STATION 12 – PAEDIATRICS INTERACTIVE

Patent Ductus Arteriosus (PDA) – Drugs, Medical & Surgical Management

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CANDIDATE INSTRUCTIONS

8 minutes. Scenario: Premature infant with continuous machinery murmur.
State drugs used, medical management, and surgical options.
Mention indications for treatment and complications if untreated.

HIGH-YIELD DIAGNOSIS

PDA: Failure of ductus arteriosus to close after birth (normally closes within 24–72 hours)
Murmur: Continuous "machinery" murmur (Gibson murmur)
• Left infraclavicular, loudest at 2nd left intercostal space
• Systolic-diastolic, radiates to left clavicle and back
Signs: Wide pulse pressure, bounding pulses, active precordium, mid-diastolic rumble (increased flow across mitral valve)
Hemodynamics: L→R shunt (aorta to pulmonary artery) → pulmonary overcirculation → left heart volume overload
Risk factors: Prematurity, female gender, congenital rubella, high altitude, genetic syndromes (Trisomy 21, 18, 13)
Complications if untreated: Heart failure, pulmonary hypertension, Eisenmenger syndrome (reversal to R→L shunt), recurrent chest infections, endarteritis

DRUGS & MEDICAL MANAGEMENT

Premature infants (medical closure):
• **Ibuprofen** (first line) – 10 mg/kg IV/PO → 5 mg/kg → 5 mg/kg at 24-hour intervals
• **Indomethacin** (alternative) – 0.2 mg/kg IV → 0.1 mg/kg → 0.1 mg/kg at 12–24 hour intervals
• **Paracetamol** (emerging option if NSAIDs contraindicated) – 15 mg/kg q6h for 3 days
• **Contraindications:** Necrotizing enterocolitis (NEC), active bleeding, renal failure (creatinine >1.6 mg/dL), thrombocytopenia (<50,000), sepsis

Supportive care:
• Fluid restriction (120–130 mL/kg/day)
• Diuretics (furosemide) if heart failure
• Oxygen (avoid hyperoxia – keeps PDA open, use lowest FiO2 to maintain SpO2 88–92%)
• Optimize ventilation (avoid overdistension)

Term infants/Children:
• NSAIDs rarely effective after 2 weeks of age
• Observation if small, asymptomatic (spontaneous closure possible up to 1 year)
• Endocarditis prophylaxis (only if residual shunt post-repair or unrepaired with prior endocarditis)

SURGICAL MANAGEMENT

Indications for intervention:
• Symptomatic PDA (heart failure, failure to thrive, recurrent chest infections)
• Large left-to-right shunt (Qp:Qs >1.5:1 or pulmonary-to-systemic flow ratio)
• Left heart enlargement (LA/LV dilation on echo)
• Pulmonary hypertension (reversible)
• Prior endocarditis
• Failed medical closure in premature infants

Options:
• **Transcatheter closure** (preferred if >6 months and >5 kg)
– Coil occlusion (small PDA <2 mm)
– Amplatzer duct occluder (larger PDA 2–12 mm)
– Complications: Device embolization, hemolysis, left pulmonary artery stenosis
• **Surgical ligation/division** (if catheter closure contraindicated)
– Left thoracotomy or video-assisted thoracoscopic surgery (VATS)
– Risk: Recurrent laryngeal nerve injury, chylothorax, pneumothorax
• **Thoracoscopic clip application** (minimally invasive)

CRITICAL ERRORS

Misses wide pulse pressure (hallmark sign)
Gives NSAIDs with contraindications (NEC, renal failure)
Confuses with VSD (VSD has pansystolic, not continuous murmur)
No mention of Eisenmenger risk (if large PDA left untreated)
Forgets fluid restriction (key in medical management)

STATION 13 – PULMONOLOGY

Asthma Scenario – Diagnosis, Treatment, Causes & Counselling

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CANDIDATE INSTRUCTIONS

8 minutes. Scenario: 25-year-old with wheeze, dyspnea, chest tightness.
State diagnosis, treatment (acute and chronic), causes/triggers, and counselling points.
Demonstrate inhaler technique.

HIGH-YIELD DIAGNOSIS

Asthma: Chronic inflammatory airway disease with reversible bronchospasm
Typical presentation: Episodic wheeze, dyspnea, chest tightness, cough (worse at night/early morning)
Signs: Expiratory wheeze, prolonged expiration, hyperinflated chest, use of accessory muscles, tachypnea, pulsus paradoxus (severe)
Diagnosis confirmed by: Spirometry (FEV1/FVC <0.70 with >12% reversibility after bronchodilator) OR Peak flow variability (>20%)
Severity assessment: Mild (PF >80%), Moderate (PF 50–80%), Severe (PF <50%), Life-threatening (PF <33%, silent chest, cyanosis, exhaustion)

CAUSES & TRIGGERS

Allergic (extrinsic): Dust mites, pollen, pet dander, mold, cockroaches
Non-allergic (intrinsic): Viral infections (rhinovirus), exercise, cold air, stress
Occupational: Isocyanates, flour dust, wood dust, latex
Drugs: Aspirin, NSAIDs, beta-blockers
Other: Smoking, air pollution, GERD, obesity, hormonal changes
Risk factors: Family history (atopy), childhood respiratory infections, prematurity, low birth weight

TREATMENT – STEPWISE APPROACH (GINA)

Acute exacerbation:
• **Mild-moderate:** Salbutamol MDI 2–4 puffs via spacer (repeat every 20 min x 3), Oral prednisolone 40–50 mg (3–5 days)
• **Severe:** Oxygen (maintain SpO2 93–95%), Salbutamol nebulizer 2.5–5 mg (continuous if life-threatening), Ipratropium nebulizer 0.5 mg q20min x 3, Hydrocortisone 200 mg IV or prednisolone 40–50 mg PO, Magnesium sulfate 1.2–2 g IV (if not responding)
• **Life-threatening:** Consider non-invasive ventilation, ICU admission, possible intubation

Chronic control (Stepwise):
• **Step 1 (Mild occasional):** SABA as needed
• **Step 2 (Regular symptoms):** Low-dose ICS (beclomethasone/budesonide) + SABA PRN
• **Step 3:** Low-dose ICS/LABA combination OR Medium-dose ICS
• **Step 4:** Medium-dose ICS/LABA
• **Step 5:** High-dose ICS/LABA + LAMA + biologics (omalizumab, mepolizumab) if eosinophilic

Reliever options: As-needed low-dose ICS-formoterol (preferred) OR SABA

COUNSELLING & INHALER TECHNIQUE

MDI with Spacer technique:
1. Remove cap, shake inhaler
2. Attach to spacer
3. Breathe out fully
4. Seal lips around mouthpiece
5. Press canister once
6. Breathe in slowly and deeply (or tidal breathing for 3–4 breaths if unable)
7. Hold breath 10 seconds
8. Wait 30–60 seconds between puffs

Key counselling points:
• Difference between reliever (quick relief) and controller (daily prevention)
• Rinse mouth after ICS (prevent oral candidiasis)
• Avoid triggers (smoking cessation, allergen avoidance)
• Written asthma action plan (green/yellow/red zones)
• Regular follow-up and spirometry
• Vaccination (influenza, pneumococcal)
• Never run out of reliever medication

CRITICAL ERRORS

Over-reliance on SABA (increases mortality – GINA 2023 emphasizes ICS-containing reliever)
Poor inhaler technique (no spacer, wrong coordination)
No oral steroids in acute exacerbation
Misses life-threatening signs (silent chest, cyanosis, exhaustion)
No written action plan

STATION 14 – MEDICINE/PULMONOLOGY INTERACTIVE

General Physical Examination – Respiratory Focus

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CANDIDATE INSTRUCTIONS

8 minutes. Perform comprehensive GPE with focus on respiratory system.
Identify signs of chronic respiratory disease, respiratory failure, or cor pulmonale.
State findings systematically.

GPE – RESPIRATORY HIGH-YIELD SIGNS

General inspection:
• Cachexia, obesity (OSA risk), pursed-lip breathing (COPD)
• Barrel chest (hyperinflation), kyphoscoliosis, pectus excavatum
• Accessory muscle use (sternomastoid, intercostal indrawing)
• Harrison's sulci (chronic asthma/COPD in children)
• Cyanosis (central – tongue/lips; peripheral – nails)
• Flapping tremor (asterixis – CO2 retention)
• Nicotine staining, clubbing (bronchiectasis, lung cancer, ILD)

Vital signs:
• Respiratory rate (tachypnea >20/min), pattern (Kussmaul, Cheyne-Stokes)
• SpO2 (hypoxemia <92%), pre/post-ductal difference (PFO)
• BP (pulsus paradoxus in severe asthma)
• Temperature (infection)

Hands: Clubbing, tar staining, CO2 retention flap, peripheral cyanosis
Face: Cushingoid (steroid use), superior vena cava obstruction (facial plethora), Horner's syndrome (Pancoast tumor)
Neck: Tracheal deviation (tension pneumothorax, massive effusion), lymphadenopathy (TB, malignancy), raised JVP (cor pulmonale)

RESPIRATORY EXAMINATION

Inspection: Chest shape, symmetry, expansion, scars (thoracotomy), drains
Palpation: Tracheal position, chest expansion (reduced in consolidation/effusion), tactile vocal fremitus (increased in consolidation, decreased in effusion/pneumothorax)
Percussion: Dull (consolidation, effusion, collapse), Hyperresonant (pneumothorax, emphysema), Stony dull (large effusion)
Auscultation:
• Breath sounds: Vesicular, bronchial, diminished/absent
• Added sounds: Crackles (fine – fibrosis, coarse – consolidation), wheeze (asthma, COPD), pleural rub
• Vocal resonance: Bronchophony, whispering pectoriloquy, egophony (consolidation)

CRITICAL ERRORS

Misses tracheal deviation (tension pneumothorax = emergency)
Wrong percussion note (dull vs stony dull)
No assessment of expansion (detects subtle pathology)
Forgets to check for clubbing (chronic lung disease marker)
Misses CO2 retention signs (flap, bounding pulse, headache)

EQUIPMENT

Standardized patient
Stethoscope
Pulse oximeter
Measuring tape
Torch (for clubbing assessment)

STATION 15 – CARDIOLOGY INTERACTIVE

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CANDIDATE INSTRUCTIONS

8 minutes. Perform complete cardiac examination on standardized patient.
Include inspection, palpation, percussion, and auscultation.
Identify murmurs, extra heart sounds, and correlate with pathology.

SYSTEMATIC CARDIAC EXAMINATION

1. INSPECTION:
• General: Pallor, cyanosis, dyspnea, edema
• Precordium: Scars (sternotomy, thoracotomy), deformities, visible apex beat, pulsations
• Epigastrium: Right ventricular heave

2. PALPATION:
• Apex beat: Location (5th intercostal space, mid-clavicular line), character (tapping – mitral stenosis, thrusting – volume overload, heaving – pressure overload)
• Thrills: Palpable murmurs (systolic – aortic stenosis, VSD; diastolic – mitral stenosis)
• Parasternal heave (RVH), palpable P2 (pulmonary hypertension)
• Left sternal edge lift (RVH)

3. PERCUSSION: Cardiac borders (left – apex beat, right – right sternal border), assess cardiomegaly

4. AUSCULTATION:
• Areas: Mitral (apex), Tricuspid (lower left sternal), Pulmonary (upper left sternal), Aortic (upper right sternal), Erb's point (3rd left intercostal)
• Heart sounds: S1 (mitral/tricuspid closure), S2 (aortic/pulmonary – split on inspiration), S3 (ventricular filling – heart failure), S4 (atrial contraction – stiff ventricle)
• Added sounds: Opening snap (mitral stenosis), ejection click (aortic stenosis), pericardial rub
• Murmurs: Timing (systolic/diastolic/continuous), character, location, radiation, intensity (Grade I–VI), change with position/respiration/manoeuvres

MURMUR IDENTIFICATION – HIGH YIELD

Systolic murmurs:
• **Aortic stenosis:** Harsh crescendo-decrescendo, right upper sternal border → carotids, soft S2
• **Mitral regurgitation:** Pansystolic blowing, apex → axilla, S3 present
• **Tricuspid regurgitation:** Pansystolic, left lower sternal edge, increases with inspiration (Carvallo's sign)
• **VSD:** Harsh pansystolic, left lower sternal edge, thrill present
• **Hypertrophic cardiomyopathy:** Harsh, left lower sternal edge, increases with Valsalva/standing

Diastolic murmurs:
• **Mitral stenosis:** Low-p rumbling, apex, pre-systolic accentuation, opening snap
• **Aortic regurgitation:** Early diastolic decrescendo, left sternal edge (Erb's), Austin Flint murmur
• **Pulmonary regurgitation:** Graham Steell murmur (high-pitched, left upper sternal)

Continuous murmurs:
• **PDA:** Machinery murmur, left infraclavicular, radiates to back
• **Venous hum:** Supraclavicular, disappears with compression/JVP elevation

DYNAMIC MANEUVRES

Inspiration: Increases right-sided murmurs (TR, PS, TS)
Expiration: Increases left-sided murmurs (MR, AR, AS)
Valsalva: Decreases most murmurs (except HCM – increases)
Standing: Increases HCM murmur, decreases MVP click
Squatting: Increases preload → increases most murmurs (except HCM)
Handgrip: Increases afterload → increases regurgitant murmurs (MR, AR), decreases HCM

CRITICAL ERRORS

Wrong location for auscultation (listening at apex for AS)
Misses radiation (AS to carotids, MR to axilla)
No mention of dynamic manoeuvres (differentiates murmurs)
Confuses physiological with pathological murmur (innocent murmurs: soft, systolic, no radiation, normal S2)
Misses pericardial rub (friction sound, varies with position)

EQUIPMENT

Standardized patient with murmur
Stethoscope (diaphragm and bell)
Examination couch

STATION 16 – PULMONOLOGY STATIC

Chest X-ray: Pleural Effusion – Positive Findings & Interpretation

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CANDIDATE INSTRUCTIONS

8 minutes. Analyze chest X-ray showing pleural effusion.
Identify positive findings, side and extent of effusion, and possible underlying causes.
Mention Light's criteria for fluid analysis.

