Asthma

Trials

- National Asthma Education and Prevention Program (NAEPP) Expert Panel Report III (EPR III)

Epidemiology

- 5-10% of the adult population in the US
- Steadily increasing in incidence over the years for unknown reasons
- Onset is usually childhood but can begin at any age
- M&M has increased over the years

Mechanism

- Increased Underlying Risk
  - Genetics
  - Ethnicity: AA>Caucasian
  - Presence of Atopy: asthma + allergic rhinoconjunctivitis + eczema
  - Social Status: Poor & Developing Countries > Affluent & Industrial Countries
  - “Hygiene Hypothesis”: It has been suggested that early in life the absence of exposure to infections, other children, etc aka “country living” skews pts towards a Th2 phenotype (which worsen asthma) vs Th1 phenotype (which improve asthma)

- Exposure to a Precipitant
  - **Allergens**
    - Outdoor (usually seasonal)
      - Winter: Mountain Cedar Tree
      - Spring: All Trees
      - Summer: Grass
      - Fall: Weeds
    - Indoor (usually perennial)
      - All Year Round: indoor mold, house dust mite, cockroach, animal dander
  - **Pollutants**: environmental, smoke, etc
  - **Exercise**-Triggered Asthma (vs Exercise Induced Bronchospasm) pt usually has other asthma precipitants
    - NB some pts especially adults do not have a known allergen

- Acute on Chronic HYPER-RESPONSIVE REVERSIBLE (at least partially) INFLAMMATORY response to a stimulus resulting in AIRWAY OBSTRUCTION from main bronchus down to most distal subunits along with underlying REMODELING
  - **Acute Reversible Airway Obstruction** due to degranulation and release of preformed protein mediators (histamine, heparin, serotonin, proteases, tryptase, etc) as well as rapid synthesis and release of lipid mediators (leukotrienes, prostaglandins, cytokines, chemotactic factors, growth factors, etc) from Mast Cells and Neutrophils
    - (1) bronchoconstriction 2/2 bronchial smooth muscle contraction
    - (2) mucus build-up to inspissated mucus plugs due to stimulation of mucosal goblet cells and submucosal mucus glands
    - (3) edema and inflammatory infiltrate
  - **Chronic Irreversible Airway Obstruction** (aka Airway Remodeling) due to Mast Cell release of late mediators (above) during the acute response which attract Eosinophils which in turn release additional substances which trigger remodeling
    - (1) smooth muscle hypertrophy
    - (2) mucosal goblet cell and submucosal mucus gland hypertrophy
    - (3) epithelial denudation, thickened basement membrane, angiogenesis, inflammatory infiltrate, fibrosis, etc

DDx (“not all that wheezes is asthma”)

- Asymptomatic GERD: PPIs are shown not to be helpful in asthma control if a pt has asypm GERD
- **Other** Inflammatory Conditions (eg. COPD, CF, Bronchiectasis, Bronchiolitis Obliterans, Diffuse Panbronchiolitis)
  - Bronchiolitis Obliterans or Obliterative Bronchiolitis
    - Pt: young w/ h/o RA, connective tissue dz or fume exposure
    - Imaging: hypodense areas
    - Tx: high dose steroids but despite Tx the disease is progressive w/ most pts dying from respiratory failure
  - Diffuse Panbronchiolitis
    - Pt: non-smoking Asian men w/ h/o chronic sinusitis
    - Imaging: diffuse small centrilobular nodular opacities
    - Tx: long term macrolides
- **Infammation**
  - **NSAID**: aspirin inhibits cyclooxygenase but NOT lipoygenase thus tipping the balance to bronchoconstrictive leukotrienes rather than bronchodilative prostaglandins 1hr after ingestion (“Samter’s Triad” asthma, severe recurrent rhinitis/sinusitis, nasal polyps), pts can be de-sensitized to ASA if they need to take it
  - **Opiates**: cause histamine release
- **Bronchospasing**
Exercise Induced Bronchospasm [vs Exercise Triggered Asthma]: pt has NO other asthma precipitants only exercise, usually seen in only elite athletes, ICS do NOT work, essentially a DOE, heat/water loss (desiccation) from airways esp in cold/dry weather occurs AFTER exercise (short more intense sprints > long less intense jogs) w/ DOE, etc resolving spontaneously, do not avoid exercise as being fit decreases the bronchospasm, Tx w/ SABA 15min b/f exercise, warm up before exercising, avoid cold/dry weather

