COPD

Cline: ch. 37
Brady pp 583; 694
(+/or other standard paramedic text)

Objectives

After this lecture you should be able to discuss :

- Etiology , frequency, and mortality of COPD
- Clinical presentation of COPD
- Methods of quick and accurate recognition and appropriate aggressive management of decompensating COPD
- Common first line therapies as well as some less common treatments
- Complications of COPD disease state and of the treatments for COPD

Assessment and Treatment Goals

- Presentation
  - Acute deterioration
    - Reason for deterioration
    - Complications which may be present
- Correct tissue hypoxia to prevent rapid deterioration and respiratory failure:
  - Recognition - quick and accurate
  - Intervention - aggressive and prompt

Epidemiology

Chronic obstructive pulmonary disease (COPD)

- Affects 32 million persons in US
- 5% of the US population
- 7-10% of the elderly population
- Uncommon below age 40
- Sixth leading cause of death in the world
- Fourth leading cause of death in US
- One of the only two leading causes of death that is actually increasing

Etiology

Chronic obstructive pulmonary disease (COPD)

- tobacco abuse
- Other
  - cystic fibrosis
  - alpha-1 antitrypsin deficiency
  - bronchiectasis
  - air pollution
  - occupational exposure (eg, coalmining)

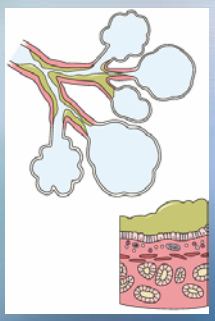
Pathophysiology

COPD

- Mixture of 3 separate disease processes:
  - Chronic bronchiti
  - Emphysema
  - Asthma

Chronic Bronchitis

- Excessive mucus production
  - hyperplasia of mucus-producing glands
  - with *airway obstruction*
- Endothelium damage
  - impaired clearing of bacteria and mucus
- Pulmonary capillary bed
  - relatively *un*damaged
  - (in contrast to emphysema)



Chronic Bronchitis V/Q Mismatch

Normal blood flow to *poorly ventilated lung*

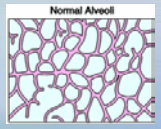
- Hypoxemia
  - Polycythemia (excessive RBCs)
- Increased CO_2 retention
  - Respiratory acidosis develops
    - pulmonary artery vasoconstriction
    - cor pulmonale (with signs of right CHF)
- Appears cyanotic ("blue bloaters")

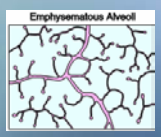
Emphysema Pathophysiology

- Gradual *airway destruction* of
  - alveolar septae
    - not available for gas exchange
  - pulmonary capillary bed
    - greater resistance to pulmonary blood flow [pulmonary hypertension]
      - leading to RCHF
  - leading to decreased ability to oxygenate blood

History

- Worsening dyspnea
- Productive cough [chronic bronchitis]
- Progressive exercise intolerance
- Wheezing
- Chest tightness
- Altered in mental status
- CHF failure symptoms
  - edema and weight gain





History Suggesting Risk of Impending Respiratory Failure

- ALOC
- Decreased respiratory effort
- Attack lasting greater than several (2) days
- Steroid dependence
- Prior intubation

Physical in Emphysema

Gen :

- Very thin
- Barrel chest
- Tripod sitting position
- +/- Cyanosis

Physical Exam in Emphysema

GEN :
- Very thin; barrel chest
- Pursed lips
- Tripod sitting position
- Diaphoresis
- +/- Cyanosis

NECK:
- NVD [RCHF (ie, cor pulmonale)]

CHEST:
- hyperresonant
- use of accessory muscles of respiration
- rhonchi and wheezing

CV:
- heart sounds are distant

EXTR:
- edema [RCHF]

Physical Signs of Impending Respiratory Failure

O2 - Hypoxic signs
- Agitation
- Tachycardia
- Hypertension
- +/- Cyanosis

CO2 - Hypercapnea signs
- ALOC ; stupor
- Decreased respiratory effort
- Rhonchi and wheezing

CHEST:
- Breath sounds are distant
- Quiet * ; no wheezing

* *"a quiet chest is a dangerous chest"*

Diagnosis

- Clinical impression
- Ancillary tests
  - PEFR
    - peak expiratory flow rate
    - Objective measurement of airflow obstruction
    - More accurate than clinical impression
      - For assessing severity
      - For trending response to therapy
    - Quick test distinguishing COPD from CHF
      - PEFR less than 200 mL : probably COPD exacerbation

