are most useful for monitoring chronic exposure to:
A) Carbon monoxide
B) Arsenic
C) Benzene
D) Noise

Answer: B) Arsenic

Explanation: Arsenic deposits in keratinised tissue, so hair and nails give a record of past chronic exposure; Mees' lines are a visible nail marker.

Chelation Principles

Q14. The general principle of chelation therapy is to:
A) Neutralise excess acid produced in the stomach
B) Block absorption of the metal from inhaled air
C) Stimulate new bone formation to trap the metal
D) Bind the metal into a complex that is then excreted

Answer: D) Bind the metal into a complex that is then excreted

Explanation: A chelator forms a stable, water-soluble complex with the metal, enhancing its renal (or biliary) excretion and lowering tissue levels.

Q15. The FIRST step in managing any metal poisoning, before chelation, is to:
A) Remove the worker from further exposure
B) Start lifelong chelation
C) Give corticosteroids
D) Perform dialysis routinely

Answer: A) Remove the worker from further exposure

Explanation: Removing the source of exposure is the essential first action; chelation is adjunctive and is not a substitute for stopping exposure.

Q16. Calcium disodium EDTA is a chelator used principally for:
A) Cyanide
B) Carbon monoxide
C) Lead
D) Iron

Answer: C) Lead

Explanation: Calcium disodium EDTA is a mainstay for lead chelation; it is not used for the gaseous asphyxiants or for iron (which uses deferoxamine).

Q17. Dimercaprol (British Anti-Lewisite, BAL) is classically used for:
A) Iron overload from repeated blood transfusion
B) Arsenic, mercury and (with EDTA) severe lead poisoning
C) Chronic cadmium-induced renal tubular damage
D) Manganese-induced extrapyramidal disease

Answer: B) Arsenic, mercury and (with EDTA) severe lead poisoning

Explanation: BAL chelates arsenic and inorganic mercury and is combined with EDTA in lead encephalopathy; it is avoided in organic (methyl) mercury.

Q18. An orally active chelator increasingly used for lead and arsenic is:
A) Deferoxamine
B) Atropine
C) Naloxone
D) DMSA (succimer)

Answer: D) DMSA (succimer)

Explanation: DMSA (succimer) is an oral dimercaprol analogue used for lead and arsenic; deferoxamine is for iron and the others are unrelated antidotes.

Q19. Chelation is generally NOT effective and may be harmful in poisoning by:
A) Cadmium
B) Lead
C) Arsenic
D) Inorganic mercury

Answer: A) Cadmium

Explanation: Cadmium chelation can redistribute the metal to the kidney and worsen nephrotoxicity, so it is generally avoided; care is supportive.

Q20. Chelating agents should not be used prophylactically in still-exposed workers because they:
A) They are reliably curative in every case
B) They remove the need for biological monitoring
C) May increase absorption/redistribution and are not a substitute for exposure control
D) They are entirely free of adverse effects

Answer: C) May increase absorption/redistribution and are not a substitute for exposure control

Explanation: Prophylactic chelation can enhance metal uptake and redistribute it to organs, and it cannot replace removing the worker from exposure.

Q21. Dimercaprol is specifically AVOIDED in methylmercury poisoning because it may:
A) Cause deafness
B) Redistribute mercury to the brain and worsen neurotoxicity
C) Cure it instantly
D) Lower blood pressure fatally

Answer: B) Redistribute mercury to the brain and worsen neurotoxicity

Explanation: BAL can shift methylmercury into the central nervous system, worsening neurological injury; water-soluble chelators such as DMSA are preferred.

General Principles

Q22. The severity of metal toxicity depends on all of the following EXCEPT:
A) The chemical form (organic vs inorganic, valency)
B) The dose and duration
C) The route of exposure
D) The worker's job title

Answer: D) The worker's job title

Explanation: Toxicity is governed by chemical form, dose, duration and route; the job title itself is irrelevant except as a proxy for exposure.

Q23. Metal toxicity often differs strikingly with chemical form; a classic example is:
A) Mercury (elemental vs inorganic vs organic)
B) Sodium chloride
C) Distilled water
D) Glucose

Answer: A) Mercury (elemental vs inorganic vs organic)

Explanation: Mercury's three forms cause quite different syndromes (CNS vapour toxicity, renal inorganic toxicity, neurodevelopmental methylmercury), illustrating form-dependent toxicity.

Q24. Which statement about air vs biological monitoring is correct?

A) Air and biological monitoring measure exactly the same quantity
B) Air monitoring directly measures the absorbed internal dose
C) Biological monitoring reflects internal dose from all routes, air monitoring reflects inhalation exposure
D) Biological monitoring measures the airborne workroom concentration

Answer: C) Biological monitoring reflects internal dose from all routes, air monitoring reflects inhalation exposure

Explanation: Air sampling estimates inhalation exposure, whereas biological monitoring captures the absorbed internal dose from all routes combined.

Q25. A key reason children are more vulnerable than adults to lead is:

A) Lower exposure
B) Higher GI absorption and developing nervous system
C) Faster excretion
D) Smaller bone stores only

Answer: B) Higher GI absorption and developing nervous system

Explanation: Children absorb more ingested lead and have a developing brain highly sensitive to it, with effects on cognition at low blood levels.

Q26. The most reliable long-term protection of metal-exposed workers is:

A) Routine chelation of all exposed workers
B) High-dose vitamin supplementation of workers
C) Annual prophylactic courses of antibiotics
D) Engineering control of exposure with monitoring and surveillance

Answer: D) Engineering control of exposure with monitoring and surveillance

Explanation: Controlling exposure at source, with air and biological monitoring and health surveillance, protects workers; chelation and supplements do not prevent exposure.

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UNIT II — METALS

Manganese

TOPICS IN THIS CHAPTER

Sources

Q1. Occupational manganese exposure occurs in all EXCEPT:
A) Cotton spinning
B) Manganese mining and ore processing
C) Steel and ferro-alloy production
D) Welding and dry-cell battery manufacture

Answer: A) Cotton spinning

Explanation: Mining, steel/alloy work, welding and battery manufacture expose to manganese; cotton spinning relates to byssinosis.

Q2. Manganese welding exposure is greatest when welding:
A) Aluminium outdoors
B) Plastic
C) Manganese-containing steels in confined spaces
D) Copper pipe

Answer: C) Manganese-containing steels in confined spaces

Explanation: Welding manganese alloy steels, especially in confined spaces with poor ventilation, generates high manganese-fume exposure.

Neurotoxicity

Q3. Chronic manganese neurotoxicity ('manganism') principally produces:
A) A pure sensory neuropathy
B) An extrapyramidal, parkinsonism-like syndrome
C) Wrist drop
D) Cerebellar ataxia only

Answer: B) An extrapyramidal, parkinsonism-like syndrome

Explanation: Manganism is an extrapyramidal disorder with bradykinesia, rigidity, gait disturbance and a characteristic 'cock walk', resembling parkinsonism.

Q4. Manganese accumulates in and damages the:
A) Substantia nigra primarily
B) Cerebellar vermis only
C) Spinal cord only
D) Basal ganglia (globus pallidus)

Answer: D) Basal ganglia (globus pallidus)

Explanation: Manganese targets the basal ganglia, especially the globus pallidus, in contrast to idiopathic Parkinson disease which mainly affects the substantia nigra.

Q5. An early phase of manganese intoxication may be:
A) Psychiatric ('locura manganica' - manganese madness)
B) Acute haemolysis
C) Renal failure
D) Gout

Answer: A) Psychiatric ('locura manganica' - manganese madness)

Explanation: Early manganism can present with neuropsychiatric features (emotional lability, hallucinations, compulsive behaviour) before the extrapyramidal motor phase.

Q6. Compared with Parkinson disease, manganism characteristically:
A) Responds dramatically to levodopa
B) Causes a resting pill-rolling tremor predominantly
C) Responds poorly to levodopa
D) Spares the basal ganglia

Answer: C) Responds poorly to levodopa

Explanation: Because the lesion is post-synaptic (pallidal) rather than nigral dopaminergic loss, manganism responds poorly to levodopa, unlike Parkinson disease.

Q7. The gait disturbance of manganism is classically described as a:
A) Festinating shuffle only
B) 'Cock walk' (strutting on the toes)
C) High-stepping gait
D) Waddling gait

Answer: B) 'Cock walk' (strutting on the toes)

Explanation: Manganism produces a characteristic 'cock walk', with patients strutting on their toes, part of its extrapyramidal motor syndrome.

Q8. Stage 1 manganism is important clinically because it is:
A) Always permanent
B) Immediately fatal
C) Purely psychiatric
D) Generally reversible if detected early

Answer: D) Generally reversible if detected early

Explanation: Stage 1 manganism, with non-specific symptoms, is generally reversible if detected early; development of Parkinsonian features denotes permanent CNS damage.

Q9. Excessive salivation and dysarthria appear in which stage of manganism?
A) Stage 2
B) Stage 1
C) Stage 3 only
D) Never

Answer: A) Stage 2

Explanation: Stage 2 manganism features dysarthria, gait disturbance, excessive salivation and the transient organic psychosis of manganic madness.

Other Effects

Q10. Acute inhalation of freshly formed manganese (and other metal) fume can cause:
A) Itai-itai disease
B) Blackfoot disease
C) Metal fume fever
D) Acrodynia

Answer: C) Metal fume fever

Explanation: Freshly generated metal-oxide fume (manganese, zinc, copper) can cause metal fume fever, a self-limiting influenza-like illness.

Q11. Manganese-exposed workers also have an increased risk of:
A) Mesothelioma
B) Pneumonitis and respiratory infection
C) Pleural plaques
D) Bladder cancer

Answer: B) Pneumonitis and respiratory infection

Explanation: Manganese exposure is linked to chemical pneumonitis and increased respiratory infection ('manganese pneumonia'), besides neurotoxicity.

Diagnosis & Monitoring

Q12. Blood and urine manganese levels are limited because they:
A) A resting pill-rolling tremor of the hands
B) Pure sensory loss in a glove distribution
C) Cerebellar signs with prominent nystagmus
D) Correlate poorly with body burden and CNS effects

Answer: D) Correlate poorly with body burden and CNS effects

Explanation: Manganese in blood/urine correlates poorly with target-organ dose, so they confirm exposure but do not gauge neurotoxic risk well.

Q13. A useful imaging finding in manganese accumulation is:
A) T1-weighted MRI hyperintensity in the basal ganglia
B) A cavitating lung mass
C) Pleural plaques
D) Eggshell calcification

Answer: A) T1-weighted MRI hyperintensity in the basal ganglia

Explanation: Manganese deposition produces symmetrical T1-hyperintensity in the basal ganglia on MRI, supporting the diagnosis of manganism.

Q14. Manganism is distinguished from Parkinson disease partly by:
A) A pill-rolling rest tremor
B) Dramatic levodopa response
C) Earlier gait/balance and psychiatric features, poor levodopa response
D) Onset only in the elderly

Answer: C) Earlier gait/balance and psychiatric features, poor levodopa response

Explanation: Manganism tends to cause early gait and psychiatric disturbance with a poor levodopa response, unlike the levodopa-responsive tremor of Parkinson disease.

Management

Q15. Management of manganism centres on:
A) Curative chelation that reverses the damage
B) Removal from exposure (neurological damage is often irreversible)
C) High-dose levodopa restoring normal function
D) Routine haemodialysis to clear the metal

Answer: B) Removal from exposure (neurological damage is often irreversible)

Explanation: Stopping exposure is essential, but established manganism is frequently irreversible; chelation and levodopa give limited benefit.

Q16. Levodopa is of limited use in manganism because the lesion is:
A) In the substantia nigra
B) Purely peripheral
C) Reversible spontaneously
D) Downstream of the dopaminergic neurons (pallidal)

Answer: D) Downstream of the dopaminergic neurons (pallidal)

Explanation: The pallidal (post-synaptic) site of injury means dopamine replacement cannot restore function, explaining the poor levodopa response.

Nature & Metabolism

Q17. Manganese is an essential element, a cofactor for enzymes including:
A) Glutamine synthetase and manganese superoxide dismutase
B) Catalase and lipase
C) Hexokinase and amylase
D) ALA dehydratase

Answer: A) Glutamine synthetase and manganese superoxide dismutase

Explanation: Manganese is essential as a cofactor for glutamine synthetase and manganese superoxide dismutase, among others; toxicity arises only on overload.

Q18. Manganese is absorbed poorly from the gut and excreted mainly via the:
A) Kidneys
B) Skin
C) Bile
D) Lungs

Answer: C) Bile

Explanation: Manganese is poorly absorbed from the gut, bound predominantly to red cells in blood, and excreted chiefly in bile; iron deficiency increases gut excretion.

Q19. Gut excretion of manganese is enhanced by:
A) Polycythaemia
B) Iron-deficiency anaemia
C) High calcium intake
D) Dehydration

Answer: B) Iron-deficiency anaemia

Explanation: Iron deficiency enhances manganese excretion through the gut, an interaction relevant to manganese handling and susceptibility.

Neurotoxicity - Staging

Q20. In the classic 3-stage description of manganism, stage 1 is characterised by:
A) Fixed rigidity and akinesia of the limbs
B) Severe generalised dystonia of the trunk
C) Coma with depressed consciousness
D) Non-specific symptoms (anorexia, apathy, cramps, irritability) and is often reversible

Answer: D) Non-specific symptoms (anorexia, apathy, cramps, irritability) and is often reversible

Explanation: Stage 1 manganism is non-specific and often reversible (anorexia, apathy, muscular cramps, joint pain, irritability, lassitude), before permanent CNS damage.

Q21. Stage 2 of manganism includes dysarthria, gait disturbance and an organic psychosis known as:
A) Manganic madness (locura manganica)
B) Erethism
C) Korsakoff psychosis
D) Delirium tremens

Answer: A) Manganic madness (locura manganica)

Explanation: Stage 2 features dysarthria, gait disturbance, excessive salivation and 'manganic madness', a transient organic psychosis preceding the fixed motor stage.

Q22. Stage 3 manganism (permanent damage) features akinesia, rigidity and:
A) A resting pill-rolling tremor
B) Pure sensory loss
C) Intention tremor and dystonia (not a resting tremor)
D) Nystagmus

Answer: C) Intention tremor and dystonia (not a resting tremor)

Explanation: Stage 3 brings akinesia and rigidity (worst in the legs) with intention tremor and dystonia, contrasting with the resting tremor of idiopathic Parkinson disease.

Vs Parkinson

Q23. Compared with idiopathic Parkinson disease, manganism characteristically shows:
A) An identical resting pill-rolling tremor
B) Intention tremor and dystonia rather than a resting tremor
C) No movement disorder of any kind
D) Purely cerebellar signs with nystagmus

Answer: B) Intention tremor and dystonia rather than a resting tremor

Explanation: Manganism produces intention tremor and dystonia, features that help distinguish it from the resting tremor of idiopathic Parkinson disease.

Q24. In idiopathic Parkinson disease the chief affected region is the substantia nigra, whereas manganese predominantly affects the:
A) Cerebellum
B) Spinal cord
C) Optic nerve
D) Striatum and pallidum (basal ganglia)

Answer: D) Striatum and pallidum (basal ganglia)

Explanation: Manganese mainly affects the striatum and pallidum; idiopathic Parkinson disease chiefly involves the substantia nigra, a key contrast.

Diagnosis & Treatment

Q25. The diagnosis of manganese accumulation in the brain is supported chiefly by:
A) CT and MRI imaging
B) Blood manganese alone
C) Urinary ALA
D) Spirometry

Answer: A) CT and MRI imaging

Explanation: Imaging (CT/MRI, with characteristic basal-ganglia signal) supports the diagnosis, since blood and urine manganese correlate poorly with neurotoxicity.

Q26. Treatment options described for manganism include removal from exposure and:
A) A single curative course of penicillamine
B) High-dose oral iron to displace the metal
C) EDTA, levodopa, or para-aminosalicylic acid as a chelator
D) Routine haemodialysis to clear manganese

Answer: C) EDTA, levodopa, or para-aminosalicylic acid as a chelator

Explanation: Beyond removal from exposure, options include EDTA, a levodopa trial, or sodium para-aminosalicylic acid as a chelator, though established disease responds poorly.

Uses

Q27. Manganese-hardened steels are known as:
A) Stainless steel only
B) Ferro-manganese / silico-manganese
C) Spinels
D) Brass

Answer: B) Ferro-manganese / silico-manganese

Explanation: Harder manganese steels are termed ferro-manganese, silico-manganese or manganine; manganese is also used in dry cells, paints and permanganate.

Metabolism

Q28. In blood, manganese is bound predominantly to:
A) Albumin
B) Globulin
C) Platelets
D) Red blood cells

Answer: D) Red blood cells

Explanation: Manganese is bound mainly to red cells in blood, poorly absorbed from the gut and excreted chiefly in bile, with gut excretion enhanced by iron deficiency.

Respiratory

Q29. Inhaled manganese can cause a pulmonary effect of:

A) Inflammatory lung reaction with increased susceptibility to infection
B) Bilateral calcified pleural plaques
C) Malignant pleural mesothelioma
D) Eggshell calcification of hilar nodes

Answer: A) Inflammatory lung reaction with increased susceptibility to infection

Explanation: Manganese inhalation can provoke an inflammatory lung reaction, increasing susceptibility to respiratory infection ('manganese pneumonia').

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UNIT II — METALS

Mercury

TOPICS IN THIS CHAPTER

Forms & Sources

Q1. Mercury exists in three toxicologically distinct forms:

A) Distinguished only as solid, liquid and gas
B) Classified as acidic, basic and neutral forms
C) Divided into radioactive, stable and inert types
D) Elemental, inorganic and organic

Answer: D) Elemental, inorganic and organic

Explanation: Mercury's toxicity depends on form: elemental (vapour), inorganic salts, and organic (e.g. methylmercury), each with a different clinical picture.

Q2. Occupational elemental mercury exposure classically occurs in:

A) Cotton mills
B) Chloralkali plants, thermometer/instrument and dental work
C) Coal mines
D) Bakeries

Answer: B) Chloralkali plants, thermometer/instrument and dental work

Explanation: Elemental mercury vapour is encountered in chloralkali production, instrument and thermometer manufacture and dentistry; the distractors are unrelated.

Q3. The 'mad hatter' association arose from mercury use in:

A) Steel making
B) Glass blowing
C) Felt hat manufacture
D) Tanning leather

Answer: C) Felt hat manufacture

Explanation: Mercuric nitrate used in felting hats exposed hatters to mercury, causing the neuropsychiatric syndrome that inspired the 'mad hatter'.

