Asthma chronic inflammatory disorder of the airways in which inflammation causes varying degrees of obstruction and hyperactivity of the airway.
This inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and cough.
The airway obstruction may reverse spontaneously or with treatment. The course of asthma is unpredictable.
Incidence 30 million people in US affected (2006), About 4500 people die of asthma annually.
Patient's at greatest risk: African American adolescents, Lower mean socioeconomic groups

Triggers

Allergens

Most common allergens: dust mite, pollen, grass, and animal dander
Some people develop an exaggerated IgE response to certain allergens.
IgE is left on mast cells, and when a 2nd exposure occurs the allergen triggers degranulation (the process by which cytoplasmic granules (as of mast cells) release their contents) even years after initial exposure.

Respiratory Infections

One of the most common precipitating factors of an acute asthma attack.
Infections cause inflammatory changes in the tracheobronchial system and alter the mucociliary mechanism.
The infected bronchial system has an increase in the hyperresponsiveness of the bronchus, causing narrowing and asthma symptoms.
This type of asthma may last 2-8 weeks after the infection is over and or may last several months.

Nose and Sinus Problems

About 30% of asthmatics have chronic sinus and nasal problems.
These problems cause allergic rhinitis, (which can be seasonal or perennial) and nasal polyps.
These problems cause inflammation which in turn can affect the bronchus.

Exercise

Exercise induced/exacerbated: during physical exertion is called exercise-induced asthma (EIA).
Typically EIA occurs after several minutes after activity and is characterized by bronchospasm, SOB, cough, and or wheezing.
There are several treatments used prior to activity to prevent EIA.

Cold Temperature

Cold dry air can constrict the bronchus and cause bronchospasm and therefore trigger an asthma attack.
This can especially occur when in combination with other triggers.

Drug and Food Additives

About 12-15% of people with asthma have what is termed **asthma triad- nasal polyps, asthma, and sensitivity to ASA/NSAIDs.**
Some asthmatics will begin wheezing within 2 hours of ingestion.
Common drugs: beta blockers, ACE inhibitors, ASA, NSAIDs.
Common foods: yellow dye #5, vitamins, fruit, beer, wine, peanut/oil, monosodium gluconate, wheat

GERD

may cause stomach acid reflux into the esophagus that can be inhaled/ aspirated into the lungs and cause reflex bronchoconstriction.
Patients with hiatal hernia, excessive stress, middle obesity are at greater risk.

Emotional Stress

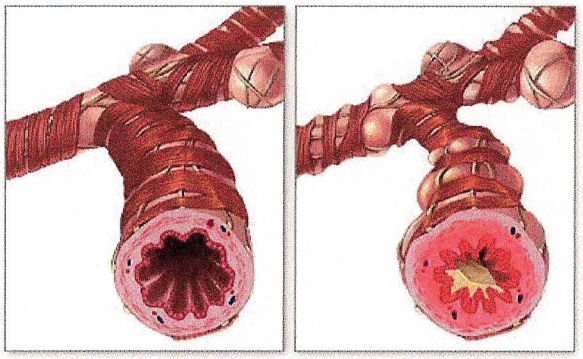
Asthma is **not** a psychosomatic disease.
Psychological factors can interact with the asthmatic response to worsen the disease process.
Panic and anxiety can trigger an attack.