Diagnosis - Additional Tests

- Pulse oximetry :
  - Useful in assessing
    - Severity
    - Response to therapy
      - Should be corrected to above 90%
  - ***Not*** useful in predicting overall illness or clinical outcome
  - Not useful in assessing acuteness

Diagnosis - Tests

- ABGs provide clues :
  - Severity
  - Acuteness
    - In chronic CO2 retention
      - renal compensation
      - pH usually near normal
    - In acute CO2 excess
      - rising pCO2 : ominous
      - pH below 7.3
      - acute uncompensated respiratory deterioration

Diagnosis - Further Testing

- Electrolyte Disorders:
  - sodium retention
  - low potassium (beta agonists cause
  - low magnesium drop in both)
- Chest x-ray
  - hyperinflation [air trapping]
  - flat hemidiaphragms [air trapping]
  - small appearing heart
- Electrocardiogram
  - R/O new ischemia / infarct
  - RV strain; abnormal p waves; MAT

Treatment

- Depends upon the clinical presentation
  - O2 , IV, M3 (continuous pulse oximetry)
- Primary goal: correct hypoxia
  - Balancing
    - O2 sat > 90
    - Against progressively rising pCO2 and suppression of the hypoxic drive
- Treat bronchospasm
  - Acute : beta-adrenergic agonists
  - Sustained : anti-inflammatories (prednisone,…)

EMC 360

Assessing Need for ET

- Can be established quickly across the room
  - Confused [brain]
  - Central cyanosis [skin]
  - Cannot hold head up [muscle]
- Can be established quickly at the bedside
  - Ask patient to hold nebulizer in his or her hand
    - So weak or so sleepy that the nebulizer falls
- If the above are present +/or immediate therapy is failing to improve patient's condition, then consider immediate intubation
  - regardless of O2 sat or pCO2 levels

EMC 360

Increased CO2 Levels

CO2 levels rising during treatment :

- Reversal of hypoxic arterial vasoconstriction
  - Perfusing high CO2 areas
  - Decreased respiratory [hypoxic] drive
- Marked elevation :
  - Deterioration of mental status (sleepy; stupor)
  - CO2 narcosis
    - inhibits respiratory drive
    - worsening O2 delivery ; worsening hypoxia
- Deciding to decrease O2 delivery:
  - wrong; will only worsen of hypoxia

EMC 360

Treatment , continued

- Supply enough O2
  - If oxygen leads to deterioration
    - patient's condition is so unstable
    - intubation likely needed anyway
- Medications
- Other
  - CPAP or biphasic positive airway pressure (BiPAP)
  - Heliox (ie, mixture of helium and oxygen)

EMC 360

Medications

- Bronchodilators
  - Albuterol
  - Terbutaline
  - Epinephrine
  - Magnesium
- Anticholinergics
  - Ipratropium (Atrovent)
- Anti-inflammatories
  - Corticosteroids
    - Oral prednisone
    - IV methylprednisolone
  - Leukotriene inhibitors

EMC 360

Bronchodilators

- MOA:
  - Act directly on beta 2-receptors
  - relax bronchial smooth muscle
    - relieving bronchospasm
    - reducing airway resistance
- Continuous may be superior to intermittent
  - Cochrane review / *severe* asthma (only 15% of COPD pts have significantly reversible bronchospasm)

EMC 360

Bronchodilators , continued

- Albuterol
  - Beta-agonist of choice
  - Aerosol superior to IV
  - Albuterol superior to Levalbuterol (Xopinex)
    "Xopinex: twice the money for half the drug"
  - Dose< : 1.25 - 5mg [5mg/mL]
  - MDI with spacer is equal in effectiveness to nebulizer
- Epinephrine
  - Dose : 0.1- 0.3mL [1:1000]

More Bronchodilators

- Terbutaline
  - Dose:
    - 0.25 mg (0.25 mL of 1 mg/mL conc.) SC
    - not to exceed 0.5 mg SC q4h
  - Caution in coronary disease [may be safer than Epi]
    - intracellular shunting of potassium
    - may decrease serum K levels
- Magnesium
  - Dose:
    - 1-2 grams over 30 min.
  - Recommendation
    - Only for severe asthma
    - Not in mild to moderate
  - Replete lost stores from adrenergic stress +/or diuretics

