Approach to Respiratory Distress

1. GENERAL APPROACH TO SHORTNESS OF BREATH

Initial Approach:
- AIRWAY:
  - Ensure upper airway *patent* → patient speaking (patent) vs gurgle/stridor (not patent)
    - If GCS → tone of upper airway soft tissue → assume possible obstruction
  - Management:
    - Look for causes:
      - Blood/vomit in mouth → Suction
      - Swelling from trauma/allergy → Medication
      - GCS
  - If unable to clear obstruction → prepare for Intubation
- BREATHEING:
  - Ensure oxygenating (sats ≥92%) & ventilation
    - Look → Resp Distress (tripoding, tracheal tug, intercostal/supraventricular indrawing)
    - Listen → presence/absence of breath sounds
  - Management:
    - Give O₂ → via nasal cannula vs non-rebreather mask
    - If not responding → positive airway pressure (CPAP/BiPAP) vs intubation/mechanical ventilation
- CIRCULATION:
  - Management:
    - 2 large bore IVs (18G+) with IVFs if hypotensive
- Investigations:
  - ECG
  - CXR (portable if unstable)
  - Bedside US

Differential Dx → focus on deadly
- 4 categories
  - UPPER AIRWAY:
    - Foreign body, Swelling, Blood, Vomit
  - LUNGS
    - Pneumonia, Pneumothorax (PTX), PE, Asthma/COPD
  - HEART
    - ACS, Pulmonary edema, Pericardial effusion, Tamponade
  - METABOLIC (Acidosis)
    - Sepsis, DKA, Drugs

Physical Exam:
- UPPER AIRWAY
  - Signs of resp distress with stridor
  - Look for blood/vomit in oral cavity
- LUNGS
  - Signs of resp distress without stridor
  - Pneumonia → crackles, bronchial breath sounds
  - PTX → ↓breath sounds
  - Asthma/COPD → wheezes, ↓breath sounds
- HEART:
  - Pulmonary edema → pink/frothy sputum, crackles
  - Pericardial effusion → muffled heart sounds
  - Tamponade → hypotension
- ACIDOSIS:
  - Tachypnea without symptoms of resp distress (normal O₂ sat)
2. APPROACH TO ANAPHYLAXIS

Terminology:
- Allergic reaction = body reacting to previously sensitized allergen
- Can occur in different body systems & range from mild to severe (anaphylaxis)
- Anaphylaxis = multi-system involvement (medical emergency)

Pathophysiology:
- Previous sensitized allergen = production of IgE antibodies → bind mast cells/basophils → degranulation & release of mediators (histamine, leukotrienes, prostaglandins) → symptoms
  - Tx based on inhibiting mediators
- Multi-system involvement by above mediators
  - LUNGS → bronchoconstriction & mucous secretion
  - CVS → vasodilation
  - GI → diarrhea & cramping
  - SKIN → swelling (urticarial = superficial; angioedema = deep)

Assessment & Treatment:
- AIRWAY → can have ++swelling
  - Assess oropharynx for swelling of lips, tongue, mucous membranes
  - Assess for difficulty swallowing/throat tightness & listen for stridor (mainly inspiratory)
  - Tx = early Epinephrine (0.3-0.5cc 1:1000 solution IM q5-10min) & monitor airway closely
    - If no swelling/worsening → definitive airway (intubation)
    - Safety Note → never give 1:1000 Epi IV!!
- BREATHING → wheezing from bronchoconstriction
  - Assess for respiratory distress (in-drawing, tracheal tug, abdominal breathing, tripoding)
  - Tx = bronchodilators (Ventolin & Epinephrine) + O2 if hypoxic
- CIRCULATION → BP & syncope from vasodilation & contractility (potential for arrhythmias)
  - Put on cardiac monitor, get ECG
  - Tx = IVF (1-2L)
    - If still hypotensive → vasopressor = Epinephrine (0.5-1cc 1:10000 IV infusion over 10-15min) or Dopamine
- Target Mediators:
  - Anti-histamines = main target
    - H1-blockers (Diphenhydramine 50mg IV/PO/IM q4h)
    - H2-blockers (Ranitidine 150mg PO/50mg IV q8h)
  - Note: if anaphylaxis → give Epi early, before antihistamines
- 2nd phase reaction phenomenon
  - Recurrence of symptoms 6-72h post initial reaction without re-exposure to allergens
  - Can be the same reaction, worse, or less severe
  - Hypothesized that steroids may 2nd reaction (controversial)
    - If given → Prednisone 50mg PO daily x 5d