Atypical U1: Virus/Mycoplasm induces inflammation which lowers the threshold of subepithelial vagal receptors to irritants particularly air pollutants resulting in parasympathetic mediated bronchoconstriction

BB: block sympathetic mediated bronchodilation of airways

Reactive Airway Dysfunction Syndrome (RADS) suspect if pt improves when home/work, on vacation, etc

- Fumes, Chemical Dust, etc
- GERD (innervations of esophagus are closely related to innervations of airways therefore irritation from GERD can result in bronchospasm, in addition reflux can be severe enough that it reaches airways)

Other: Cold Air, Emotional State, Menstruation, Change in Weather, Sulfites in foods

Structural Airway Obstruction

- Vocal Cord Dysfunction (VCD): abnormal adduction during active ventilation esp inspiration, consider in difficult to treat asthmatic esp elite athletes, often 2/2 GERD, postnasal drip, prior intubation, Dx w/ fiberoptic laryngoscopy but suggested by flattening of flow-volume loop, Tx w/ speech therapy and treat GERD and postnasal drip (NB often VSD AND asthma coexist)
- Tracheobronchomalacia: loss of cartilage that keeps airways open during expiration, 2/2 pressure necrosis from prolonged intubation or chest wall trauma or inflammatory disease of cartilaginous structures, Tx w/ stent placement
- Foreign Body Aspiration
- Tumor or LAD
- Vascular Rings or Laryngeal Webs
- Tracheal/Bronchial Stenosis
- Choanal Atresia

Allergic Broncho Pulmonary Aspergillosis (ABPA) (refer to “Fungus” notes)

Churg-Strauss Syndrome (CSS) (refer to “Vasculitis” notes)

ACE-I induced Coughing (very common)

S/S

- Expiratory Wheezing (NB “not all asthma wheezes”, cough variant asthma is very common esp in children), Chest Tightness, Dyspnea/SOB
  - manifest w/in 30min of exposure
  - worse during late night and early morning
  - many times pt is asymptomatic and has a benign normal PEEx b/t episodes
- If atopic then asthma + eczema + rhinoconjunctivitis
- If ASA then asthma + nasal polyps + rhinosinusitis (“Santer’s Triad”)

Complications

- Remodeling
- Status Asthmaticus

Dx

- FHx
- H&P
- PFTs (to confirm S/S, 3 maneuvers for >6sec w/ results w/in 5% of each other)
  - If Normal PFTs (seen in early mild dz) then Bronchoprovocation Test (increasing doses of methacholine {Ach+ which bronchoconstricts} are administered to pt until FEV1: drops by >20% if the “Provocative Concentration” (PC20) needed is <8mg/mL then hyperresponsiveness of airways is diagnosed but really it is mainly not for diagnosis but to exclude asthma in pts with somewhat atypical Sx (like only cough) and nl spirometry b/c the test has a high NPV
    - Obstructive PFTs (↓FEV1, ↓FVC, ↓FEV1/FVC, ↓FEF25-75%) Spirometry, concave flow-volume loops
    - Most pts when asymptomatic and stable and mild have nl lung volumes but they can change (↑ RV 2/2 air trapping, ↑ TLC, ↓ VC) Helium Dilution Technique / Body Plethysmography NB dyspnea correlates w/ increased RV and reduction of IC
    - (↑ DLCO asthma vs. ↓DLCO emphysema vs. =DLCO bronchitis)
- Bronchodilator Test: short acting beta-2 agonist is administered to pt and if FEV1 increases by >12% AND > 200mL then reversibility of obstructive airways is diagnosed
- Normal PFTs
- Peak Flow Meter which measures PEFR (greatest velocity that can be obtained during a forced exhalation starting with the lungs fully inflated), varies with age/gender/race/height but in average 500L/min is normal, good surrogate for degree of obstruction, have pt document personal best aka when they have no sx so as to compare to when pt is sick, Walgreens has a good one called the Peak-O-Meter that costs about $30 and also measures FEV1