Anticholinergic Therapy

Ipratropium

- MOA:
  - chemically related to atropine
  - Anticholinergic :
    - inhibit vagally mediated reflexes (bronchial smooth muscle muscarinic receptor)
    - In COPD, vagal tone may be abnormally high (increased by 50%)
- Dose
  - 0.5 mg / nebulizer treatment
  - mixed with albuterol as part of first treatment
- Peaks
  - 1-2 hr ; duration: 4 hr

Anti-inflammatory Therapy

Leukotriene inhibitors (or modifying agents)

- Leukotriene
  - 1000 times more potent bronchoconstrictor than histamine
  - produce edema and mucus
- Additive effect on:
  - Beta agents
  - Corticosteroids (don't actually suppressleukotrienes)
- No place in emergency management at this time
  - No RCT in ED or prehospital

Corticosteroid Anti-inflammatory Therapy

- MOI
  - Anti-inflammation
  - Up-regulation of beta receptors
  - Immediate vasoconstriction of bronchial tissue

Corticosteroid Therapy

Effectiveness

- Effective in accelerating recovery from acute
- Effective accelerating recovery from acute exacerbations
- PO steroids equally effective as IV
- High dose (360mg IV) no better than usual doses (Methylprednisolone 125mg IV; Prednisone 80mg po QD)
- May not a dramatic clinical improvement in the ED
  - Early dosing is critical
    - 70% compliance by ED physicians
    - Some effect begins by 6-8 h into therapy

Inhaled Corticosteroid Therapy

- MOI
  - Same as PO or IV
  - Maybe: earlier local vasoconstriction and suppression of mucus production
- Used in chronic asthma
- Possible prehospital / ED use
  - Early dosing is critical
  - Ease of administration

CPAP and BiPAP

- CPAP
- Continuous Positive Air Pressure
- Frequently set : 10 cm of H2O continuous pressure
- Delivered throughout respiratory cycle
- Rationale in COPD
  - Improves inspiration
    - intrinsic PEEP (due to obstruction, lung does not fully empty)
    - by over-riding intrinsic ["afterload"] PEEP
  - Decreases the work of breathing

CPAP

- Intrinsic ["afterload"] PEEP
  - In respiratory failure due to COPD, intrinsic PEEP : about 5 cm H2O
- Usual starting CPAP level:
  - 5 –10 cm H2O
  - Why not always start at 10: risk of pneumothorax
- Both CPAP and BiPAP require
  - alert, cooperative patient
  - no excessive secretions

Bi PAP

- Senses when an inspiratory effort is beginning
- and delivers additional pressure during inspiration
- When inspiratory flow stops, pressure returns to the CPAP level
- In respiratory failure, begin with
  - expiratory level (EP) : 2-5 cm H2O
  - inspiratory level (IP) : 10-15 cm H2O

COPD Complications

- Structural:
  - Incidence of pneumothorax due to bleb formation is relatively high
  - consider pneumothorax in all patients with COPD who have increased shortness of breath
- Metabolic
  - adrenal crisis
    - In long-term steroid use, possibility of withdrawal-crisis
- Co morbid disease
  - coronary artery disease
  - pneumonia

Patient Education

- Dangers of smoking
  - Smoking cessation improves prognosis
  - Improvement in quality of life still attainable
- Do not wait until "you're about to pass out"
  (Don't wait for near respiratory failure)
  - Call 911 early during exacerbation

Pitfalls in Management

- Always R/O cardiac ischemia
  - In hypoxia and distress, may overlook underlying ischemia
- Administer as much oxygen as necessary to avoid hypoxia
  - If the patient retains excessive carbon dioxide, intubate

More Pitfalls

- Post intubation excessive rate and volume
- Excessive tidal volume
  - Decreased venous return
    - shock
- Excessive rate
  - Rapid respiratory rate induces an alkalosis (in a previously acidotic patient)
    - Causing an intracellular shift in potassium (Low K because: bronchodilator +/- diuretic use)
    - Potentially dangerous hypokalemic arrhythmias

Summary

We have discussed :

- Pathophysiology and presentations of COPD
- Methods of quick and accurate recognition and appropriate aggressive management of decompensating COPD
- Common first line and less common treatments such as CPAP and BiPAP
- Complications of COPD disease
- Complications and pitfalls of the treatments for COPD