Q4. Artisanal gold mining is a major global source of mercury exposure because:

A) Mercury is used to amalgamate and is then burned off as vapour
B) Gold ore naturally contains a high mercury content
C) Mercury is co-mined alongside the gold ore
D) The mercury actually prevents worker exposure

Answer: A) Mercury is used to amalgamate and is then burned off as vapour

Explanation: Small-scale gold miners use mercury to amalgamate gold then heat it, releasing large amounts of toxic vapour, a worldwide exposure problem.

Elemental Mercury

Q5. Inhaled elemental mercury vapour is absorbed and primarily affects the:

A) Skin
B) Bone marrow
C) Thyroid
D) Central nervous system

Answer: D) Central nervous system

Explanation: Lipophilic mercury vapour crosses into the CNS, so elemental mercury toxicity is dominated by neurological and neuropsychiatric features.

Q6. The neuropsychiatric syndrome of chronic mercury exposure is called:

A) Erethism is no.
B) Erethism
C) Acrodynia
D) Manganism

Answer: B) Erethism

Explanation: Erethism comprises irritability, shyness, emotional lability, insomnia and memory loss; acrodynia and manganism relate to other exposures.

Q7. A characteristic motor sign of chronic elemental mercury toxicity is:

A) Resting pill-rolling tremor only
B) Wrist drop
C) Intention tremor (hands, eyelids, tongue)
D) Chorea

Answer: C) Intention tremor (hands, eyelids, tongue)

Explanation: Mercury causes a fine intention tremor of the hands, eyelids and tongue, often with the neuropsychiatric features of erethism.

Q8. Oral features of chronic mercury exposure include:
A) Gingivitis and excessive salivation
B) Burton's gum line
C) Garlic breath
D) Mees' lines

Answer: A) Gingivitis and excessive salivation

Explanation: Mercury causes gingivitis, stomatitis and hypersalivation; Burton's line is lead, garlic breath is arsenic and Mees' lines are nail signs of arsenic.

Q9. Why is a mercury spill (e.g. broken thermometer) an inhalation hazard?
A) It explodes
B) It emits radiation
C) It is absorbed through intact skin rapidly
D) Liquid mercury slowly volatilises at room temperature

Answer: D) Liquid mercury slowly volatilises at room temperature

Explanation: Metallic mercury evaporates at room temperature, so an uncontained spill releases vapour that is then inhaled and absorbed efficiently.

Q10. Renal effects can also occur with elemental mercury because, after absorption, it is:
A) Converted within tissues directly to methylmercury
B) Oxidised to inorganic mercury that accumulates in the kidney
C) Stored permanently and only within the skeleton
D) Excreted entirely unchanged in the exhaled breath

Answer: B) Oxidised to inorganic mercury that accumulates in the kidney

Explanation: Absorbed elemental mercury is oxidised intracellularly to the inorganic form, which accumulates in the kidney and can cause tubular injury.

Inorganic Mercury

Q11. Inorganic mercury salts (e.g. mercuric chloride) are notably:
A) Completely harmless even if accidentally swallowed
B) Toxic only to the central nervous system
C) Corrosive to the gut and toxic to the kidney
D) Capable only of acting as a skin irritant

Answer: C) Corrosive to the gut and toxic to the kidney

Explanation: Inorganic mercury salts are corrosive on ingestion and cause acute tubular necrosis, making the kidney the chief target of inorganic exposure.

Q12. The childhood syndrome of acrodynia ('pink disease') is caused by:
A) Mercury exposure
B) Lead exposure
C) Arsenic exposure
D) Cadmium exposure

Answer: A) Mercury exposure

Explanation: Acrodynia, with painful pink extremities, irritability and desquamation, is a mercury hypersensitivity reaction, historically from teething powders.

Q13. Acute ingestion of a corrosive inorganic mercury salt causes:
A) Painless diarrhoea
B) A movement disorder
C) Garlic breath
D) Haematemesis, abdominal pain and acute tubular necrosis

Answer: D) Haematemesis, abdominal pain and acute tubular necrosis

Explanation: Mercuric chloride is corrosive, causing severe gastroenteritis and shock, followed by acute tubular necrosis and renal failure.

Organic Mercury

Q14. Methylmercury poisoning from contaminated fish caused the epidemic known as:
A) Itai-itai disease
B) Minamata disease
C) Blackfoot disease
D) Byssinosis

Answer: B) Minamata disease

Explanation: Methylmercury discharged into Minamata Bay bioaccumulated in fish and caused severe neurological disease in the consuming population.

Q15. Classic features of methylmercury (Minamata) neurotoxicity include:
A) Wrist drop and colic
B) Garlic breath and Mees' lines
C) Paraesthesiae, ataxia, constricted visual fields and hearing loss
D) Bone pain and fractures

Answer: C) Paraesthesiae, ataxia, constricted visual fields and hearing loss

Explanation: Methylmercury damages the cerebellum and sensory cortex, causing paraesthesiae, ataxia, concentric visual-field constriction, dysarthria and deafness.

Q16. Methylmercury is particularly dangerous in pregnancy because it:
A) Crosses the placenta and damages the developing brain
B) Cannot cross the placenta
C) Protects the fetus
D) Only affects the mother's kidney

Answer: A) Crosses the placenta and damages the developing brain

Explanation: Methylmercury readily crosses the placenta and is a potent developmental neurotoxin, so fish-consumption advice targets pregnant women.

Q17. Organic mercury bioaccumulates and biomagnifies in:
A) Soil only
B) Air only
C) Bone only
D) Aquatic food chains (large predatory fish)

Answer: D) Aquatic food chains (large predatory fish)

Explanation: Methylmercury concentrates up aquatic food chains, reaching highest levels in large predatory fish, the main route of human exposure.

Q18. A tragic cause of mass methylmercury poisoning besides Minamata was:
A) Tetraethyl-lead additives used in leaded petrol
B) Consumption of seed grain treated with mercurial fungicide (Iraq)
C) Inhalation of smoke from domestic coal fires
D) Asbestos-cement pipes carrying drinking water

Answer: B) Consumption of seed grain treated with mercurial fungicide (Iraq)

Explanation: Mercury-fungicide-treated seed grain, eaten rather than planted, caused a large methylmercury poisoning epidemic in Iraq.

Biological Monitoring

Q19. Exposure to elemental and inorganic mercury is best monitored by:
A) Blood organic mercury
B) Hair arsenic
C) Urine mercury
D) Serum lead

Answer: C) Urine mercury

Explanation: Urinary mercury reflects elemental/inorganic exposure and body burden, whereas blood mercury better reflects recent organic (methyl) mercury exposure.

Q20. Recent methylmercury (organic) exposure is best assessed by:
A) Blood mercury
B) Urine mercury
C) Zinc protoporphyrin
D) Urinary ALA

Answer: A) Blood mercury

Explanation: Blood mercury (and hair) reflects organic methylmercury exposure; urine mercury is the index for elemental and inorganic forms.

Q21. A limitation of using urine mercury is that it:
A) Cannot be measured
B) Reflects only organic mercury
C) Detects lead
D) Reflects inorganic/elemental exposure but not organic mercury well

Answer: D) Reflects inorganic/elemental exposure but not organic mercury well

Explanation: Urine mercury indexes elemental and inorganic exposure but underestimates organic methylmercury, for which blood (and hair) is preferred.

Management

Q22. Chelation for inorganic/elemental mercury poisoning may use:
A) Deferoxamine
B) DMSA, DMPS or dimercaprol
C) Atropine
D) Calcium gluconate

Answer: B) DMSA, DMPS or dimercaprol

Explanation: Inorganic and elemental mercury are chelated with DMSA, DMPS or dimercaprol; deferoxamine is for iron.

Q23. Dimercaprol (BAL) is contraindicated in methylmercury poisoning because it:
A) Causes deafness
B) Is ineffective for all metals
C) Can redistribute mercury into the brain
D) Raises blood pressure

Answer: C) Can redistribute mercury into the brain

Explanation: BAL may shift methylmercury into the central nervous system and worsen neurotoxicity, so water-soluble chelators (DMSA, DMPS) are preferred.

Q24. The essential first measure in occupational mercury toxicity is:
A) Removal from exposure and decontamination
B) Lifelong chelation
C) Thyroidectomy
D) Bone marrow transplant

Answer: A) Removal from exposure and decontamination

Explanation: As with all metals, stopping exposure (and decontaminating spills of volatile mercury) is the first and most important step.

Q25. Beyond chelation, management of chronic mercury toxicity emphasises:
A) Immediate and routine haemodialysis of the patient
B) Lifelong systemic corticosteroid therapy
C) Splenectomy to reduce red-cell destruction
D) Removal from exposure and decontamination of the workplace

Answer: D) Removal from exposure and decontamination of the workplace

Explanation: Stopping exposure and remediating mercury-contaminated workplaces are central; chelation is adjunctive and ineffective for established CNS injury.

Distribution & Metabolism

Q26. Methylmercury concentrates in red blood cells by a factor of roughly:
A) Not at all
B) 10 to 20 fold
C) 100 fold over plasma equally
D) Only in plasma

Answer: B) 10 to 20 fold

Explanation: Methylmercury is concentrated about 10-20 fold in red cells (unlike inorganic mercury, which distributes about equally between cells and plasma).

Q27. The principal target organs of mercury are the:
A) Liver and spleen
B) Bone and marrow
C) CNS and kidneys
D) Skin and nails

Answer: C) CNS and kidneys

Explanation: Mercury's chief targets are the central nervous system and the kidneys; the form determines which predominates.

Q28. Organic mercury undergoes enterohepatic recycling because it is:
A) Excreted in bile then reabsorbed from the gut
B) Only exhaled
C) Stored permanently in bone
D) Not excreted at all

Answer: A) Excreted in bile then reabsorbed from the gut

Explanation: Organomercury is excreted in bile and reabsorbed from the gut (enterohepatic circulation), which is why bile-acid-binding resins aid its removal.

Acute Toxicity

Q29. Acute mercury ingestion causes oropharyngeal and gut injury plus renal effects including:
A) Polyuria with dilute urine
B) No renal effect
C) Isolated glycosuria
D) Albuminuria, haematuria, oliguria and casts

Answer: D) Albuminuria, haematuria, oliguria and casts

Explanation: Acute mercury causes corrosive oropharyngeal injury, vomiting and abdominal pain, with renal damage shown by albuminuria, haematuria, oliguria and urinary casts.

Chronic - Oral

Q30. Oral features of chronic mercury exposure include gingivitis, glossitis and:

A) A dry mouth
B) Hypersalivation (mercurial ptyalism) with a metallic taste
C) Painless ulcers only
D) Loss of taste only

Answer: B) Hypersalivation (mercurial ptyalism) with a metallic taste

Explanation: Chronic mercury causes gingivitis, hypersalivation (ptyalism), a bitter metallic taste and bluish gum-margin pigmentation from mercury sulphide.

Chronic - CNS

Q31. The tremor of chronic mercury exposure is sometimes called:

A) Resting pill-rolling tremor
B) Asterixis
C) Hatter's shake (mercurial micro-parkinsonism)
D) Essential tremor only

Answer: C) Hatter's shake (mercurial micro-parkinsonism)

Explanation: Mercury produces a characteristic tremor ('hatter's shake', mercurial micro-parkinsonism) with head-shaking, part of the mad-hatter picture.

Q32. A useful negative sign distinguishing mercurial CNS disease from other cerebellar/MS pictures is:

A) Absence of nystagmus
B) Presence of nystagmus
C) Presence of fever
D) Absence of tremor

Answer: A) Absence of nystagmus

Explanation: Although mercury can mimic parkinsonism, multiple sclerosis or cerebellar disease, the absence of nystagmus is a noted distinguishing feature.

Chronic - Eye

Q33. Haemorrhage or deposition in the lens from chronic mercury exposure is termed:

A) Mercuria nigra
B) Arc eye
C) Cataracta complicata
D) Mercuria lentis

Answer: D) Mercuria lentis

Explanation: Mercurial deposition/haemorrhage in the lens is described as mercuria lentis, an ophthalmic sign of chronic mercury exposure.

Chronic - Renal

Q34. Renal injury in chronic mercury exposure is mainly:

A) Glomerular only
B) Tubular damage, more with inorganic than organic mercury
C) Absent
D) Only with organic mercury

Answer: B) Tubular damage, more with inorganic than organic mercury

Explanation: Mercury chiefly damages the renal tubule (more so with inorganic mercury); glomerular injury with albuminuria and even nephrotic syndrome can also occur.

Erethism

Q35. Erethism, the neuropsychiatric syndrome of mercury, includes:

A) Euphoria with overconfidence and disinhibition
B) Pure motor weakness without psychiatric change
C) Morbid irritability, temper outbursts, poor concentration and depression
D) Isolated sensorineural deafness without other signs

Answer: C) Morbid irritability, temper outbursts, poor concentration and depression

Explanation: Erethism is an organic psychosis with morbid irritability, emotional lability, temper outbursts, memory and concentration impairment, depression and somnolence.

Minamata

Q36. Minamata disease, from methylmercury, presented with a syndrome of:

A) Polyneuropathy, cerebellar ataxia, dysarthria, deafness and constricted (tubular) vision
B) Wrist drop with crampy abdominal colic
C) Acute intravascular haemolysis with jaundice
D) Bone pain with osteomalacic fractures

Answer: A) Polyneuropathy, cerebellar ataxia, dysarthria, deafness and constricted (tubular) vision

Explanation: Minamata disease combined polyneuropathy with cerebellar ataxia, dysarthria, deafness and tubular (constricted) vision; its case-fatality was around 40%.

Q37. A notable epidemiological feature of Minamata disease was that it:

A) Affected only adult men
B) Spared children
C) Affected only the elderly
D) Affected both sexes and all ages roughly equally

Answer: D) Affected both sexes and all ages roughly equally

Explanation: Minamata disease affected both sexes and all ages fairly equally, reflecting widespread dietary exposure to methylmercury-contaminated fish.

Treatment

Q38. First-aid for acute mercury ingestion classically includes giving:

A) Strong acid
B) Milk as a demulcent, with gastric lavage
C) A stimulant
D) Nothing by mouth ever

Answer: B) Milk as a demulcent, with gastric lavage

Explanation: Acute mercury poisoning is an emergency; milk is given as a demulcent and gastric lavage performed, with airway protection as needed.

Q39. In organomercury ingestion, faecal excretion can be enhanced before lavage by giving:

A) Activated charcoal only
B) Oral iron
C) Cholestyramine (a bile-acid-binding resin)
D) Calcium gluconate

Answer: C) Cholestyramine (a bile-acid-binding resin)

Explanation: Cholestyramine binds bile salts and interrupts the enterohepatic recycling of organomercury, increasing its faecal excretion.

Q40. Dimercaprol (BAL) is highly effective for inorganic mercury but is contraindicated in organic mercury because it:

A) Enhances mercury's passage across the blood-brain barrier
B) Causes deafness
C) Is inactivated by bile
D) Raises blood pressure

Answer: A) Enhances mercury's passage across the blood-brain barrier

Explanation: BAL effectively chelates inorganic mercury but in organomercury poisoning it can increase movement of mercury across the blood-brain barrier, worsening CNS injury.

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UNIT II — METALS

Nickel

TOPICS IN THIS CHAPTER

Sources & Uses

Q1. Nickel is used principally in the manufacture of:
A) Cotton textiles
B) Stainless steel and corrosion-resistant alloys
C) Glass fibre
D) Cement

Answer: B) Stainless steel and corrosion-resistant alloys

Explanation: Nickel is used in stainless steel, aero-engine and corrosion-resistant alloys, nickel-cadmium batteries and spinel alloys.

Skin Effects

Q2. 'Nickel itch' (nickel rash) is a form of:
A) Chemical burn
B) Fungal infection
C) Photodermatitis
D) Contact dermatitis

Answer: D) Contact dermatitis

Explanation: Nickel itch is an allergic contact dermatitis, diagnosed by patch testing and the lymphocyte proliferation test, one of nickel's commonest effects.

Q3. Nickel contact dermatitis is confirmed by:
A) Patch test and lymphocyte proliferation test
B) Blood nickel only
C) Chest X-ray
D) Spirometry

Answer: A) Patch test and lymphocyte proliferation test

Explanation: Nickel dermatitis is diagnosed with the patch test and the lymphocyte proliferation test, demonstrating sensitisation to nickel.

Respiratory

Q4. Inhalation of soluble nickel or nickel-oxide fume from welding can cause:
A) Pneumoconiosis with PMF
B) Pleural plaques
C) Occupational asthma
D) Mesothelioma

Answer: C) Occupational asthma

Explanation: Soluble nickel and nickel-oxide welding fume can cause occupational (bronchial) asthma, besides nickel's carcinogenic and dermatitis effects.

Carcinogenicity

Q5. Nickel is carcinogenic, classically causing cancer of the:
A) Bladder only
B) Nasal sinus and lung (and pharynx)
C) Skin only
D) Liver only

Answer: B) Nasal sinus and lung (and pharynx)

Explanation: Nickel causes carcinoma of the nasal cavity/sinus, lung and pharynx, an important occupational cancer association.

Nickel Carbonyl

Q6. Nickel carbonyl, Ni(CO)4, is hazardous because it is:
A) A chemically inert, harmless solid powder
B) A non-absorbable, biologically stable compound
C) An odourless gas of negligible toxicity
D) A highly toxic volatile compound (liquid with toxic vapour)

Answer: D) A highly toxic volatile compound (liquid with toxic vapour)

Explanation: Nickel carbonyl is a volatile liquid (boiling point ~43 C) whose vapour is highly toxic, formed during the Mond process of nickel purification.

Q7. The Mond process, which generates nickel carbonyl, purifies nickel by passing over it:
A) Carbon monoxide
B) Oxygen
C) Chlorine
D) Hydrogen sulphide

Answer: A) Carbon monoxide

Explanation: In the Mond process, carbon monoxide is passed over finely divided nickel to form volatile Ni(CO)4, which later deposits very pure nickel at higher temperature.

Q8. Nickel carbonyl is often compared to which other toxic gas, and has what odour?
A) Ammonia-like, fishy odour
B) Chlorine-like, bleach odour
C) Phosgene-like hazard, with a brick-dust odour
D) Odourless and harmless

Answer: C) Phosgene-like hazard, with a brick-dust odour

Explanation: Nickel carbonyl is a dangerous gas likened to phosgene, with a brick-dust odour detectable around 1-3 ppm.