Asthma Pathophysiology

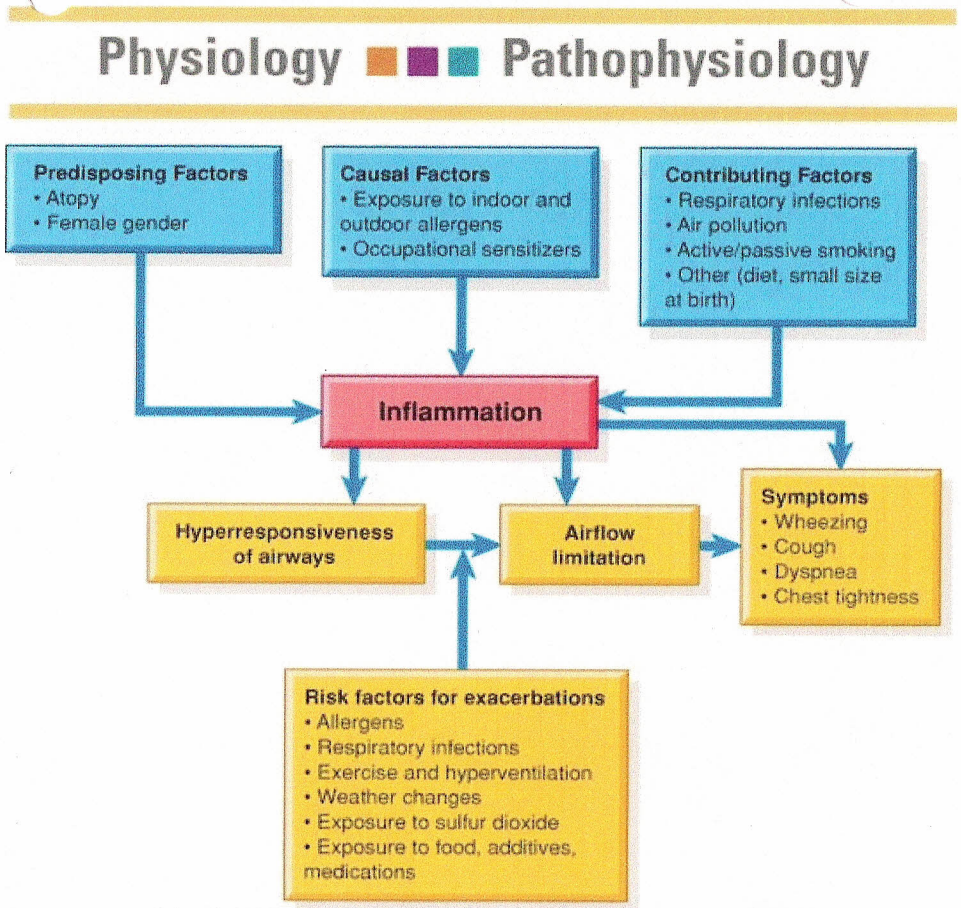
Hallmarks of Patho
Airway inflammation
Nonspecific hyperirritability
Hyperresponsiveness

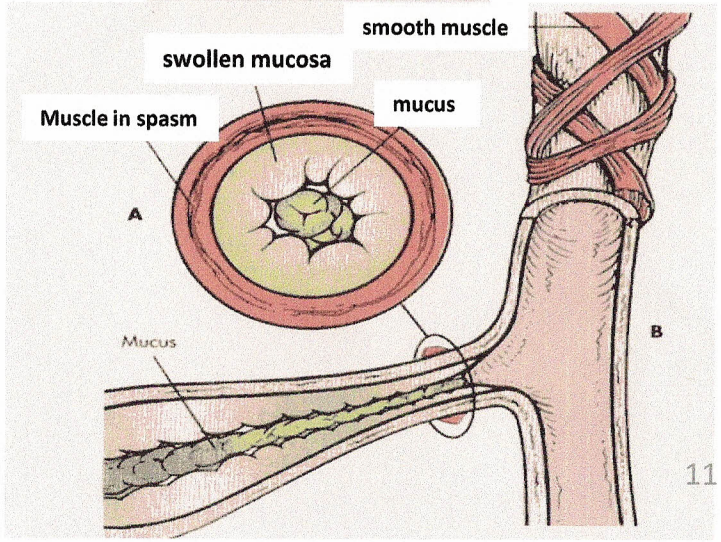
Inflammatory mediators (histamine, prostaglandins and leukotrienes) are released (by mast cells) in response to the trigger(s).
Chemotactic factors are produced that result in bronchial infiltration by neutrophils, eosinophils and lymphocytes. This ultimately will allow epithelial cell damage. Airway "remodeling" may occur in response to chronic inflammation, causing further airway narrowing.

- These (mediators, chemotactics) cause bronchial smooth muscle spasm, vascular congestion, increased vascular permeability, edema formation, production of thick tenacious mucus, impaired mucociliary function, thickening of the airway walls, increased contractile response of the bronchus.
- All of theses changes with the epithelial cell damage produce hyperresponsiveness and obstruction.
- Airway obstruction increases resistance to air flow and decreases flow rates, primarily expiratory flow.
- Impaired expiration causes hyperinflation distal to obstructions and increases the workload of breathing.
- Airway resistance becomes uneven.
- Hyperventilation is eventually triggered by lung receptors responding to increased lung volume from air trapping and obstruction.
- Gas pressures rise, causing ↓perfusion of the alveoli.
- The result is early hypoxemia without CO_2 retention
  $PaCO_2$ decreases and ph increases (respiratory alkalosis).
- More severe conditions result in increase in CO_2 retention and respiratory acidosis. This signals respiratory failure.