Imaging
• CXR: usually normal but sometimes mucus plugs, bronchial markings, flattening of diaphragm, hyperinflation, and pneumothorax can be seen
• CT: airway thickening, mucus plugs, etc

• BAL
  • Creola Bodies (mucus plugs also contain whorls of desquamated epithelia)
  • Curschmann Spirals (mucus casts)
  • Charcot-Leyden Crystals (crystals of eosinophil membrane protein)
  • Eosinophils
  • Endobronchial Bx (histology above)

• Other
  • Fractional excretion of NO (FeNO) is proportional to airway inflammation and sputum eosinophilia
  • If considering allergic as cause
    • CBC: eosinophilia
    • IgE: elevated but if >1000 then suggests ABPA
    • Skin Testing: intradermal injection of suspected allergen and saline control in forearm or back
    • RadioAllergo Sorbent Test (RAST): place pt's serum over a solid phase carrier embedded w/ the suspected allergen, incubate, wash serum off, add radioactive or enzymatic (horseradish peroxidase) labeled anti-IgE Ab, more specific but less sensitive than skin testing therefore RAST is indicated in those pts who are so exquisitely sensitive to an Ag that skin testing may yield a systemic reaction

Tx (bronchodilators and anti-inflammatories)
• Education
  • Explaining Disease Process
  • Explaining Difference b/t Acute and Chronic Tx
  • Monitoring Disease thru PEFR and FEV1 not so much Sx (esp if severe asthma, h/o exacerbations, or pt poorly perceives obstruction)
    • good to monitor at home not for diagnosis due to wide variety of types
    • refer to charts which factor in age, height, gender for nl rates or compare to pts best PEFR in past to determine how they are managing their asthma
    • Peak flows are cheaper devices but they are more effort dependent than true spirometry therefore good for home monitoring but when in clinic true spirometry is better
  • Avoid Triggers and/or Immunotherapy aka Desensitization
  • Action Plan (refer below)
  • Medication Technique
    • MDI (Meter Dose Inhaler) high velocity liquid propellant (inhale deep and slowly over 3sec)
      • w/ value holding chamber (eg Aerochamber) decreases deposition of drug into mouth b/c holds onto large particles while allowing small articles to go into airway at a slower speed which don't hit posterior pharynx but go down trachea (spacers was the first form but now not used anymore)
      • the propellant used to be CFC but they are now being changed to HFA b/c less harmful to ozone
      • BUT tell pts that if they use an HFA inhaler that they (1) shake it and (2) prime it twice if not used w/in 2wks
    • DPI (Dry Powder Inhaler) high velocity powdered propellant (inhale deep and fast over 1sec)
      • b/c you have to inhale deep and fast it is not recommended in severely obstructed pts b/c so hard to do
      • likely delivers more medicine than MDI
    • Neb (Nebulizer) low velocity liquid propellant
      • Med delivery is much better than MDI and DPI
  • Acute Treatment ("Relievers" or "Rescuers")
    • Short Acting Beta-2 Agonist (SABA): albuterol (AccuNeb, ProAir, Proventil, Ventolin, VoSpire) MDI/DPI/NEB, pirbuterol (Maxair) MDI, levalbuterol (Xopenex) NEB
      • Mechanism: bronchodilates after 3 minutes of inhalation and last 3 hours
      • SES: tremor, palpitations, etc
      • Combo: albuterol + ipratropium (Combivent, DuoNeb) MDI/NEB (concomitant inhalation of anticholinergics increases SABA delivery is controversial and asthma UNLIKE COPD is not a very vagal mediated process hence anticholinergics are not that helpful)
      • Most drugs are aerosolized for delivery but only 10% actually reach lung the remaining 90% are swallowed resulting in systemic side effects therefore newer drugs are being designed which are poorly absorbed by GI and/or inactivated by first pass hepatic metabolism
      • Albuterol isomer is bullshit
      • Asthma Clinical Research Network Trial showed there are beta-receptor polymorphisms and those pts (1/6 of asthmatics) who do NOT have the wild type amino acid at residue 16 (Gly/Gly) but have the polymorphism (Arg/Arg) have less of a response to SABAs and might have more harmful effects
    • Short Acting Cholinergic Antagonist: ipratropium Br (Atrovent) NEB/MDI
      • Mech: blocks parasympathetic bronchoconstriction
      • SES: dry mouth
- Ipratra: 1" M2 2" M3 (short acting 6hrs)
- M2 is a feedback inhibition receptor therefore you don’t want to block it
- M3 is the true constriction receptor therefore you want to block it
  - Corticosteroids
    - Systemic (PO)
    - Mech: reduce expression and action of pro-inflammatory mediators and increase expression of anti-inflammatory mediators with effect after one week and lasting for months