Discharge Instructions:
- Rx:
  - Antihistamines (Diphenhydramine + Ranitidine) x 3d
  - ±Prednisone
  - Epi-pen with proper instructions on correct usage
- If allergen known → avoidance
- If allergen unknown → allergy testing
3. APPROACH TO PNEUMONIA

Terminology:
- Pneumonia = inflammation of lung parenchyma
  - Alveolar airspaces filled with exudate & inflammatory cells
  - Causes → infection, chemical exposure

Classification:
- Important to classify as organisms involved depend on **patient & setting**
  - Bacterial → Gram +ve, Gram -ve, anaerobes
  - Viral
  - Fungal
- Based on **PATIENT**:
  - Healthy
  - Unhealthy (co-morbidities) = ↑susceptibility to infections/specific organisms
    - Immunocompromized → DM, HIV, antirejection meds, steroids
    - Existing lung problems → COPD, asthma
    - Aspiration risk → seizures, strokes
- Based on **SETTING**:
  - Community-acquired → patient lives independently in own home
  - Hospital-acquired → infection from hospital setting (>48h from admission)
  - Health-care acquired → patient from LTC, nursing home

**Causes** (based on classification):
- **Community Acquired = CAP:**
  - HEALTHY Patient
    - Bacterial:
      - Typical (classic Sx) → S.pneumoniae (most common), M.catarrhalis, H.influenza
      - Atypical (non-classic Sx) → mycoplasma, chlamydia
    - Viral → influenza, RSV
  - UNHEALTHY Patient
    - Potential for “healthy” patient organisms (above) plus:
      - HIV = PCP, TB
      - Asplenic = encapsulated organisms (ie. Strep)
      - On chemotherapy = ↑general susceptibility to infection
      - Existing lung problems = ↑general susceptibility to infection, ↑resistance d/t multiple courses of abx for previous infection
      - Aspiration risk = anaerobes (Klebsiella, Fusobacterium)
- **Hospital/Health-Care Acquired (Nosocomial) = HAP:**
  - Bacterial (most common):
    - Can get community organisms (ie. S.pneumoniae, H.influenza)
    - More worrisome = Pseudomonas, Klebsiella, E.Coli, Staph/MRSA
  - Viral (influenza) can occur but more rare

**Presentation of Pneumonia:**
- **TYPICAL**:
  - Hx: Fever, cough, ↑sputum production, confused
  - Px: ↑HR, ↑RR, resp distress, confused
    - Resp exam: adventitia (crackles, wheeze, rales, rhonchi), ↓breath sounds, dullness to percussion
    - If pleural effusion → friction rub
- **ATYPICAL** (more common with extremes of age):
  - Hx: confusion, dyspnea, shock
  - +/- fever, cough, ↑sputum

**Diagnosis:**
- Labs:
- **WBC (↑ Neut), ↑ plat (acute phase reaction)**
- **Na, ↓ HCO₃, ↑ BUN/Cr**
- VBG/ABG for those in resp failure
- **Imaging → ESSENTIAL for diagnosis**
  - CXR → portable (if too sick) vs PA & Lat