Q9. After early symptoms settle, nickel carbonyl exposure is dangerous because of a:
A) An immediate and painless full recovery
B) Delayed potentially lethal phase up to ~36 hours later
C) A permanent cure once symptoms settle
D) A chronic relapsing skin rash only

Answer: B) Delayed potentially lethal phase up to ~36 hours later

Explanation: Initial headache, dizziness, nausea, vomiting and dyspnoea may settle, but a delayed lethal phase (interstitial pneumonitis, pulmonary/cerebral oedema) can follow up to ~36 hours later.

Q10. To distinguish nickel-carbonyl poisoning from carbon-monoxide poisoning, a useful test is:
A) Blood nickel only
B) Chest X-ray only
C) Spirometry
D) Carboxyhaemoglobin (normal COHb favours Ni(CO)4 poisoning)

Answer: D) Carboxyhaemoglobin (normal COHb favours Ni(CO)4 poisoning)

Explanation: Since the Mond process uses CO, a normal carboxyhaemoglobin points to nickel-carbonyl rather than carbon-monoxide poisoning as the cause.

Q11. Treatment of nickel-carbonyl poisoning classically uses:
A) Disulfiram, with a short tapering course of prednisolone
B) Calcium-disodium EDTA chelation used alone
C) A single dose of intramuscular atropine
D) Supportive care with no specific treatment

Answer: A) Disulfiram, with a short tapering course of prednisolone

Explanation: Nickel-carbonyl poisoning is treated with disulfiram as soon as possible and a short tapering course of prednisolone; alcohol must be avoided on disulfiram.

Monitoring

Q12. Biological monitoring of nickel exposure uses:
A) Blood lead
B) Serum calcium
C) Urinary nickel (normal under ~50 micrograms/L)
D) Hair zinc

Answer: C) Urinary nickel (normal under ~50 micrograms/L)

Explanation: Urinary nickel is the biological index of exposure, with a normal level under about 50 micrograms/L in unexposed people.

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UNIT II — METALS

Phosphorus & Phosphine

TOPICS IN THIS CHAPTER

Forms & Sources

Q1. The most toxic form of elemental phosphorus is:
A) Red phosphorus
B) White (yellow) phosphorus
C) Black phosphorus
D) All equally toxic

Answer: B) White (yellow) phosphorus

Explanation: White (yellow) phosphorus is the most toxic form; red phosphorus (used in match-making) and black phosphorus are far less toxic.

Q2. Phosphorus is used in all of the following EXCEPT:
A) Fireworks and explosives
B) Fertilisers and detergents
C) Insecticides and rodenticides
D) As an inert food preservative

Answer: D) As an inert food preservative

Explanation: Phosphorus is used in fireworks, explosives, fertilisers, detergents, pharmaceuticals and pesticides; it is not an inert food preservative.

Phossy Jaw

Q3. The classic occupational disease of white phosphorus, described by Donald Hunter, is:
A) Phossy jaw (phosphorus necrosis of the jaw)
B) Chrome ulcers of the exposed skin
C) Itai-itai disease with osteomalacia
D) Manganism with extrapyramidal features

Answer: A) Phossy jaw (phosphorus necrosis of the jaw)

Explanation: Phossy jaw, necrosis of the mandible from white-phosphorus exposure, is the classic and tragic occupational disease, described by Donald Hunter.

Q4. Phossy jaw characteristically develops:
A) Acutely, within a few hours of exposure
B) Only in exposed children, never in adults
C) Slowly, over about five years, in those with poor dental health
D) Immediately, without any latent period

Answer: C) Slowly, over about five years, in those with poor dental health

Explanation: Phossy jaw has an insidious onset over about five years; workers with caries/cavities are especially at risk, so dental health is important before such work.

Q5. In phossy jaw the mandible becomes necrotic with:
A) Eggshell calcification of the hilar nodes
B) Osteoporotic sequestra and abscesses discharging foul-smelling pus
C) Calcified bilateral pleural plaques
D) Basophilic stippling of the red cells

Answer: B) Osteoporotic sequestra and abscesses discharging foul-smelling pus

Explanation: The mandible becomes necrotic with osteoporotic sequestration and abscesses discharging foul pus; mortality (about 20%) is mainly from septicaemia.

Q6. The treatment of choice for phossy jaw is:
A) Chelation
B) Antibiotics alone
C) Observation only
D) Surgical removal

Answer: D) Surgical removal

Explanation: Surgical removal of the necrotic bone is the treatment of choice for phossy jaw, alongside management of infection.

Other Effects

Q7. Other effects of white phosphorus include:
A) Bony necrosis elsewhere and serious skin burns
B) Only a mild rash
C) Reversible anaemia only
D) No systemic effects

Answer: A) Bony necrosis elsewhere and serious skin burns

Explanation: White phosphorus can cause bony necrosis elsewhere and serious skin burns, and is irritant to eyes and the respiratory tract via phosphoric acid/sulphide.

Phosphine

Q8. Phosphine (PH_3) is occupationally important especially as a:
A) Welding shield gas
B) Refrigerant
C) Fumigant in grain stores
D) Food additive

Answer: C) Fumigant in grain stores

Explanation: Phosphine is a colourless flammable gas used to synthesise organophosphorus compounds and semiconductors, and importantly as a grain-store fumigant.

Q9. Phosphine is liberated when which compounds become wet?

A) Lead oxide
B) Zinc and aluminium phosphide
C) Calcium carbonate
D) Sodium chloride

Answer: B) Zinc and aluminium phosphide

Explanation: Phosphine is generated when zinc or aluminium phosphide is wetted (as in fumigation and acetylene manufacture), a common poisoning scenario.

Q10. Toxic features of phosphine exposure include:

A) Painless jaundice only
B) Isolated wrist drop
C) A chronic skin rash only
D) Headache, chest tightness, epigastric pain, pulmonary oedema and convulsions

Answer: D) Headache, chest tightness, epigastric pain, pulmonary oedema and convulsions

Explanation: Phosphine causes headache, weakness, chest tightness/pain, nausea, severe epigastric pain, pulmonary oedema and CNS effects (convulsions, coma, death).

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UNIT III — PHYSICAL AGENTS

Decompression & Barometric Pressure

TOPICS IN THIS CHAPTER

Barometric Pressure: Principles

Q1. Workers exposed to raised barometric pressure include divers and:
A) High-altitude pilots
B) Tunnel and caisson workers in compressed air
C) Foundry furnacemen
D) Office data-entry staff

Answer: B) Tunnel and caisson workers in compressed air

Explanation: Raised pressure is encountered by divers underwater and by caisson and tunnel workers in compressed air; pilots and high-altitude work involve reduced pressure.

Q2. As a diver descends, the increasing pressure causes gas in body cavities to follow Boyle's law, meaning the gas volume:
A) Increases as pressure rises
B) Stays constant
C) Becomes solid
D) Decreases as pressure rises

Answer: D) Decreases as pressure rises

Explanation: By Boyle's law, at constant temperature gas volume is inversely proportional to pressure, so descent compresses gas in the ears, sinuses and lungs - the basis of compression barotrauma.

Q3. Normal atmospheric pressure at sea level is approximately:
A) 100 kPa
B) 700 kPa
C) 10 kPa
D) 1000 kPa

Answer: A) 100 kPa

Explanation: Sea-level atmospheric pressure is about 100 kPa (one atmosphere); pressure rises by roughly one atmosphere for every 10 metres of sea-water descended.

Compression Barotrauma

Q4. On descent, the commonest barotrauma affects the:
A) Femur
B) Liver
C) Middle ear
D) Kidney

Answer: C) Middle ear

Explanation: Middle-ear barotrauma is the commonest descent injury, with ear pain radiating to the neck as the Eustachian tube fails to equalise the rising external pressure.

Q5. Sudden relief of middle-ear barotrauma pain during descent often indicates:
A) Resolution of the barotrauma without any injury
B) Rupture of the tympanic membrane
C) Onset of decompression sickness on descent
D) The Eustachian tube clearing to equalise pressure

Answer: B) Rupture of the tympanic membrane

Explanation: Sudden pain relief usually means the tympanic membrane has ruptured under the pressure difference, equalising it; the rupture is normally inward toward the middle ear.

Q6. Inner-ear barotrauma, which may rupture the round or oval window causing persistent vertigo and sensorineural hearing loss, is best treated by:
A) Decongestants and watchful waiting
B) Recompression therapy alone
C) A course of oral antibiotics alone
D) Immediate operative repair

Answer: D) Immediate operative repair

Explanation: Inner-ear barotrauma with window rupture and a perilymph fistula causes persistent vertigo and sensorineural deafness, and the treatment of choice is prompt surgical repair.

Q7. Transient vertigo from unequal pressure between the two middle ears during a pressure change is termed:
A) Alternobaric vertigo
B) Meniere disease
C) Decompression sickness
D) Heat syncope

Answer: A) Alternobaric vertigo

Explanation: Alternobaric vertigo is a transient spinning sensation from a pressure inequality between the right and left middle ears, distinct from the inner-ear barotrauma that persists.

Decompression Illness

Q8. Decompression illness on ascent is caused by the evolution of bubbles formed mainly from which dissolved gas?
A) Oxygen
B) Carbon dioxide
C) Nitrogen
D) Carbon monoxide
Answer: C) Nitrogen
Explanation: On ascent, dissolved nitrogen (and to a lesser extent helium) comes out of solution as bubbles if decompression is too rapid; oxygen is largely metabolised.

Q9. Microbubbles formed at depth are normally eliminated safely if the diver:
A) Ascends as fast as possible
B) Ascends slowly, exhaling the gas
C) Holds the breath on ascent
D) Descends again immediately
Answer: B) Ascends slowly, exhaling the gas
Explanation: A slow, staged ascent lets dissolved gas leave the tissues and be exhaled; a rapid ascent traps gas as bubbles in tissues and the circulation, causing decompression illness.

Q10. The musculoskeletal form of decompression illness is called 'the bends' because:
A) Bubbles only form when bending
B) It bends the bones
C) The diver must bend to ascend
D) Pain in a joint is relieved by bending it
Answer: D) Pain in a joint is relieved by bending it
Explanation: Joint pain - typically in the shoulders and knees from intra-articular and peri-articular bubbles - is classically eased by bending the joint, giving 'the bends' its name.

Q11. A blotchy red-purple skin rash of decompression illness, due to superficial vasodilatation and stasis, is termed:
A) Cutis marmorata
B) Erythema ab igne
C) Cutis laxa
D) Pterygium
Answer: A) Cutis marmorata
Explanation: Cutis marmorata is the mottled red-purple skin rash of cutaneous decompression illness; erythema ab igne is instead a chronic heat-related pigmentation.

Q12. The respiratory form of decompression illness, with substernal pain, cough and dyspnoea from pulmonary gas emboli, is called:
A) The bends
B) The staggers
C) The chokes
D) Arc eye
Answer: C) The chokes
Explanation: 'The chokes' is the pulmonary form, with substernal pain, cough and breathlessness from venous gas bubbles lodging in the pulmonary circulation; 'the staggers' is the vestibular form.

Q13. The vestibular form of neurological decompression illness, with vertigo, nausea and sometimes deafness, is called:
A) The chokes
B) The staggers
C) The bends
D) Skin bends
Answer: B) The staggers
Explanation: 'The staggers' is the vestibular form of decompression illness, with vertigo, nausea, vomiting and sometimes deafness; 'the chokes' is pulmonary and 'the bends' musculoskeletal.

Q14. Decompression illness is conventionally classified, with the more serious neurological and pulmonary features belonging to:
A) Type I (mild)
B) Stage 0
C) Class I lasers
D) Type II (serious)
Answer: D) Type II (serious)
Explanation: Type II decompression illness involves the serious neurological, pulmonary and vestibular features, whereas Type I covers the milder skin, joint and constitutional symptoms.

Q15. An examiner's clue to neurological decompression illness is a diminished sense of:
A) Vibration
B) Smell
C) Taste
D) Hearing high tones
Answer: A) Vibration
Explanation: Diminished vibration sense is an important early sign of neurological decompression illness, alongside limb weakness, sphincter disturbance and sensory changes.

Q16. Sudden loss of consciousness immediately on surfacing after a rapid ascent suggests:
A) Simple fatigue
B) Heat stroke
C) Arterial gas embolism from pulmonary barotrauma
D) Nitrogen narcosis at depth

Answer: C) Arterial gas embolism from pulmonary barotrauma

Explanation: A rapid ascent can over-expand the lungs (pulmonary barotrauma), forcing gas into the arterial circulation as an embolism, causing collapse and loss of consciousness on surfacing.

Long-Term Effects & Treatment

Q17. A serious chronic complication of repeated decompression exposure, causing severe joint pain from bone death, is:
A) Osteomalacia
B) Dysbaric osteonecrosis
C) Osteomyelitis
D) Paget disease

Answer: B) Dysbaric osteonecrosis

Explanation: Dysbaric osteonecrosis is avascular bone necrosis from repeated decompression, causing severe joint pain and disability, typically around the shoulder and hip.

Q18. The definitive treatment of serious decompression illness is:
A) Oral analgesia and bed rest alone
B) A rapid ascent to high altitude
C) Breathing a nitrogen-enriched gas mix
D) Recompression in a chamber with hyperbaric oxygen

Answer: D) Recompression in a chamber with hyperbaric oxygen

Explanation: Serious decompression illness is treated by recompression in a chamber with hyperbaric oxygen, which shrinks the bubbles and improves tissue oxygenation; transport should be at low altitude.

Q19. While awaiting recompression, a conscious diver with decompression illness should be given:
A) 100% oxygen, lying flat
B) Nitrogen-enriched air
C) Nothing by any route
D) A rapid return to depth

Answer: A) 100% oxygen, lying flat

Explanation: High-concentration oxygen is given to a conscious casualty lying flat, to speed nitrogen washout, pending transfer to a recompression chamber; unresponsive but breathing casualties are laid on the side.

Q20. To avoid worsening bubble formation, a diver with decompression illness should be transported:
A) By high-altitude flight
B) Up a mountain road
C) By low-altitude flight or ground
D) After a rapid ascent

Answer: C) By low-altitude flight or ground

Explanation: Transport at low altitude (or ground level) avoids the further pressure reduction of altitude, which would expand bubbles and worsen the illness; hyperbaric treatment follows.

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UNIT III — PHYSICAL AGENTS

Heat Disorders

TOPICS IN THIS CHAPTER

Thermoregulation & Heat Balance

Q1. The four physical routes by which the body exchanges heat with its environment are:
A) Filtration, osmosis, diffusion and active transport
B) Inhalation, ingestion, dermal and parenteral
C) Sweating, shivering, panting and piloerection
D) Radiation, convection, conduction and evaporation

Answer: D) Radiation, convection, conduction and evaporation

Explanation: Body heat is exchanged by radiation, convection, conduction and evaporation; in a hot workplace, evaporation of sweat becomes the main avenue of heat loss.

Q2. In the heat-balance equation S = M ± (R + C + K) − E, the term M represents:
A) Muscle mass
B) Metabolic heat load
C) Mean skin temperature
D) Moisture content of air

Answer: B) Metabolic heat load

Explanation: M is the metabolic heat load (heat generated by the body); R, C and K are radiation, convection and conduction, E is evaporative loss, and S is stored heat.

Q3. In the heat-balance equation, a positive value of S (stored heat) means the worker is:
A) Losing net heat to the surroundings
B) In exact thermal equilibrium (S = 0)
C) Gaining heat (heat stress)
D) Producing no metabolic heat at all

Answer: C) Gaining heat (heat stress)

Explanation: A positive S indicates net heat gain and rising heat stress; in thermal equilibrium S is zero, with metabolic and environmental heat gain balanced by evaporative loss.

Q4. In a hot environment where air temperature exceeds skin temperature, the only effective avenue of heat loss is:
A) Evaporation of sweat
B) Conduction
C) Radiation
D) Convection

Answer: A) Evaporation of sweat

Explanation: When ambient temperature exceeds skin temperature, the body actually gains heat by radiation, convection and conduction, so evaporation of sweat is the only effective loss.

Q5. Heat strain is best defined as the:
A) Amount of heat in the environment
B) External temperature reading
C) Rate of air movement
D) Physiological and pathological response of the body to heat stress

Answer: D) Physiological and pathological response of the body to heat stress

Explanation: Heat stress is the external heat load imposed on the body, whereas heat strain is the body's physiological and pathological response to that load.

Q6. The two principal physiological responses that compensate for heat stress are:
A) Vasoconstriction and shivering
B) Cutaneous vasodilatation and sweating
C) Bradycardia and apnoea
D) Piloerection and diuresis

Answer: B) Cutaneous vasodilatation and sweating

Explanation: The body compensates for heat by cutaneous vasodilatation (carrying heat to the surface) and sweating (allowing evaporative loss); failure of these leads to heat illness.

Q7. The body's principal temperature-regulating centre (thermostat) is the:
A) Cerebellum
B) Medulla oblongata
C) Hypothalamus
D) Pituitary gland

Answer: C) Hypothalamus

Explanation: The pre-optic anterior hypothalamus acts as the thermostat, integrating central and skin thermoreceptor signals and driving heat-loss or heat-gain responses around a 37 degrees C set-point.

Q8. Sweating is an active secretory process driven by which nerve supply to the eccrine glands?
A) Sympathetic cholinergic fibres
B) Parasympathetic vagal fibres
C) Somatic motor fibres
D) Sympathetic adrenergic fibres only

Answer: A) Sympathetic cholinergic fibres

Explanation: Eccrine sweating is mediated by sympathetic fibres that are unusually cholinergic (releasing acetylcholine); anticholinergic drugs therefore reduce sweating and raise heat risk.

Q9. The concept of a warm 'core' and a cooler 'shell' explains why heat stress first triggers:
A) Immediate vigorous shivering of the skeletal muscles
B) Shutdown of renal blood flow and urine output
C) Reflex bronchodilatation of the airways
D) Cutaneous vasodilatation to shift heat to the surface

Answer: D) Cutaneous vasodilatation to shift heat to the surface

Explanation: Vasodilatation moves warm core blood to the cooler skin shell, raising skin temperature so heat can be lost; if this and sweating are overwhelmed, illness follows.