Expiratory obstruction: Air spaces distend beyond the terminal bronchioles
Factors causing expiratory obstruction





Asthma Manifestations – S&S **Most common syptoms: Wheezing, SOB,** breathlessness, **chest tightness,** nasal flaring, and **cough,** may occur particularly at night (related to circadian rhythms) and in the early morning.

Onset may be abrupt or gradual.

Pt may be asymptomatic w/ normal or abnormal pulmonary function bt attacks

As exacerbation progresses, diaphoresis, tachycardia, and widened pulse pressure may occur along w/ hypoxemia & central cyanosis (a late sign of poor oxygenation).

Severe, life-threatening hypoxemia can occur in asthma, but it is relatively uncommon.

The hypoxemia is secondary to a V/Q mismatch and readily responds to supplemental O2.

Wheezing is an unreliable sign to gauge the severity of an attack.

Some people may have no audible wheezing because of marked reduction in the airflow. This is known as "tightness".

Wheezing usually occurs first on exhalation, but progresses during both phases of inspiration.

Cough may be the only symptom w/ some pts

Bronchospasm may not be severe enough to cause airflow obstruction, but it can ↑bronchial tone and cause irritation and stimulation of the cough receptors.

Cough may be nonproductive. Secretions may be thick, tenacious, white, or gelatinous.

Physical Examination

Wheezing or decreased/absent breath sounds
(this may indicate atelectasis or pneumothorax)
Anxiety
Restlessness
BP, P, R increases
Pulsus paradoxus greater than 12 mm HG
Percussion indicates hyperresonance
Accessory muscle use
Nasal flaring

Status Asthmaticus when severe bronchospasm is **not reversed by usual measures**

If it continues hypoxemia worsens, expiratory flows and volumes decrease further, and the individual can develop respiratory acidosis and failure.

Asthma is life threatening at this point.

A silent chest (no audible air movement) and a PCO2 over 70 mm HG are ominous signs, generally pt is intubated or trached, placed on vents and admitted to ICU.

Additional manifestations:
- Fear of suffocation
- Increased work of breathing
- Diaphoresis (absence may indicate dehydration)
- Sternocleidomastoid, intercostal, and supraclavicular muscle retractions Peak flows are less than 100 L/minute.
- Quiet chest
- Neck vein distention
- Pulsus paradoxus of 40 mm Hg or higher
- Hypertension, tachycardia, ventricular arrhythmia

Asthma Complications

Pneumothorax –
partial/complete collapse of lung due to + pressure in the pleural space
Pneumomediastium –
abnormal state characterized by presence of gas (as air) in mediastinum
Atelectasis – collapse of alveoli
Pneumonia
Status asthmaticus – severe, unreversed bronchospasm

Status Asthmaticus Complications

Pneumothorax
Pneumomediastium
Acute cor pulmonle –
enlargement of right ventricle from hypertrophy or dilation
Right ventricular failure
Respiratory arrest
Death

Diagnosis of Asthma

Physical exam

Diagnostics

Spirometry (PFT) reveals:

↓expiratory flow rate, forced expiratory volume (FEV),
↓ forced vital capacity (FVC); FEV1
↑functional residual capacity (FRC)
↑ total lung capacity (TLC)

ABGs: hypoxemia, w/early respiratory alkalosis & late respiratory acidosis (2^{nd} to obstruction of expiratory air flow w/ air trapping and ↑lung volumes)

Peak flows are done at home, for mild asthma and as a management tool

Eosinophils in the sputum and serum eosinophilia

(> or equal to 5% of the total white blood cell)

In symptomatic pts a chest x-ray shows hyperinflation & ↑IgE lvls show allergy

Allergy testing: to determine sensitivity to antigens;

Sputum specimen for Gram's stain & culture to R/O other infections.

Treatment

Acute Attacks

Oxygen: mask, NC
Monitoring: BP, P, R, pulse oximetry, ABGs, cardiac monitoring
Nebulizers: bronchodilators and anti-inflammatory medications
IVs
Aminophyllin drip
I&O
IV anti-inflammatory agents: steroids
SQ B agonists: epinephrine
Hospitalization if necessary

Control /Prevention

The goal is **control and prevention** of asthma symptoms.
Teach about medications, dosing and techniques
Importance of hydration, dehydration exacerbates asthma.
Diet: dairy may exacerbate asthma. Small frequent well nourished meals.
Avoid icy cold beverages.