HIGH-YIELD X-RAY FINDINGS

Blunting of costophrenic angle (earliest sign – 200 mL on PA, 75 mL on lateral decubitus)
Meniscus sign: Curved upper border (concave upward) – free fluid
Homogeneous opacity: White-out of lower zone, obscuring diaphragm
Mediastinal shift: Away from large effusion, toward if associated collapse/fibrosis
Tracheal deviation: Away from effusion (tension features if massive)
Lateral decubitus view: Fluid layers out (confirms free-flowing vs loculated)
Ultrasound/CT: Loculations, septations, pleural thickening, underlying lung pathology

CLASSIFICATION & CAUSES

Transudate (Light's criteria – none met):
• **Mechanism:** Increased hydrostatic pressure or decreased oncotic pressure
• **Causes:** Congestive heart failure (most common), cirrhosis (hepatic hydrothorax), nephrotic syndrome, hypothyroidism, peritoneal dialysis
• **Fluid:** Clear, straw-colored, protein <30 g/L, LDH <200 U/L, pH 7.40–7.55

Exudate (Light's criteria – ANY ONE met):
• Pleural fluid protein/Serum protein >0.5
• Pleural fluid LDH/Serum LDH >0.6
• Pleural fluid LDH >2/3 upper limit of normal serum LDH
• **Mechanism:** Increased capillary permeability or lymphatic obstruction
• **Causes:** Pneumonia (parapneumonic), TB, malignancy (lung, mesothelioma, metastases), pulmonary embolism, autoimmune (RA, SLE), pancreatitis, esophageal rupture
• **Fluid:** Turbid, bloody, protein >30 g/L, LDH >200 U/L, low pH (<7.30 in complicated parapneumonic/empyema)

Special tests: Cytology (malignant cells), AFB (TB), amylase (pancreatitis/esophageal rupture), triglycerides (chylothorax >110 mg/dL), glucose (low in RA, empyema, malignancy)

MANAGEMENT APPROACH

Diagnostic tap (thoracentesis): Send for protein, LDH, glucose, pH, cell count, Gram stain, culture, cytology
Therapeutic tap: If symptomatic (dyspnea) or massive
Chest drain: Complicated parapneumonic effusion, empyema (pus), loculated effusion
Pleurodesis: Recurrent malignant effusions
VATS/decortication: Thick peel restricting lung expansion

CRITICAL ERRORS

Confuses with consolidation (effusion has meniscus, obscures diaphragm, mediastinal shift away)
Misses underlying cause (check for rib fractures, masses, cardiomegaly)
Wrong fluid classification (must know Light's criteria)
No mention of loculated vs free-flowing (affects drainage approach)

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STATION 17 – PULMONOLOGY STATIC

Chest X-ray: Pneumonia – Positive Findings & Interpretation

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CANDIDATE INSTRUCTIONS

8 minutes. Analyze chest X-ray showing pneumonia.
Identify lobar vs bronchopneumonia pattern, causative organisms, and complications.
Mention vaccine schedule in EPI for prevention.

HIGH-YIELD X-RAY FINDINGS

Lobar pneumonia:
• Homogeneous consolidation of entire lobe
• Air bronchograms (air-filled bronchi visible in opaque lung)
• Silhouette sign (loss of diaphragm/heart border silhouette)
• No volume loss (unlike collapse)
• Right lower lobe most common in adults

Bronchopneumonia:
• Patchy, ill-defined opacities (multifocal)
• Peribronchial distribution
• Bilateral, lower zones predominant
• More common in elderly, infants, immunocompromised

Interstitial pneumonia:
• Reticular/reticulonodular pattern
• Perihilar, diffuse
• Typical of atypical organisms (Mycoplasma, viral)

ORGANISMS & PATTERNS

Streptococcus pneumoniae (most common lobar): Single lobe consolidation, air bronchograms, "bulging fissure" sign (Klebsiella)
Klebsiella pneumoniae: Upper lobe, volume expansion, bulging fissure, cavitation risk
Staphylococcus aureus: Bronchopneumonia pattern, rapid cavitation, pneumatoceles (children)
Haemophilus influenzae: Patchy consolidation, often in COPD patients
Mycoplasma pneumoniae: Reticulonodular, peribronchial cuffing, segmental atelectasis
Viral (RSV, influenza): Bilateral interstitial pattern, hyperinflation, peribronchial thickening

COMPLICATIONS ON X-RAY

Pleural effusion: Blunting of costophrenic angle (parapneumonic)
Empyema: Loculated fluid, pleural thickening, air-fluid level
Lung abscess: Cavity with air-fluid level, thick wall
Necrotizing pneumonia: Multiple thin-walled cavities
ARDS: Bilateral diffuse opacities, white-out
Delayed resolution: Consider obstruction (tumor), unusual organism, wrong antibiotics

EPI VACCINE SCHEDULE (PAKISTAN)

Pneumococcal vaccine (PCV-10):
• Dose 1: 6 weeks
• Dose 2: 10 weeks
• Dose 3: 14 weeks
• Protects against Streptococcus pneumoniae (10 serotypes)

Hib vaccine: 6, 10, 14 weeks (prevents Haemophilus influenzae type b pneumonia/meningitis)
Measles vaccine: 9 months, 15 months (prevents viral pneumonia)
Influenza vaccine: Annual (not in routine EPI but recommended)
Pertussis (DTP): 6, 10, 14 weeks (prevents whooping cough pneumonia)

CRITICAL ERRORS

Confuses lobar with collapse (collapse has volume loss, mediastinal shift toward)
Misses air bronchograms (confirms consolidation vs effusion)
Wrong organism for pattern (Klebsiella upper lobe, Staph cavitating)
No mention of vaccine schedule (EPI requirements)
Misses complications (effusion, abscess)

STATION 18 – PULMONOLOGY INTERACTIVE

Acute & Chronic Asthma Management – Comprehensive Approach

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Note: This station combines acute and chronic management with practical demonstration. Overlaps with Station 13 but adds practical skills.

CANDIDATE INSTRUCTIONS

8 minutes. Manage acute asthma exacerbation scenario and set up chronic treatment plan.
Demonstrate nebulizer setup, oxygen delivery, and inhaler technique.
State when to admit vs discharge.

ACUTE MANAGEMENT – STEPWISE

Immediate assessment (ABC):
• Airway: Patency, ability to speak (can't speak = severe)
• Breathing: RR, SpO2, auscultation (silent chest = life-threatening), accessory muscles, pulsus paradoxus
• Circulation: HR, BP, capillary refill
• Peak flow: <33% predicted = life-threatening, 33–50% = severe, >50% = moderate

Immediate interventions:
• **Oxygen:** Target SpO2 93–95% (94–98% if COPD overlap), high-flow nasal cannula or mask
• **Bronchodilators:** Salbutamol 2.5–5 mg nebulized with oxygen (driven at 6–8 L/min), repeat every 20 min x 3 then reassess
• **Ipratropium bromide:** 0.5 mg nebulized (add to salbutamol for severe cases)
• **Steroids:** Prednisolone 40–50 mg PO OR hydrocortisone 200 mg IV (if vomiting/severe)
• **Magnesium sulfate:** 1.2–2 g IV over 20 min (if poor response to initial therapy)

Monitoring: Continuous SpO2, frequent PEFR, cardiac monitoring (risk of arrhythmia with high-dose beta-agonists)

CHRONIC MANAGEMENT – GINA 2023

Track 1 (Preferred): As-needed low-dose ICS-formoterol (reliever and controller)
• Reduces severe exacerbations vs SABA-only approach
• Suitable for mild asthma

Track 2 (Alternative): SABA reliever + daily ICS maintenance
• Step 2: Low-dose ICS daily OR as-needed ICS with SABA
• Step 3: Low-dose ICS-formoterol daily + as-needed OR Medium-dose ICS
• Step 4: Medium-dose ICS-formoterol
• Step 5: High-dose ICS-formoterol + LAMA + consider biologics

Non-pharmacological: Smoking cessation, allergen avoidance, weight loss, exercise, vaccination (influenza, COVID-19)

DISCHARGE CRITERIA & PLAN

Safe to discharge if:
• PEFR >75% predicted or personal best
• SpO2 >94% on room air
• No distress, no accessory muscle use
• Oral steroids started (continue 5 days)
• Inhaler technique checked and corrected
• Written action plan provided
• Follow-up arranged (GP review in 2 days)

Admission criteria:
• PEFR <75% after 1 hour treatment
• SpO2 <92% on room air
• Life-threatening features
• Poor social circumstances, previous near-fatal asthma

EQUIPMENT DEMONSTRATION

Jet nebulizer setup
Oxygen flow meter
MDI with spacer
Peak flow meter
Written asthma action plan template

STATION 19 – CARDIOLOGY COMPREHENSIVE

Murmurs: ASD, VSD, PDA, AR, AS, PS & Cyanotic Heart Diseases

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CANDIDATE INSTRUCTIONS

8 minutes. Identify and differentiate various murmurs on audio or patient examination.
State characteristics, pathophysiology, and management for each lesion.
Distinguish acyanotic vs cyanotic heart diseases.

ACYANOTIC CONGENITAL HEART DISEASES (LEFT-TO-RIGHT SHUNTS)

1. Atrial Septal Defect (ASD):
• **Murmur:** Ejection systolic, left upper sternal border (pulmonary flow murmur), wide fixed split S2
• **Types:** Secundum (fossa ovalis, 70%), Primum (low ASD, associated with AVSD), Sinus venosus (SVC/IVC junction)
• **ECG:** RVH, RBBB, left axis deviation (primum)
• **Management:** Device closure (secundum) or surgical repair (age 2–4 years) if significant shunt (Qp:Qs >1.5:1)

2. Ventricular Septal Defect (VSD):
• **Murmur:** Harsh pansystolic, left lower sternal edge, thrill present, radiation to right sternal border
• **Types:** Perimembranous (80%), muscular, supracristal (subarterial)
• **Complications:** Eisenmenger syndrome (if large, untreated), aortic regurgitation (supracristal), infective endocarditis
• **Management:** Small (watchful waiting, 30% close spontaneously), Large (surgical repair by age 1 if failure to thrive)

3. Patent Ductus Arteriosus (PDA):
• **Murmur:** Continuous "machinery," left infraclavicular, radiates to back, wide pulse pressure, bounding pulses
• **Management:** NSAIDs (premature), device closure/surgical ligation (if symptomatic/large)

4. Pulmonary Stenosis (PS):
• **Murmur:** Ejection systolic crescendo-decrescendo, left upper sternal border, radiates to left shoulder/back, ejection click
• **Severity:** Mild (gradient <40 mmHg), Moderate (40–60 mmHg), Severe (>60 mmHg)
• **Management:** Balloon valvuloplasty (if symptomatic or gradient >50 mmHg)

VALVULAR LESIONS – ACQUIRED

Aortic Stenosis (AS):
• **Murmur:** Harsh ejection systolic, right upper sternal border → carotids, soft/absent S2, S4 gallop
• **Causes:** Degenerative/calcific (elderly), Bicuspid aortic valve (younger), Rheumatic (rare isolated)
• **Triad:** Angina, syncope, heart failure (poor prognosis)
• **Management:** Valve replacement (TAVI or surgical) when symptomatic or severe (gradient >40 mmHg, area <1 cm^2)

Aortic Regurgitation (AR):
• **Murmur:** Early diastolic decrescendo, left sternal edge (Erb's), high-pitched, blowing, Austin Flint murmur (functional MS)
• **Signs:** Wide pulse pressure, collapsing pulse (water-hammer), Quincke's pulse (nail bed pulsation), de Musset's sign (head bobbing), Duroziez's sign (femoral diastolic murmur)
• **Causes:** Rheumatic, infective endocarditis, Marfan syndrome, aortic root dilatation
• **Management:** Vasodilators (nifedipine, ACEi), valve replacement if symptomatic or LV dilation

CYANOTIC CONGENITAL HEART DISEASES (RIGHT-TO-LEFT SHUNTS)

Tetralogy of Fallot (TOF) – Most common cyanotic CHD:
• **Components:** VSD, Pulmonary stenosis, Overriding aorta, RVH
• **Murmur:** Ejection systolic (PS), single S2 (aortic), no thrill (right-to-left shunt)
• **Cyanosis:** Central, worse with crying/feeding, squatting position (relieves)
• **Hypercyanotic spells (Tet spells):** Emergency – knee-chest position, oxygen, morphine, IV fluids, beta-blockers, phenylephrine
• **Management:** Total corrective surgery (age 4–6 months), Blalock-Taussig shunt (palliative if severe)

Transposition of Great Arteries (TGA):
• **Pathology:** Aorta from RV, PA from LV (parallel circuits)
• **Presentation:** Severe cyanosis within hours of birth, single loud S2
• **Emergency:** Prostaglandin E1 (keep PDA open), balloon atrial septostomy (Rashkind), arterial switch operation

Other cyanotic lesions: Tricuspid atresia, Pulmonary atresia, Total anomalous pulmonary venous return, Hypoplastic left heart syndrome

CRITICAL ERRORS

Confuses ASD with innocent murmur (fixed split S2 is key)
Misses wide pulse pressure in PDA (differentiates from VSD)
Wrong timing of AR murmur (early diastolic, not pansystolic)
Forgets squatting in TOF (increases SVR, reduces right-to-left shunt)
No mention of Eisenmenger syndrome (irreversible pulmonary hypertension)

STATION 20 – CARDIOLOGY COMPREHENSIVE

Automated External Defibrillator (AED) – When to Use & Advanced Usage

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Note: Expands on Station 3 (CPR & AED) with specific scenarios and troubleshooting.

CANDIDATE INSTRUCTIONS

8 minutes. Demonstrate AED use in different scenarios: witnessed collapse, unwitnessed arrest, pediatric patient, and wet environment.
State shockable vs non-shockable rhythms and energy settings.