**Treatment:**
- **GENERAL:**
  - Airway → if not protecting/significant resp distress = Intubate
  - Breathing → if hypoxic = give O₂
  - Circulation → if hypotensive/tachycardic = IVF (RL or NS)
- **SPECIFIC → Antibiotics** (choice dependent on local, prevalence, and resistance organisms)
  - **CAP:**
    - HEALTHY:
      - PO = Macrolide vs doxycycline
      - IV = Fluoroquinolone; Ceftriaxone + macrolide
    - UNHEALTHY (co-morbid):
      - Immunosuppressed or COPD → IV fluoroquinolone; ceftriaxone + macrolide
      - HIV = worried about PCP → Septra
      - Aspiration = worried about anaerobes → Clindamycin
  - **HAP → need broader & stronger**
    - IV Ceftriaxone, Fluoroquinolone +/- Vanco (if MRSA)
  - **Viral** → usually in context of pandemic
    - Refer to local guidelines for use of antivirals
- **Risk Stratification** = send home vs admit vs ICU → 2 tools
  - **CURB 65 →** determines overall risk of death at 30d (each = 1pt)
    - **Scoring**
      - Confusion
      - Urea > 7mol/L
      - RR > 30bpm
      - BP (SBP < 90 or DBO < 60)
      - Age ≥65
    - 0-1 → outpatient treatment
    - 2-3 → consider short hospitalization vs close outpatient F/U
    - 4-5 → hospitalization +/- ICU
      - 5 = ~60% death at 30d
  - **Pneumonia Severity Index** (PORT score)
    - Components
      - Demographics → age, gender, residence (ie. nursing home)
      - Co-morbidities → cancer, liver disease, CHF, CVD
      - Px → altered MS, ↑HR, ↑RR, ↓BP, ↑or↓Temp
      - Labs & Imaging

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4. **APPROACH TO PNEUMOTHORAX**

**Terminology:**
- Pneumothorax (PTX) = air in pleural cavity (potential space between parietal and visceral pleura)
- **Effects:**
  - 1st → Can impair oxygenation + ventilation
  - 2nd → if 1-way valve leaking air into pleural space → can shift mediastinum to contralateral side →
    → venous return to heart → ↓BP → cardiac arrest

**Causes:**
- **SPONTANEOUS:**
  - Occurs when blebs or bullae rupture in lungs
  - 1° → in patient with NO underlying lung disease
• Usually affects thin & tall patients
• ↑risk in smokers & genetic factors (ie. marfan's syndrome)
  o 2° → in patient WITH underlying lung disease
  ▪ Most common = COPD, asthma
  ▪ Can also occur with infections → TB, PCP

• TRAUMATIC:
  o Puncture of pleura leading to air into space
  o Iatrogenic → biopsy, thoracentesis, intercostal nerve blocks
  o Trauma/Penetrating injury → open wound to pleura, rib fractures puncturing pleura
  o More likely to lead to TENSION pneumothorax (d/t ↑likelihood of 1-way valve)

Presentations:
• SPONTANEOUS:
  o Hx → CP in affected side = severe, sudden onset, stabbing/sharp, radiation to shoulder, pleuritic
  o Px → SOB, ↑RR, ↓O2 sat, ↓Breath sounds (BS) affected side

• TRAUMATIC:
  o Hx → trauma, recent procedures
  o Px → as above + evidence of open wounds

• TENSION:
  o Px → ++resp distress, contralateral tracheal deviation, ↑JVP, ↓BP, ↓BS on affected side

Investigations = Diagnostic Imaging → CXR, US, CT
• Important → tension PTX is a clinical diagnosis (no time for imaging)
• CXR:
  o Can visualize the outline of pleura d/t detachment
  o Expiratory film → sensitivity of picking up PTX (especially if small)
  o Supine patients → no air rising to apex of lungs (any air will rise to most superior aspect = chest wall) → will instead get deep sulcus sign (air collecting in inferior sulci at angle where diaphragm joins chest wall)
  o Limitations:
    ▪ Difficult to detect small PTX & in supine patients

• US:
  o High sensitivity and specificity for Dx of PTX

• CT:
  o High sensitivity and specificity
  o Will also indicate if any underlying lung/chest pathology is present
  o Limitation = requires stable patient

Treatment:
• Goals:
  o 1st = release air accumulated within pleural space in order to maintain ventilation
  o 2nd = stop further accumulation of air into pleural space
• SPONTANEOUS & IATROGENIC PTX:
  o Small (<3cm from apex):
    ▪ If hemodynamically stable → conservative tx (observe in ED for few hours then D/C with frequent f/u)
  o Large:
    ▪ Need to release air as patient likely symptomatic
    ▪ Requires Chest tube → 5th ICS in Anterior Axillary Line