Adequate rest and sleep
Avoid triggers
Decrease stress
Contact HCP or go to ED for uncontrolled or danger symptoms.
Medication treatments today are based on the number of attacks the pt has.
The treatments are based on "**steps**".

Step I- Mild Intermittent:

Symptoms no more frequent than 2X weekly,
and are asymptomatic w/ normal PEFR bt exacerbations
•Exacerbations are brief (hours to days)
•Intensity of exacerbation varies
•Nocturnal symptoms: no more than twice monthly
Medications: No daily medications
Rescue_medication: short-acting inhaled B2 agonist.

Step 2: Mild Persistent

Symptoms > 2 X week, but less than once per day.
Exacerbations may affect activity
Nocturnal symptoms more frequent than 2 X month
Daily Medications: Anti-inflammatory agents such as low-dose inhaled glucocorticoids or cromolyn or sustained-released theophylline. Leukotrienes modifiers may be considered for ages> 12 years of age.
Rescue medications:
Short-acting inhaled B2 agonist
Daily use or increasing use indicates need for additional long-term therapy.

Step 3 Moderate persistent

Symptoms: Daily asthma symptoms, exacerbated with activity; may last for days; uses daily B2 agonists.
Nocturnal symptoms: more frequent than once weekly.
Daily medications: one or 2 daily medications: anti-inflammatory agent (medium dose of inhaled glucocorticoid) and or medium-dose inhaled glucocorticoid and a long acting bronchodilator.
Rescue Medications:
Short acting inhaled B2 agonist
Daily use or increased use indicates need for additional long-term therapy.

Step 4 Severe Persistent

Symptoms: continual symptoms, limited activity and frequent exacerbation.
Nocturnal symptoms: frequent
Daily Medications: two daily medications: anti-inflammatory agents (high-dose inhaled glucocorticoid) and long-acting bronchodilator (inhaled or B2 agonists) or theophyllin **and** oral glucocorticoid.
Rescue Medications:
Short-acting, inhaled B2 agonists.
Daily use or increasing use indicates need for additional long-term therapy

Patient Teaching majority of asthma medications are administered by inhalation. Inhalation technique must be correct for pt to receive medication.

MDI (Two common MDI: Servent and Flovent)

Techniques for administration of aerosol MDI

Closed mouth technique:
Patient shakes inhaler, exhales, lips around actuator, pump canister and inhale. Patient holds breath for a count of 10 (seconds) then exhales.

Open mouth technique:
As above, but pt holds actuator about 3 inches away from their mouth.

Spacer or Aero Chamber:
a tube attaches to the actuator and canister. The rx is dispersed in the tube so that pt gets entire dose. Also used for clients with poor coordination.

Auto inhaler: MDI that are "breath-activated" and require no coordination.

Combivent MDI inhaler containing Atrovent and Proventil

Diskus - Serevent Diskus

breath-activated and have no aerosol. These have a powder that is scraped off microscopically and inhaled.

Has a counter that tells the client how many doses are in the discus.

Developed in response to the concern of aerosol environmental issues.

Miscellaneous Instructions

The metered dose inhaler (except discus) should be cleaned by rinsing it with warm water.

If using more than one inhaler, use the B2 agonist first, wait 5 minutes, than use the anti-inflammatory.

Rinse mouth after each inhaled anti-inflammatory.

Teach the importance not to overuse inhaler.

Medications

Non-prescription (OTC): should be avoided unless directed by the HCP. Some OTC may increase risks for side effects/ toxic effects of prescription.

Peak Flows

Check the peak flow and record the values daily or as needed.

Rationale: Pts w/ asthma frequently do not perceive changes in their breathing, they're used to breathing at lower lung capacity.

The best of 3 attempts is utilized. Three zones are taught to the client: green, yellow and red.

Pt takes a deep breath and blows into the device as hard as they can (FEV1). Air loss from the nose must be controlled.

Length of breathing is not measured in peak flow measurement

Green zone means pt is within 80-100% of his best value.

Yellow zone means pt is within 60-80% of the best value. Strategies may be employed by the patient based on the asthma plan to return the pt to the green zone.

Red zone means pt is <60% of his personal best. This is the danger zone and the pt must take definitive action. A plan should be in place prior to this happening.