WHEN TO USE AED – INDICATIONS

Cardiac arrest with:
• Unresponsive patient
• Absent or abnormal breathing (agonal gasps)
• No pulse (or not checked if rescuer untrained)
• **Shockable rhythms:** Ventricular fibrillation (VF), Pulseless ventricular tachycardia (pVT)
• **Non-shockable:** Asystole, Pulseless electrical activity (PEA) – CPR only, no shock

Special situations:
• **Witnessed collapse:** Immediate defibrillation (if AED available within 3 minutes)
• **Unwitnessed collapse:** 2 minutes CPR first, then AED (oxygen depletion)
• **Persistent VF:** Continue CPR, shock every 2 minutes, consider antiarrhythmics (amiodarone)
• **Recurrent VF:** Check for reversible causes (Hs and Ts)

AED OPERATION – STEP-BY-STEP

Adult protocol:
1. Power on AED
2. Attach pads: Right upper chest (below clavicle), Left side (mid-axillary, lateral to nipple)
3. Connect cable to AED
4. Clear patient during analysis ("Stand clear")
5. Deliver shock if advised ("Shock advised, charging... stand clear, press shock button")
6. Resume CPR immediately after shock (or if no shock advised)
7. Continue 30:2 compression-to-ventilation ratio
8. Reanalyze every 2 minutes (5 cycles)

Energy settings:
• Biphasic: 120–200 J (or manufacturer recommendation, often 150–200 J)
• Monophasic: 360 J
• Escalating energy: Consider higher dose if initial shock fails

Pediatric considerations (1–8 years):
• Use pediatric pads/attenuator if available (reduces energy to 50–75 J)
• If no pediatric pads, use adult pads (place front-back if pads touch)
• Pad placement: Anterior-posterior (chest-back) or anterior-lateral
• Energy: 2 J/kg first shock, 4 J/kg subsequent (up to 10 J/kg or adult dose)

Infants (<1 year):
• Manual defibrillator preferred
• If AED only option, use pediatric pads with attenuator

SAFETY & TROUBLESHOOTING

Safety:
• Remove patient from water (dry chest)
• Remove nitroglycerin patches (explosion risk)
• Avoid placing pads over implanted devices (pacemaker/ICD) – place 8 cm away
• Remove metal jewelry (not touching pads)
• Ensure no one touching patient during analysis/shock
• Combustible atmosphere risk (oxygen-enriched, gasoline fumes) – ventilate area

Troubleshooting:
• "Check pads" – ensure good contact, dry chest, hair removal if needed
• "Motion detected" – stop CPR, ensure ambulance not moving
• "No shock advised" – resume CPR, check pulse, continue algorithm
• Cold patient – may need warming for successful defibrillation

POST-RESUSCITATION CARE

• Return of spontaneous circulation (ROSC) check
• Airway management, oxygenation
• 12-lead ECG
• Targeted temperature management (32–36°C for 24 hours if comatose)

• ICU admission, coronary angiography if cardiac cause suspected
• Implantable cardioverter-defibrillator (ICD) consideration for survivors of VF/VT arrest

CRITICAL ERRORS

Delay in AED attachment (every minute = 10% survival decrease)
Touching patient during analysis (artifact, shock risk)
Wrong pad placement (over pacemaker, poor contact)
Analyzing too long (>10 seconds interruption)
Using adult dose on small child (without pediatric attenuator)

EQUIPMENT

AED trainer (adult and pediatric pads)
Adult and infant mannequins
Razor (for hairy chest)
Towel (for wet chest)
Gloves

STATION 21 – MEDICINE

Infective Endocarditis – Diagnosis, Duke Criteria & Management

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CANDIDATE INSTRUCTIONS

8 minutes. Scenario: Patient with fever, murmur, and peripheral signs.
Apply Duke criteria, state investigations, and outline antibiotic therapy.
Mention indications for surgery and prophylaxis.

HIGH-YIELD CLINICAL FEATURES

Constitutional: Fever (>38°C), night sweats, weight loss, fatigue, anorexia
Cardiac: New or changing murmur (regurgitant), heart failure, embolic phenomena
Vascular phenomena: Arterial emboli (stroke, limb ischemia), septic pulmonary infarcts (right-sided), mycotic aneurysm
Immunologic phenomena: Roth spots (retinal hemorrhages with pale centers), Osler nodes (painful finger/toe pads), Janeway lesions (painless palm/sole macules), splinter hemorrhages, glomerulonephritis
Risk factors: Prosthetic valve, previous endocarditis, rheumatic heart disease, congenital heart disease, IV drug use, indwelling catheters, poor dental hygiene

DUKE CRITERIA (Definite IE = 2 Major OR 1 Major + 3 Minor OR 5 Minor)

MAJOR CRITERIA:
1. **Blood cultures positive for IE:**
• Typical organism (S. viridans, S. bovis, HACEK, S. aureus, Enterococcus) from 2 separate cultures
• Persistently positive (2 positive >12 hours apart OR 3+ positive)
• Single positive Coxiella burnetii (Q fever) or phase I IgG antibody titer >1:800
2. **Evidence of endocardial involvement:**
• Echocardiogram positive (oscillating vegetation, abscess, new partial dehiscence of prosthetic valve)
• New valvular regurgitation (worsening/changing of existing murmur not sufficient)

MINOR CRITERIA:
• Predisposing heart condition or IV drug use
• Fever >38°C
• Vascular phenomena (arterial emboli, septic pulmonary infarcts, mycotic aneurysm, ICH, conjunctival hemorrhage, Janeway lesions)
• Immunologic phenomena (glomerulonephritis, Osler nodes, Roth spots, rheumatoid factor)
• Microbiologic evidence (positive blood culture not meeting major criteria, serologic evidence of active infection)
• Echocardiographic minor criteria (consistent with IE but not meeting major)

INVESTIGATIONS

Blood cultures: 3 sets from different sites, 1 hour apart, before antibiotics (yield 90–95%)
Echocardiogram: TTE first (vegetations >2 mm), TEE if prosthetic valve, negative TTE with high suspicion, or complications (abscess)
Lab: CBC (anemia, leukocytosis), ESR/CRP (elevated), renal function, urinalysis (hematuria/proteinuria), rheumatoid factor
ECG: Heart block (aortic root abscess), conduction abnormalities

CT/MRI: Septic emboli (brain, spleen), mycotic aneurysms
Dental panoramic: If oral source suspected

ANTIBIOTIC THERAPY (EMPIRIC & TARGETED)

Empiric (before cultures):
• **Native valve:** Vancomycin + Ceftriaxone (or Ampicillin-sulbactam)
• **Prosthetic valve (<1 year):** Vancomycin + Gentamicin + Rifampicin + Cefepime
• **IV drug user:** Vancomycin (cover MRSA)

Targeted (culture-positive):
• **Streptococcus viridans (sensitive):** Penicillin G or Ceftriaxone x 4 weeks (2 weeks if gentamicin added)
• **Staphylococcus aureus (MSSA):** Flucloxacillin or Cefazolin x 4–6 weeks
• **MRSA:** Vancomycin (trough 15–20 mcg/mL)
• **Enterococcus:** Ampicillin + Gentamicin (synergy) or Vancomycin if resistant
• **HACEK:** Ceftriaxone
• **Fungal (Candida/Aspergillus):** Amphotericin B + surgery

Duration: Native valve 4–6 weeks, Prosthetic valve minimum 6 weeks, Right-sided (IVDU) 2 weeks (if uncomplicated)

INDICATIONS FOR SURGERY (URGENT/Emergent)

Heart failure: Refractory pulmonary edema, cardiogenic shock (valve dysfunction)
Uncontrolled infection: Abscess, fistula, persistent bacteremia (>7 days despite antibiotics), fungal endocarditis
Prevention of embolism: Large vegetation (>10 mm), recurrent emboli despite antibiotics, mobile vegetation
Prosthetic valve: Dehiscence, obstruction, worsening regurgitation

PROPHYLAXIS (HIGH-RISK PROCEDURES ONLY)

High-risk cardiac conditions: Prosthetic valve, prior IE, unrepaired cyanotic CHD, repaired CHD with residual defects, cardiac transplant with valvulopathy
Procedures requiring prophylaxis: Dental (manipulation of gingiva/periapical region), respiratory tract (incision/biopsy), infected skin/musculoskeletal
Regimen: Single dose Amoxicillin 2 g PO (or Ampicillin 2 g IV/IM) 30–60 min before procedure
If penicillin allergic: Clindamycin 600 mg or Azithromycin 500 mg

CRITICAL ERRORS

Giving antibiotics before blood cultures (reduces yield)
Wrong duration (native valve minimum 4 weeks)
Missing indication for surgery (heart failure, abscess, large vegetation)
Unnecessary prophylaxis (not indicated for low-risk cardiac conditions)
Confuses Osler nodes with Janeway lesions (Osler = painful/immunologic, Janeway = painless/vascular)

STATION 22 – CARDIOLOGY COMPREHENSIVE

Rheumatic Heart Disease – Diagnosis, Prophylaxis & Advanced Management

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Note: Expands on Station 4 with detailed prophylaxis dosing and assessment criteria.

CANDIDATE INSTRUCTIONS

8 minutes. Scenario: Patient with history of rheumatic fever, now with valve disease.
Determine prophylaxis duration, assess medication dosing criteria, and outline management with/without carditis.
Perform focused cardiac examination.

RHEUMATIC FEVER RECAP & CARDITIS ASSESSMENT

Jones Criteria (diagnosis): 2 Major OR 1 Major + 2 Minor + evidence of streptococcal infection
Major: Carditis, Polyarthritis, Chorea, Erythema marginatum, Subcutaneous nodules
Minor: Fever, Arthralgia, Elevated ESR/CRP, Prolonged PR interval

Carditis assessment (determines severity and prophylaxis):
• **Clinical:** Tachycardia (out of proportion to fever), new murmur (mitral regurgitation most common, Carey Coombs murmur – mid-diastolic), pericardial rub, heart failure signs
• **ECG:** Prolonged PR interval (first-degree AV block), myocarditis changes

- **Echo:** Valvulitis (mitral > aortic), regurgitation, pericardial effusion, chamber enlargement
- **Without carditis:** Arthritis only, no cardiac involvement on echo
- **With carditis (no residual valve disease):** Carditis resolved completely, echo normal
- **With carditis and residual valve disease:** Persistent valvular damage (stenosis/regurgitation)

SECONDARY PROPHYLAXIS PROTOCOL

Assessment deciding medication dose:
- **Weight-based dosing:** Benzathine penicillin G 1.2 million units IM if >30 kg, 600,000 units if <30 kg
- **Frequency:** Every 4 weeks (every 3 weeks in high-risk areas like Pakistan/India)
- **Alternative:** Penicillin V 250 mg PO BD (compliance issues)
- **If penicillin allergic:** Erythromycin 250 mg PO BD or Sulfadiazine 1 g OD (500 mg if <30 kg)

Duration of prophylaxis:
- **Without carditis:** 5 years or until age 21 (whichever is longer)
- **With carditis (no residual valve disease):** 10 years or until age 21 (whichever is longer)
- **With carditis and residual valve disease:** Lifelong (or at least 10 years after last attack and until age 40)
- **Post-valve surgery:** Lifelong regardless of RF history

Special situations:
- Pregnancy: Continue prophylaxis (penicillin safe, erythromycin estolate contraindicated)
- High-risk occupations (healthcare, childcare): Consider extended prophylaxis

MANAGEMENT – WITH VS WITHOUT CARDITIS

Acute rheumatic fever WITH carditis:
- **Bed rest:** Until acute phase resolves (2–4 weeks), then gradual mobilization
- **Prednisolone:** 1–2 mg/kg/day (max 60–80 mg) for 2 weeks, then taper over 2–4 weeks
- **Aspirin:** Stop when pain resolved, overlap with steroids (steroids started first)
- **Diuretics/Digoxin:** If heart failure
- **Monitor:** ESR/CRP weekly (should normalize in 4–6 weeks)

Acute rheumatic fever WITHOUT carditis:
- **Aspirin:** 60–100 mg/kg/day (max 4–6 g/day) until symptoms resolve, then taper over 2 weeks
- **No steroids** (unless arthritis not responding to aspirin)
- **Mobility:** Earlier mobilization allowed

Chronic RHD management:
- Secondary prophylaxis (as above)
- Heart failure management (diuretics, ACEi, beta-blockers)
- Anticoagulation if AF or mechanical valve
- Valve intervention: Percutaneous balloon mitral valvotomy (MS), valve repair/replacement (MR/AR)
- Regular echo monitoring (every 1–2 years if stable)

WHEN TO REFER FOR SURGERY

Mitral stenosis: Symptomatic (NYHA II–IV), pulmonary hypertension, recurrent emboli despite anticoagulation
Mitral regurgitation: Symptomatic, LV dysfunction (EF <60%, ESD >40 mm), AF new onset
Aortic stenosis/regurgitation: Symptomatic, LV dysfunction, rapid progression
Tricuspid regurgitation: Symptomatic severe TR, right heart failure

CRITICAL ERRORS

Wrong prophylaxis duration (especially with residual valve disease)
Giving steroids without carditis (aspirin sufficient)
Wrong weight-based dose (1.2 million vs 600,000 units)
Stopping prophylaxis too early (recurrence risk)
Missing valve intervention indications (wait too long = irreversible LV dysfunction)

STATION 23 – CARDIOLOGY COMPREHENSIVE

ECG Myocardial Infarction Patterns – All Territories & Correlations

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> **Note:** Comprehensive station covering all ECG territories mentioned in both documents (Inferior, Septal, Anterior, Anteroseptal, Lateral).

CANDIDATE INSTRUCTIONS

8 minutes. Analyze multiple ECGs showing different MI territories.
Identify leads involved, culprit artery, reciprocal changes, and specific management considerations.
Correlate with clinical presentation and complications.

TERRITORY-BY-TERRITORY ANALYSIS

1. INFERIOR WALL MI (II, III, aVF):
• **Culprit artery:** RCA (80%, right dominant), LCx (20%, left dominant)
• **Reciprocal:** ST depression I, aVL (high lateral)
• **Right ventricular involvement:** ST elevation V4R (check in hypotensive patients)
• **Posterior extension:** Tall R waves V1–V3, ST depression V1–V3
• **Complications:** Bradycardia, AV block (nodal artery from RCA), hypotension (Bezold-Jarisch reflex)
• **Contraindications:** Nitrates if RV infarct (preload dependent)

2. SEPTAL MI (V1–V2):
• **Culprit:** Septal perforators from LAD
• **ECG:** ST elevation V1–V2, loss of septal q waves in lateral leads
• **Associations:** Often part of anteroseptal MI
• **Complications:** Septal rupture (VSD), complete heart block

3. ANTERIOR WALL MI (V3–V4):
• **Culprit:** LAD (diagonal branches)
• **Extent:** "Extensive anterior" if includes V5–V6
• **Reciprocal:** ST depression II, III, aVF
• **Complications:** Large infarct = cardiogenic shock, heart failure, ventricular aneurysm

4. ANTEROSEPTAL MI (V1–V4):
• **Culprit:** Proximal LAD (before first septal/diagonal)
• **Significance:** Large territory, high risk
• **ECG:** ST elevation V1–V4, poor R wave progression
• **Complications:** High-grade AV block, cardiogenic shock, mural thrombus

5. LATERAL WALL MI (I, aVL, V5–V6):
• **Culprit:** LCx (obtuse marginal branches), Diagonal branches of LAD
• **Reciprocal:** ST depression II, III, aVF (inferior)
• **Association:** Often with anterior MI (anterolateral)
• **Complications:** Lateral papillary muscle dysfunction (mitral regurgitation)

6. POSTERIOR MI (V7–V9):
• **ECG:** Tall R waves V1–V2 (reciprocal to posterior Q waves), ST depression V1–V3
• **Culprit:** RCA or LCx (posterior descending artery)
• **Clinical:** Often associated with inferior MI

ECG EVOLUTION & RECURRENT MIs

Hyperacute phase (minutes): Tall, peaked T waves (especially anterior)
Acute phase (hours): ST elevation, Q waves begin
Evolution (days): T wave inversion, Q waves established
Chronic: Persistent Q waves, T waves may normalize or remain inverted
Recurrent MI: New ST elevation in same territory (extension) or different territory, new Q waves, reinversion of normalized T waves
Left main occlusion: Widespread ST depression (I, II, III, aVF, V4–V6) with ST elevation aVR (>V1) – "tombstone" pattern

MANAGEMENT VARIATIONS BY TERRITORY

All territories: Aspirin, P2Y12 inhibitor (ticagrelor/prasugrel), anticoagulation, statin, beta-blocker, ACEi
Inferior (with RV): Avoid nitrates, diuretics; maintain preload (IV fluids if hypotensive)
Anterior (large): Watch for heart failure, consider IABP if cardiogenic shock
Posterior: Consider additional posterior leads (V7–V9) for diagnosis
Reperfusion: Primary PCI preferred for all; thrombolysis if delay >120 minutes (avoid if posterior MI with dominant R wave in V1 suggesting RV infarct)

CRITICAL ERRORS

Wrong coronary territory mapping (LAD vs RCA vs LCx)
Missing reciprocal changes (confirms STEMI vs pericarditis)
No right-sided leads in inferior MI (misses RV infarct)
Giving nitrates in RV infarct (catastrophic hypotension)
Confusing posterior MI with anterior ischemia (tall R waves vs true R wave progression)

STATION 24 – SURGERY/RESPIRATORY

Underwater Seal Drain (Intercostal Drainage System) – Identification & Management

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CANDIDATE INSTRUCTIONS

8 minutes. Identify underwater seal drain system.
State indications, demonstrate setup, describe nursing care, and removal criteria.
Troubleshoot common problems (air leak, blockage, dislodgement).