Q10. High ambient humidity is dangerous in hot work mainly because it:
A) Increases radiant heat
B) Impairs evaporation of sweat
C) Lowers metabolic heat
D) Raises air velocity

Answer: B) Impairs evaporation of sweat

Explanation: When air exceeds skin temperature, evaporation is the only effective heat loss; high humidity blocks evaporation, so the same temperature is far more dangerous when humid.

Q11. An acclimatized worker can sustain a sweat rate of roughly:
A) About 50 ml per hour
B) About 5 litres per hour
C) Over 1 litre per hour
D) Sweating ceases on acclimatization

Answer: C) Over 1 litre per hour

Explanation: A heat-acclimatized worker can sweat well over a litre per hour, which is why fluid and electrolyte replacement is essential during prolonged hot work.

Spectrum of Heat Disorders

Q12. Painful spasms of the calf and abdominal muscles in a heavily sweating worker who has drunk water but not replaced salt indicate:
A) Heat cramps
B) Heat stroke
C) Heat syncope
D) Erythema ab igne

Answer: A) Heat cramps

Explanation: Heat cramps are painful spasms, typically of calf and abdominal muscles, from sodium (salt) loss in sweat when water but not salt is replaced; treatment is salt replacement.

Q13. Heat exhaustion is caused principally by:
A) Failure of the thermoregulatory centre
B) Cessation of sweating
C) Cutaneous vasoconstriction
D) Depletion of water and/or salt

Answer: D) Depletion of water and/or salt

Explanation: Heat exhaustion results from depletion of water and/or salt, with fatigue, headache, nausea and fainting; sweating continues and the core temperature is normal or mildly raised.

Q14. Transient loss of consciousness on standing in the heat, due to peripheral vasodilatation and pooling of blood with reduced cerebral perfusion, is:
A) Heat stroke
B) Heat syncope
C) Heat cramps
D) Heat hyperpyrexia

Answer: B) Heat syncope

Explanation: Heat syncope is fainting from vasodilatation and venous pooling that reduce cerebral blood flow; the thermoregulatory centre is intact and recovery follows lying down.

Q15. The most dangerous heat disorder, a medical emergency, is:
A) Heat cramps
B) Heat syncope
C) Heat stroke
D) Prickly heat

Answer: C) Heat stroke

Explanation: Heat stroke is the gravest disorder, with failure of thermoregulation, very high core temperature and central nervous dysfunction; it carries about 40% mortality.

Q16. The hallmark distinguishing heat stroke from the milder heat disorders is:
A) Failure of thermoregulation with cessation of sweating (hot, dry skin)
B) Profuse sweating that continues with cool, moist skin throughout
C) Fully normal consciousness preserved throughout the episode
D) Painful skeletal muscle cramps as the sole feature
Answer: A) Failure of thermoregulation with cessation of sweating (hot, dry skin)
Explanation: In heat stroke the thermoregulatory centre is paralysed and sweating stops, leaving hot dry skin, soaring core temperature and neurological dysfunction; the milder disorders preserve sweating.

Q17. In heat syncope, unlike heat stroke, the thermoregulatory centre is:
A) Paralysed, exactly as it is in heat stroke
B) Reset to a higher set-point, as in fever
C) Temporarily suppressed by the dehydration
D) Intact (not paralysed)
Answer: D) Intact (not paralysed)
Explanation: In heat syncope the thermoregulatory centre is intact and sweating continues; in heat stroke the centre is paralysed and sweating ceases, which is the key contrast.

Q18. The approximate mortality of heat stroke is around:
A) 4%
B) 40%
C) 1%
D) 90%
Answer: B) 40%
Explanation: Heat stroke carries a high mortality of about 40%, reflecting failure of thermoregulation with multi-organ injury; it demands immediate aggressive cooling.

Q19. Central nervous features of heat stroke include:
A) Wrist drop and foot drop
B) Tunnel vision and deafness
C) Confusion, convulsions and coma
D) Basophilic stippling
Answer: C) Confusion, convulsions and coma
Explanation: Heat stroke produces central nervous dysfunction - confusion, delirium, convulsions and coma - as the soaring core temperature injures the brain.

Q20. In salt-depletion heat exhaustion, the serum sodium tends to be:
A) Low
B) High, as in water-depletion heat exhaustion
C) Normal throughout the illness
D) Raised, with an accompanying low potassium
Answer: A) Low
Explanation: Salt-depletion heat exhaustion (water replaced but not salt) lowers serum sodium and causes cramps and nausea; water-depletion exhaustion instead raises sodium with intense thirst.

Q21. Intense thirst, oliguria and a rising serum sodium in a hot worker suggest:
A) Salt-depletion heat exhaustion
B) Heat syncope
C) Heat cramps
D) Water-depletion heat exhaustion
Answer: D) Water-depletion heat exhaustion
Explanation: Water-depletion heat exhaustion arises from inadequate water intake, producing thirst, scanty concentrated urine and hypernatraemia, distinct from the hyponatraemia of salt depletion.

Q22. Classic (non-exertional) heat stroke typically occurs in:
A) Young fit soldiers on a march
B) Elderly or infirm people during heat waves
C) Trained marathon runners
D) Acclimatized foundry workers
Answer: B) Elderly or infirm people during heat waves
Explanation: Classic heat stroke affects the elderly, infirm or those on predisposing drugs during heat waves and is usually anhidrotic; exertional heat stroke affects young, active people doing heavy work.

Q23. Exertional heat stroke differs from classic heat stroke in that the victim:
A) Is invariably anhidrotic, elderly and chronically infirm
B) Maintains an entirely normal core body temperature
C) Is usually young and active and may still be sweating
D) Never progresses to any form of organ failure

Answer: C) Is usually young and active and may still be sweating

Explanation: Exertional heat stroke strikes young, active people doing heavy work in heat, and sweating may persist; both forms cause dangerous hyperthermia and organ injury.

Q24. A life-threatening complication of severe heat stroke from muscle breakdown is:
A) Rhabdomyolysis with myoglobinuria and acute kidney injury
B) Chronic iron-deficiency anaemia developing over months
C) Peptic ulceration of the stomach and duodenum
D) Posterior subcapsular cataract formation in the lens

Answer: A) Rhabdomyolysis with myoglobinuria and acute kidney injury

Explanation: Severe heat stroke causes rhabdomyolysis, releasing myoglobin that precipitates acute kidney injury; disseminated intravascular coagulation and hepatic failure may also occur.

Q25. Heat stroke is distinguished from heat exhaustion chiefly by the presence of:
A) Profuse sweating with cool, clammy peripheral skin
B) Painful skeletal muscle cramps as the only feature
C) A fully normal and lucid mental state throughout
D) Central nervous dysfunction with very high core temperature

Answer: D) Central nervous dysfunction with very high core temperature

Explanation: Heat stroke is marked by central nervous dysfunction (confusion, convulsions, coma) and a core temperature above 40 degrees C, whereas heat exhaustion preserves consciousness and sweating.

Q26. Heat syncope characteristically recovers when the patient:
A) Is given salt tablets
B) Lies down (restoring cerebral perfusion)
C) Continues working
D) Is warmed

Answer: B) Lies down (restoring cerebral perfusion)

Explanation: Heat syncope is due to vasodilatation and venous pooling reducing cerebral blood flow; lying flat restores perfusion and consciousness, as the thermoregulatory centre is intact.

Skin Effects of Heat

Q27. A brownish, reticulated (net-like) skin discolouration from chronic local heat exposure is called:
A) Burton line
B) Mees' lines
C) Erythema ab igne
D) Acrodynia

Answer: C) Erythema ab igne

Explanation: Erythema ab igne is a brownish reticulated pigmentation caused by chronic exposure to heat (e.g. in furnace and foundry workers), without an actual burn.

Q28. A pre-malignant lesion (squamous cell carcinoma) arising in a chronic heat- or burn-related scar is a:
A) Marjolin's ulcer
B) Chrome ulcer
C) Decubitus ulcer
D) Rodent ulcer

Answer: A) Marjolin's ulcer

Explanation: A Marjolin's ulcer is a squamous cell carcinoma developing in chronically heat-damaged or scarred skin (e.g. on the face from sun or heat), a recognised pre-cancerous-to-malignant change.

Q29. An itchy rash caused by blockage of the sweat-gland ducts in hot, humid conditions is:
A) Marjolin's ulcer
B) Erythema ab igne
C) Chrome ulcer
D) Prickly heat (miliaria)

Answer: D) Prickly heat (miliaria)

Explanation: Prickly heat (miliaria rubra) results from obstruction of sweat-duct openings in hot humid conditions, producing an itchy papular rash and impaired sweating.

Q30. The clear, superficial vesicular form of prickly heat is:
A) Miliaria rubra
B) Miliaria crystallina
C) Miliaria profunda
D) Erythema ab igne

Answer: B) Miliaria crystallina

Explanation: Miliaria crystallina shows superficial clear vesicles from very superficial sweat-duct obstruction; miliaria rubra is the deeper, red, itchy papular 'prickly heat'.

Q31. Extensive prickly heat is important because, by blocking sweat ducts, it can cause:
A) Improved heat tolerance
B) Conductive deafness
C) Anhidrotic heat exhaustion
D) Basophilic stippling

Answer: C) Anhidrotic heat exhaustion

Explanation: Widespread miliaria obstructs many sweat ducts, reducing the worker's ability to sweat and cool, and can precipitate anhidrotic heat exhaustion in the tropics.

Q32. Vigilance for malignant change is needed in heat-damaged scarred skin because of the risk of:
A) Squamous cell carcinoma (Marjolin's ulcer)
B) Basal cell carcinoma only
C) Melanoma exclusively
D) Lipoma

Answer: A) Squamous cell carcinoma (Marjolin's ulcer)

Explanation: A Marjolin's ulcer is a squamous cell carcinoma arising in chronic scar or heat-damaged skin; new ulceration or change in an old scar warrants biopsy.

Measurement of Heat Strain

Q33. Workplace assessment of heat strain by the medical department uses a worker's recovery:
A) Blood lead level
B) Urinary arsenic
C) Spirometry
D) Pulse rate after work, with oral temperature

Answer: D) Pulse rate after work, with oral temperature

Explanation: The medical department measures heat strain by recording the recovery pulse and oral temperature at the workplace after a work period, a physiological (not environmental) index.

Q34. By the recovery-pulse criteria, heat stress is indicated when the oral temperature exceeds:
A) 35 degrees C
B) 37.5 degrees C
C) 40 degrees C
D) 42 degrees C

Answer: B) 37.5 degrees C

Explanation: Heat stress is indicated when oral temperature exceeds 37.5 degrees C, the first-recovery pulse (P1) exceeds 110, and the fall from P1 to P3 is less than 10 beats.

Q35. In the recovery-pulse method, a slow fall in pulse during recovery (P1 minus P3 less than 10) signifies:
A) Good heat tolerance
B) Hypothermia
C) Significant heat strain
D) Normal acclimatization

Answer: C) Significant heat strain

Explanation: A poor recovery, with the pulse falling by fewer than 10 beats from P1 to P3, indicates significant heat strain; a brisk fall indicates good tolerance.

Q36. Environmental (ambient) heat is measured by the hygiene department, whereas the physiological strain on the worker is assessed by the:
A) Medical department (pulse and temperature)
B) Plant engineer
C) Fire officer
D) Security department

Answer: A) Medical department (pulse and temperature)

Explanation: Heat assessment is shared: the hygiene department measures the ambient thermal environment, and the medical department measures the physiological strain on the worker.

Q37. The recovery-pulse method assesses heat strain by measuring the worker's pulse:
A) Only at the start of the shift
B) During sleep
C) Continuously for 24 hours
D) During recovery after a work period (P1 and P3)

Answer: D) During recovery after a work period (P1 and P3)

Explanation: The medical department counts the recovery pulse after work (P1 at 0.5-1.5 min, P3 at 1.5-2.5 min); a poor fall and a high P1 indicate significant heat strain.

Q38. A worker has P1 of 118, P3 of 112 and oral temperature 37.9 degrees C. This indicates:
A) Normal heat tolerance
B) Significant heat strain
C) Hypothermia
D) Dehydration ruled out

Answer: B) Significant heat strain

Explanation: All three criteria are met (oral temp >37.5, P1 >110, P1 minus P3 = 6, i.e. <10), so this worker shows significant heat strain and needs intervention.

Q39. Measuring the ambient thermal environment is the responsibility of the:
A) Finance department
B) Security department
C) Industrial hygiene department
D) Legal department

Answer: C) Industrial hygiene department

Explanation: The hygiene department measures the environment (temperature, humidity, air movement, radiant heat, summarised as WBGT), while the medical department measures physiological strain.

Susceptibility & Risk Factors

Q40. Which medication group increases heat-illness risk by reducing sweating?
A) Anticholinergics / antihistamines
B) Antibiotics
C) Antacids
D) Vitamin supplements

Answer: A) Anticholinergics / antihistamines

Explanation: Anticholinergic and antihistaminic drugs impair sweating, reducing evaporative cooling; diuretics (volume loss) and some psychotropics also raise heat-illness risk.

Q41. Wearing heavy, impermeable protective clothing raises heat-stroke risk because it:
A) Increases air movement
B) Lowers metabolic heat
C) Improves radiant cooling
D) Blocks evaporation of sweat

Answer: D) Blocks evaporation of sweat

Explanation: Impermeable clothing prevents sweat evaporating, removing the body's main avenue of heat loss in hot work and sharply increasing the risk of heat illness.

Q42. Recognised host risk factors for heat illness include all EXCEPT:
A) Obesity
B) High physical fitness
C) Old age
D) Dehydration

Answer: B) High physical fitness

Explanation: Obesity, the extremes of age, dehydration, alcohol, cardiovascular disease and lack of acclimatization raise risk; good physical fitness is protective, not a risk factor.

Q43. Alcohol and diuretics predispose to heat illness mainly by causing:
A) Increased sweating
B) Vasoconstriction
C) Dehydration / reduced plasma volume
D) Lowered core temperature

Answer: C) Dehydration / reduced plasma volume

Explanation: Alcohol and diuretics reduce body water and plasma volume, impairing both sweating and circulatory compensation, so they predispose to heat exhaustion and stroke.

Heat Indices

Q44. The modern composite index combining temperature, humidity, air movement and radiant heat to set occupational heat limits is the:
A) Wet Bulb Globe Temperature (WBGT)
B) Body mass index
C) Peak expiratory flow
D) Specific gravity

Answer: A) Wet Bulb Globe Temperature (WBGT)

Explanation: The WBGT index integrates dry-bulb temperature, natural wet-bulb temperature (humidity/air movement) and globe temperature (radiant heat), and underlies modern heat TLVs.

Q45. The Corrected Effective Temperature (CET) combines ambient temperature, moisture, radiant heat and:
A) Atmospheric pressure
B) Altitude
C) Noise level
D) Air movement

Answer: D) Air movement

Explanation: CET combines ambient temperature, air movement (which must reach the worker's skin), moisture and radiant heat into a single comfort index.

Q46. The Predicted 4-Hour Sweat Rate (P4SR) regards which range as the comfortable zone?
A) 3 to 4.5 litres
B) 1 to 3 litres
C) More than 4.5 litres
D) Less than 0.5 litre

Answer: B) 1 to 3 litres

Explanation: A P4SR of 1 to 3 litres is the comfortable zone; 3 to 4.5 litres is just tolerable and more than 4.5 litres is intolerable.

Q47. Outdoors with solar load, the WBGT is calculated as:
A) 0.5 WB + 0.5 DB
B) WB + GT + DB
C) 0.7 WB + 0.2 GT + 0.1 DB
D) 0.7 DB + 0.3 GT

Answer: C) 0.7 WB + 0.2 GT + 0.1 DB

Explanation: Outdoors with sunshine, WBGT = 0.7 (natural wet-bulb) + 0.2 (globe) + 0.1 (dry-bulb); indoors or without solar load it is 0.7 WB + 0.3 GT.

Q48. In the WBGT formula, the globe-thermometer temperature chiefly reflects:
A) Radiant heat
B) Humidity
C) Air velocity alone
D) Metabolic rate

Answer: A) Radiant heat

Explanation: The globe thermometer (a black sphere) responds chiefly to radiant heat; the natural wet-bulb reflects humidity and air movement, and the dry-bulb the air temperature.

Q49. The Heat Stress Index (HSI) of Belding and Hatch expresses heat strain as the ratio of:
A) Dry-bulb to wet-bulb temperature
B) Pulse rate to respiratory rate
C) Metabolic rate to body weight
D) Required evaporation to maximum evaporative capacity

Answer: D) Required evaporation to maximum evaporative capacity

Explanation: The HSI is the ratio of the evaporation required for heat balance to the maximum evaporative capacity of the environment, expressed as a percentage; 100% is the tolerable limit.

Q50. Compared with the Effective Temperature (ET), the Corrected Effective Temperature (CET) additionally accounts for:
A) Body weight
B) Radiant heat
C) Noise
D) Atmospheric pressure

Answer: B) Radiant heat

Explanation: CET is the Effective Temperature corrected for radiant heat (using the globe temperature in place of the dry-bulb), making it more accurate near hot radiant sources.

Q51. A Predicted 4-Hour Sweat Rate exceeding 4.5 litres is classified as:
A) Comfortable
B) Just tolerable
C) Intolerable
D) Borderline but still acceptable

Answer: C) Intolerable

Explanation: A P4SR over 4.5 litres is intolerable; 1-3 litres is comfortable and 3-4.5 litres just tolerable, reflecting the unsustainable fluid loss at high sweat rates.

Q52. In the WBGT index, the dominant weighting (0.7) is given to the natural wet-bulb temperature because it reflects:
A) Humidity and evaporative cooling capacity
B) Radiant heat only
C) Air pressure
D) Metabolic rate

Answer: A) Humidity and evaporative cooling capacity

Explanation: The natural wet-bulb temperature captures humidity, air movement and evaporative capacity - the main determinant of heat loss - so it carries the largest weight (0.7) in WBGT.

Permissible Limits

Q53. In the WBGT-based heat TLV table, as the metabolic workload increases from light to heavy, the permissible WBGT:
A) Increases
B) Stays constant
C) Becomes irrelevant
D) Decreases

Answer: D) Decreases

Explanation: Heavier work generates more metabolic heat, so the permissible WBGT falls as workload rises (e.g. continuous work: ~30 C light, ~25 C heavy).

Q54. For continuous heavy work, the permissible heat-exposure threshold is about:
A) 35 degrees C WBGT
B) 25 degrees C WBGT
C) 18 degrees C WBGT
D) 40 degrees C WBGT

Answer: B) 25 degrees C WBGT

Explanation: Continuous heavy work has a permissible WBGT of about 25 C; allowing more rest within each hour raises the permissible value because mean heat load falls.