• TENSION PTX (unstable patients):
  o 1st = Needle decompression with large bore IV needle (14/16G) → 2nd ICS in midclavicular line (MCL)
    ▪ Should hear gush of air → will allow enough time to insert chest tube
    ▪ Hemodynamic status should improve post decompression
  o 2nd = chest tube (directions as above)
  o TRAUMATIC Tension PTX:
    ▪ Often has air leak from outside (open wound) or inside (tear in tracheobronchial tree)
    ▪ Open PTX = sucking chest wound
• **Initial Tx** = 3-way occlusive dressing over wound → dressing pulled to chest wall on inspiration (stopping air entry to pleural space from wound) & allows air to exit through open side of dressing on expiration = 1-way valve effect

  * **Ultimate Tx** = chest tube + definitive repair in OR
    - Persistent air leak from tracheobronchial tree
  * Initial Tx = chest tube (5th ICS AAL)
    - However will continue to have persistent PTX
  * Ultimate Tx = definitive repair in OR

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5. **APPROACH TO ACUTE CORONARY SYNDROMES (ACS)**

**Cause → blockage** of coronary arteries
- Can lead to permanent damage (cell death) to myocardium = **MYOCARDIAL INFARCTION**
  - If ST elevation on ECG = **STEMI**
  - If no ST elevation on ECG = **NSTEMI**
- Can lead to ↓ blood supply to myocardium (no cell death) = **MYOCARDIAL ISCHEMIA**

**Presentations:**
- **Hx** = most important aspect of evaluation
  - RF for ACS = **RF for CAD**
    - Smoking, DM, HTN, FHx of CAD (at young ages), ↑ cholesterol
    - RFs ↑ chance of ACS but NOT required to Dx ACS
  - CP → onset (timing & what pt doing), quality, location, severity, duration (new or recurrent), frequency (similar previous episodes), radiation (jaw, shoulder, arm, back), ↑ / ↓ factors (pleuritic, positional, affect of exercise/rest)
  - Associated Sx → SOB, diaphoresis, N/V, presyncope
  - Hx worrisome for ACS (TYPICAL Sx):
    - Retrosternal chest pressure
    - Radiation pain to both arms/shoulders
    - Exertional pain better with rest
    - Not pleuritic or positional
    - Association with nausea, diaphoresis
    - Patient states similarity of pain to previous MI
  - ATYPICAL Sx:
    - CP → mild discomfort, isolated in shoulder/arm/jaw/epigastric, isolated SOB or diaphoresis, pain sharp/pleuritic/reproducible on palpation, no association with exercise & no improvement with rest
    - Have low threshold for investigation due to atypical presentations
    - More likely in → elderly, women, DM

- **Px:**
  - Large range of presentations → from sick to well
    - Sick → cardiac arrest, unstable vitals (↓ / ↑ BP, ↓ / ↑ HR, ↑ RR)
  - Cardiac → listen for S1/S2, presence of EHS/murmurs, ↑ JVP
  - Resp → listen for crackles, presence of breath sounds

**Investigations:**
- **ECG** = cornerstone Dx for ACS
  - Approach = rate, rhythm, axis
  - In potential ACS patient → look for ST elevation & ST depression +/- T inversion
  - ST elevation → indicates acute infarct
    - Causes of non-ACS ST elevations:
      - Benign early repolarization, LBBB, LVH, LV aneurysm, pericarditis
    - Acute infarct follows 2 rules
      - **TERRITORIAL** → each coronary artery + branches supply specific areas to heart
        - If 1 branch blocked → territory supplied by this artery = no O2 delivery
        - Look at 12 lead ECG in territories:
Inferior = Leads 2, 3, aVF
  • Supplied mainly by RCA
Lateral = Leads 1, aVL, V5, V6
  • Supplied by branches of LAD + Left circumflex
Anterior = Leads V1, V2, V3, V4
  • Supplied mainly by LAD
  
  Want to determine if ST elevations belong to same territories
  
  RECIPROCAL Changes
  
  o If ST elevations occur in 1 territory \( \rightarrow \) get ST depression in opposite territory & vice-versa
  