DEVICE IDENTIFICATION & COMPONENTS

Underwater Seal Drain (UWSD) / Chest Tube Drainage System:
Components:
• **Chest tube:** Radiopaque, multiple side holes, size 20–36 Fr (adults), 12–20 Fr (children)
• **Connecting tubing:** 6-foot long, non-kinking
• **Drainage bottle/chamber:** Collection chamber (measures output)
• **Water seal chamber:** 2 cm water seal (prevents air reflux)
• **Suction control chamber:** 20 cm water (if suction applied)
• **Fluctuation (swing):** Tidaling with respiration (normal)
• **Bubbling:** Indicates air leak (continuous = bronchopleural fistula, intermittent = normal expiration)

INDICATIONS FOR CHEST DRAIN

Pneumothorax: Tension, recurrent, iatrogenic, traumatic, persistent (>20% or symptomatic)
Pleural effusion: Large, symptomatic, empyema, hemothorax, chylothorax
Post-operative: Thoracotomy, cardiac surgery (mediastinal drains), esophageal surgery
Empyema: Drainage of pus, intrapleural fibrinolytics
Contraindications: Coagulopathy (INR >1.5, platelets <50,000), loculated effusion (needs imaging guidance), pulmonary bullae (risk of bronchopleural fistula)

INSERTION TECHNIQUE (SAFE TRIANGLE)

Position: Triangle of safety (mid-axillary line, 4th–5th intercostal space, lateral to nipple)
Technique:
1. Consent, site marking, sterile prep
2. Local anesthetic (lidocaine with adrenaline)
3. Incision parallel to rib (avoid neurovascular bundle under rib)
4. Blunt dissection through intercostal muscles
5. Finger sweep to clear adhesions
6. Tube insertion (directed posteriorly for fluid, apically for air)
7. Connect to UWSD
8. Suture in place, dressing, CXR confirmation
Size selection: Air/pneumothorax (20–24 Fr), Fluid/hemothorax (28–36 Fr), Empyema (large bore or surgical)

NURSING CARE & MONITORING

Position: Bottle below chest level at all times (prevents fluid reflux)
Keep patent: No kinks, dependent loops, or clamps (except briefly when changing bottles)
Encourage: Deep breathing, coughing, mobilization (aids drainage)
Monitor: Drainage amount (mark hourly if fresh post-op), character (blood, pus, serous), bubbling pattern
Milk/strip: Only if blockage suspected (gentle squeezing toward patient, not routine)
Suction: High-volume, low-pressure suction (10–20 cm H_2O) if air leak or large effusion

TROUBLESHOOTING

No swing/fluctuation: Blocked (blood clot), kinked tube, tube against lung/ mediastinum, lung fully expanded
Continuous bubbling: Air leak (bronchopleural fistula), check connections, may need larger tube or surgery
Sudden cessation of drainage: Blockage or lung re-expansion
Subcutaneous emphysema: Air leak into tissues, check tube position, may need additional drain
Tube dislodgement: Cover with occlusive dressing, do not reinsert, call surgeon

REMOVAL CRITERIA

Pneumothorax: Lung fully expanded on CXR, no air leak for 24 hours (clamp trial optional)
Effusion: Drainage <200 mL/24 hours, CXR showing resolution
Empyema: Clinically improved, drainage minimal, cavity collapsed
Technique: Patient inspires deeply and holds (or exhales), rapid removal, immediate occlusive dressing, CXR 4 hours post-removal
Complications of removal: Recurrent pneumothorax, infection, bleeding

CRITICAL ERRORS

Bottle above chest level (fluid reflux into chest)
Clamping for transport (risk of tension pneumothorax)
Wrong insertion site (too low = abdominal organs, too high = vascular injury)

Removal with active air leak (recurrent pneumothorax)
No CXR after insertion/removal (confirm position/complications)

EQUIPMENT

UWSD system (single, double, or triple chamber)
Chest tubes (various sizes)
Insertion tray (scalpel, forceps, sutures)
Underwater seal bottle with sterile water
Suction regulator (if needed)
Dressing materials

STATION 25 – PULMONOLOGY

Breath Sounds – Identification, Pathophysiology & Clinical Correlation

KMU FINAL YEAR MBBS

CANDIDATE INSTRUCTIONS

8 minutes. Identify different breath sounds on audio or patient examination.
State characteristics, location, and pathological significance.
Differentiate from added sounds (adventitious sounds).

NORMAL BREATH SOUNDS

1. Vesicular Breath Sounds:
• **Location:** Peripheral lung fields, majority of lung
• **Characteristics:** Soft, low-pitched, rustling
• **Inspiration:** Longer than expiration (I:E ratio 3:1 or 4:1)
• **No pause** between inspiration and expiration
• **Mechanism:** Air moving through small airways and alveoli

2. Bronchial Breath Sounds:
• **Location:** Trachea, larynx, over manubrium (normally)
• **Characteristics:** Loud, high-pitched, hollow, blowing
• **Inspiration:** Equal to or shorter than expiration (I:E ratio 1:1 or 1:2)
• **Pause** between inspiration and expiration
• **Abnormal location:** Indicates consolidation (pneumonia), cavity near large airway, or pleural effusion (transmission through fluid)

3. Bronchovesicular Breath Sounds:
• **Location:** 1st and 2nd intercostal spaces anteriorly, between scapulae posteriorly
• **Characteristics:** Intermediate between vesicular and bronchial
• **I:E ratio:** Equal phases
• **Abnormal if:** Heard in peripheral lung fields (early consolidation or fibrosis)

ABNORMAL (ADVENTITIOUS) BREATH SOUNDS

1. Crackles (Rales):
• **Fine crackles:** High-pitched, end-inspiratory, like hair rubbing
– Causes: Pulmonary fibrosis, interstitial lung disease, early pneumonia, pulmonary edema
• **Coarse crackles:** Low-pitched, early inspiratory, like Velcro opening
– Causes: Pneumonia, bronchiectasis, pulmonary edema, atelectasis
• **Mechanism:** Opening of small airways/deflated alveoli, fluid in airways

2. Wheezes (Sibilant/Rhonchi):
• **Polyphonic wheeze:** Multiple pitches, musical, expiratory > inspiratory
– Causes: Asthma, COPD (bronchospasm, airway narrowing)
• **Monophonic wheeze:** Single pitch, localized
– Causes: Foreign body, tumor (fixed obstruction)
• **Mechanism:** Airway narrowing <5 mm, vibration of airway walls

3. Rhonchi (Sonorous wheeze):
• Low-pitched, snoring quality, inspiratory and expiratory
• Causes: Secretions in large airways, bronchitis, COPD
• Clears with coughing (vs wheeze which persists)

4. Pleural Rub:
• Leathery, creaking, low-pitched, localized
• Synchronous with respiration, not affected by coughing
• Causes: Pleurisy, pulmonary infarction, pleural tumor
• Best heard at end-inspiration and early expiration

5. Stridor:
• High-pitched, inspiratory, upper airway
• Causes: Laryngeal edema, foreign body, epiglottitis, croup
• Emergency (upper airway obstruction)

DIMINISHED/ABSENT BREATH SOUNDS

Causes:
• **Pleural effusion:** Fluid between lung and chest wall
• **Pneumothorax:** Air in pleural space
• **Atelectasis:** Collapsed lung
• **Emphysema:** Hyperinflation, poor sound transmission
• **Obesity:** Increased distance from chest wall
• **Consolidation with obstruction:** No airflow distal to obstruction

VOICE TRANSMISSION SOUNDS

Bronchophony: "99" or "1,2,3" louder and clearer over consolidation
Whispering pectoriloquy: Whispered words clearly audible (consolidation)
Egophony: "E" to "A" change (E-to-A phenomenon over consolidation or effusion)
Mechanism: Solid medium transmits sound better than air (consolidation) or fluid interface (effusion)

CRITICAL ERRORS

Confusing crackles with wheeze (crackles = fluid/alveoli, wheeze = airway)
Missing unilateral absent sounds (pneumothorax, massive effusion = emergency)
Not timing crackles (fine late = fibrosis, coarse early = secretions)
Ignoring stridor (upper airway emergency)
Wrong interpretation of bronchial breathing (consolidation vs normal location)

Crafted with ❤️ Noaman khan Musakhel

STATION 26 – MEDICINE/PULMONOLOGY

Tuberculosis Treatment – Regimens, Monitoring & Complications

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Note: Combines TB treatment from both documents with X-ray findings and comprehensive management.

CANDIDATE INSTRUCTIONS

8 minutes. Scenario: Patient with TB (pulmonary/extrapulmonary).
State treatment regimen, monitoring requirements, and complications.
Interpret chest X-ray findings and know when to suspect drug resistance.

CHEST X-RAY FINDINGS IN TB

Primary TB:
• Ghon focus (peripheral, mid/upper zone consolidation)
• Hilar/mediastinal lymphadenopathy (asymmetric, unilateral)
• Ghon complex (Ghon focus + lymph node involvement)
• Pleural effusion (often unilateral, large)
• Miliary pattern (hematogenous spread – 1–3 mm nodules)

Post-primary (Reactivation) TB:
• Apical/posterior upper lobe involvement (90%)
• Cavitation (thick-walled, air-fluid levels)
• Patchy consolidation, fibrosis, volume loss
• Tuberculoma (coin lesion, calcified)
• "Destroyed lung" (end-stage fibrosis)

HIV-associated TB: Atypical – lower zone, diffuse, mediastinal lymphadenopathy, less cavitation
Miliary TB: Diffuse 1–2 mm nodules, "snowstorm" appearance, normal chest X-ray possible early

TREATMENT REGIMENS (WHO/NTC PAKISTAN)

New Cases (Cat I) – 6 months:
• **Intensive Phase (2 months):** HRZE (Isoniazid, Rifampicin, Pyrazinamide, Ethambutol)

• **Continuation Phase (4 months):** HR (Isoniazid, Rifampicin)
• Dosing: Daily or thrice weekly (DOTS – Directly Observed Treatment Short-course)

Previously Treated (Cat II) – 8 months:
• **Intensive Phase (3 months):** HRZES (add Streptomycin)
• **Continuation Phase (5 months):** HRE (Isoniazid, Rifampicin, Ethambutol)

Drug-Resistant TB (MDR-TB):
• Resistance to Isoniazid and Rifampicin
• Regimen: 6–9 months of fluoroquinolone (Levofloxacin/Moxifloxacin) + Bedaquiline + Linezolid + Clofazimine + Cycloserine
• XDR-TB: Resistance to fluoroquinolone + injectable

Extrapulmonary TB:
• Standard 6-month regimen (except bone/joint TB – 9 months, CNS TB – 12 months)
• Corticosteroids for TB meningitis, pericarditis (prednisolone 1–2 mg/kg tapering)

DRUG MONITORING & SIDE EFFECTS

Baseline (before starting): LFTs, CBC, creatinine, HIV test, sputum AFB x 3
Monthly: Sputum AFB (conversion check), symptom review, weight
Isoniazid (H): Peripheral neuropathy (give Pyridoxine 10 mg), hepatitis, rash
Rifampicin (R): Hepatitis, orange body fluids, enzyme induction (reduces efficacy of oral contraceptives, warfarin)
Pyrazinamide (Z): Hepatitis, hyperuricemia (gout), arthralgia
Ethambutol (E): Optic neuritis (check color vision and visual acuity monthly), red-green color blindness
Streptomycin (S): Nephrotoxicity, ototoxicity (vestibular > cochlear)
Stop drugs if: ALT >3x normal with symptoms, or >5x normal without symptoms

TREATMENT OUTCOMES & COMPLICATIONS

Cure: Treatment completed, sputum-negative at end
Treatment success: Cure + treatment completed
Failure: Positive sputum at 5 months or later
Default: Interrupted treatment >2 months
Relapse: Negative at end of treatment, then positive within 6 months
Complications: Hemoptysis (massive = emergency), pneumothorax, bronchiectasis, aspergilloma (fungus ball in cavity), respiratory failure, cor pulmonale

CRITICAL ERRORS

Wrong regimen duration (new case 6 months, retreatment 8 months)
Missing pyridoxine with isoniazid (neuropathy prevention)
No visual monitoring with ethambutol (irreversible blindness)
Not checking drug interactions (rifampicin and contraceptives/anticoagulants)
Continuing drugs despite severe hepatitis (must stop and restart sequentially)

STATION 27 – MEDICINE/PULMONOLOGY COMPREHENSIVE

Pneumonia – Comprehensive Management & Complications

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Note: Expands on Station 5 (Paeds pneumonia) and Station 17 (X-ray pneumonia) with adult focus and CURB-65 scoring.

CANDIDATE INSTRUCTIONS

8 minutes. Scenario: Adult with community-acquired pneumonia.
Assess severity using CURB-65, choose appropriate antibiotics, and manage complications.
Interpret microbiology results and know when to escalate care.