    - SEs: (refer)
  - Chronic Treatment ("Controllers" or "Preventers")
    - Long Acting Beta-2 Agonist (LABA): formoterol (Foradil, Perforomist), salmeterol (Serevent) DPI
      - Mech: bronchodilates after 3 hours of inhalation and lasts 12 hours + decreases bacterial adhesion to airway epithelial cells resulting in less infections
      - SEs: ?
      - SMART Trial LABAs increase morality from severe exacerbations b/c LABAs downregulate expression of beta-receptors therefore when you use SABAs they don’t work as well (recently FDA placed a "Black Box Warning") therefore the most important thing is to get pts under control minimizing risk of exacerbation if they are on a LABA and they should never be used alone aka w/o an ICS
      - Combo: formoterol + budesonide (Symbicort) MDI, salmeterol + fluticasone (Advair) MDI/DPI
    - Long Acting Cholinergic Antagonists: tiotropium Br (Spiriva) DPI
      - Tio: 1" M3 2" M2 (long acting 24hrs)
    - Inhaled Corticosteroids (ICS): flunisolide (AeroBid) MDI, mometasone (Asmanex) DPI, triamcinolone (Azmacort) MDI/NEB, fluticasone (Flovent) MDI/DPI, budesonide (Pulmicort) DPI/NEB, beclomethasone (QVAR) MDI/DPI/NEB
    - INH (?)
      - Mech: reduce expression and action of pro-inflammatory mediators and increase expression of anti-inflammatory mediators with effect after 4-6weeks w/ max effect after 6months (ICS also decrease the amount of mucus produced)
      - SEs: thrush (rinse mouth w/ water), dysphonia/hoarseness 2/2 laryngeal muscle steroid myopathy (use a spacer, lower dose, use DPI), and very rarely systemic SEs
      - More important than any other long-term medicine
      - FeNO can predict responsiveness and guide treatment
      - Compliance is a big issue b/c pts don’t see the benefit therefore educate pts
      - 1% of asthma pts are "steroid insensitive" b/c they have a genetic abnormality that causes impaired glucocorticoid receptor production, Tx very high dose systemic steroids, IVIG or MTX
      - There is some evidence that ICS actually reverse remodeling
      - There is some evidence that ICS upregulate beta-receptors therefore augments SABA/LABA effects which is why steroids help even early on (before the anti-inflammatory effects kick in at 6hrs)
    - Leukotriene Receptor Antagonists (LTRA): montelukast (Singulair), zafirlukast (Accolate)
      - Systemic (PO)
      - Mech: competitive inhibitors of leukotriene receptor (bronchoconstrictors) with effect after 3 hours
      - SEs: montelukast (N/V) vs zafirlukast (N/V, mild hepatic, mild drug-drug interaction)
      - good in aspirin induced asthma and for prophylaxis for exercise induced asthma, very hit or miss drug in that in some pts it does great but in others it does nothing
      - There was the belief that the use of these agents DEVELOPED chynestraus but now the thinking is that when added for steroid sparing effect the decrease dose of steroid simply unmasked the underlying vasculitis which can ONLY be treated with steroids
      - NB Zileuton (Zyflo CR) competitive inhibitor of 5-lipoxygenase, not used much anymore b/c of SEs (N/V, severe hepatic, significant drug-drug interaction)
    - Mast Cell Stabilizers (MCS): cromolyn Na (Intal), nedocromil Na (Tailade)
      - INH (MDI)
      - Mech: unknown but somehow they inhibit degranulation of mast cells
      - SEs: cromolyn (cough, pharyngitis) vs nedocromil (cough, dysgeusia, dysphonia)
      - Essentially good when you know the exact trigger (aka exercise, specific allergen, etc) b/c it should only be given right before hand as kind of a prophylactic
      - Good in young pts b/c you don’t want to use steroids
    - Methylenxanthines: theophylline (Elixophyllin, Uniphyl)
      - Systemic (PO)
      - Mech: increase cAMP by inhibiting phosphodiesterase and/or competitive antagonism of Gi receptor resulting in bronchodilation
      - SEs: CNS (anorexia, anxiety, HA, insomnia, tremors, AMS, seizures, coma), CV (hypotN, arrhythmias esp MAT), GI (N/V, D, GERD), Renal (hypok)
      - Check plasma concentrations (nl 8-12µg/mL) as you can develop seizures/arrhythmias w/o other lesser SEs and other drugs can increase/decrease concentrations (overall very narrow TI)
      - Good in pts hard to control
      - Good in pts w/ nocturnal asthma
    a. Anti-IgE: omalizumab (Xolair)
      i. SC injection
ii. Mech: binds IgE preventing it from stimulating inflammatory cells
iii. SEs: malignant neoplasms and anaphylaxis
iv. consider in severe allergic type asthma where IgE/eosinophilia is moderately high (not too high b/c then not that effective)