Pursed–lip breathing: Prolonged exhalation to prevent bronchiolar collapse and air trapping. Pt is taught to inhale through the nose slowly, and then exhale slowly through pursed lips. Done for retraining purposes **not for an acute attack.**

Diaphragmatic breathing or abdominal breathing focuses on using the diaphragm instead of the accessory muscles to achieve maximum inhalation and to slow the respiratory rate. This technique increases lung expansion.

Pt supine or semi-Fowler's placing one hand on the chest and the other on the abdomen. The client observes which hand moves during inspiration. The abdomen should protrude on inhalation with diaphragmatic breathing and contract on exhalation.

Prevention
Goal is control and prevention of acute exacerbations
Teach about meds
Importance of hydration,
Avoid dairy
Avoid icy cold bev.
Adequate rest and sleep
Avoid triggers
Decrease stress
Contact HCP or go to ED for uncontrolled/danger symptoms.

Teaching
MDI technique
Avoid OTC
Ck Peak flow Zones
Asthma diary & Plan
Pursed lip breathing
Diaphragmatic breating
SE of Albuterol (can sub Zofinex

Risk
African American adolescents
Lower socioeconomic groups

Triggers
Allergens/pollution
Resp infxns
Nasal/sinus problems
Exercise
Cold dry air
Rx & food additives
GERD
Emotional stress

Maintenance Tx/Meds
All steps get Rescue: short-acting inhaled B2 agonist
Step I- Mild Intermittent:
No daily medications
.Step 2: Mild Persistent
Daily: low-dose inhaled glucocorticoids or cromolyn or sustained-released theophylline. Leukotrienes modifiers may be considered for > 12 yo
Step 3 Moderate persistent
Daily: 1-2 daily meds:
Med dose inhaled glucocorticoid or med dose inhaled glucocorticoid &long acting bronchodilator.
Step 4 Severe Persistent
2 daily meds: high-dose inhaled glucocorticoid and Inhaled B2 agonists or theophyllin **and** oral glucocorticoid.

Asthma

Hallmark Patho: airway inflammation, hyperirritability, hyperresponsiveness.
Chronic inflammatory disorder of the lungs that causes varying degrees of recurrent airway obstruction (of expiratory air flow w/ air trapping and ↑lung volumes) **and hyperactivity. Reversible (sometimes spontaneously). Exagerrated IgE response. Reversilbility distinguishes it from COPD**

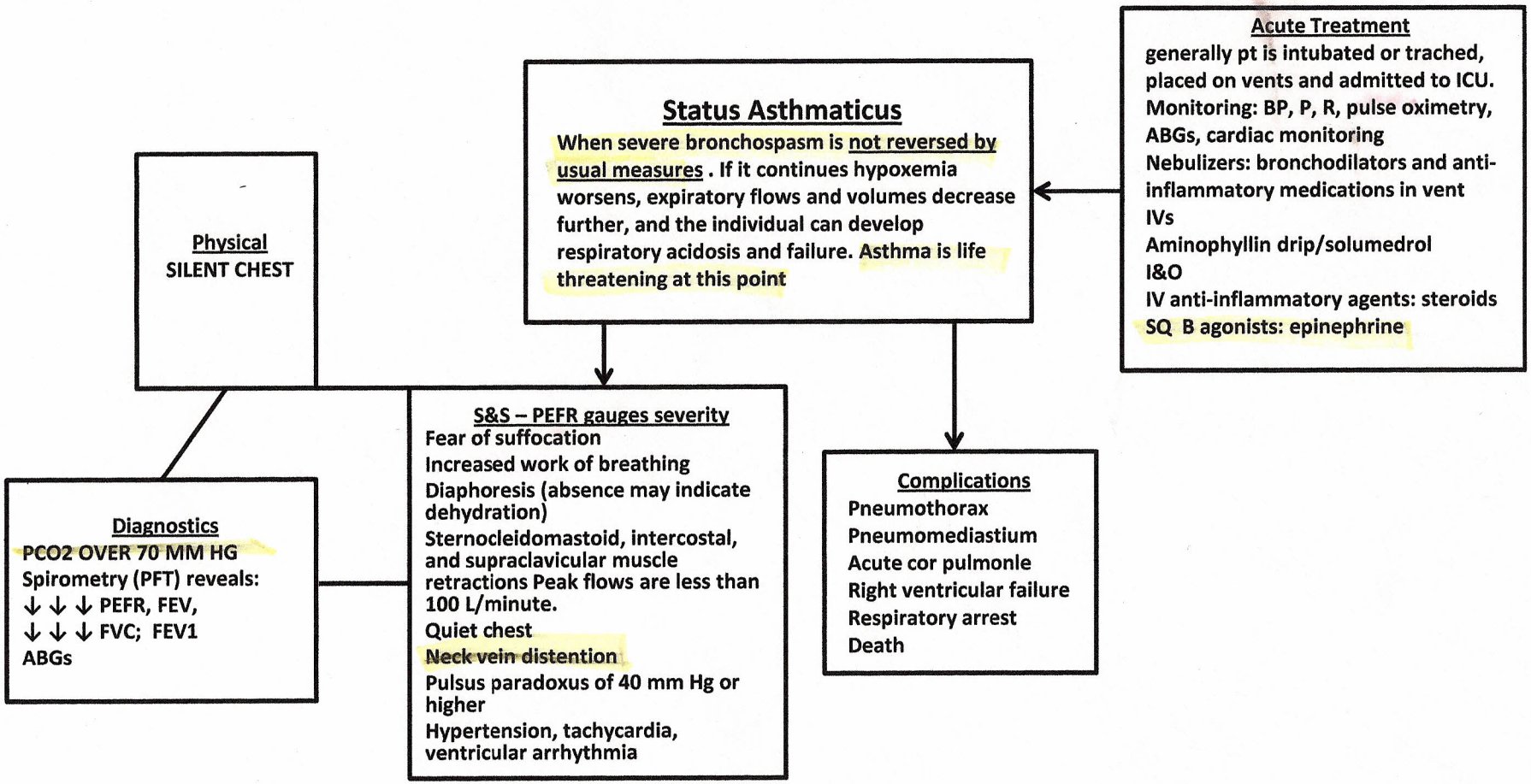
Physical
Wheezing or decreased/absent
Anxiety
Restlessness
Up BP, HR, RR
Pulses paradoxus > 12mm/HG
Hyperresonance on percussion (air trapping)
Acessory muscle use