  Inferior = opposite to Lateral
  
  Anterior = opposite to Posterior (requires 15 lead ECG with posterior leads = V7, V8, V9)

ST depression +/- T inversion
  
  ▪ ST depression = denotes ischemia \( \rightarrow \) not territorial or reciprocal
  
  ▪ If no ST depression then look for T-wave inversion

Non-EKG modalities:
  
  o Imaging \( \rightarrow \) to exclude other Dx of CP
    1. CXR:
      ▪ Look for PTX, pneumonia, aortic dissection
    2. Bedside US:
      ▪ Look for pericardial effusions,
    3. CT:
      ▪ Look for PE, aortic dissection

BW:
  
  Routine:
  
  ▪ CBC \( \rightarrow \) Hg (Sx may be present with anemia)
  
  ▪ Lytes & creatinine \( \rightarrow \) idea of baseline function, may direct future Tx choices
  
  Specific = cardiac enzymes \( \rightarrow \) ↑ if myocardial death

  • Trop I = most sensitive
  
  ▪ However can take hours to appear abnormal on BW
  
  ▪ Most institutions choose to draw levels at 6-8h post event

  ▪ Delayed –ve Trop CAN r/o MI but cannot r/o angina
  
  ▪ Early –ve Trop CANNOT r/o MI as may be too early

Treatment:
  
  ▪ STEMI \( \rightarrow \) 5 overall principles
    1. O2 \( \rightarrow \) To achieve O2 sat >92%
    2. Antiplatelet \( \rightarrow \) ASA, Clopidogrel, Ticagrelor, Glycoprotein 2B/3A inhibitor
    3. Antithrombin \( \rightarrow \) Unfractionated Heparin, LMWH, fondaparinux
    4. Open vessel (main Tx) \( \rightarrow \) PCI, CABG
    5. Symptom treatment \( \rightarrow \) morphine, nitrates

  ▪ NSTEMI
    1. Risk stratify with TIMI Score \( \rightarrow \) with non Dx ECG for ACS or normal 1st set of cardiac enzymes
      ▪ Looks at multiple RFs for death, MI, and ischemia needing revascularization in next 14d
      ▪ Includes (each 1 point):
        ▪ Age ≥65
        ▪ ≥3 CAD RFs (as above in Hx)
        ▪ Known CAD
        ▪ ASA use in past 7d
        ▪ Severe angina (>2 episodes in past 24h)
• ST segment changes (>0.5mm)
• Positive cardiac markers
  ▪ ↑ score = more aggressive Tx
  o Tx = same as STEMI (with ↓ incidence of requirement to open vessel)

6. APPROACH TO PULMONARY EMBOLUS (PE)

Terminology:
• PE = Clot in pulmonary arterial tree → in main, segmental, or sub-segmental arteries
• Why is PE bad:
  o RESP problems → V/Q mismatch (areas of lung with ventilation with no perfusion) → hypoxemia
  o CARDIAC problems → ↑ pulmonary vascular resistance (PVR) → RV strain +/- failure → CV collapse
• Often originate from deep vein thrombosis (DVT) with migration to lungs
  o Below knee → from anterior/posterior tibial and/or peroneal veins
  o Behind knee → popliteal veins
  o Above knee → superficial/deep/common femoral veins, external iliac veins

Causes → Virchow’s Triad
• STASIS → any conditions attributing to immobilization of patient
  o Surgeries, leg casts, long flights/train rides, intubation
• HYPERCOAGULABILITY
  o Congenital → protein C/S deficiency, Factor 5 Leiden, etc.
  o Acquired → HRT, OCP
  o Illness → SLE, malignancy, pregnancy, IBD
• ENDOTHELIAL INJURY
  o Central venous catheters, surgery

Presentation:
• Hx:
  o CP → pleuritic, associated with cough +/- hemoptysis
  o SOB → poor exercise tolerance, anxiety-like sx d/t tachycardia
  o Can be asymptomatic
  o If severe = CV collapse → ranging from syncope to cardiac arrest
• Px:
  o VITALS → ↑ HR, ↑ RR (most common), ↓ BP, ↑ temp, ↓ O2 sat
  o CARDIAC → +/- EHS (S3/S4)
  o RESP → Rales
  o PERIPHERIES → signs of DVT in lower limbs (swelling, erythema, warmth)