b. Research: anti-TNFs, anti-IL4/5/13, anti-CD23
c. Other: Bronchial Thermoplasty (radiofrequency destruction of airway smooth muscle, decreases Sx and Hz of exacerbations but does not change FEV1)

Treatment Strategy

- Initial Diagnosis: Classify Severity (the intensity of the disease process)

<table>
<thead>
<tr>
<th>Severity Stage</th>
<th>Day Sx</th>
<th>Night Sx</th>
<th>Interference w/ NI Daily Activity</th>
<th>FEV1 Predicted</th>
<th>Treatment</th>
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</thead>
<tbody>
<tr>
<td>Mild Intermittent</td>
<td>&lt;2d/wk</td>
<td>&lt;2x/mo</td>
<td>None</td>
<td>&gt;80%</td>
<td>Step 1</td>
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<tr>
<td>Mild Persistent</td>
<td>&gt;2d/wk but not Qd</td>
<td>3-4x/mo</td>
<td>Minor</td>
<td>&gt;80%</td>
<td>Step 2</td>
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<td>Moderate Persistent</td>
<td>Qd</td>
<td>&gt;1x/wk</td>
<td>Some</td>
<td>60-80%</td>
<td>Step 3-4</td>
</tr>
<tr>
<td>Severe Persistent</td>
<td>Throughout the Day</td>
<td>Nightly</td>
<td>Much</td>
<td>&lt;60%</td>
<td>Step 5-6</td>
</tr>
</tbody>
</table>