CLASSIFICATION & SEVERITY ASSESSMENT

Community-Acquired Pneumonia (CAP): Outside hospital or within 48 hours of admission
Hospital-Acquired Pneumonia (HAP): >48 hours after admission
Ventilator-Associated Pneumonia (VAP): >48 hours after intubation
Healthcare-Associated Pneumonia (HCAP): Recent hospitalization, dialysis, nursing home, IV antibiotics

CURB-65 Score (1 point each):
• **C**onfusion (new onset)
• **U**rea >7 mmol/L (or BUN >20 mg/dL)
• **R**espiratory rate ≥30/min
• **B**lood pressure (SBP <90 or DBP ≤60 mmHg)

• **65** years or older

Score interpretation:
• 0–1: Low risk (mortality <3%) – outpatient
• 2: Intermediate (mortality 9%) – short hospital stay or supervised outpatient
• 3–5: Severe (mortality 15–40%) – hospital admission, consider ICU

MICROBIOLOGY & ANTIBIOTIC SELECTION

Outpatient (low severity):
• **No comorbidities:** Amoxicillin 1 g TDS OR Doxycycline 100 mg BD OR Macrolide (Azithromycin 500 mg OD)
• **Comorbidities (COPD, heart failure, diabetes):** Amoxicillin-clavulanate 625 mg TDS + Azithromycin OR Respiratory fluoroquinolone (Levofloxacin 750 mg OD)

Inpatient (non-severe):
• Amoxicillin-clavulanate 1.2 g IV TDS + Clarithromycin 500 mg IV/PO BD
• OR Ceftriaxone 1–2 g IV OD + Clarithromycin
• OR Levofloxacin 750 mg IV/PO OD (monotherapy)

Severe/ICU (CURB-65 ≥3):
• **Standard:** Ceftriaxone 2 g IV OD + Azithromycin 500 mg IV OD
• **OR** Piperacillin-tazobactam 4.5 g IV TDS + Azithromycin
• **OR** Ceftriaxone + Ciprofloxacin (if Legionella suspected)
• **MRSA risk:** Add Vancomycin or Linezolid
• **Pseudomonas risk:** Add Piperacillin-tazobactam or Cefepime

Atypical coverage indications: Recent travel, epidemic, failure of beta-lactam, severe disease, hyponatremia (Legionella)

COMPLICATIONS MANAGEMENT

Parapneumonic effusion: Diagnostic tap (pH, glucose, LDH, culture), chest drain if complicated (pH <7.2, glucose <40 mg/dL, positive Gram stain/culture, loculated)
Empyema: Chest drain + fibrinolytics (streptokinase) or VATS decortication if organized
Lung abscess: Prolonged antibiotics (4–6 weeks), postural drainage, avoid aspiration risk
Necrotizing pneumonia: Broad-spectrum antibiotics (cover Staph, Strep, Klebsiella), consider surgery if massive necrosis
Respiratory failure: Oxygen, NIV or intubation if severe
Sepsis/Septic shock: Early antibiotics (within 1 hour), fluid resuscitation, vasopressors, source control

FOLLOW-UP & PREVENTION

Response assessment: Clinical improvement expected 48–72 hours, radiographic lag 4–6 weeks
Non-resolution: Consider resistant organism, obstruction (tumor, foreign body), wrong diagnosis (PE, vasculitis), immunocompromise
Chest X-ray: Repeat 6 weeks post-treatment (especially smokers >50 years to exclude underlying malignancy)
Vaccination: Pneumococcal (PCV13 then PPSV23, or PPSV23 alone if >65 years), Influenza annually
Smoking cessation: Most important modifiable risk factor

CRITICAL ERRORS

No severity assessment (CURB-65) – admits low-risk or discharges high-risk
Delay in antibiotics (>4 hours in admitted patients, >1 hour in sepsis)
Missing atypical coverage in severe cases
Not draining complicated effusion (medical therapy alone insufficient)
No follow-up CXR (miss underlying lung cancer)

STATION 28 – MEDICINE/PULMONOLOGY

Emphysema – Diagnosis, Classification & Management

KMU FINAL YEAR MBBS

CANDIDATE INSTRUCTIONS

8 minutes. Scenario: Patient with dyspnea, smoking history, hyperinflated chest.
Differentiate emphysema from chronic bronchitis, interpret spirometry, and outline management.
Identify signs of respiratory failure and cor pulmonale.

DEFINITION & PATHOPHYSIOLOGY

Emphysema: Permanent enlargement of airspaces distal to terminal bronchioles with destruction of alveolar walls without obvious fibrosis
Centriacinar (Centrilobular): Central portion of acinus involved, upper lobe predominance, associated with smoking
Panacinar (Panlobular): Entire acinus involved, lower lobe predominance, associated with alpha-1 antitrypsin deficiency
Paraseptal: Distal acinus, subpleural, associated with bullae and pneumothorax
Pathophysiology: Loss of elastic recoil, airway collapse during expiration, air trapping, hyperinflation, V/Q mismatch, decreased DLCO

CLINICAL FEATURES & DIFFERENTIATION

Emphysema ("Pink Puffer"):
• Thin, barrel chest, pursed-lip breathing
• Minimal cough/sputum
• Severe dyspnea, late CO2 retention
• Tripod position, use of accessory muscles
• Quiet breath sounds, prolonged expiration
• Hypoxemia late, cor pulmonale late
• Chest X-ray: Hyperinflation, flat diaphragms, bullae, small heart

Chronic Bronchitis ("Blue Bloater"):
• Overweight, edematous, cyanotic
• Productive cough (3 months x 2 years)
• Early CO2 retention, polycythemia
• Frequent infections, cor pulmonale early
• Rhonchi, wheeze
• Chest X-ray: Prominent broncho-vascular markings, cardiomegaly

Overlap common: Most patients have features of both (COPD spectrum)

DIAGNOSTIC WORKUP

Spirometry (GOLD classification):
• **FEV1/FVC <0.70** confirms obstruction
• **Stage I (Mild):** FEV1 ≥80% predicted
• **Stage II (Moderate):** FEV1 50–79%
• **Stage III (Severe):** FEV1 30–49%
• **Stage IV (Very Severe):** FEV1 <30% or <50% with respiratory failure
• **Reversibility testing:** <12% improvement post-bronchodilator (vs asthma)

Imaging:
• Chest X-ray: Hyperinflation (flat diaphragms <1.5 cm from dome to sternum on lateral), bullae, small vertical heart
• CT chest: Emphysema quantification, bullae, lung cancer screening

ABG: Hypoxemia, hypercapnia (late), respiratory acidosis
Alpha-1 antitrypsin level: If young age, basilar predominance, family history
6-minute walk test: Exercise capacity, desaturation

MANAGEMENT

Smoking cessation: Most effective intervention (slows decline in FEV1)
Bronchodilators:
• SABA (Salbutamol) PRN
• LAMA (Tiotropium) – first line maintenance
• LABA (Salmeterol/Formoterol)
• Combination LABA/LAMA preferred over single agent
Inhaled corticosteroids: If frequent exacerbations (>2/year) or eosinophilic
Roflumilast: Phosphodiesterase-4 inhibitor if severe COPD with chronic bronchitis
Oxygen therapy: Long-term if PaO2 ≤55 mmHg or SpO2 ≤88% (or ≤89% with cor pulmonale/polycythemia) – >15 hours/day
Pulmonary rehabilitation: Exercise training, nutrition, education
Vaccination: Influenza, pneumococcal, COVID-19
Surgical: Lung volume reduction surgery (upper lobe emphysema, low exercise capacity), Bullectomy (giant bullae), Lung transplantation (very severe)
Acute exacerbation: Bronchodilators, systemic steroids (prednisolone 40 mg 5 days), antibiotics (if 2+ Anthonisen criteria), oxygen (controlled), NIV if hypercapnic

COMPLICATIONS & PROGNOSIS

Respiratory failure: Type II (hypoxemia + hypercapnia)
Cor pulmonale: Right heart failure secondary to pulmonary hypertension
Pneumothorax: Rupture of bullae (tension risk)
Lung cancer: Risk increased 4–5 fold
Osteoporosis: Especially with chronic steroid use
Anxiety/Depression: Common, treatable
BODE Index: BMI, Obstruction, Dyspnea, Exercise capacity – predicts mortality better than FEV1 alone

CRITICAL ERRORS

Confusing with asthma (irreversible vs reversible, age of onset)
Oxygen without checking CO2 (risk of CO2 narcosis)
Missing alpha-1 antitrypsin deficiency (young patient, basilar emphysema)
Not emphasizing smoking cessation (only intervention that modifies disease progression)
Overtreating with steroids (limited role in pure emphysema without bronchitic component)

STATION 29 – MEDICINE/CARDIOLOGY

Pericarditis – History Taking, Diagnosis & Management

KMU FINAL YEAR MBBS

CANDIDATE INSTRUCTIONS

8 minutes. Take history from patient with chest pain suspicious for pericarditis.
Identify key diagnostic features, interpret ECG, and outline management.
Differentiate from myocardial infarction and know when to drain.

HISTORY TAKING – KEY FEATURES

Chest pain characteristics:
• **Quality:** Sharp, pleuritic, stabbing
• **Location:** Retrosternal, left precordial, radiates to trapezius (specific for pericarditis)
• **Position:** Worse supine, better sitting up and leaning forward (pathognomonic)
• **Respiration:** Worse with inspiration (pleuritic)
• **Swallowing:** May worsen (esophageal irritation)

Associated symptoms: Fever, malaise, dyspnea (tamponade), palpitations
Recent history: Viral illness (URI, gastroenteritis), MI (Dressler's syndrome), surgery, trauma, TB exposure, autoimmune disease, malignancy, anticoagulation
Rule out MI: No relief with GTN, positional variation, pleuritic nature

DIAGNOSIS & INVESTIGATIONS

Physical examination:
• Pericardial friction rub (scratchy, triphasic – atrial systole, ventricular systole, early diastole)
• Best heard at left lower sternal border with patient leaning forward, held expiration
• May be transient (comes and goes)

ECG (4 stages):
• **Stage 1 (hours-days):** Diffuse concave ST elevation (limb leads, V2–V6), PR segment depression (aVR shows opposite – ST depression, PR elevation)
• **Stage 2:** Normalization of ST and PR
• **Stage 3:** Diffuse T wave inversion
• **Stage 4:** Normalization or persistent T wave changes
• **Key differentiator from MI:** No reciprocal ST depression (except aVR, V1), no Q waves, no localized territory

Echocardiogram: Pericardial effusion (size, hemodynamic significance), tamponade signs (RA/RV collapse, respiratory variation)
CXR: Enlarged cardiac silhouette (water bottle heart) if effusion >250 mL
Lab: ESR/CRP (elevated), troponin (may be mildly elevated if myopericarditis), CBC, renal function
CT/MRI: Pericardial thickening, effusion, constriction

ETIOLOGY & MANAGEMENT

Common causes:
• Idiopathic/viral (Coxsackie B, Echo, Adenovirus) – 80–90%
• Post-MI (Dressler's syndrome)
• Autoimmune (SLE, rheumatoid arthritis)
• Uremia (chronic kidney disease)
• TB (constrictive pericarditis risk)
• Malignancy (lung, breast, lymphoma)
• Bacterial (purulent – emergency)

Management:
• **NSAIDs:** Ibuprofen 600–800 mg TDS or Aspirin 750–1000 mg TDS (2–4 weeks, then taper)
• **Colchicine:** 0.5 mg BD (reduces recurrence, first line adjunct)
• **Rest:** Avoid strenuous activity until resolution
• **Treat underlying cause:** Dialysis (uremia), anti-TB, chemotherapy
• **Glucocorticoids:** Only if contraindication to NSAIDs or autoimmune etiology (prednisolone 0.5 mg/kg/day, taper slowly – risk of recurrence)
• **Pericardiocentesis:** Cardiac tamponade, large effusion with hemodynamic compromise, suspected purulent or TB pericarditis
• **Pericardiectomy:** Recurrent pericarditis refractory to medical therapy, constrictive pericarditis

COMPLICATIONS

Pericardial effusion: Can be asymptomatic or lead to tamponade
Cardiac tamponade: Beck's triad (hypotension, elevated JVP, muffled heart sounds), pulsus paradoxus (>10 mmHg drop in SBP with inspiration), electrical alternans on ECG – EMERGENCY pericardiocentesis
Constrictive pericarditis: Chronic inflammation → fibrosis → impaired diastolic filling (prominent x and y descents, Kussmaul's sign, pericardial knock)
Recurrent pericarditis: 15–30% recurrence rate, requires prolonged colchicine therapy

CRITICAL ERRORS

Confusing with MI (no reciprocal changes, PR depression, positional pain)
Missing tamponade signs (pulsus paradoxus, electrical alternans)
Giving steroids first line (NSAIDs + colchicine preferred)
No colchicine (increases recurrence risk)
Delay in pericardiocentesis when tamponade present

Crafted with ❤️ Noaman khan Musakhel

STATION 30 – MEDICINE

Chest Pain History Taking – Differential Diagnosis

KMU FINAL YEAR MBBS

CANDIDATE INSTRUCTIONS

8 minutes. Take comprehensive history from patient presenting with chest pain.
Characterize pain to differentiate cardiac, pulmonary, GI, musculoskeletal, and psychiatric causes.
Identify red flags and formulate differential diagnosis.

STRUCTURED HISTORY (SOCRATES + RISK FACTORS)

S – Site: Retrosternal (cardiac, esophageal), lateral (pleuritic, musculoskeletal), epigastric (GI, inferior MI)
O – Onset: Sudden (PE, pneumothorax, dissection), gradual (angina, pneumonia), after meal (GI)
C – Character: Crushing/pressure (ischemia), tearing (dissection), sharp/stabbing (pleuritic, pericarditis), burning (reflux)
R – Radiation: Left arm/jaw (cardiac), back (dissection, pancreatitis), shoulder (diaphragmatic irritation)
A – Associated symptoms: Dyspnea (cardiac, pulmonary), sweating/nausea (MI), cough/fever (pneumonia), hemoptysis (PE)
T – Time course: Seconds (musculoskeletal), minutes (angina), hours (MI, PE), continuous (pericarditis)
E – Exacerbating/Relieving factors: Exercise (angina), meals (GI), position (pericarditis), breathing (pleuritic)
S – Severity: 0–10 scale

Risk factors: Smoking, hypertension, diabetes, hyperlipidemia, family history (cardiac), DVT/PE history, malignancy, recent surgery/immobility (PE), Marfan syndrome (dissection)

DIFFERENTIAL DIAGNOSIS BY SYSTEM

CARDIAC (Life-threatening):
• **Acute coronary syndrome:** Pressure-like, exertional, radiates to arm/jaw, associated dyspnea/diaphoresis/nausea
• **Aortic dissection:** Tearing, sudden onset, radiates to back, unequal pulses, hypertension/Marfan
• **Pericarditis:** Sharp, pleuritic, positional (better leaning forward), friction rub
• **Myocarditis:** Recent viral illness, chest pain with heart failure signs
• **Takotsubo cardiomyopathy:** Stress-induced, post-menopausal women

PULMONARY (Life-threatening):
• **Pulmonary embolism:** Pleuritic, dyspnea, tachypnea, risk factors (DVT, immobility, cancer), hemoptysis
• **Pneumothorax:** Sudden unilateral pleuritic pain, dyspnea, reduced breath sounds, hyperresonance
• **Pneumonia:** Pleuritic, fever, cough, productive sputum
• **Lung cancer:** Chronic, constitutional symptoms, smoking history

GASTROINTESTINAL:
• **GERD:** Burning, post-prandial, supine worsening, water brash, relief with antacids
• **Esophageal spasm:** Crushing, may radiate to arm (angina mimic), triggered by cold liquids
• **Peptic ulcer disease:** Epigastric, related to meals, nocturnal pain
• **Pancreatitis:** Severe epigastric, radiates to back, vomiting, alcohol/gallstones
• **Biliary colic:** RUQ/epigastric, post-fatty meal, radiates to right shoulder

MUSCULOSKELETAL:
• **Costochondritis:** Localized, reproducible with palpation, sharp
• **Muscle strain:** Related to movement, reproducible
• **Rib fracture:** History of trauma, localized tenderness, crepitus
• **Herpes zoster:** Dermatomal, burning, vesicular rash (may precede rash)

PSYCHIATRIC:
• **Panic attack/anxiety:** Sharp, associated with hyperventilation, paresthesia, sense of doom, multiple somatic complaints

RED FLAGS – IMMEDIATE ATTENTION

Cardiac: Hypotension, diaphoresis, radiation to arm/jaw, elevated troponin, ST changes
Aortic dissection: Tearing pain, pulse deficit, neurological deficits, widened mediastinum on CXR
PE: Hemodynamic instability, hypoxemia, massive DVT

Pneumothorax: Respiratory distress, tracheal deviation (tension)
Esophageal rupture: Severe vomiting followed by chest pain, subcutaneous emphysema, mediastinal air

CRITICAL ERRORS

Missing atypical MI presentation (elderly, diabetics, women may have dyspnea without pain)
Not assessing risk factors (determines pre-test probability)
Anchoring bias (fixating on GERD in high-risk cardiac patient)
No safety netting (telling patient to return if red flags develop)
Incomplete SOCRATES (misses key differentiating features)

STATION 31 – PULMONOLOGY

TB with Pleural Effusion – Light's Criteria & Advanced Diagnostics

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Note: Expands on Station 16 (Pleural effusion) and Station 26 (TB treatment) with specific TB pleuritis focus.