Investigations:
• Goal = to r/o alternative Dx & rule in PE
• CXR → Look for pneumonia, pleural effusion, pneumothorax, CHF
  o PE CXR findings (rare):
    ▪ Hampton’s Hump = wedge shaped hyperdensity representing area of pulmonary infarct
    ▪ Majority of patients with PE = normal CXR
• ECG:
  o Look for STEMI, pericarditis
  o PE ECG findings:
    ▪ Most common = sinus tachycardia (~40% pts)
    ▪ Flipped T in V1-V4 (~30% pts)
    ▪ RBBB (~20% pts)
    ▪ S1Q3T3 pattern (~20% pts) = prominent S in Lead 1, Q in lead 3, inverted T in lead 3
• BW:
  o CBC, lytes, BUN, Cr → for baseline values
  o Troponin
  o D-Dimer = fibrin degradation product (can ↑ in PE)
- See below

- **CONFIRMATORY TESTING** for Dx
  - VQ scan → looks at:
    - Ventilation → inhalation of air with radionucleotide and entry to air spaces in lungs
    - Perfusion → injection of IV radionucleotide to look at blood flow in lungs
    - In PE → part of lung affected will have **ventilation without perfusion**
    - Best used in patients with normal lungs
    - Not as sensitive or specific as CT scan
  - CT with contrast → imaging of choice to Dx PE
    - Will detect filling defects
    - Indicate regions of pulmonary infarction
  - Doppler US → used in patients with suspicion of DVT
    - Can also indicate alternative Dx → ie. Cellulitis, Baker’s cyst

### Approach to PE:
- **Risk stratification** → Very Low vs Low vs High
  - VERY LOW risk → PERC Rule
    - 8 criteria
      - Age > 50
      - HR > 100
      - O2 sat <94%
      - Prior DVT/PE
      - Recent trauma or surgery
      - Hemoptysis
      - Exogenous estrogen use
      - DVT Sx
    - If patient has none of above with low clinical suspicion of PE → no further w/u indicated
    - Total w/u may include only → CXR, ECG, basic BW (NO D-dimer)
  - LOW risk → D-Dimer
    - Scored via **Well’s Score** → based on clinical presentation with point scoring
      - Components:
        - DVT Sx (3)
        - HR > 100 (1.5)
        - Recent immobilization >3d or surgery in past 30d (1.5)
        - Previous VTE (1.5)
        - Hemoptysis (1)
        - Malignancy (1)
    - Scoring:
      - ≤4 = low risk
        - D-Dimer -ve = no further w/u indicated
        - D-Dimer +ve = further w/u with imaging required (follow high-risk)
      - >4 = high risk (see below)
  - HIGH risk → Imaging (cannot r/o with negative D-Dimer)
    - VQ scan
    - CT → high sensitivity
    - Doppler US → to r/o DVT

- **Treatment**:
  - Initial = ABCs
    - Ensure airway patent
    - Ventilation & oxygenation adequate → supplemental O2
    - Not hypotensive → IVF +/- vasopressors
  - If hemodynamically stable → **anticoagulation** (heparin, LMWH bridging to warfarin, oral anticoagulants)
  - If massive PE causing CV compromise (unstable) → **thrombolytics**
7. APPROACH TO ASTHMA

Terminology:
- Asthma = Chronic inflammation of airways → leading to airway edema and bronchoconstriction → airflow obstruction → ↓ ventilation (↑CO₂) + ↓ oxygenation (↓O₂) → respiratory arrest
- **Triggers** of asthma exacerbations:
  - Allergens
  - Smoking
  - URTI

RF for **severe** exacerbations:
- Previous asthma exacerbations requiring ICU admission/intubation
- Multiple hospital admissions
- Poor baseline lung function