- Follow-Up: Classify Control based on Impairment & Risk
  - NB this means you treat NOT based on severity as classified above but based on the goal of complete control with minimal impairment & risk (this is a subtle but important difference)
    - Impairment: frequency/intensity of Sx (Sx <2x/wk, NO Nocturnal Sx, NO Limitation of Activity, SABAs <2x/wk, ni PEFR or FEV1) and functional limitations (normal activity level)
    - Risk: likelihood of either: Asthma exacerbations (<2x/y), progressive decline in lung function or risk of adverse effects from medications
    - NB important to note that some pts can have minimal impairment but still be at risk hence two different criteria
      - Determine if you need to step up, maintain, or if tolerated for 3mo consider stepping down
      - Goal: “Rule of Two’s” (<2d/wk, <2x/mo, <2x10 drop in FEV1, <2inhalers/yr)
      - Frequency of Clinical F/U visits: Poor Control (Qmo) vs Maintaining Control (Q2-6mos depending on which step they are on)
      - Action Plan (refer to exacerbation section)
      - Influenza Vaccine
  - Exacerbations: Classify Severity
    - Hx: baseline PEFRs, past ED visits requiring hospitalizations and ICU care w/ intubation (best predictor of risk of death)
    - Current: duration of Sx, exact use of meds including amount of systemic steroids, known precipitants.
    - PEX: tachypnea, tachycardia, diaphoresis, cyanosis, telegraphic fragmented speech, absence of breath sounds w/ loss of wheezing, accessory muscle use w/ abdominal paradox, pulsus paradoxus (a decrease in SBP by >20mm, 2/2 lung overexpansion aka breath stacking which compresses the heart during inspiration), pursed lips w/ prolonged expiratory period, signs of barotrauma (PTX) including asymmetric breath sounds, tracheal deviation, Hamman’s crunch, et al
    - Dx: more important than ABGs (remember pt will be hypoxicem as expected but ALSO hypocarbic 2/2 tachypnea therefore if is eu/hypercarbic then concerning for respiratory muscles tiring out) and CXR (not helpful unless you suspect PNA or PTX) are O2Sat and PFTs (check PEFR compare to personal best and then check at 1hr (biggest predictor of hospitalization) then follow Q8hrs)
    - NB even mild asthmatics can get severe exacerbations
    - Below is also the pt’s Action Plan

<table>
<thead>
<tr>
<th>Severity Stage</th>
<th>Symptoms</th>
<th>Predicted FEV1 or PEFR of Baseline</th>
<th>Treatment Algorithm</th>
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<tbody>
<tr>
<td>Mild Exacerbation Green Zone</td>
<td>During physical activity</td>
<td>&gt;70%</td>
<td>Stay Home</td>
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<td>• CombiVent</td>
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<td>• if on ICS then QUADRUPLE (do NOT just double as shown not to be</td>
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<tr>
<td><strong>Exacerbation</strong></td>
<td><strong>Zone</strong></td>
<td><strong>Level</strong></td>
<td><strong>Action</strong></td>
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<td>Mod Exacerbation Yellow Zone</td>
<td>At rest</td>
<td>40-70%</td>
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<td>Go to ER/Office Visit and the go Home</td>
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<td>DuONeb</td>
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<td>Prednisone x7d (no taper needed)</td>
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<td>Humidified 100% O2 w/ Venturi Mask</td>
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<td>Set up appointment w/ MD to titrate chronic treatment</td>
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<td>If no improvement then refer below</td>
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<td>Severe Exacerbation Red Zone</td>
<td>At rest w/ concerning PEx findings like accessory muscle use, pulsur paradociousus, tachycardia, hypoxemia, hypercarbia, etc</td>
<td>&lt;40%</td>
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<td>Go to ER and then Admission</td>
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<td>&quot;&quot; and consider increasing steroid and SABA dose to 1mg/kg and 5mg</td>
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<td>Heliox via Venturi Mask (helium-oxygen mixture, helium lowers density of air therefore lowers viscosity of air so that it can pass more easily aka more laminar thru obstructed airways, you can actually hook up to Neb and use it to drive MedNeb) → BiPAP → Intubation w/ MV (if refractory hypoxemia, respiratory acidosis, or hypotension)</td>
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<td>Give a dose of Singular</td>
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<td>Give a PPI b/c increased reflux w/ SABAs</td>
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<td>MgSO4 2g IV over 2hrs (bronchodilates)</td>
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<td>Consider empiric PNA antibiotics if suspected</td>
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<td>IVF (pts are often dehydrated 2/2 resp losses and hydrating decreases pulmonary mucus viscosity)</td>
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<td>NO mucolytics (can actually cause bronchospasming)</td>
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<td>NO epinephrine (not proven to be of benefit)</td>
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<td>NO sedation</td>
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<td>Consider Admission if…</td>
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<td>severe exacerbation does not fall to mild after 3hrs of Tx = “status asthmaticus”</td>
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<td>any h/o ICU admit w/ intubation</td>
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<td>pneumonia, pneumothorax, etc</td>
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<td>other comorbidities</td>
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<td>Discharge once &gt;70%</td>
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