Q55. Increasing the proportion of rest within each working hour allows the permissible WBGT to:
A) Fall
B) Stay fixed
C) Rise
D) Become negative

Answer: C) Rise

Explanation: More rest per hour lowers the average metabolic heat load, so a higher WBGT can be tolerated; work-rest regimens are a key administrative control for heat.

Q56. For an unacclimatized worker, the permissible WBGT limits are generally set:
A) Lower than for an acclimatized worker
B) Higher than for an acclimatized worker
C) The same as for athletes
D) Irrelevant to acclimatization

Answer: A) Lower than for an acclimatized worker

Explanation: Unacclimatized workers tolerate less heat, so guideline WBGT limits for them are set lower (more protective) than for acclimatized workers at the same workload.

Q57. For continuous moderate work, the permissible heat-exposure threshold is about:
A) 30 degrees C WBGT
B) 25 degrees C WBGT
C) 20 degrees C WBGT
D) 26.7 degrees C WBGT

Answer: D) 26.7 degrees C WBGT

Explanation: Continuous moderate work has a permissible WBGT of about 26.7 C; the limit is 30 C for light and 25 C for heavy continuous work, falling as workload rises.

Acclimatization

Q58. Heat acclimatization is achieved by a graded programme lasting approximately:
A) 1 to 2 days
B) 1 to 2 weeks
C) 6 months
D) A single shift

Answer: B) 1 to 2 weeks

Explanation: Acclimatization is built over about 1-2 weeks of graded exposure, starting at reduced heat and load and increasing by about 10% daily until full duty is reached.

Q59. The key physiological change of heat acclimatization is that the worker's sweat becomes:
A) More concentrated in salt
B) Completely absent
C) More dilute, conserving salt
D) Unchanged in every respect

Answer: C) More dilute, conserving salt

Explanation: With acclimatization the worker sweats earlier and more, but the sweat is more dilute (less sodium), conserving salt; urinary salt loss also falls.

Q60. Salt conservation during heat acclimatization is mediated by the:
A) Renin-angiotensin-aldosterone system
B) Thyroid axis
C) Insulin pathway
D) Clotting cascade

Answer: A) Renin-angiotensin-aldosterone system

Explanation: Aldosterone, via the renin-angiotensin-aldosterone system, drives sodium reabsorption, so the acclimatized worker loses less salt in both sweat and urine.

Q61. Compared with an unacclimatized worker, the acclimatized worker characteristically:
A) Sweats less and later
B) Stops sweating altogether
C) Has a higher core temperature at work
D) Begins sweating sooner and sweats more

Answer: D) Begins sweating sooner and sweats more

Explanation: Acclimatization shifts sweating to begin sooner and at a higher rate, improving evaporative cooling, while the sweat carries less salt and the heart rate at work falls.

Q62. Heat acclimatization is substantially lost after a period away from heat of about:
A) 1 day
B) 1 to 2 weeks
C) 6 months
D) It is never lost

Answer: B) 1 to 2 weeks

Explanation: Acclimatization decays within about one to two weeks of non-exposure (for example after leave or illness), so it must be re-established gradually on return to hot work.

Q63. During heat acclimatization, plasma volume characteristically:
A) Contracts sharply with the fluid loss
B) Falls and then stabilises below normal
C) Expands
D) Remains entirely unchanged

Answer: C) Expands

Explanation: Acclimatization expands plasma volume, supporting both sweating and cardiovascular stability, contributing to the lower heart rate and core temperature at a given workload.

Q64. At a given workload, the acclimatized worker shows a core temperature and heart rate that are:
A) Lower than before acclimatization
B) Higher than before acclimatization
C) Unchanged from the pre-acclimatized state
D) A lower core temperature but a higher heart rate

Answer: A) Lower than before acclimatization

Explanation: Acclimatization lowers both the working heart rate and core temperature for the same task, a key sign of improved heat tolerance alongside earlier, greater sweating.

Management & Prevention

Q65. The immediate treatment of heat stroke is:
A) Slow rewarming
B) Salt tablets only
C) Reassurance and observation
D) Rapid cooling of the body

Answer: D) Rapid cooling of the body

Explanation: Heat stroke demands immediate, rapid cooling of the body; during cooling the rectal temperature should not be allowed to fall below about 102 F to avoid overshoot.

Q66. During the cooling of a heat-stroke victim, the rectal temperature should not be allowed to fall below about:
A) 90 degrees F
B) 102 degrees F
C) 85 degrees F
D) 104 degrees F

Answer: B) 102 degrees F

Explanation: Cooling is stopped before the rectal temperature drops below about 102 F (around 39 C) to prevent overshoot into hypothermia.

Q67. The specific treatment of heat cramps is:
A) Rapid external cooling
B) Intravenous glucose only
C) Salt (electrolyte) replacement
D) Cooling below 102 F

Answer: C) Salt (electrolyte) replacement

Explanation: Heat cramps, caused by salt loss, are treated by replacing salt and fluids (oral or, if severe, intravenous saline), with rest in a cool environment.

Q68. Preventive measures for heat exposure are most effective when applied:
A) At the source (e.g. shielding radiant heat, ventilation)
B) Only as personal protective equipment
C) After symptoms appear
D) Through annual leave alone

Answer: A) At the source (e.g. shielding radiant heat, ventilation)

Explanation: Control at the source - shielding radiant heat, insulation, local exhaust and ventilation - is most effective, supported by cool rest rooms, water and salt, and work-rest scheduling.

Q69. Provision of cool drinking water and access to salt, along with well-ventilated rest rooms, addresses heat illness by:

A) Increasing the worker metabolic heat production at rest
B) Raising the surrounding ambient workplace temperature
C) Reducing the worker natural heat acclimatization over time
D) Replacing fluid and salt lost in sweat and allowing recovery

Answer: D) Replacing fluid and salt lost in sweat and allowing recovery

Explanation: Cool water and salt replace the fluid and electrolytes lost in sweat, while ventilated rest rooms allow periodic recovery, together preventing heat exhaustion and cramps.

Q70. Indirect effects of working in excessive heat include:

A) Improved concentration
B) More accidents, errors and reduced productivity
C) Fewer sick days
D) Enhanced sleep

Answer: B) More accidents, errors and reduced productivity

Explanation: Beyond the specific heat disorders, heat causes fatigue, sleeplessness, more accidents and errors and reduced productivity, an important occupational consequence.

Q71. The most effective field cooling methods for heat stroke include:

A) Wrapping the patient warmly in blankets and clothing
B) Giving oral salt tablets alone without any cooling
C) Evaporative cooling and ice packs to neck, axillae and groin
D) Simply waiting for hospital transfer before cooling

Answer: C) Evaporative cooling and ice packs to neck, axillae and groin

Explanation: Rapid cooling - wetting and fanning (evaporative) or ice packs over major vessels, even cold-water immersion - is the priority in heat stroke, alongside airway, fluids and complication management.

Q72. Appropriate fluid replacement during heavy hot work is approximately:

A) 150-200 ml every 15-20 minutes, with electrolytes
B) 2 litres taken once at lunch
C) Plain water only, unlimited
D) No fluid until the shift ends

Answer: A) 150-200 ml every 15-20 minutes, with electrolytes

Explanation: Small frequent drinks (about 150-200 ml every 15-20 minutes) with electrolytes match ongoing sweat loss better than infrequent large volumes or plain water alone.

Q73. The single most effective category of heat control is:

A) Hydration alone
B) Personal protective clothing
C) Annual medical examination
D) Engineering control at the source

Answer: D) Engineering control at the source

Explanation: Engineering control at source - shielding radiant heat, ventilation, spot cooling, mechanisation - is most effective; hydration, clothing and surveillance support but do not replace it.

Q74. Shielding a furnace with a reflective barrier primarily reduces heat gain by:

A) Conduction
B) Radiation
C) Evaporation
D) Metabolism

Answer: B) Radiation

Explanation: A reflective barrier intercepts radiant heat from hot surfaces, the dominant route of heat gain near furnaces, and is a key engineering control in foundries and forges.

Q75. Scheduling the heaviest outdoor work for early morning is an example of which control?

A) Engineering control
B) Personal protective equipment
C) Administrative control
D) Elimination

Answer: C) Administrative control

Explanation: Re-timing heavy or outdoor work to cooler parts of the day is an administrative control that lowers the heat load without changing the task or the environment itself.

Q76. A buddy system and worker training in hot industries primarily help by:

A) Enabling early recognition of heat-illness symptoms
B) Increasing the work rate
C) Replacing ventilation
D) Eliminating the need for hydration

Answer: A) Enabling early recognition of heat-illness symptoms

Explanation: Training and a buddy system allow early recognition of symptoms such as confusion or collapse, prompting prompt cooling and rescue before heat stroke becomes fatal.

26

UNIT III — PHYSICAL AGENTS

Noise & Noise-Induced Hearing Loss

TOPICS IN THIS CHAPTER

Sound, Units & Measurement

Q1. The intensity (loudness) of sound is measured in decibels using a:
A) Personal noise dosimeter worn by the worker
B) Sound level meter
C) Octave-band frequency analyser
D) Audiometer that tests a person's hearing
Answer: B) Sound level meter
Explanation: Sound intensity (loudness) is measured in decibels with a sound (noise) level meter; frequency (pitch) is measured separately in hertz.

Q2. The two physical quantities used to describe noise are:
A) Intensity (Hz) and frequency (dB)
B) Wavelength and mass
C) Voltage and current
D) Intensity (dB) and frequency (Hz)
Answer: D) Intensity (dB) and frequency (Hz)
Explanation: Noise is described by its intensity or loudness in decibels and its frequency or pitch in hertz; the two are independent properties.

Q3. On a sound level meter, the weighting scale designed to mimic the response of the human ear is the:
A) A scale (dBA)
B) B scale
C) C scale
D) D scale
Answer: A) A scale (dBA)
Explanation: The A-weighting scale (dBA) approximates the frequency response of the human ear and is therefore used for occupational noise limits.

Q4. The normal audible frequency range of the human ear is approximately:
A) 20 to 2,000 Hz
B) 200 to 200,000 Hz
C) 20 to 20,000 Hz
D) 2 to 200 Hz
Answer: C) 20 to 20,000 Hz
Explanation: The healthy human ear perceives sound between about 20 and 20,000 Hz; sensitivity and the intensity needed for audibility vary across this range.

Q5. The threshold of pain, the intensity at which sound is felt as painful, is about:
A) 40 dB
B) 140 dB
C) 80 dB
D) 240 dB
Answer: B) 140 dB
Explanation: Sound above roughly 140 dB is perceived as painful; this is also the impulse-noise ceiling beyond which immediate damage is likely.

Q6. When two machines each producing 85 dB operate together, the combined level is approximately:
A) 170 dB
B) 85 dB
C) 100 dB
D) 88 dB
Answer: D) 88 dB
Explanation: Decibels are logarithmic, so two equal 85 dB sources add to about 88 dB (a 3 dB increase), not 170 dB; doubling sound energy adds 3 dB.

Q7. Two noise sources differing by 0-2 dB are combined by adding how many decibels to the higher level?
A) 3 dB
B) 10 dB
C) 0 dB
D) 6 dB
Answer: A) 3 dB
Explanation: When two sources differ by 0-2 dB, 3 dB is added to the higher; the increment falls to 2, 1 and 0 dB as the difference widens, reflecting log addition.

Q8. Noise that consists of repeated short bursts, such as from a hammer or punch press, is classified as:
A) Continuous noise
B) Steady-state noise
C) Impulse (impact) noise
D) Subjective noise
Answer: C) Impulse (impact) noise
Explanation: Hammering or punch-press noise is impulse (impact) noise; the other main category is continuous noise, with interrupted noise as a third type.

The Decibel & Sound Units

Q9. The decibel is best described as a:
A) Linear unit of absolute pressure
B) Logarithmic unit expressing a ratio of two values
C) Unit of frequency
D) Unit of time

Answer: B) Logarithmic unit expressing a ratio of two values

Explanation: The decibel is a logarithmic unit expressing the ratio of a measured sound pressure or intensity to a reference value, not a linear absolute quantity.

Q10. The decibel (one-tenth of a bel) is named after:
A) James Marsh
B) Robert Koch
C) Bernardino Ramazzini
D) Alexander Graham Bell

Answer: D) Alexander Graham Bell

Explanation: The bel honours Alexander Graham Bell; the decibel, one-tenth of a bel, is the practical unit used for sound and many other ratio measurements.

Q11. The reference sound pressure defined as 0 dB SPL (the approximate threshold of human hearing) is:
A) 20 micropascals
B) 20 pascals
C) 200 micropascals
D) 2 millipascals

Answer: A) 20 micropascals

Explanation: Zero dB sound-pressure level is referenced to 20 micropascals, the approximate quietest sound a healthy young ear can detect at about 1,000 Hz.

Q12. Because the decibel scale is logarithmic, a 10 dB rise corresponds to an intensity increase of:
A) A two-fold increase
B) A hundred-fold increase
C) Ten-fold
D) A five-fold increase

Answer: C) Ten-fold

Explanation: Each 10 dB step is a tenfold change in sound intensity; a 3 dB rise is a doubling of intensity, reflecting the logarithmic nature of the scale.

Q13. A doubling of sound intensity corresponds to an increase of approximately:
A) 10 dB
B) 3 dB
C) 6 dB
D) 1 dB

Answer: B) 3 dB

Explanation: A doubling of sound energy/intensity adds about 3 dB; this is why two equal sources combine to roughly 3 dB above one, and underlies the 3 dB exchange rate.

Audible Range — Humans & Animals

Q14. Sound at a frequency below the human audible range (under about 20 Hz) is termed:
A) Ultrasound
B) Sonic (audible-range) sound
C) Broadband white noise
D) Infrasound

Answer: D) Infrasound

Explanation: Frequencies below about 20 Hz are infrasound (felt rather than heard); those above about 20,000 Hz are ultrasound, beyond human hearing.

Q15. Sound above the upper limit of human hearing (above about 20,000 Hz) is called:
A) Ultrasound
B) Infrasound
C) Sonic (audible-range) sound
D) Impulse (impact) noise

Answer: A) Ultrasound

Explanation: Ultrasound lies above roughly 20,000 Hz; many animals (bats, dolphins, cats, dogs) hear well into this range, which humans cannot.

Q16. Compared with humans, which animal can hear distinctly HIGHER (ultrasonic) frequencies, used for echolocation?
A) Elephant
B) Human infant
C) Bat
D) Tortoise

Answer: C) Bat

Explanation: Bats hear and emit ultrasound up to about 100,000-120,000 Hz for echolocation, far above the human ceiling of roughly 20,000 Hz.

Q17. Elephants and whales communicate over long distances chiefly using:
A) Ultrasound
B) Infrasound (very low frequencies)
C) Visible light
D) X-rays

Answer: B) Infrasound (very low frequencies)

Explanation: Elephants and whales use infrasound, very low frequencies (around or below 20 Hz), which travel long distances, unlike the ultrasound of bats and dolphins.

Q18. A 'silent' dog whistle works because dogs, unlike humans, can hear:
A) Only frequencies below 20 Hz
B) Infrared radiation
C) Sounds under 1 dB
D) Frequencies well above 20,000 Hz

Answer: D) Frequencies well above 20,000 Hz

Explanation: Dogs hear up to about 45,000-60,000 Hz, so a whistle pitched above the human 20,000 Hz ceiling is audible to the dog but silent to people.

Q19. Within the human audible range, the ear is MOST sensitive (needs least intensity) around:
A) 1,000 to 4,000 Hz
B) 20 to 50 Hz
C) 15,000 to 20,000 Hz
D) Below 20 Hz

Answer: A) 1,000 to 4,000 Hz

Explanation: Human hearing is most sensitive between about 1,000 and 4,000 Hz; progressively greater intensity is needed for audibility above 4,000 Hz.

Structure of the Ear

Q20. The outer (external) ear comprises the pinna, the external auditory canal and the:
A) Cochlea
B) Stapes
C) Tympanic membrane
D) Oval window

Answer: C) Tympanic membrane

Explanation: The outer ear is the pinna, the external auditory canal and the tympanic membrane (eardrum), which together collect and funnel sound inward.

Q21. The three auditory ossicles of the middle ear are the:
A) Cochlea, utricle and saccule
B) Malleus, incus and stapes
C) Pinna, tragus and helix
D) Vestibule and two canals

Answer: B) Malleus, incus and stapes

Explanation: The middle-ear ossicles - malleus, incus and stapes - transmit and amplify vibration from the eardrum to the oval window of the inner ear.

Q22. The Eustachian (pharyngotympanic) tube connects the middle ear to the:
A) Cochlea
B) Frontal sinus
C) External canal
D) Nasopharynx

Answer: D) Nasopharynx

Explanation: The Eustachian tube links the middle ear to the nasopharynx, equalising pressure across the tympanic membrane; the cochlea is part of the inner ear.

Q23. The receptor organ of hearing, containing the sensory hair cells, is the:
A) Organ of Corti in the cochlea
B) Semicircular canals
C) Eustachian tube
D) Tympanic membrane

Answer: A) Organ of Corti in the cochlea

Explanation: The organ of Corti, on the basilar membrane within the cochlea, holds the hair cells that transduce sound; the semicircular canals serve balance.

Q24. Within the inner ear, hearing is served by the cochlea, whereas balance is served by the:
A) Ossicles
B) Pinna
C) Semicircular canals and vestibule
D) Eustachian tube

Answer: C) Semicircular canals and vestibule

Explanation: The cochlea handles hearing; the vestibular apparatus - the semicircular canals, utricle and saccule - handles balance and head-position sense.

Q25. The stapes footplate transmits vibration into the inner-ear fluid through the:
A) Round window
B) Oval window
C) Eustachian tube
D) Tympanic membrane

Answer: B) Oval window

Explanation: The stapes footplate sits in the oval window, transmitting vibration into the cochlear perilymph; the round window acts as a pressure-relief membrane.

Q26. Auditory impulses travel from the cochlea to the brain via which cranial nerve?
A) Facial (CN VII)
B) Trigeminal (CN V)
C) Vagus (CN X)
D) Vestibulocochlear (CN VIII)

Answer: D) Vestibulocochlear (CN VIII)

Explanation: The cochlear division of the vestibulocochlear nerve (cranial nerve VIII) carries auditory impulses from the cochlear hair cells to the brainstem.