Presentation:
- Hx:
  - SOB + chest tightness → worse on exertion
  - Audible wheezes
  - ↑ use of puffers
- Px:
  - General APPEARANCE → position, LOC, colour, VS (varies based on degree of exacerbation)
    - **Mild** exacerbation:
      - Can lay down comfortably, normal LOC, normal colour (pink), ↑↑RR/HR, normal O₂ sat
    - **Moderate** exacerbation:
      - Will sit upright, normal LOC, normal colour (pink), ↑↑↑RR/HR, ↓O₂ sat
    - **Severe** exacerbation:
      - “Tripod” positioning leaning forward, confused +/- ↓LOC, cyanotic (blue) + diaphoretic, ↑↑↑HR or ↓HR, ↓BP, ↓↓O₂ sat
- RESP:
  - **Mild**:
    - Full sentences, no in-drawing, bilateral AE but ↓, + wheezes
  - **Moderate**:
    - 3-4 word dyspnea, +/- in-drawing (intercostal, supraclavicular), tracheal tug, loud wheezes, ↓AE
  - **Severe**:
    - Single word dyspnea, ↑ in-drawing/tracheal tugging, +/- abdo breathing, quiet on auscultation with ↓↓AE & no wheezing

Investigations:
- CXR → to r/o pneumonia, PTX, pneumomediastinum
  - Not necessary if Dx clear on Hx (ie. known allergen, URTI trigger, etc)
- BW → not indicated in patients with routine exacerbations
- ECG → if suspicious for MI/ACS
- **FEV1** → bedside test to measure forced expiratory volume in 1 sec (requires alert patient)
  - Compare measured value to expected value (as %) → for risk stratification
    - **Mild** exacerbation → **50-70%** expected FEV1
    - **Moderate** exacerbation → **25-50%** expected FEV1
    - **Severe** exacerbation → <**25%** expected FEV1
  - Use in conjunction with clinical information to determine severity of exacerbation

Treatment:
- GENERAL = ABCs:
  - Avoid hypoxia → give supplemental O₂ to maintain sats >92%
  - Avoid hypotension → give IVF
- **RAPID ACTING B2-AGONISTS** = **mainstay** of asthma tx
Action = bronchodilation
o **Salbutamol** (Ventolin) = main choice → various delivery methods (Neb, MDI, IV)
  - MDI for Mild-Moderate exacerbation
    - 4-6 puffs q20-40min
  - Neb for Severe exacerbation (as unable to coordinate breathing efficiently enough for MDI)
    - 3 Neb doses back to back (1 Neb = 4-6 MDI puffs)
  - Adverse Effects:
    - ↑ HR, ↓ K

- **ANTICHOLINERGICS:**
  o Action = bronchodilation
  o **Ipratropium** (Atrovent) → various delivery methods (Neb, MDI)
    - MDI for Mild
    - Neb for Severe
  o Given with Ventolin in 1st 3 rounds of Tx → dosing as above

- **EPINEPHRINE** → given if exacerbation triggered by **allergic** reaction
  o Action = bronchodilator, vasopressor

- **STEROIDS:**
  o Action = ↓ airway inflammation & edema
  o Can be given IV/PO → IV only for those who can’t tolerate PO intake
    - IV = Hydrocortisone 100-200mg
    - PO = Prednisone/Prednisolone 50mg OR Dexamethasone 16mg

- **ANTIBIOTICS:**
  o Only for those with documented **pneumonia** on CXR

- **SPECIFIC for Severe Exacerbations:**
  o **Magnesium** 2g IV over 30min → smooth muscle relaxant (bronchodilation)
  o BiPAP → no large RCTs to support use; depends on local centre preferences
  o Intubation + mechanical ventilation:
    - Indications = ↓ LOC, ++ exhaustion, cardiac or respiratory arrest
    - Intubated asthmatic = **very difficult** patient (d/t many reasons including inducing bronchospasm, difficult to maintain resp requirements, etc)

**Discharge:**
- Requirements:
  o In no resp distress
  o FEV1 > 75% predicted (at least 2h after last tx with bronchodilator)
- Medications:
  o **Salbutamol** puffer + aerochamber → 1-2 puffs q4h
  o **Steroids** → Prednisone/Prednisolone 50mg daily x 4d OR Dexamethasone 16mg x 1d (if given in ED)

**8. APPROACH TO COPD**

**Terminology:**
- COPD = chronic inflammation of tracheobronchial tree → leading to airflow obstruction → resp failure → death