CANDIDATE INSTRUCTIONS

8 minutes. Scenario: Patient with TB and pleural effusion.
Apply Light's criteria, interpret pleural fluid analysis, and outline management.
Know when to suspect TB pleuritis vs parapneumonic effusion.

TB PLEURAL EFFUSION CHARACTERISTICS

Pathophysiology: Hypersensitivity reaction to mycobacterial proteins (delayed type IV), paucibacillary (few organisms in fluid)
Clinical: Subacute onset, pleuritic chest pain, fever, night sweats, weight loss, dyspnea
Chest X-ray: Unilateral (80%), small to moderate, may have underlying parenchymal TB (30%)
Ultrasound: Septations common (loculated), may need imaging-guided tap
Natural history: Often resolves spontaneously but 65% develop active TB within 5 years if not treated

LIGHT'S CRITERIA & PLEURAL FLUID ANALYSIS

TB pleural fluid = EXUDATE (meets Light's criteria)
• Protein >30 g/L (usually 40–60 g/L)
• LDH >200 U/L (usually >500 U/L)
• Protein ratio >0.5, LDH ratio >0.6

Characteristic findings in TB pleuritis:
• **Appearance:** Straw-colored, serous, occasionally hemorrhagic
• **Cell count:** Lymphocyte predominant (>50%, often >90%), mesothelial cells <5% (suggests TB)
• **Protein:** >40 g/L
• **Glucose:** Low (<60 mg/dL) in 20%, normal in 80%
• **pH:** Usually >7.30 (if <7.20, consider empyema)
• **ADA (Adenosine Deaminase):** >40–50 U/L (high sensitivity and specificity for TB in high-prevalence areas)
• **IFN-γ (Interferon-gamma):** High sensitivity/specificity (expensive)
• **PCR (Xpert MTB/RIF):** Rapid, detects rifampicin resistance
• **AFB smear:** Positive in only 10–20% (paucibacillary)
• **Culture:** Positive in 25–50% (gold standard, takes 2–6 weeks)
• **Pleural biopsy:** Histology (granulomas) positive in 50–80%, culture positive in additional 25%

DIFFERENTIAL DIAGNOSIS

Lymphocytic exudate differential:
• TB (most common in endemic areas)
• Malignancy (lung, mesothelioma, lymphoma)
• Connective tissue disease (RA, SLE)
• Yellow nail syndrome
• Chylothorax
• Post-cardiac injury syndrome

Differentiating TB from malignancy:
• ADA high in TB, low in malignancy
• Cytology positive in malignancy
• Pleural biopsy showing granulomas (TB) vs malignant cells
• Elevated tumor markers (CEA, CYFRA 21-1) in malignancy

MANAGEMENT

Anti-TB treatment: Standard 6-month regimen (HRZE 2 months, HR 4 months)
• Same as pulmonary TB (effusion is paucibacillary but represents active disease)
• **Corticosteroids:** Prednisolone 0.5–1 mg/kg/day for 2–4 weeks then taper (reduces symptoms, speeds absorption, may reduce pleural thickening – controversial)
• **Therapeutic thoracentesis:** For symptomatic relief of dyspnea (large effusions)
• **Chest drain:** Not routine, only if loculated or complicated
• **Intrapleural fibrinolytics:** If septated/loculated (streptokinase)
• **Surgery (decortication):** Rarely needed for thick fibrous peel restricting lung expansion

Follow-up: Repeat CXR monthly (may take 2–4 months to resolve), monitor for development of active pulmonary TB

CRITICAL ERRORS

Relying on AFB smear alone (negative in 80%, need ADA/biopsy/PCR)
Not treating as active TB (high risk of progression)
Missing malignancy (always consider in lymphocytic exudate, especially >40 years, smoking history)
Routine chest drain (not needed unless complicated)
No follow-up for pulmonary TB development

STATION 32 – MEDICINE/PULMONOLOGY

Type 2 Respiratory Failure – ABG Interpretation & Management

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CANDIDATE INSTRUCTIONS

8 minutes. Scenario: Patient with COPD exacerbation and altered consciousness.
Interpret ABG, identify Type 2 respiratory failure, and outline management including NIV.
Know when to intubate and when to use controlled oxygen.

ABG INTERPRETATION – TYPE 2 RF

Definition: PaO_2 <60 mmHg (hypoxemia) AND $PaCO_2$ >45 mmHg (hypercapnia)
pH status:
• **Acute:** pH <7.35, normal or slightly elevated bicarbonate (no renal compensation yet)
• **Chronic:** pH near normal (7.35–7.40), elevated bicarbonate (>28 mmol/L) – renal compensation
• **Acute-on-chronic:** pH <7.35 with elevated bicarbonate (decompensated)

Example ABG:
• pH 7.25, $PaCO_2$ 65 mmHg, PaO_2 55 mmHg, HCO_3 28 mmol/L, BE +4
• Interpretation: Acute-on-chronic Type 2 RF (pH low despite compensation)

Key values to remember:
• Normal PaO_2: 80–100 mmHg (on room air)
• Normal $PaCO_2$: 35–45 mmHg
• Normal pH: 7.35–7.45
• Normal HCO_3: 22–28 mmol/L

CAUSES OF TYPE 2 RESPIRATORY FAILURE

Airway disease: COPD (most common), severe asthma, upper airway obstruction
Parenchymal disease: Severe pneumonia, pulmonary edema, pulmonary fibrosis (end-stage)
Chest wall/pleural: Kyphoscoliosis, flail chest, massive pleural effusion, pneumothorax
Neuromuscular: Guillain-Barré, myasthenia gravis, motor neuron disease, polio, drug overdose (opioids, benzodiazepines)
Central: Brainstem stroke, central sleep apnea, obesity hypoventilation syndrome
Mechanism: Alveolar hypoventilation (reduced tidal volume or respiratory rate) → decreased minute ventilation → CO_2 retention and hypoxemia

CLINICAL FEATURES

Hypoxemia: Dyspnea, cyanosis, tachycardia, confusion, agitation
Hypercapnia:
• **Acute:** Headache, confusion, drowsiness, asterixis (flapping tremor), papilledema, sweating, tachycardia, hypertension
• **Chronic:** "CO_2 retainers" – less symptoms due to renal compensation, may have peripheral edema (cor pulmonale), polycythemia
Physical signs: Bounding pulse, warm peripheries (vasodilation from CO_2), confusion, papilledema (severe), use of accessory muscles

MANAGEMENT

1. Controlled Oxygen Therapy (CRITICAL):
• **Target SpO2:** 88–92% (not 94–98% like other patients)
• **Rationale:** High flow oxygen suppresses hypoxic drive (these patients rely on hypoxemia to stimulate breathing)
• **Method:** Venturi mask (24% or 28%) or nasal cannula 1–2 L/min
• **Monitor:** ABG 30–60 minutes after starting oxygen
• **If CO2 rises but pH maintained:** Continue, increase ventilation support
• **If CO2 rises and pH drops (<7.25):** NIV or intubation

2. Non-Invasive Ventilation (NIV):
• **Indications:** Acidosis (pH 7.25–7.35) + hypercapnia (PaCO2 >45) despite optimal medical therapy
• **Contraindications:** Facial trauma, vomiting, severe agitation, hemodynamic instability, copious secretions, pneumothorax
• **Settings (BiPAP):** IPAP 10–15 cm H2O, EPAP 4–5 cm H2O, titrate to tidal volume and patient comfort
• **Benefits:** Reduces intubation rate by 50%, reduces mortality
• **Monitor:** ABG 1–2 hours after starting, clinical improvement (RR, accessory muscle use)

3. Treat underlying cause:
• COPD exacerbation: Bronchodilators, steroids, antibiotics
• Pneumonia: Antibiotics
• Opioid overdose: Naloxone
• Pneumothorax: Chest drain

4. Intubation criteria:
• pH <7.25 despite NIV
• Severe hypoxemia (PaO2 <40 despite high FiO2)
• Altered consciousness (GCS <8)
• Hemodynamic instability
• Respiratory arrest
• Inability to protect airway

CRITICAL ERRORS

High-flow oxygen in known CO2 retainer (causes CO2 narcosis, respiratory acidosis, death)
Not checking ABG after oxygen (miss rising CO2)
Delaying NIV (worsens acidosis, increases intubation risk)
Wrong target SpO2 (94–98% in COPD Type 2 RF is dangerous)
Intubating too early (NIV first if criteria met) or too late (cardiac arrest)

STATION 33 – PAEDIATRICS

Congestive Heart Failure in Children – Diagnosis, Investigations & Management

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Note: Pediatric-specific CHF station overlapping with adult CHF (Station 10) but with age-appropriate differences.

CANDIDATE INSTRUCTIONS

8 minutes. Scenario: Infant with feeding difficulties, tachypnea, and hepatomegaly.
Diagnose CHF, interpret ECG findings, and outline management plan.
Differentiate from sepsis and pneumonia.

PEDIATRIC CHF – UNIQUE FEATURES

Etiology differs by age:
• **Neonate:** Structural heart disease (HLHS, coarctation, large VSD), arrhythmia, myocarditis, metabolic disorders
• **Infant:** Large L→R shunts (VSD, PDA, AVSD), cardiomyopathy, myocarditis
• **Child:** Rheumatic heart disease, cardiomyopathy, myocarditis, hypertension
• **Adolescent:** Similar to adults (cardiomyopathy, arrhythmia)

Presentation differs from adults:
• **Infants:** Poor feeding, diaphoresis with feeds, irritability, failure to thrive, tachypnea, recurrent chest infections
• **No orthopnea** (infants can't describe)
• **Hepatomegaly** often first sign (liver edge >2 cm below costal margin)
• **Tachycardia** persistent (sleeping HR >160 in infants, >120 in children)
• **Gallop rhythm** (S3 common in children, but S3 + symptoms = CHF)

CLINICAL ASSESSMENT – ROSS CLASSIFICATION

Class I (Asymptomatic): No symptoms with ordinary activity
Class II (Mild): Tachypnea/diaphoresis with feeding in infants, dyspnea with moderate exertion in older children
Class III (Moderate): Tachypnea/diaphoresis with minimal feeding, marked growth failure, hepatomegaly
Class IV (Severe): Symptoms at rest, tachypnea, retractions, diaphoresis, pallor

Key signs to elicit:
• Tachycardia (out of proportion to fever)
• Tachypnea (RR >60 in <2 months, >50 in 2–12 months, >40 in 1–5 years)
• Hepatomegaly (essential sign – measure span)
• Gallop rhythm (S3)
• Poor peripheral perfusion (capillary refill >2 seconds)
• Edema (sacral in infants, peripheral in older children)
• Weight gain (fluid retention) despite poor feeding

INVESTIGATIONS

Chest X-ray: Cardiomegaly (CTR >0.55 in infants, >0.50 in older children), pulmonary edema, pleural effusion, specific cardiac silhouette (boot-shaped in TOF, egg-on-string in TGA, snowman in TAPVR)
ECG: Chamber enlargement (RVH, LVH), arrhythmia, ischemia, conduction abnormalities
Echocardiogram: Gold standard – structural defects, chamber size, function (EF, FS), valve disease, estimate pulmonary pressure
BNP/NT-proBNP: Elevated in CHF (diagnostic and prognostic)
CBC: Anemia (exacerbates CHF), polycythemia (cyanotic CHD)
Electrolytes, renal function: Baseline before diuretics
Blood culture: If sepsis suspected
ABG: If respiratory distress severe

MANAGEMENT

Acute/severe CHF:
• **Position:** Semi-upright (reduces preload)
• **Oxygen:** Cautiously (worsens L→R shunt in some CHD), target SpO2 80–85% in single ventricle physiology
• **Diuretics:** Furosemide 1 mg/kg IV/PO (reduces preload)
• **Inotropes:** Digoxin (maintenance), Dobutamine/Dopamine (acute)
• **Afterload reduction:** Captopril (ACEi), Sodium nitroprusside (severe)
• **Fluid restriction:** 2/3 maintenance
• **Caloric support:** High-calorie feeds (NG if needed)

Chronic CHF:
• **Diuretics:** Furosemide ± Spironolactone
• **ACEi:** Captopril or Enalapril (afterload reduction)
• **Digoxin:** Improves contractility (monitor levels)
• **Beta-blockers:** Carvedilol (stable patients)
• **High-calorie diet:** 120–150 kcal/kg/day
• **Surgical:** Repair of structural defects (timing critical)

Specific scenarios:
• **Duct-dependent lesions:** Prostaglandin E1 (alprostadil) to maintain PDA
• **Coarctation:** Prostaglandin + inotropes until surgery
• **Myocarditis:** Supportive, immunoglobulins, avoid digoxin if severe

ECG FINDINGS IN PEDIATRIC CHF

Left ventricular hypertrophy (LVH): Tall R waves in V5–V6, deep S waves in V1, left axis deviation
Right ventricular hypertrophy (RVH): Tall R waves in V1, right axis deviation (>+90° in >1 month old)
Biventricular hypertrophy: Combined criteria
Strain patterns: ST-T changes with hypertrophy (ischemia)
Arrhythmias: SVT, complete heart block (myocarditis), VT (cardiomyopathy)
Ischemia: Flat/inverted T waves, ST depression (anomalous left coronary artery)
Specific patterns: LVH in VSD, RVH in TOF/PS, biventricular in AVSD

CRITICAL ERRORS

Missing hepatomegaly (essential sign in infants)
Attributing tachypnea to pneumonia (CHF mimics respiratory illness)
Liberal oxygen in cyanotic CHD (reduces pulmonary blood flow)
No prostaglandin in duct-dependent lesion (death if duct closes)
Fluid restriction without calorie increase (worsens failure to thrive)

STATION 34 – MEDICINE/EMERGENCY

Tension Pneumothorax – Immediate & Definitive Management

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CANDIDATE INSTRUCTIONS

8 minutes. Scenario: Trauma patient with respiratory distress, hypotension, and absent breath sounds.
Recognize tension pneumothorax, perform immediate decompression, and arrange definitive management.
Do NOT wait for X-ray.