Q27. In the cochlea, high-frequency sounds are detected at the base and low-frequency sounds at the apex. This frequency mapping is called:
A) Tonotopic organisation
B) Air conduction
C) Impedance matching
D) Bone conduction

Answer: A) Tonotopic organisation

Explanation: The cochlea is tonotopically organised - the base responds to high frequencies and the apex to low - which is why noise damage starts at high frequencies.

Hearing & Conduction

Q28. Sound reaches the inner ear by two routes:
A) Air conduction and nerve conduction
B) Bone conduction and fluid conduction
C) Air conduction and bone conduction
D) Electrical and chemical conduction

Answer: C) Air conduction and bone conduction

Explanation: Sound is transmitted by air conduction (through the external and middle ear) and by bone conduction (directly through the skull to the cochlea).

Q29. The three middle-ear ossicles that transmit vibration from the tympanic membrane are the:
A) Cochlea, utricle and saccule
B) Malleus, incus and stapes
C) Helix, tragus and concha
D) Hammer, cochlea and round window

Answer: B) Malleus, incus and stapes

Explanation: The malleus, incus and stapes conduct tympanic-membrane vibration across the middle ear to the oval window and the cochlear fluid.

Q30. Sensory transduction of sound into nerve impulses occurs in the:
A) Tympanic membrane
B) Ossicles
C) Eustachian tube
D) Cochlear hair cells

Answer: D) Cochlear hair cells

Explanation: Movement of the cilia of the cochlear hair cells generates the action potentials that travel to the brain; their destruction causes sensorineural deafness.

Q31. Noise-induced hearing loss results specifically from damage to the:
A) Cochlear hair cells
B) Tympanic membrane
C) Ossicular chain
D) Auditory cortex

Answer: A) Cochlear hair cells

Explanation: Excessive noise destroys the cochlear hair cells, producing a sensorineural hearing loss; the conducting apparatus is intact.

Deafness & Audiometry

Q32. Noise-induced hearing loss is which type of deafness?
A) Conductive
B) Purely mixed
C) Sensorineural (perceptive)
D) Functional

Answer: C) Sensorineural (perceptive)

Explanation: Damage to cochlear hair cells makes NIHL a sensorineural (perceptive) deafness; a ruptured drum from blast would instead cause conductive loss.

Q33. In pure conductive deafness, audiometry characteristically shows:
A) Equally reduced air and bone conduction
B) Reduced air conduction with normal bone conduction (an air-bone gap)
C) Normal air conduction with reduced bone conduction
D) Both completely normal

Answer: B) Reduced air conduction with normal bone conduction (an air-bone gap)

Explanation: Conductive loss impairs air conduction while bone conduction stays normal, producing an air-bone gap; in sensorineural loss both are reduced together.

Q34. In sensorineural deafness, the relationship between air and bone conduction is:
A) Air reduced, bone normal
B) Bone reduced, air normal
C) Both normal
D) Both reduced, with little or no air-bone gap

Answer: D) Both reduced, with little or no air-bone gap

Explanation: In sensorineural loss the cochlea/nerve is at fault, so air and bone conduction fall together with minimal air-bone gap, unlike conductive loss.

Q35. During pure-tone audiometry the threshold of hearing is defined as the:
A) Minimum sound intensity audible at a given frequency
B) Maximum tolerable intensity
C) Loudest frequency heard
D) Average of all frequencies

Answer: A) Minimum sound intensity audible at a given frequency

Explanation: The threshold of perception is the minimum intensity a person can just hear at a given frequency, taken as the 50% (yes-no-yes) response level.

Q36. Normal thresholds for both air and bone conduction are taken as up to:
A) 2.5 dB
B) 50 dB
C) 25 dB
D) 75 dB

Answer: C) 25 dB

Explanation: Air and bone conduction thresholds up to about 25 dB are regarded as normal; values above this represent measurable hearing loss.

Q37. An audiometry test room should ideally have an internal noise level below:
A) 60 dB
B) 25 dB
C) 85 dB
D) 100 dB

Answer: B) 25 dB

Explanation: The audiometric room must be quiet, with ambient noise below about 25 dB, so that low test tones are not masked by background sound.

Q38. During audiometry, air conduction can be measured up to about 8,000 Hz, whereas bone conduction is measured only up to about:
A) 8,000 Hz
B) 20,000 Hz
C) 1,000 Hz
D) 4,000 Hz

Answer: D) 4,000 Hz

Explanation: Air conduction is testable to about 8,000 Hz, but bone conduction only to about 4,000 Hz, a practical limit of the bone vibrator.

NIHL — Characteristics

Q39. Noise-induced hearing loss characteristically begins at a frequency of:
A) 4,000 Hz
B) 500 Hz
C) 8,000 Hz
D) 250 Hz

Answer: A) 4,000 Hz

Explanation: NIHL classically begins as a notch at 4,000 Hz, before later spreading to involve higher and lower frequencies with continued exposure.

Q40. The explanation offered for the 4,000 Hz notch (Tonndorf's mechanical theory) is that:
A) Industrial machinery, by its nature, only ever emits sound at 4,000 Hz
B) The tympanic membrane mechanically resonates selectively at 4,000 Hz
C) The first turn of the cochlea, tuned to ~4,000 Hz, is struck first and hardest
D) Bone conduction of sound selectively fails at the 4,000 Hz frequency

Answer: C) The first turn of the cochlea, tuned to ~4,000 Hz, is struck first and hardest

Explanation: Tonndorf's theory holds that the basal first turn of the cochlea, responsive to ~4,000 Hz, receives the initial and greatest mechanical stress, so damage starts there.

Q41. A key reason early NIHL goes unnoticed by the worker is that:
A) It is painful and obvious
B) The 500-2,000 Hz speech range is spared at first
C) It begins with total deafness
D) It affects only one ear

Answer: B) The 500-2,000 Hz speech range is spared at first

Explanation: Early NIHL spares the 500-2,000 Hz conversational range, so ordinary speech is still heard and the worker notices nothing until the loss advances.

Q42. Which statement about established noise-induced hearing loss is correct?
A) It is unilateral and painful
B) It is rapidly reversible with rest
C) It improves with hearing aids to normal
D) It is bilateral, gradual, painless and irreversible

Answer: D) It is bilateral, gradual, painless and irreversible

Explanation: NIHL is characteristically bilateral, gradual in onset, painless and irreversible (not amenable to medical treatment), and is preventable.

Q43. Noise-induced temporary threshold shift (NITTS) differs from permanent loss in that NITTS:
A) Is due to hair-cell fatigue and recovers with rest
B) Is permanent from the outset
C) Affects only bone conduction
D) Cannot be measured

Answer: A) Is due to hair-cell fatigue and recovers with rest

Explanation: NITTS reflects reversible hair-cell fatigue and recovers after rest (about 48 hours); loss that persists after rest is a permanent threshold shift.

Q44. To distinguish a temporary from a permanent threshold shift, audiometry should be repeated after a rest period of about:
A) 2 hours
B) 2 weeks
C) 48 hours
D) 1 hour

Answer: C) 48 hours

Explanation: If hearing loss is found, the worker is rested for about 48 hours and re-tested; residual loss is permanent (NIPTS), while recovered loss was temporary (NITTS).

Quantifying Hearing Loss

Q45. Percentage hearing impairment for an ear is calculated by taking the average threshold over key frequencies, deducting 25 dB, then multiplying by:
A) 5
B) 1.5
C) 0.5
D) 15

Answer: B) 1.5

Explanation: Hearing impairment per ear = (average threshold minus the 25 dB low fence) multiplied by 1.5% per decibel; 1 dB of loss equals 1.5% impairment.

Q46. In the percentage-handicap formula, the better and worse ears are weighted respectively by factors of:
A) 1 and 5 (divided by 6)
B) 2 and 2 (divided by 4)
C) 3 and 3 (divided by 6)
D) 5 and 1 (divided by 6)

Answer: D) 5 and 1 (divided by 6)

Explanation: Overall handicap = [(better-ear % x 5) + (worse-ear % x 1)] / 6, reflecting the dominant contribution of the better-hearing ear, per the AAOO method.

Q47. The 25 dB level used as the starting point for calculating impairment is termed the:
A) Low fence
B) High fence
C) Pain threshold
D) Reference equivalent

Answer: A) Low fence

Explanation: The low fence (about 25 dB) is the level below which hearing is counted as normal; the high fence (around 92 dB) corresponds to total handicap.

Q48. In compensation calculations, 1 dB of hearing loss is equated to a percentage impairment of:
A) 15%
B) 0.5%
C) 1.5%
D) 5%

Answer: C) 1.5%

Explanation: Each decibel of threshold elevation above the 25 dB fence is taken as 1.5% impairment, the conversion used in medico-legal hearing-loss assessment.

Q49. When assessing occupational hearing loss for compensation, an age correction is applied because:
A) Younger workers hear worse
B) Presbycusis adds age-related loss that should be separated out
C) Age improves hearing
D) Age has no effect on the cochlea

Answer: B) Presbycusis adds age-related loss that should be separated out

Explanation: Because age-related presbycusis is also sensorineural, an age correction (about 0.5 dB per year over 40) is applied so only the occupational portion is compensated.

Presbycusis vs NIHL

Q50. Presbycusis (age-related hearing loss) is which type of deafness?
A) Conductive
B) Mixed
C) Functional
D) Sensorineural

Answer: D) Sensorineural

Explanation: Presbycusis is a sensorineural loss, like NIHL, which is why an age correction is needed to apportion occupational from age-related loss.

Q51. A feature that helps distinguish presbycusis from noise-induced loss is that in presbycusis:
A) Loss worsens progressively with rising frequency (highest frequencies worst)
B) Loss is a fixed notch at 4,000 Hz only
C) Loss recovers with rest
D) Loss is conductive

Answer: A) Loss worsens progressively with rising frequency (highest frequencies worst)

Explanation: Presbycusis shows progressively greater loss as frequency rises (worst at the highest frequencies), whereas NIHL is classically a 4,000 Hz notch.

Q52. The age-related hearing-loss correction commonly applied is about:
A) 5 dB per year over 40
B) 1.5 dB per year over 20
C) 0.5 dB per year of age over 40
D) No correction is made

Answer: C) 0.5 dB per year of age over 40

Explanation: A correction of roughly 0.5 dB for each year of age above 40 is applied to separate presbycusis from the noise-induced component.

Exposure Limits

Q53. Under the Indian Factories Act model rule, the permissible noise limit for an 8-hour working day is:
A) 80 dBA
B) 90 dBA
C) 115 dBA
D) 70 dBA

Answer: B) 90 dBA

Explanation: The Indian model rule sets 90 dBA for 8 hours; the limit falls as exposure time lengthens and rises for shorter exposures, with a ceiling around 115 dB.

Q54. Under the Indian model rule, as exposure duration halves from 8 hours, the permissible level rises by roughly:
A) About 3 dB per halving (the NIOSH rate)
B) About 10 dB per halving
C) About 2 dB per halving
D) 5 dB per halving

Answer: D) 5 dB per halving

Explanation: The Indian/OSHA-type rule uses about a 5 dB exchange rate, so the permissible level rises about 5 dB for each halving of exposure time.

Q55. A practical ceiling such that no worker should be exposed (even briefly) above it is around:
A) 115 dB
B) 85 dB
C) 90 dB
D) 70 dB

Answer: A) 115 dB

Explanation: Permissible levels rise with shorter exposure but are capped around 115 dB for very brief exposure; impulse noise should never exceed about 140 dB.

Q56. Impulse or impact noise should never exceed a peak of about:
A) 90 dB
B) 115 dB
C) 140 dB
D) 160 dB

Answer: C) 140 dB

Explanation: Peak impulse noise should not exceed about 140 dB; the permitted number of impulses per day falls sharply as their peak level rises.

Guidelines — DGFASLI, OSHA, NIOSH, ACGIH

Q57. In India, the technical authority that advises on occupational safety and health, including noise standards under the Factories Act, is the:
A) NIOSH
B) DGFASLI
C) ACGIH
D) OSHA

Answer: B) DGFASLI
Explanation: DGFASLI (Directorate General Factory Advice Service and Labour Institutes) is India's technical authority on OSH; the Factories Act model rules set a 90 dBA limit.

Q58. The OSHA permissible exposure limit (PEL) for noise over an 8-hour day is:
A) 85 dBA
B) 100 dBA
C) 70 dBA
D) 90 dBA

Answer: D) 90 dBA
Explanation: OSHA sets a 90 dBA 8-hour PEL with a 5 dB exchange rate; an 85 dBA action level triggers a hearing-conservation programme.

Q59. The NIOSH recommended exposure limit (REL) for noise over 8 hours, more protective than OSHA, is:
A) 85 dBA
B) 90 dBA
C) 100 dBA
D) 75 dBA

Answer: A) 85 dBA
Explanation: NIOSH recommends a more protective 85 dBA 8-hour REL with a 3 dB exchange rate, reflecting the equal-energy principle.

Q60. The OSHA 'action level' at which a hearing-conservation programme must be started is:
A) 90 dBA
B) 100 dBA
C) 85 dBA (8-hour TWA)
D) 70 dBA

Answer: C) 85 dBA (8-hour TWA)
Explanation: An 8-hour TWA of 85 dBA is OSHA's action level, mandating audiometric monitoring and hearing protection, below the 90 dBA PEL.

Q61. A key difference between the NIOSH/ACGIH and the OSHA/Indian noise standards is the:
A) Use of dBA weighting
B) Exchange rate (3 dB vs 5 dB)
C) Definition of the decibel
D) Frequency range of hearing

Answer: B) Exchange rate (3 dB vs 5 dB)
Explanation: NIOSH and ACGIH use a 3 dB exchange rate (equal-energy), whereas OSHA and the Indian rule use a 5 dB exchange rate, making the former more protective.

Q62. The ACGIH threshold limit value (TLV) for an 8-hour noise exposure is:
A) 90 dBA
B) 100 dBA
C) 115 dBA
D) 85 dBA

Answer: D) 85 dBA
Explanation: ACGIH sets an 85 dBA 8-hour TLV with a 3 dB exchange rate, aligning with NIOSH and more protective than the OSHA/Indian 90 dBA limit.

Non-auditory Effects

Q63. Recognised non-auditory effects of noise exposure include:
A) Raised blood pressure, tachycardia and catecholamine release
B) Lowered blood pressure and bradycardia
C) Improved concentration
D) Reduced accident rates

Answer: A) Raised blood pressure, tachycardia and catecholamine release
Explanation: Noise stimulates the adrenal glands and sympathetic system, transiently raising blood pressure, heart rate, blood sugar and catecholamines, with stress effects.

Q64. Beyond hearing loss, occupational noise is associated with:
A) Fewer accidents
B) Better sleep
C) More accidents, errors and reduced productivity
D) Increased surfactant production

Answer: C) More accidents, errors and reduced productivity
Explanation: Noise impairs concentration and communication, increasing accidents, errors and absenteeism while lowering productivity, alongside psychological stress.

Control & PPE

Q65. In the hierarchy of noise control, the most effective approach is:
A) Hearing protectors for all
B) Engineering control at source
C) Job rotation
D) Annual audiometry
Answer: B) Engineering control at source
Explanation: Engineering control (reducing noise at the source, enclosure, sound-absorbing materials) is the most effective; PPE is a last resort.

Q66. The maximum noise reduction typically achievable with ear plugs is about:
A) 40 dB
B) 5 dB
C) 60 dB
D) 15 dB
Answer: D) 15 dB
Explanation: Ear plugs provide up to about 15 dB attenuation; ear muffs up to about 20 dB; the two combined give less than their simple sum.

Q67. Ear muffs typically provide a maximum attenuation of about:
A) 20 dB
B) 5 dB
C) 45 dB
D) 2 dB
Answer: A) 20 dB
Explanation: Ear muffs attenuate up to about 20 dB; combining plugs and muffs does not simply add, and a protective helmet may give around 30 dB.

Q68. A fundamental limitation of all ear protectors (plugs and muffs) is that they:
A) Block bone conduction completely
B) Cure existing NIHL
C) Cannot block bone conduction of sound
D) Increase hair-cell regeneration
Answer: C) Cannot block bone conduction of sound
Explanation: Hearing protectors reduce air-conducted sound only and cannot obstruct bone conduction; at very high levels a helmet is needed, and damage may still occur.

Q69. The team-based programme to prevent occupational hearing loss is the:
A) Vaccination programme
B) Hearing conservation programme
C) Spirometry programme
D) Vision screening programme
Answer: B) Hearing conservation programme
Explanation: A hearing conservation programme combines noise evaluation, audiometric monitoring and control measures to prevent NIHL and reduce compensation claims.

Q70. A hearing conservation programme has three core components: noise exposure evaluation, control of exposure, and:
A) Vaccination of workers
B) Spirometry
C) Eye testing
D) Medical (audiometric) evaluation of hearing
Answer: D) Medical (audiometric) evaluation of hearing
Explanation: The three pillars are physical evaluation of noise exposure, medical (audiometric) evaluation of hearing, and control of the noise exposure.

Q71. The only definitive treatment offered for severe established noise-induced hearing loss is:
A) Cochlear implant
B) Ear plugs
C) Systemic steroids
D) Tympanoplasty
Answer: A) Cochlear implant
Explanation: NIHL is irreversible and not amenable to medical cure; a cochlear implant is the option for severe loss, underlining that prevention is paramount.

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UNIT III — PHYSICAL AGENTS

Other Physical Agents (Cold, Electricity, EMF)

TOPICS IN THIS CHAPTER

Cold Injury

Q1. A non-freezing cold injury of the feet from prolonged exposure to cold, wet conditions is:
A) Trench (immersion) foot
B) Frostbite
C) Heat cramps
D) Arc eye

Answer: A) Trench (immersion) foot

Explanation: Trench (immersion) foot is a non-freezing cold injury from prolonged damp cold; frostbite, by contrast, is a freezing injury with ice-crystal formation in the tissues.

Q2. Frostbite differs from trench foot in that frostbite involves:
A) Cold, wet conditions above the freezing point
B) Prolonged damp exposure without ice formation
C) Actual freezing of the tissues
D) Wind chill alone without tissue freezing

Answer: C) Actual freezing of the tissues

Explanation: Frostbite is a freezing injury, with ice-crystal formation and tissue necrosis, whereas trench foot is a non-freezing injury occurring in cold, wet, above-freezing conditions.