- **Causes:**
  - Smoking (majority), alpha1-antitrypsan deficiency
  - Natural **progression** → lung function ↓ after each acute exacerbation of COPD (AECOPD)
  - **Triggers:**
    o Lung problems → pneumonia, PE, PTX
    o Medication → ran out of puffers
    o Social → continuation of smoking

- **Evaluation:**
  - Hx:
    o CURRENT attack:
      - Onset, current Sx (SOB, cough, exercise intolerance), associated Sx (PND, orthopnea, CP)
- **Triggers:**
  - Pneumonia → fever, chills, sputum changes (colour, amount)
  - PE → immobilization, previous VTE, etc.
  - PTX → abrupt onset SOB
  - Run out of puffers, ↑smoking

  - **BASELINE Function:**
    - Able to walk around the block, walk up stairs, O2 dependent (rest vs exertion)
    - Puffer use → name & frequency of use

  - **PREVIOUS Attacks:**
    - Frequency of exacerbations → last exacerbation, previous steroid use, usual course of illness (# of days, requiring hospitalization/ICU, needing intubation)

  - Px = Similar to approach with CC of SOB
    - VITALS → ↓O2 sat, ↑HR, ↑temp (if infection = trigger)
    - GENERAL → Signs resp distress = tripod positioning, cyanosis, tracheal tugging, intercostal in-drawing, abdominal breathing
    - CARDIAC → HS/EHS, murmurs, cardiac apex, JVP
    - RESP → AE & symmetry, presence of crackles/rales, pleural rubs
    - PERIPHERIES → signs of DVT in legs (if PE = trigger), peripheral edema (if CHF = trigger)

- **Investigations:**
  - BW:
    - CBC → for signs of infection (↑WBC), hemoconcentration (↑Hg)
    - Lytes, BUN, Cr → for electrolyte abnormalities & renal insufficiency
    - VBG +/- ABG → look for CO2 retention (↑pCO2), compare to baseline value if available for severity of current illness
    - Others:
      - If concerned of cardiac insult → Trop, CK, ECG

  - ECG:
    - ↑HR during acute exacerbation → typically Multifocal Atrial Tachycardia (MAT)
    - Also look for cardiac cause mimicking AECOPD (ie. STEMI)

  - Imaging:
    - CXR → most common
      - Typical COPD CXR → hyperinflation with flattened diaphragm
      - Identify triggers → pneumonia, PE, PTX
    - CT → used if suspicion of PE as trigger
    - Bedside US → used to evaluate for PTX

- **Treatment:**
  - Initiated once other caused of SOB excluded
  - O2:
    - Prevent hypoxia → titrate to O2 sats of 90% (via NP or venturi mask)

  - BRONCHODILATORS:
    - Salbutamol (main tx) → via Nebs/MDI
      - Given back-to-back x 3 initially
      - AE = ↑HR, ↓K
    - Ipratropium → added for additional bronchodilation
      - Given as single initial dose

  - STEROIDS:
    - Methylprednisone IV OR Prednisone PO
    - Important to begin early as can take hours for effect

  - ANTIBIOTICS:
    - Indication:
      - Pneumonia on CXR, or
      - Sx consistent with infection = fever, ↑sputum production, change in sputum colour
    - Either Doxycycline, Fluoroquinolone, or Cephalosporin

  - BiPAP → for mod-severe exacerbation
    - Can only be used in awake patients
- Use **early** in treatment plan
- Use useful in preventing intubation in those with severe exacerbations

**INTUBATION:**
- Indications:
  - Severe exacerbation without access to BiPAP
  - Failing BiPAP/\(\downarrow\)O2 sat despite on BiPAP
  - Worsening acidosis (\(\uparrow\)CO2)

**Disposition:**
- **D/C home if:**
  - Normal O2 sat at rest **AND** on exertion
  - Meds = 10-14d PO **steroids** +/- abx if indicated
- **Admission if:**
  - \(\downarrow\)O2 sat with exertion/ambulation
  - Requiring continuous BiPAP \(\rightarrow\) admit to ICU

Summarized by Alex Mungham, Dr. Stella Yiu