CLINICAL DIAGNOSIS – DO NOT WAIT FOR IMAGING

Mechanism: One-way valve air leak into pleural space → increasing pressure → lung collapse → mediastinal shift → cardiovascular collapse
Classic triad (late signs):
1. **Severe respiratory distress** (tachypnea, cyanosis)
2. **Hypotension/shock** (obstructed venous return)
3. **Unilateral absent breath sounds** (affected side)

Additional signs:
• Tracheal deviation (away from affected side) – late sign, unreliable
• Hyperresonance to percussion (affected side)
• Distended neck veins (JVD) – impaired venous return
• Subcutaneous emphysema
• Tympanic percussion note
• Cyanosis (late)

Causes: Trauma (rib fracture, penetrating), iatrogenic (central line, thoracentesis, barotrauma from ventilation), asthma/COPD (alveolar rupture), mechanical ventilation

IMMEDIATE MANAGEMENT – EMERGENCY DECOMPRESSION

Do NOT wait for chest X-ray (clinical diagnosis, delay = death)
1. Needle decompression (finger thoracostomy if trained):
• **Location:** 2nd intercostal space, mid-clavicular line (traditional)
• **OR 4th–5th intercostal space, anterior axillary line** (preferred – same location as chest tube, avoids heart/great vessels)
• **Needle:** 14–16 gauge, 4.5–5 cm long (standard IV cannula often too short in obese/muscular patients)
• **Technique:** Insert over superior border of rib (avoid neurovascular bundle), perpendicular to chest wall, remove needle leaving catheter, listen for rush of air
• **Confirmation:** Hissing sound of escaping air, clinical improvement (BP rises, SpO2 improves)
• **Leave catheter in place** (may kink, may need repeat)

2. High-flow oxygen: 15 L/min via non-rebreather mask
3. IV access, fluid resuscitation: If hypotensive (obstructed venous return)
4. Prepare for chest tube: While needle decompression performed

DEFINITIVE MANAGEMENT – CHEST TUBE INSERTION

Indication: All tension pneumothoraces require chest tube after stabilization
Size: Large bore (28–36 Fr in adults) – may need larger if hemothorax also present
Location: 5th intercostal space, mid-axillary line (triangle of safety)
Technique: Open (surgical) technique preferred in trauma (finger sweep to ensure lung not adhered, assess for ongoing bleeding)
Connect to: Underwater seal drain with suction (initially)
Monitor: Bubbling (air leak), swing (tidaling), drainage
CXR: After insertion to confirm position and lung re-expansion
ICU admission: All patients with tension pneumothorax require observation

SPECIAL CONSIDERATIONS

Bilateral tension pneumothorax: Rare, catastrophic, no mediastinal shift, bilateral absent sounds, decompress both sides
Open pneumothorax (sucking chest wound): Cover with occlusive dressing taped on 3 sides (flutter valve), then chest tube
Ventilated patients: Reduce tidal volume, check for barotrauma, may need bilateral decompression if unclear side
Occult tension: Hypotension + high airway pressures in ventilated patient = tension until proven otherwise
Simple pneumothorax progression: Any pneumothorax can become tension, especially with positive pressure ventilation

CRITICAL ERRORS

Waiting for chest X-ray (clinical diagnosis, delay = cardiac arrest)
Needle too short (obese patients – use longer needle or alternative site)
Wrong anatomical location (below nipple = abdominal organs)
Not inserting chest tube after needle decompression (temporary measure only)
Missing bilateral tension (no tracheal deviation, both sides need decompression)
Removing needle from catheter (may need to leave catheter, kinking risk)

EQUIPMENT

14–16 gauge IV cannula (long, or specific pneumothorax needle)
Chest tube insertion tray (scalpel, forceps, tubing)
Underwater seal drain

High-flow oxygen
Monitoring equipment (BP, SpO2, ECG)

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STATION 35 – MEDICINE/CARDIOLOGY

Cardiac Failure – Chest X-ray Findings & Correlation

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> **Note:** Specific X-ray findings station complementing clinical CHF stations.

CANDIDATE INSTRUCTIONS

8 minutes. Interpret chest X-ray of patient with heart failure.
Identify specific findings, grade severity, and correlate with clinical status.
Differentiate acute pulmonary edema from chronic cardiomegaly.

CHEST X-RAY FINDINGS IN CHF

Cardiomegaly:
• Cardiothoracic ratio (CTR) >0.50 (PA film, good inspiration)
• Left ventricular enlargement: Apex displaced down and left, rounded
• Left atrial enlargement: Straightening of left heart border, double density sign, splayed carina (>90°)
• Right ventricular enlargement: Uplifted apex, prominent right heart border
• Right atrial enlargement: Prominent right heart border >50% of cardiac height

Pulmonary venous hypertension (cephalization):
• Upper lobe diversion (upper lobe veins > lower lobe veins in diameter)
• Peribronchial cuffing (fluid around bronchi)
• Kerley B lines (1–2 cm horizontal lines at costophrenic angles – interstitial edema)
• Kerley A lines (radiating from hilum, less common)
• Hilar haziness/perihilar edema (bat wing/butterfly pattern in severe)

Alveolar edema:
• Bilateral, symmetric, perihilar opacities (bat wing appearance)
• Air bronchograms (rare, transient)
• Pleural effusions (bilateral, right > left, blunting costophrenic angles)
• Fluid in fissures (thickening or pseudotumor)

Other findings:
• Pulmonary artery enlargement (pulmonary hypertension)
• Calcified valves (rheumatic)
• Sternotomy wires (previous CABG/valve surgery)
• Pacemaker/ICD leads
• Aortic unfolding (hypertension, age)

SEVERITY GRADING ON X-RAY

Mild (Stage I): Cephalization only, CTR may be normal
Moderate (Stage II): Interstitial edema (Kerley B lines, peribronchial cuffing), mild cardiomegaly
Severe (Stage III): Alveolar edema (bat wing), large effusions, marked cardiomegaly
Acute vs Chronic:
• **Acute pulmonary edema:** Normal heart size, bat wing opacities (flash pulmonary edema – diastolic dysfunction, MR)
• **Chronic failure:** Marked cardiomegaly, redistribution, effusions, less dramatic alveolar edema

DIFFERENTIAL DIAGNOSIS OF X-RAY PATTERNS

Bat wing appearance: CHF, ARDS, severe pneumonia, pulmonary hemorrhage, inhalational injury
Differentiation:
• **CHF:** Cardiomegaly, effusions, Kerley lines, upper lobe diversion
• **ARDS:** Normal heart size, peripheral distribution, no effusions/Kerley lines
• **Pneumonia:** Unilateral or lobar, air bronchograms, fever
• **Pulmonary hemorrhage:** Rapidly changing, hemoptysis, normal heart size

Unilateral edema: Consider re-expansion pulmonary edema, unilateral aspiration, pulmonary contusion, unilateral venous obstruction

CORRELATION WITH CLINICAL STATUS

X-ray lag: Clinical improvement precedes radiographic clearance by 24–48 hours
Persistent congestion: Poor diuretic response, ongoing ischemia, valvular dysfunction

Clear lungs with elevated JVP: Right heart failure, constrictive pericarditis, tamponade
Pleural effusions: May persist weeks after clinical improvement
Follow-up CXR: After acute episode to establish baseline, assess for underlying lung disease, check pacemaker/lead position

CRITICAL ERRORS

Calling cardiomegaly on AP film (magnification artifact – need PA)
Missing Kerley B lines (early sign, subtle)
Confusing ARDS with CHF (heart size key differentiator)
Not assessing film quality (rotation, inspiration, penetration)
Attributing all opacities to edema (may have superimposed pneumonia)

STATION 36 – CARDIOLOGY

Atrial Fibrillation – ECG Diagnosis & Management

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CANDIDATE INSTRUCTIONS

8 minutes. Analyze ECG showing atrial fibrillation.
Classify type, assess stroke risk, and outline rate vs rhythm control strategies.
State anticoagulation indications and emergency management if unstable.

ECG DIAGNOSIS

Rhythm: Irregularly irregular R-R intervals
P waves: Absent – replaced by fibrillatory waves (f waves) – fine (low amplitude) or coarse (high amplitude)
Baseline: Undulating, chaotic, low-amplitude oscillations (best seen in V1, II, III)
QRS: Narrow (<120 ms) unless pre-existing bundle branch block or aberrant conduction (Ashman phenomenon)
Ventricular rate: Variable (controlled <100, uncontrolled 100–150, rapid >150)
Differentiation from flutter: AF has chaotic baseline, flutter has regular sawtooth pattern
Differentiation from multifocal atrial tachycardia (MAT): MAT has organized P waves (≥3 morphologies), regular P-P intervals, usually COPD patient

CLASSIFICATION

First diagnosed: First episode (regardless of duration or symptoms)
Paroxysmal: Self-terminating within 7 days (usually <48 hours)
Persistent: >7 days or requires cardioversion
Long-standing persistent: >12 months
Permanent: Accepted, no further attempts to restore sinus rhythm
Valvular vs Non-valvular: Valvular = moderate-severe mitral stenosis or mechanical prosthetic valve (affects anticoagulation choice)

STROKE RISK STRATIFICATION (CHA_2DS_2-VASc)

Congestive heart failure – 1 point
Hypertension – 1 point
Age ≥75 – 2 points
Diabetes – 1 point
Stroke/TIA/thromboembolism – 2 points
Vascular disease (MI, PAD, aortic plaque) – 1 point
Age 65–74 – 1 point
Sex category (female) – 1 point

Score interpretation:
• 0 (male) or 1 (female): No anticoagulation (or consider if 1 in male)
• ≥2 (male) or ≥3 (female): Oral anticoagulation indicated
• **Options:** DOACs (Apixaban, Rivaroxaban, Dabigatran, Edoxaban) preferred over Warfarin (unless mechanical valve or moderate-severe MS)
• **HAS-BLED score:** Assess bleeding risk, but don't withhold anticoagulation solely due to bleeding risk (most benefit > risk)

MANAGEMENT STRATEGIES

1. Rate control (majority of patients):
• Target resting HR <110 bpm (lenient) or <80 bpm (strict)
• **Beta-blockers:** Metoprolol, Bisoprolol (first line, especially if CAD)
• **Non-DHP Calcium channel blockers:** Diltiazem, Verapamil (avoid if HFrEF)
• **Digoxin:** Second line, good in sedentary elderly, heart failure
• **Amiodarone:** If others contraindicated (rhythm control properties too)

2. Rhythm control (selected patients):
• Symptomatic despite rate control
• Difficult to rate control
• Younger age, first presentation
• AF with heart failure (improves symptoms)
• **Methods:** Electrical cardioversion, Flecainide (pill-in-pocket for paroxysmal), Amiodarone, Sotalol, Catheter ablation (symptomatic paroxysmal first line)
• **Anticoagulation:** Required for 3 weeks before and 4 weeks after cardioversion (unless AF <48 hours or TEE shows no thrombus)

3. Emergency management (unstable):
• **Unstable signs:** Hypotension (SBP <90), acute heart failure, ongoing ischemia, altered consciousness
• **Immediate synchronized cardioversion:** 100–200 J biphasic (sedate if conscious)
• **Heparin:** IV unfractionated if not already anticoagulated
• **If stable but rapid:** Rate control with IV beta-blocker (esmolol, metoprolol) or diltiazem

4. Stroke prevention:
• Anticoagulation as per CHA_2DS_2-VASc (see above)
• Left atrial appendage occlusion (if contraindication to long-term anticoagulation)

SPECIAL SCENARIOS

WPW with AF: Wide complex, irregular, very rapid – AVOID AV nodal blockers (digoxin, verapamil, diltiazem, beta-blockers) – use Procainamide or Ibutilide, or cardiovert
Post-operative AF: Common after cardiac surgery, usually self-limiting, rate control, anticoagulation if prolonged
Thyrotoxicosis: Treat hyperthyroidism, beta-blockers, anticoagulate (high stroke risk)
Alcohol binge ("holiday heart"): Usually self-limiting, abstain from alcohol
Lone AF: <60 years, no structural heart disease, low stroke risk (but still use CHA_2DS_2-VASc)

CRITICAL ERRORS

Cardioverting without anticoagulation (stroke risk)
Giving AV nodal blockers in WPW (accelerates conduction to ventricles → VF)
Not anticoagulating high-risk patients (CHA_2DS_2-VASc ≥2)
Using rhythm control in permanent AF (futile)
Delaying cardioversion in unstable patient (synchronized shock immediately)

STATION 37 – CARDIOLOGY COMPREHENSIVE

STEMI – Diagnosis, ECG Findings & Complete Management

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Note: Comprehensive STEMI station integrating all MI ECG patterns and management.

CANDIDATE INSTRUCTIONS

8 minutes. Analyze ECG showing STEMI.
Identify territory, culprit artery, and outline complete management from door to balloon.
Mention complications and secondary prevention.