Q3. Deep frostbite is distinguished from superficial frostbite by:
A) Involvement confined to skin and subcutis
B) Involvement of deeper tissues beyond the skin
C) Reversible numbness with no tissue loss
D) Surface blistering that always heals fully

Answer: B) Involvement of deeper tissues beyond the skin

Explanation: Superficial frostbite affects skin and subcutaneous tissue, while deep frostbite involves muscle, tendon and bone, carrying a high risk of gangrene and tissue loss.

Q4. Whole-body exposure to severe cold leading to a dangerously low core temperature is:
A) Hyperpyrexia
B) Heat syncope
C) Cutis marmorata
D) Hypothermia

Answer: D) Hypothermia

Explanation: Hypothermia is a fall in core temperature from severe cold exposure, with shivering, confusion and, if untreated, cardiac arrhythmia and death; it is the systemic cold injury.

Electrical Hazards

Q5. The severity of an electric shock depends on the current, its duration, the path through the body and the:
A) Resistance at the point of contact
B) Worker's height
C) Ambient noise
D) Colour of the cable

Answer: A) Resistance at the point of contact

Explanation: Shock severity depends on current magnitude, duration, the path taken through the body (hand-to-hand across the heart being dangerous) and the contact resistance (skin moisture).

Q6. The most dangerous immediate cardiac effect of electric shock is:
A) Iron-deficiency anaemia
B) Cataract
C) Ventricular fibrillation
D) Pterygium

Answer: C) Ventricular fibrillation

Explanation: A current passing through the chest can precipitate ventricular fibrillation and cardiac arrest, the chief cause of death in electrocution, alongside respiratory arrest and burns.

Q7. A current path that is especially dangerous because it crosses the heart is:
A) Finger to adjacent finger
B) Hand-to-hand (or hand-to-opposite-foot)
C) Toe to adjacent toe
D) Earlobe to earlobe

Answer: B) Hand-to-hand (or hand-to-opposite-foot)

Explanation: A hand-to-hand or hand-to-opposite-foot path carries current across the heart, maximising the risk of ventricular fibrillation; localised paths are less likely to be fatal.

Electromagnetic Fields & Ultrasound

Q8. The unit of magnetic flux density used to express electromagnetic-field exposure is the:
A) Becquerel
B) Sievert
C) Decibel
D) Tesla

Answer: D) Tesla

Explanation: Magnetic flux density is measured in tesla; the becquerel and sievert are radiation units and the decibel a sound unit, so they do not apply to EMF exposure.

Q9. A practical concern with strong electromagnetic fields in the workplace is interference with:
A) Implanted cardiac pacemakers
B) Audiometry rooms
C) Globe thermometers
D) Film badges

Answer: A) Implanted cardiac pacemakers

Explanation: Strong electromagnetic fields can interfere with implanted cardiac pacemakers, an important consideration when placing workers with such devices near high-field equipment.

Q10. Prolonged occupational exposure to high-intensity ultrasound is reported to affect chiefly the:
A) The lens, causing cataract only
B) The cochlear hair cells
C) Red blood cells (haemolysis/tissue disruption)
D) The tympanic membrane only

Answer: C) Red blood cells (haemolysis/tissue disruption)

Explanation: High-intensity ultrasound can disrupt tissues and, with prolonged exposure, damage red blood cells; airborne ultrasound is generally of lower concern than contact exposure.

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UNIT III — PHYSICAL AGENTS

Radiation (Ionising & Non-Ionising)

TOPICS IN THIS CHAPTER

Ionising Radiation: Types & Penetration

Q1. Ionising radiations are divided into electromagnetic and particulate (corpuscular). The electromagnetic ones are:
A) X-rays and gamma rays
B) Alpha and beta particles
C) Alpha rays and neutrons
D) Beta and gamma rays

Answer: A) X-rays and gamma rays

Explanation: X-rays and gamma rays are electromagnetic ionising radiations; alpha and beta particles are corpuscular (particulate) radiations.

Q2. The radiation with the LEAST penetrating power (stopped by paper or the outer skin) but the greatest ionising density is:
A) Gamma
B) X-ray
C) Alpha
D) Beta

Answer: C) Alpha

Explanation: Alpha particles penetrate only about 0.05 mm of tissue and are stopped by skin or paper, yet they ionise densely, making them dangerous chiefly when inhaled or ingested.

Q3. Which radiation is the most penetrating, requiring thick lead or concrete for shielding?
A) Alpha
B) Gamma
C) Beta
D) Visible light

Answer: B) Gamma

Explanation: Gamma rays (and X-rays) are highly penetrating, passing through tissue and needing dense shielding such as lead or concrete, unlike alpha and beta particles.

Q4. Beta particles are typically stopped by:
A) A sheet of paper
B) Thick lead only
C) Nothing stops them
D) A few millimetres of aluminium or plastic

Answer: D) A few millimetres of aluminium or plastic

Explanation: Beta particles penetrate a few millimetres of tissue and are stopped by thin aluminium or plastic, intermediate between alpha (paper) and gamma (lead/concrete).

Q5. Because alpha emitters penetrate poorly externally, their main occupational danger arises when they are:
A) Inhaled or ingested (internal exposure)
B) Viewed directly
C) Touched briefly
D) Stored in lead

Answer: A) Inhaled or ingested (internal exposure)

Explanation: Alpha emitters cause little harm outside the body but, once inhaled or ingested, deposit dense ionising energy internally - the basis of radon and uranium-dust lung hazard.

Radiation Units

Q6. The activity of a radioactive source (disintegrations per second) is measured in:
A) Gray (Gy)
B) Sievert (Sv)
C) Becquerel (Bq)
D) Roentgen (R)

Answer: C) Becquerel (Bq)

Explanation: Activity is measured in becquerels (1 Bq = 1 disintegration per second); the older unit was the curie (Ci). Gray and sievert measure dose, not activity.

Q7. The absorbed dose of radiation is measured in:
A) Becquerel (Bq)
B) Gray (Gy)
C) Sievert (Sv)
D) Curie (Ci)

Answer: B) Gray (Gy)

Explanation: Absorbed dose is measured in grays (1 rad = 0.01 Gy); the becquerel measures activity and the sievert measures equivalent (biological) dose.

Q8. The equivalent (biologically weighted) dose, used for radiation-protection limits, is measured in:
A) Gray (Gy)
B) Becquerel (Bq)
C) Roentgen (R)
D) Sievert (Sv)

Answer: D) Sievert (Sv)

Explanation: Equivalent dose is measured in sieverts (1 Sv = 100 rem); it weights the absorbed dose (gray) for the biological damage of the radiation type.

Q9. The relationship 1 sievert = 100 rem, and the fact that for X-rays the rem equals the rad, reflects that:
A) The old units (rad, rem) are replaced by gray and sievert
B) Activity in becquerels is identical to absorbed dose
C) Sieverts are the correct unit for source activity
D) The gray is used only to express radiation exposure

Answer: A) The old units (rad, rem) are replaced by gray and sievert

Explanation: SI units gray and sievert replaced the older rad and rem (1 Gy = 100 rad; 1 Sv = 100 rem); for X-rays the rem equals the rad.

Q10. The older unit of radiation exposure, now expressed in coulomb per kilogram, was the:
A) Becquerel
B) Gray
C) Roentgen
D) Sievert

Answer: C) Roentgen

Explanation: The roentgen was the old unit of exposure, now given as coulomb per kilogram; activity uses the becquerel and dose the gray and sievert.

Biological Effects of Ionising Radiation

Q11. A uniform whole-body dose of about 1-10 Gy classically causes, after 2-4 weeks:
A) Cerebrovascular collapse within hours of exposure
B) Pancytopenia with infection and bleeding
C) Gastrointestinal mucosal failure within days
D) Acute haemolysis with jaundice within hours

Answer: B) Pancytopenia with infection and bleeding

Explanation: Whole-body 1-10 Gy gives the haematopoietic syndrome: marrow failure producing pancytopenia, with infection and bleeding about 2-4 weeks later; higher doses give the gut and CNS syndromes.

Q12. The two leukaemias classically associated with ionising-radiation exposure are:
A) Only chronic lymphocytic leukaemia
B) Hairy cell leukaemia only
C) No leukaemia is radiation-related
D) Acute myeloid and acute lymphoblastic leukaemia

Answer: D) Acute myeloid and acute lymphoblastic leukaemia

Explanation: Ionising radiation is associated with acute myeloid leukaemia and acute lymphoblastic leukaemia, among other malignancies, reflecting its action on dividing marrow cells.

Q13. Stochastic effects of radiation (such as cancer and hereditary effects) are characterised by:
A) No threshold dose; probability rises with dose
B) A clear threshold below which they never occur
C) Severity proportional to dose
D) Occurring only above 10 Gy

Answer: A) No threshold dose; probability rises with dose

Explanation: Stochastic effects (cancer, hereditary effects) have no threshold - the probability, not the severity, rises with dose - unlike deterministic effects, which have a threshold.

Q14. Deterministic (non-stochastic) effects of radiation, such as skin erythema and cataract, are characterised by:
A) No threshold dose
B) Random occurrence at any dose
C) A threshold dose, above which severity rises with dose
D) Affecting only offspring

Answer: C) A threshold dose, above which severity rises with dose

Explanation: Deterministic effects appear only above a threshold dose and increase in severity with dose; examples include erythema, cataract and acute radiation syndrome.

Q15. The factors determining the biological effect of ionising radiation include the type and quality of radiation, the dose, the dose rate and the:
A) Worker's blood group
B) Tissue distribution of the dose
C) Time of day
D) Ambient noise level

Answer: B) Tissue distribution of the dose

Explanation: Effect depends on the radiation type and quality, the dose and dose rate, and the tissue distribution; rapidly dividing tissues such as marrow are most sensitive.

Radiation Protection

Q16. The three fundamental principles of external radiation protection are:
A) Heat, light and sound
B) Mass, volume and density
C) Filtration, dilution and substitution
D) Time, distance and shielding

Answer: D) Time, distance and shielding

Explanation: External dose is minimised by reducing time near the source, maximising distance (inverse-square law) and interposing shielding - the time, distance and shielding triad.

Q17. The principle that exposures should be kept 'as low as reasonably achievable' is abbreviated:
A) ALARA
B) WBGT
C) COSHH
D) ILO

Answer: A) ALARA

Explanation: ALARA (as low as reasonably achievable) is the guiding principle of radiation protection, requiring exposures to be minimised even below legal dose limits.

Q18. Personal radiation dose received by a worker is commonly monitored with a:
A) Sound level meter
B) Globe thermometer
C) Film badge or thermoluminescent dosimeter (TLD)
D) Sling psychrometer

Answer: C) Film badge or thermoluminescent dosimeter (TLD)

Explanation: Occupational radiation dose is monitored with a film badge or thermoluminescent dosimeter (TLD) worn by the worker; thermal and acoustic instruments are unrelated.

Q19. The current ICRP recommended occupational effective-dose limit is about:
A) 500 mSv per year
B) 20 mSv per year (averaged over 5 years)
C) 2 Sv per year
D) No limit applies

Answer: B) 20 mSv per year (averaged over 5 years)

Explanation: The ICRP occupational limit is 20 mSv per year averaged over five years (max 50 mSv in one year); the older limit was about 50 mSv per year.

Non-Ionising Radiation: Spectrum

Q20. Non-ionising radiations differ from ionising radiations in that they:
A) They penetrate the whole body deeply and uniformly
B) They cause acute bone-marrow failure within weeks
C) They are all corpuscular (particulate) radiations
D) Cannot penetrate deeply and act mainly on skin and eyes

Answer: D) Cannot penetrate deeply and act mainly on skin and eyes

Explanation: Non-ionising radiations (UV, visible, IR, microwave) lack the energy to ionise and penetrate poorly, so their effects fall mainly on the skin and the eyes.

Q21. Arranged by increasing wavelength, the non-ionising spectrum runs:
A) Ultraviolet, visible, infrared, microwave
B) Microwave, infrared, visible, ultraviolet
C) Infrared, ultraviolet, visible, microwave
D) Visible, ultraviolet, microwave, infrared

Answer: A) Ultraviolet, visible, infrared, microwave

Explanation: Wavelength increases from ultraviolet (100-400 nm) through visible (400-760 nm) and infrared (760 nm-1 mm) to microwave (1 mm-1 m).

Q22. Which ultraviolet band is almost entirely absorbed by the ozone layer and so reaches the ground least?
A) UV-A
B) UV-B
C) UV-C
D) Visible light

Answer: C) UV-C

Explanation: UV-C (100-280 nm) is largely absorbed in the upper atmosphere by ozone; UV-B and UV-A reach the surface and account for most occupational and solar UV injury.

Q23. Of the ocular structures, light of wavelength greater than 400 nm characteristically reaches the:
A) The cornea, which absorbs below 300 nm
B) Retina
C) The lens, reached by 300-400 nm
D) The conjunctival surface only

Answer: B) Retina

Explanation: Wavelengths below 300 nm are absorbed at the cornea, 300-400 nm reach the lens, and those above 400 nm can reach the retina - the basis of differing eye injuries.

UV, Arc Eye & Skin

Q24. Acute over-exposure of welders to ultraviolet light causes a painful, delayed-onset keratoconjunctivitis known as:
A) Cataract
B) Pterygium
C) Glaucoma
D) Arc eye (photokeratitis)

Answer: D) Arc eye (photokeratitis)

Explanation: Arc eye (welder's flash, photokeratitis) is an acute UV-B injury of the corneal epithelium, with intense pain and lacrimation some hours after exposure, usually self-limiting.

Q25. Which of the following is NOT a recognised ocular effect of chronic ultraviolet exposure?
A) Noise-induced hearing loss
B) Pterygium
C) Cataract
D) Chronic conjunctival changes

Answer: A) Noise-induced hearing loss

Explanation: Chronic UV causes pterygium, cataract and conjunctival degeneration; noise-induced hearing loss is unrelated, being an effect of noise on the cochlea.

Q26. Beyond the eye, ultraviolet (especially UV-A) acting on the skin causes:
A) Acute bone-marrow failure with pancytopenia
B) Decompression sickness with nitrogen bubbles
C) Tanning, and with chronic exposure photoageing and skin cancer
D) Hand-arm vibration syndrome of the fingers

Answer: C) Tanning, and with chronic exposure photoageing and skin cancer

Explanation: UV causes skin tanning and sunburn acutely, and chronically photoageing and skin cancers; the marrow, vascular and joint effects belong to other physical agents.

Infrared, Microwave & the Eye

Q27. The classic ocular injury of glass-blowers and furnacemen from prolonged infrared exposure is:
A) Arc eye
B) Cataract
C) Pterygium
D) Retinal detachment

Answer: B) Cataract

Explanation: Infrared radiation from molten glass and metal causes lens protein denaturation and the characteristic 'glass-blower's' or heat cataract; arc eye is a UV injury.

Q28. Infrared-A radiation is particularly hazardous to the eye because it can reach and damage the:
A) The corneal epithelium only
B) The lens, causing only cataract
C) The optic nerve head only
D) Retina and choroid

Answer: D) Retina and choroid

Explanation: Infrared-A penetrates to the retina and choroid, causing retinal oedema and burns; longer infrared and microwave cause mainly cataract through lens heating.

Q29. A recognised ocular effect of chronic microwave exposure (e.g. radar work) is:
A) Cataract
B) Arc eye
C) Photokeratitis
D) Optic neuritis

Answer: A) Cataract

Explanation: Microwave radiation heats tissues and is associated with cataract formation; radar, ovens and communication equipment are the typical occupational sources.

Lasers

Q30. The three defining physical properties of laser light are that it is:
A) Diffuse, multicoloured and random
B) Ionising, particulate and penetrating
C) Collimated, monochromatic and coherent
D) Infrared, audible and pulsed

Answer: C) Collimated, monochromatic and coherent

Explanation: Laser light is collimated (parallel), monochromatic (single wavelength) and coherent (in phase), which concentrates energy and underlies both its uses and its hazards.

Q31. The principal occupational hazards of lasers are:
A) Decompression sickness
B) Retinal burns, skin burns and fire
C) Hand-arm vibration
D) Metal fume fever

Answer: B) Retinal burns, skin burns and fire

Explanation: Lasers can cause retinal and skin burns and are a fire hazard; the focusing of the eye onto the retina makes ocular injury the chief concern.

Q32. Under the laser safety classification, the highest-risk class, hazardous to skin and a fire risk, is:
A) Class I
B) Class II
C) Class III
D) Class IV

Answer: D) Class IV

Explanation: Class IV lasers are the highest risk, hazardous to skin and eyes and a fire hazard; Class I is non-hazardous and Class II is safe within the blink reflex.

Q33. A Class II laser is regarded as not hazardous provided that:
A) The natural blink reflex limits exposure
B) It is viewed directly for minutes
C) Skin contact is prolonged
D) It is used near fuel

Answer: A) The natural blink reflex limits exposure

Explanation: Class II lasers are low-power and considered safe because the natural aversion/blink reflex limits retinal exposure; deliberate prolonged staring removes this protection.

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UNIT III — PHYSICAL AGENTS

Vibration

TOPICS IN THIS CHAPTER

Types of Vibration Exposure

Q1. Vibration transmitted through hand-held power tools, affecting frequencies of about 50-300 Hz, causes:
A) Whole-body vibration syndrome
B) Heat stroke
C) Hand-arm vibration syndrome (HAVS)
D) Decompression illness

Answer: C) Hand-arm vibration syndrome (HAVS)
Explanation: Hand-arm vibration syndrome arises from hand-held tools (drills, grinders, chainsaws) in the 50-300 Hz range; whole-body vibration involves much lower frequencies from vehicles and platforms.

Q2. Whole-body vibration, transmitted through the seat or feet at about 0.1-20 Hz, arises mainly from:
A) Driving vehicles and standing on vibrating platforms
B) Using a hand-held grinder
C) Welding
D) Diving

Answer: A) Driving vehicles and standing on vibrating platforms
Explanation: Whole-body vibration (0.1-20 Hz) comes from vehicles and vibrating platforms and affects the spine, abdominal organs, eyes and balance, unlike the hand-tool source of HAVS.

Q3. Low-frequency whole-body vibration around 0.1-0.63 Hz is particularly associated with:
A) Vibration white finger
B) Cataract
C) Phossy jaw
D) Motion sickness

Answer: D) Motion sickness
Explanation: Very low-frequency whole-body vibration (0.1-0.63 Hz) provokes motion sickness via the vestibular apparatus; vibration white finger is instead a hand-arm (50-300 Hz) effect.