S&S – PEFR gauges severity
WHEEZING (on expiration, progresses to include inspiration), SOB, CHEST TIGHTNESS, COUGH,
Nasal flaring,
Nocturnal may be worse
Abrupt or gradual onset
As it progresses: tachycardia, diaphoresis, widened pulse pressure, along w/ hypoxemia & central cyanosis.
Pt may be asymptomatic bt attacks w/ normal or abnormal lung fxn.

Diagnostics initial*
Spirometry* (PFT) reveals:
↓PEFR, FEV,
↓ FVC; FEV1
↑FRC
↑ TLC
ABGs
Peak flows (PEFR)*
WBC Eosinophils
CXR*
CBC*
↑IgE lvls
Allergy testing
Sputum specimen & culture

Complications
Pneumothorax
Pneumomediastinum
Atelectasis
Pneumonia
Status asthmaticus

Acute Treatment
Oxygen: mask, NC
Monitoring: BP, P, R, pulse oximetry, ABGs, cardiac monitoring
Nebs
IVs
Aminophyllin drip
I&O
IV anti-inflammatory agents: steroids
SQ B agonists: epinephrine
Bedrest w/ bedpan
Hospitalization if necessary


Status Asthmaticus
When severe bronchospasm is not reversed by usual measures . If it continues hypoxemia worsens, expiratory flows and volumes decrease further, and the individual can develop respiratory acidosis and failure. Asthma is life threatening at this point
Acute Treatment
generally pt is intubated or trached, placed on vents and admitted to ICU.
Monitoring: BP, P, R, pulse oximetry, ABGs, cardiac monitoring
Nebulizers: bronchodilators and anti-inflammatory medications in vent
IVs
Aminophyllin drip/solumedrol
I&O
IV anti-inflammatory agents: steroids
SQ B agonists: epinephrine
Physical
SILENT CHEST
S&S – PEFR gauges severity
Fear of suffocation
Increased work of breathing
Diaphoresis (absence may indicate dehydration)
Sternocleidomastoid, intercostal, and supraclavicular muscle retractions Peak flows are less than 100 L/minute.
Quiet chest
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Pulsus paradoxus of 40 mm Hg or higher
Hypertension, tachycardia, ventricular arrhythmia
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Diagnostics
PCO2 OVER 70 MM HG
Spirometry (PFT) reveals:
↓ ↓ ↓ PEFR, FEV,
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ABGs