ECG CRITERIA FOR STEMI

New ST elevation at J point in 2 contiguous leads:
• ≥1 mm (0.1 mV) in all leads EXCEPT V2–V3
• ≥2 mm (0.2 mV) in men ≥40 years in V2–V3
• ≥2.5 mm in men <40 years in V2–V3
• ≥1.5 mm in women in V2–V3
• **Posterior MI:** ST depression ≥0.5 mm in V1–V3 with tall R waves (reciprocal changes)
• **Hyperacute T waves:** May precede ST elevation (early sign)
• **New LBBB** with ischemic symptoms (treat as STEMI if new onset)

Reciprocal ST depression: Confirms STEMI (vs pericarditis), helps localize (opposite wall)
• Inferior STEMI → reciprocal in I, aVL
• Anterior STEMI → reciprocal in II, III, aVF
• Posterior MI → reciprocal (ST elevation) in V1–V2

TERRITORY CORRELATION (See Station 23 for details)

Inferior (II, III, aVF): RCA (80%) or LCx (20%)
Anterior (V1–V4): LAD

Lateral (I, aVL, V5–V6): LCx or diagonal LAD
Posterior (tall R V1–V3): RCA or LCx (posterior descending)
Right ventricular (V4R): RCA (proximal)
Multivessel: Diffuse changes, cardiogenic shock

IMMEDIATE MANAGEMENT – TIME IS MUSCLE

Door-to-balloon <90 minutes, Door-to-needle <30 minutes
1. Immediate (ED):
• Aspirin 325 mg (chewed) – if not given by EMS
• P2Y12 inhibitor: Ticagrelor 180 mg or Prasugrel 60 mg (or Clopidogrel 600 mg if unavailable)
• Anticoagulation: Unfractionated heparin (bolus + infusion) or Bivalirudin
• Nitroglycerin: Sublingual or IV (if pain and SBP >90, not RV infarct)
• Morphine: 2–4 mg IV if pain uncontrolled (caution: associated with worse outcomes, use sparingly)
• Oxygen: Only if SpO2 <90%, respiratory distress, or high-risk features
• Beta-blocker: Oral within 24 hours (IV if hypertensive/tachyarrhythmic, avoid if acute heart failure, hypotension, bradycardia, heart block, RV infarct)
• Atorvastatin 80 mg
• **Primary PCI:** Emergency cardiac catheterization (preferred if available within 120 minutes)
• **Thrombolysis:** If PCI not available within 120 minutes (Tenecteplase, Reteplase, Alteplase) – contraindications must be assessed

2. Adjunctive therapy:
• ACE inhibitor (within 24 hours, especially anterior MI, EF <40%, diabetes)
• Aldosterone antagonist (Eplerenone) if EF <40% and heart failure or diabetes
• Statin (high-intensity)
• PPI if high bleeding risk (with DAPT)

COMPLICATIONS & MANAGEMENT

Arrhythmias:
• VF/pVT: Immediate defibrillation
• Bradycardia/Heart block: Atropine, pacing (temporary or permanent)
• AF: Rate control, anticoagulation

Mechanical:
• Papillary muscle rupture (acute MR) – Emergency surgery
• Ventricular septal defect – Surgical repair
• Free wall rupture (tamponade) – Pericardiocentesis, emergency surgery
• LV aneurysm – Anticoagulation, surgery if heart failure/arrhythmia

Cardiogenic shock: Inotropes, IABP, emergent revascularization
Reinfarction: Recurrent ST elevation, re-emergent symptoms – urgent angiography
Pericarditis (Dressler's): Post-MI pericarditis (now rare with reperfusion), NSAIDs + colchicine (avoid high-dose aspirin if on DAPT)

SECONDARY PREVENTION

DAPT duration: Aspirin lifelong + P2Y12 inhibitor (Ticagrelor/Prasugrel/Clopidogrel) for 12 months (minimum)
Beta-blocker: Lifelong (reduces mortality)
ACEi/ARB: Lifelong (especially if EF <40%, diabetes, anterior MI)
Statin: High-intensity (Atorvastatin 80 mg or Rosuvastatin 40 mg)
Risk factor modification: Smoking cessation, BP control (<130/80), Diabetes control (HbA1c <7%), Weight management, Exercise rehabilitation
ICD consideration: If EF <35% post-MI despite optimal medical therapy (40 days post-MI, 90 days if revascularized)

CRITICAL ERRORS

Delay in reperfusion (door-to-balloon >90 min)
Missing RV infarct (nitrates contraindicated)
No DAPT loading (stent thrombosis risk)
Giving beta-blocker in RV infarct (hypotension)
Stopping DAPT early (stent thrombosis, death)
No secondary prevention (recurrent MI)

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STATION 38 – PAEDIATRICS

Machinery Murmur – Diagnosis, Management & Surgical Options

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Note: Specific focus on PDA murmur, expanding on Station 12 and Station 19.

CANDIDATE INSTRUCTIONS

8 minutes. Identify machinery murmur on examination.
Confirm diagnosis of PDA, outline medical management, and discuss surgical options with indications.
Differentiate from other continuous murmurs.

MACHINERY MURMUR CHARACTERISTICS

Description: Continuous, machinery-like, "train in a tunnel" sound
Timing: Systolic-diastolic, continuous throughout cardiac cycle, peak at S2
Location: Left infraclavicular area, 2nd left intercostal space
Radiation: To left clavicle, left upper back, occasionally left sternal border
Intensity: Grade I–IV/VI, thrill if loud
Associated findings: Wide pulse pressure, bounding pulses (Corrigan's pulse), hyperdynamic precordium, mid-diastolic rumble at apex (increased flow across mitral valve)
Differential diagnosis of continuous murmurs:
• Venous hum (cervical, disappears with compression/JVP elevation)
• Mammary souffle (pregnancy/lactation, breast vasculature)
• Coronary AV fistula (location variable)
• Ruptured sinus of Valsalva (sudden onset, loud)
• Aortopulmonary window (similar location, rare)
• Blalock-Taussig shunt (surgical, unilateral)

HEMODYNAMIC SIGNIFICANCE ASSESSMENT

Small (silent): Detected only by echo, no murmur or minimal, no hemodynamic significance
Moderate: Classic machinery murmur, wide pulse pressure, LV volume overload (apex displaced, active), may have symptoms with large shunt
Large: Loud murmur, bounding pulses, cardiomegaly, heart failure (infants), pulmonary hypertension (if long-standing, Eisenmenger physiology)
Eisenmenger PDA: Differential cyanosis (lower body cyanosis, clubbing, normal upper body), reversed shunt (right-to-left), continuous murmur may disappear, loud P2
Echo assessment: Size of PDA, direction of shunt, estimate pulmonary pressure, LV size and function, LA enlargement

MEDICAL MANAGEMENT

Premature infants (see Station 12):
• Ibuprofen or Indomethacin (3 doses)
• Contraindications: NEC, bleeding, renal failure, thrombocytopenia, sepsis
• Success rate: 70–80% if <14 days old, lower if >14 days
• Paracetamol: Emerging option if NSAIDs contraindicated

Term infants/Children/Adults:
• Medical closure rarely successful >2 weeks of age
• Heart failure management if present (diuretics, digoxin)
• Endocarditis prophylaxis: Only if prior endocarditis or unrepaired cyanotic CHD (not routine for isolated PDA)
• Observation: Small, silent PDAs may not need closure (controversial, most close to prevent endocarditis risk)

SURGICAL OPTIONS & INDICATIONS

Indications for closure:
• Symptomatic PDA (heart failure, failure to thrive, recurrent chest infections)
• Hemodynamically significant (left heart enlargement, Qp:Qs >1.5:1)
• Prior endocarditis
• Large PDA with pulmonary hypertension (reversible)
• Patient preference (small audible PDA)
• **Contraindication:** Eisenmenger syndrome (irreversible pulmonary hypertension, right-to-left shunt)

1. Transcatheter closure (preferred if >6 months, >5 kg):
• **Coils:** Stainless steel or platinum, for small PDAs (<2 mm)
• **Amplatzer Duct Occluder:** Nitinol mesh, for larger PDAs (2–12 mm)
• **Procedure:** Femoral venous access, aortogram to size PDA, device deployment, confirm position
• **Success:** >95% closure rate
• **Complications:** Device embolization (1–3%), hemolysis (high-velocity residual shunt), left pulmonary artery stenosis, aortic coarctation (device protrusion)
• **Follow-up:** Echo at 1 day, 1 month, 6 months; Aspirin 6 months (thrombosis prevention)

2. Surgical closure:
• **Indications:** Failed device closure, very large PDA (>12 mm), unfavorable anatomy (short, wide, tortuous), associated lesions requiring surgery, infection (endarteritis)
• **Techniques:** Ligation (simple), Division (eliminates risk of recanalization), Clip application
• **Approach:** Left thoracotomy (traditional), Video-assisted thoracoscopic surgery (VATS, minimally invasive), Robot-assisted
• **Complications:** Recurrent laryngeal nerve injury (hoarseness), chylothorax (thoracic duct injury), pneumothorax, bleeding, incomplete closure
• **Outcomes:** Excellent, mortality <1%

3. Thoracoscopic clip application:
• Minimally invasive, good for infants >3 kg
• Shorter recovery, less pain
• Requires expertise

LONG-TERM OUTCOMES

After successful closure: Normal life expectancy, no activity restrictions
Residual shunt: Usually small, may close spontaneously or require second device
Pulmonary hypertension: May persist if long-standing PDA, requires follow-up

Endocarditis risk: Eliminated after complete closure (6 months)
Pregnancy: Well-tolerated after closure, no special precautions

CRITICAL ERRORS

Closing Eisenmenger PDA (fatal – right heart failure)
Missing differential cyanosis (indicates Eisenmenger, contraindication to closure)
Wrong device for PDA size (embolization risk)
No antiplatelet after device closure (thrombosis risk)
Confusing with venous hum (innocent, no treatment needed)

STATION 39 – MEDICINE/PULMONOLOGY

Oxygen Delivery Devices – Venturi Mask, NRM, MDI with Spacer, Nasal Cannula

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CANDIDATE INSTRUCTIONS

8 minutes. Identify various oxygen delivery devices.
State FiO2 ranges, indications, and demonstrate correct usage.
Know when to use each device based on patient condition.

1. VENTURI MASK (Air-Entrainment Mask)

Mechanism: Uses Bernoulli principle – high-flow oxygen through narrow jet entrains room air through side ports, delivering fixed FiO2 regardless of patient's respiratory pattern
FiO2 delivery: 24%, 28%, 31%, 35%, 40%, 50% (color-coded)
• **Blue:** 24%
• **White:** 28%
• **Orange:** 31% (commonly tested)
• **Yellow:** 35%
• **Red:** 40%
• **Green:** 50%
Flow rates: Vary by FiO2 (2–15 L/min) – must use specified flow for each color to achieve stated FiO2
Total flow delivered: 40–60 L/min (exceeds peak inspiratory flow, prevents rebreathing)
Indications:
• COPD with CO2 retention (Type 2 RF) – precise FiO2 control (28% or 24%)
• Controlled oxygen therapy required
• Variable respiratory rate/pattern (delivers consistent FiO2)
Advantages: Fixed FiO2, humidification possible, doesn't dry mucosa as much as simple mask
Disadvantages: Uncomfortable, must remove for eating, noisy, precise flow required

2. NON-REBREATHER MASK (NRM)

Components: Reservoir bag (1–1.5 L), one-way valve between bag and mask (prevents exhaled gas entering bag), one-way valves on mask sides (prevents room air entrainment during inspiration)
FiO2 delivery: 60–80% (up to 90% if tight seal and high flow)
Flow rate: 10–15 L/min (must keep reservoir bag 1/3–1/2 full during inspiration)
Indications:
• Severe hypoxemia (SpO2 <85% on room air)
• Trauma, shock, severe sepsis
• Carbon monoxide poisoning
• Pre-oxygenation before intubation
• Cluster headaches (high-flow oxygen)
Critical requirement: Reservoir bag must not collapse during inspiration (if it does, increase flow or check seal)
Advantages: Highest FiO2 without intubation
Disadvantages: Uncomfortable, poor tolerance, must be removed for eating/drinking, CO2 rebreathing if valves malfunction

3. METERED DOSE INHALER (MDI) WITH SPACER

Why use spacer:
• Increases lung deposition from 10–20% (without) to 40–60% (with)
• Reduces oropharyngeal deposition (reduces side effects – hoarseness, candidiasis with steroids)
• Allows slow, deep inhalation (better distribution)
• No need to coordinate actuation and inhalation
• Reduces environmental contamination

Two indications for spacer use:
1. **All patients using ICS** (inhaled corticosteroids) – reduces oral candidiasis and systemic absorption
2. **Patients with poor hand-breath coordination** (elderly, children, during exacerbation)

Technique (demonstrate):
1. Remove cap, shake inhaler
2. Insert into spacer
3. Breathe out gently
4. Seal lips around mouthpiece
5. Press canister once
6. Breathe in slowly and deeply (over 3–5 seconds)
7. Hold breath 10 seconds
8. Wait 30–60 seconds between puffs
9. Rinse mouth after ICS use

Types of spacers: Volumatic (large), Aerochamber (small, with mask for children), homemade (plastic bottle – emergency)
Cleaning: Monthly with detergent, air dry (reduce static)

4. NASAL CANNULA (OXYGEN PRONGS)

Mechanism: Low-flow system, FiO2 depends on patient's inspiratory flow rate and respiratory pattern
Flow rates: 1–6 L/min (higher causes nasal mucosa drying, discomfort)
FiO2 achieved: 24–44% (approximate: 24% at 1L, 28% at 2L, 32% at 3L, 36% at 4L, 40% at 5L, 44% at 6L)
Indications:
• Mild hypoxemia (SpO2 90–94%)
• Long-term oxygen therapy (chronic respiratory failure)
• Palliative care
• Patients unable to tolerate mask
• Eating/drinking while maintaining oxygen
Advantages: Comfortable, allows talking/eating, inexpensive, portable
Disadvantages: Variable FiO2, nasal irritation at high flow, mouth breathing reduces efficiency
High-flow nasal cannula (HFNC): Heated, humidified, up to 60 L/min, FiO2 up to 100%, generates PEEP (2–6 cm H2O) – used in ICU for hypoxemic respiratory failure

DEVICE SELECTION ALGORITHM

Target SpO2 88–92% (COPD/Type 2 RF): Venturi mask 24% or 28% OR nasal cannula 1–2 L/min
Target SpO2 94–98% (most patients):
• Mild hypoxemia: Nasal cannula 2–4 L/min
• Moderate: Simple face mask 5–10 L/min (40–60%)
• Severe: Non-rebreather mask 10–15 L/min (80–90%)
Critical/ICU: High-flow nasal cannula or intubation
Post-extubation: HFNC or Venturi to maintain precise FiO2

CRITICAL ERRORS

High-flow nasal cannula in Type 2 RF (uncontrolled FiO2, CO2 retention)
Wrong Venturi flow rate (delivers wrong FiO2)
Collapsed reservoir bag on NRM (patient breathing room air)
No spacer with ICS (poor deposition, side effects)
Oxygen on empty cylinder (check gauge regularly)

EQUIPMENT

Venturi mask with color-coded valves
Non-rebreather mask with reservoir bag
MDI with various spacers
Nasal cannulas (adult and pediatric)
Oxygen flow meter and tubing
Pulse oximeter

BLOCK O OSCE STATIONS – COMPLETE

38 Comprehensive Stations Covering All Topics from Both Documents

Cardiology | Paediatrics | Medicine | Pulmonology
All Overlapping Topics Integrated with Expanded Detail

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