Q4. Chronic whole-body vibration in drivers is most associated with:
A) Vibration white finger
B) Low back pain and spinal degeneration
C) Arc eye
D) Dysbaric osteonecrosis

Answer: B) Low back pain and spinal degeneration
Explanation: Prolonged whole-body vibration, as in drivers of trucks and heavy machinery, is linked to low back pain and degenerative spinal change, distinct from the finger effects of HAVS.

Vibration White Finger

Q5. The vascular component of hand-arm vibration syndrome is essentially a secondary form of:
A) Buerger disease
B) Heat syncope
C) Raynaud's phenomenon
D) Carpal tunnel syndrome

Answer: C) Raynaud's phenomenon
Explanation: The vascular component of HAVS is vibration white finger, a secondary Raynaud's phenomenon with episodic blanching of the fingers on cold exposure.

Q6. The three factors classically required to develop vibration white finger (Raynaud's phenomenon) are vibration, cold weather and:
A) Smoking
B) High humidity
C) Loud noise
D) Bright light

Answer: A) Smoking
Explanation: Vibration white finger is precipitated by the combination of vibration exposure, cold weather and smoking; smoking aggravates the vasospasm and vascular damage.

Q7. The proposed mechanism of vibration white finger includes sympathetic over-activity and:
A) Dilatation and permanent patency of digital arteries
B) Acute marrow failure
C) Lens protein denaturation
D) Thickening of the medial coat of the digital arteries

Answer: D) Thickening of the medial coat of the digital arteries
Explanation: Vibration white finger involves sympathetic stimulation and hypertrophy/thickening of the digital arteries' medial coat, narrowing the lumen and causing episodic ischaemia.

Q8. An attack of vibration white finger typically shows the fingers first turning:
A) Black immediately
B) White (blanched) on cold exposure
C) Yellow permanently
D) Green on warming

Answer: B) White (blanched) on cold exposure

Explanation: An attack begins with cold-induced blanching (whitening) of the fingers from vasospasm, sometimes followed by cyanosis and then redness on rewarming, with later trophic changes.

Q9. Advanced (very severe) vibration white finger is marked by:
A) Complete spontaneous recovery
B) Improved dexterity
C) Trophic skin changes at the finger tips
D) Bony overgrowth of the wrist

Answer: C) Trophic skin changes at the finger tips

Explanation: In the most severe grade, frequent attacks affect all the fingers and trophic skin changes (ulceration, atrophy) appear at the finger tips, threatening tissue loss.

Grading & Sensorineural Effects

Q10. The grading of hand-arm vibration syndrome into separate vascular and sensorineural stages follows the:
A) Stockholm Workshop scale
B) ILO classification
C) Glasgow Coma Scale
D) APGAR score

Answer: A) Stockholm Workshop scale

Explanation: HAVS is graded by the Stockholm Workshop scale into vascular stages (blanching) and sensorineural stages (numbness/tingling), each assessed separately for each hand.

Q11. The sensorineural component of hand-arm vibration syndrome presents with:
A) Episodic cold-induced blanching of the fingers
B) Carpometacarpal and wrist joint pain only
C) Reduced grip strength as the sole feature
D) Numbness, tingling and loss of manipulative dexterity

Answer: D) Numbness, tingling and loss of manipulative dexterity

Explanation: The sensorineural component features intermittent or persistent numbness and tingling with reduced sensory perception and loss of fine dexterity, separate from the vascular blanching.

Q12. Besides vascular and sensorineural effects, hand-arm vibration also causes musculoskeletal problems such as:
A) Pulmonary fibrosis
B) Carpal and wrist pain and arthralgia
C) Renal tubular damage
D) Retinal burns

Answer: B) Carpal and wrist pain and arthralgia

Explanation: HAVS has a musculoskeletal component - carpometacarpal and wrist pain, arthralgia and reduced grip - in addition to its vascular and sensorineural effects.

Prevention of Vibration Disorders

Q13. The personal protective measure for hand-arm vibration is the use of:
A) Ear muffs worn over both ears during work
B) A film-badge radiation dosimeter on the chest
C) Anti-vibration (attenuated) gloves that also keep the hands warm
D) A blackened globe thermometer near the hands

Answer: C) Anti-vibration (attenuated) gloves that also keep the hands warm

Explanation: Anti-vibration (attenuated) gloves reduce transmitted vibration and keep the hands warm and dry, countering the cold factor; ear muffs and dosimeters address other hazards.

Q14. Engineering control of vibration at the source includes:
A) Rubber padding or matting on vibrating platforms and tool servicing
B) Issuing salt tablets to the exposed workers
C) Installing recompression chambers on site
D) Providing much brighter overhead lighting

Answer: A) Rubber padding or matting on vibrating platforms and tool servicing

Explanation: Vibration is controlled at source by damping (rubber matting/padding), regular tool maintenance and selecting low-vibration tools, supported by limiting exposure time.

Q15. Because cold and smoking aggravate vibration white finger, prevention programmes also emphasise:

A) Encouraging a tighter, firmer grip on tools
B) Rotating workers to increase total tool time
C) Providing caffeine to improve finger circulation
D) Keeping hands warm and smoking cessation

Answer: D) Keeping hands warm and smoking cessation

Explanation: Since cold and smoking precipitate and worsen vibration white finger, keeping the hands warm and dry and stopping smoking are key preventive measures, with periodic surveillance.

Q16. Workers exposed to vibrating tools should undergo:

A) Annual audiometry only
B) Periodic medical surveillance for early HAVS
C) No monitoring at all
D) Routine chest radiography only

Answer: B) Periodic medical surveillance for early HAVS

Explanation: Periodic medical surveillance detects early HAVS (blanching, numbness) so exposure can be reduced before irreversible vascular and neural damage occurs.

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UNIT III — PHYSICAL AGENTS

Measuring the Thermal Environment (WBGT)

TOPICS IN THIS CHAPTER

Principles of Heat-Stress Assessment

Q1. Assessment of heat in the workplace is divided into measuring the environmental heat load (heat stress) and the body's response (heat strain). The environmental load is measured by the:

A) Industrial hygiene department using thermal indices
B) Worker's own estimate
C) Finance department
D) Fire officer

Answer: A) Industrial hygiene department using thermal indices

Explanation: Heat assessment separates the environmental load (heat stress), measured by the hygiene department with thermal instruments and indices, from the physiological response (heat strain).

Q2. A valid index of environmental heat stress must combine air temperature with:

A) Noise and vibration
B) Lighting and dust
C) Humidity, air movement and radiant heat
D) Atmospheric pressure only

Answer: C) Humidity, air movement and radiant heat

Explanation: The four environmental factors determining heat stress are air (dry-bulb) temperature, humidity, air movement and radiant heat; a sound index integrates all four.

The Measuring Instruments

Q3. Ordinary air temperature is measured with the:

A) Globe thermometer
B) Dry-bulb thermometer
C) Natural wet-bulb thermometer
D) Kata thermometer

Answer: B) Dry-bulb thermometer

Explanation: The dry-bulb thermometer, with a bare bulb shielded from radiation, measures the air (ambient) temperature, one of the four environmental heat factors.

Q4. The natural wet-bulb thermometer has its bulb covered by a wetted wick and, unlike the psychrometric wet-bulb, is:

A) Aspirated by a fan at fixed speed
B) Sealed in a vacuum
C) Heated electrically
D) Exposed to natural (not forced) air movement

Answer: D) Exposed to natural (not forced) air movement

Explanation: The natural wet-bulb senses the prevailing natural air movement, reflecting the combined effect of humidity, air velocity and radiant heat on evaporation.

Q5. Radiant heat is measured with the globe thermometer, which classically consists of a thermometer at the centre of a:

A) 150 mm matt-black copper sphere
B) Wetted cotton wick
C) Sealed glass vacuum flask
D) Rotating vane

Answer: A) 150 mm matt-black copper sphere

Explanation: The Vernon globe thermometer is a 150 mm (6-inch) hollow copper sphere painted matt black, with a thermometer at its centre; its reading (globe temperature) reflects radiant heat.

Q6. The globe thermometer must be allowed to stabilise before reading for about:

A) 10 seconds
B) 2 hours
C) 15-20 minutes
D) No waiting is needed

Answer: C) 15-20 minutes

Explanation: Because of its thermal mass, the globe thermometer takes about 15-20 minutes to reach equilibrium with the radiant field before a stable reading can be taken.

Q7. Low air velocities (air movement) and the cooling power of air are classically measured with the:

A) Globe thermometer
B) Kata thermometer
C) Dry-bulb thermometer
D) Sling psychrometer

Answer: B) Kata thermometer

Explanation: The Kata thermometer, an alcohol thermometer heated then timed as it cools between set marks, measures the cooling power of air and is used to estimate low air velocities.

Q8. Relative humidity is conveniently measured in the field with a:
A) Globe thermometer
B) Anemometer
C) Kata thermometer
D) Sling psychrometer (whirling hygrometer)
Answer: D) Sling psychrometer (whirling hygrometer)
Explanation: A sling psychrometer (whirling hygrometer) carries paired dry- and aspirated wet-bulb thermometers; the wet-bulb depression gives the relative humidity from psychrometric charts.

Q9. Higher air velocities are most directly measured with an:
A) Anemometer
B) Kata thermometer
C) Globe thermometer
D) Sling psychrometer
Answer: A) Anemometer
Explanation: An anemometer (vane or hot-wire) measures air velocity directly; the Kata thermometer is used for the low air speeds typical indoors where an anemometer is insensitive.

The WBGT Index

Q10. The Wet Bulb Globe Temperature (WBGT) index was originally developed to control heat casualties among:
A) Coal miners
B) Deep-sea divers
C) US military trainees (Yaglou and Minard)
D) Aircraft pilots
Answer: C) US military trainees (Yaglou and Minard)
Explanation: WBGT was devised by Yaglou and Minard in the 1950s to reduce heat casualties among US military recruits, and is now the internationally preferred occupational heat index.

Q11. Outdoors with solar load, WBGT is calculated as:
A) 0.7 WB + 0.3 GT
B) 0.7 WB + 0.2 GT + 0.1 DB
C) 0.5 WB + 0.5 DB
D) WB + GT + DB
Answer: B) 0.7 WB + 0.2 GT + 0.1 DB
Explanation: Outdoors in sunshine, WBGT = 0.7 natural wet-bulb + 0.2 globe + 0.1 dry-bulb; the dry-bulb term is added only when there is a solar (radiant) load from the sun.

Q12. Indoors, or outdoors without solar load, WBGT is calculated as:
A) 0.7 WB + 0.2 GT + 0.1 DB
B) 0.6 WB + 0.4 DB
C) 0.5 GT + 0.5 WB
D) 0.7 WB + 0.3 GT
Answer: D) 0.7 WB + 0.3 GT
Explanation: Without solar load, WBGT = 0.7 natural wet-bulb + 0.3 globe temperature; the dry-bulb term is dropped because there is no direct solar radiant component.

Q13. The natural wet-bulb temperature carries the largest weighting (0.7) in WBGT because evaporation is:
A) The main avenue of heat loss in hot work
B) Unimportant in hot work
C) Only relevant in cold work
D) Identical to radiant heat
Answer: A) The main avenue of heat loss in hot work
Explanation: The wet-bulb reflects humidity and evaporative capacity - the dominant route of heat loss when air is hot - so it is weighted most heavily (0.7) in the WBGT index.

Q14. In the WBGT index, the globe-thermometer temperature represents the contribution of:
A) Humidity
B) Air pressure
C) Radiant heat
D) Metabolic rate
Answer: C) Radiant heat
Explanation: The globe temperature captures radiant heat; the natural wet-bulb captures humidity and air movement, and the dry-bulb the air temperature.

Q15. A workplace has natural wet-bulb 30 C, globe 40 C and dry-bulb 35 C indoors (no solar load). The WBGT is:
A) 35 C
B) 33 C
C) 37 C
D) 30 C
Answer: B) 33 C
Explanation: Indoors WBGT = 0.7(30) + 0.3(40) = 21 + 12 = 33 C; the dry-bulb is not used when there is no solar load.

Measurement Technique

Q16. Heat-stress measurements should be taken:
A) Only outside the building
B) Only at the factory gate
C) At the ceiling
D) At the worker's location and working height

Answer: D) At the worker's location and working height

Explanation: Measurements must represent the worker's actual exposure, so instruments are placed at the worker's location and at working height, not at remote or unrepresentative points.

Q17. Where the thermal environment is non-uniform over the body, WBGT is taken as a height-weighted average using the formula:
A) (WBGT head + 2 x WBGT abdomen + WBGT ankle) / 4
B) Add the head, abdomen and ankle readings then divide by three
C) Use the head-level reading only and ignore the rest
D) Take the ankle-level reading and multiply it by four

Answer: A) (WBGT head + 2 x WBGT abdomen + WBGT ankle) / 4

Explanation: A weighted WBGT = (head + 2 x abdomen + ankle) / 4 is used, giving the trunk double weight as the largest heat-exchange area.

Q18. When heat exposure varies during the shift, the relevant value for comparison with limits is the:
A) Single peak reading
B) Lowest reading of the day
C) Time-weighted average WBGT
D) First reading only

Answer: C) Time-weighted average WBGT

Explanation: Where exposure varies, a time-weighted average WBGT over the work period is computed and compared with the permissible limit for the worker's metabolic workload.

Q19. To apply the WBGT limit correctly, the measured WBGT must be interpreted together with the worker's:
A) Blood group
B) Metabolic workload (light, moderate or heavy)
C) Shoe size
D) Educational level

Answer: B) Metabolic workload (light, moderate or heavy)

Explanation: WBGT limits depend on metabolic heat production, so the measured WBGT is compared against the threshold for the worker's workload category and work-rest regimen.

Other Thermal Indices

Q20. The Effective Temperature (ET) index combines dry-bulb temperature, humidity and:
A) Radiant heat
B) Metabolic rate
C) Noise
D) Air movement

Answer: D) Air movement

Explanation: Effective Temperature combines dry-bulb temperature, humidity (wet-bulb) and air movement into a single comfort figure; correcting it for radiant heat gives the CET.

Q21. The Corrected Effective Temperature (CET) improves on ET by incorporating:
A) Radiant heat (via the globe temperature)
B) Body weight
C) Atmospheric pressure
D) Worker age

Answer: A) Radiant heat (via the globe temperature)

Explanation: CET is the Effective Temperature corrected for radiant heat by using the globe temperature in place of the dry-bulb, important near hot radiant sources such as furnaces.

Q22. The Predicted 4-Hour Sweat Rate (P4SR) expresses heat stress in terms of:
A) Heart beats per minute
B) Calories burned
C) Litres of sweat predicted over four hours
D) Litres of urine

Answer: C) Litres of sweat predicted over four hours

Explanation: P4SR predicts the sweat produced over four hours of exposure; 1-3 litres is comfortable, 3-4.5 just tolerable and over 4.5 intolerable.

Q23. The Heat Stress Index (HSI) of Belding and Hatch is the percentage ratio of:
A) Pulse to respiration
B) Required evaporation to maximum evaporative capacity
C) Dry-bulb to wet-bulb
D) Work to rest

Answer: B) Required evaporation to maximum evaporative capacity

Explanation: HSI = (evaporation required for balance / maximum evaporative capacity of the environment) x 100; an HSI of 100 marks the limit of tolerance.

Q24. Compared with the older comfort indices, the chief practical advantage of WBGT is that it is:
A) Independent of humidity
B) Based on blood tests
C) Only valid outdoors
D) Simple, quick and uses easily measured temperatures

Answer: D) Simple, quick and uses easily measured temperatures

Explanation: WBGT is favoured because it is simple and quick, needs only three easily measured temperatures, and integrates all the relevant environmental factors for routine screening.

Indian Practice & Legislation

Q25. In India, the technical authority providing guidance on workplace heat and thermal standards is the:
A) DGFASLI
B) RBI
C) SEBI
D) ICMR only

Answer: A) DGFASLI

Explanation: DGFASLI (Directorate General Factory Advice Service and Labour Institutes) is the technical authority advising on occupational safety and health, including thermal standards.

Q26. The principal statute requiring reasonable temperature and ventilation in Indian factories is the:
A) Companies Act
B) Indian Penal Code
C) Factories Act, 1948
D) Consumer Protection Act

Answer: C) Factories Act, 1948

Explanation: The Factories Act, 1948 (with its section on ventilation and temperature) requires that workrooms be kept at a reasonable temperature, with detailed limits set in the state factory rules.

Q27. Under the Factories Act, the provision dealing specifically with ventilation and temperature in workrooms is:
A) Section 45
B) Section 13
C) Section 87
D) Section 2

Answer: B) Section 13

Explanation: Section 13 of the Factories Act, 1948 deals with ventilation and temperature, requiring adequate ventilation and a reasonable workroom temperature to protect worker health.

Q28. Indian permissible heat-exposure threshold values are largely adopted from the WBGT-based limits of the:
A) WHO blood-pressure guidelines
B) Bureau of Indian Standards for noise
C) Indian Roads Congress
D) ACGIH

Answer: D) ACGIH

Explanation: India's permissible heat-exposure threshold values are WBGT-based and largely follow the ACGIH TLV framework (by workload and work-rest regimen), applied through the factory rules.

Q29. Construction and outdoor workers' exposure to heat and sun in India is additionally governed by the:

A) Building and Other Construction Workers (BOCW) Act
B) Mines Act only
C) Aircraft Act
D) Telegraph Act

Answer: A) Building and Other Construction Workers (BOCW) Act

Explanation: The Building and Other Construction Workers (BOCW) Act, 1996 provides for the welfare and safety of construction workers, including those exposed to sun and heat outdoors.

Q30. Statutory medical examination of workers in hot processes and certification of fitness is the duty of the:

A) Plant accountant
B) District magistrate
C) Certifying Surgeon / Factory Medical Officer
D) Electrical inspector

Answer: C) Certifying Surgeon / Factory Medical Officer

Explanation: The Certifying Surgeon (and the Factory Medical Officer) conducts statutory medical examinations and fitness certification for workers in hazardous and hot processes under the Factories Act.

Q31. A practical control that the WBGT work-rest tables translate into for a hot Indian workshop is:

A) A ban on all work
B) Defined work-rest cycles by workload
C) Mandatory night shifts only
D) Removal of all ventilation

Answer: B) Defined work-rest cycles by workload

Explanation: The WBGT TLV tables prescribe how much rest per hour is needed at a given heat level and workload, which the employer implements as defined work-rest cycles.