AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
SCIENTIFIC ASSEMBLY
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Modern Management of Pulmonary Edema

Life-threatening acute pulmonary edema may present very rapidly. The lecturer will review the literature of pulmonary edema. Methods to distinguish among pulmonary edema, bronchospasm, and pneumonia when the patient first presents to the emergency department will be discussed. Once the diagnosis of pulmonary edema is made, proper management is critical. The role of various medications, including magnesium and Bi-PAP, will be discussed.

- Discuss a modern physiologic approach to the patient with acute pulmonary edema.
- Incorporate the newer medications and treatment modalities into a management plan based on outcomes from clinical literature.
- Discuss the role of the aortic balloon pump in the management of cardiogenic shock.

MO-54
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Room # N208
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FACULTY

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Modern Management of Pulmonary Edema

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Definition: Cardiogenic pulmonary edema may be defined as the leakage of fluid from the pulmonary capillaries and venules into the alveolar space as a result of increased hydrostatic pressure resulting from an inability of the left ventricle to effectively handle its pulmonary venous return.

Circadian Rhythm to Pulmonary Edema: 10:00 PM- 4:00 AM peak at 1:00 AM

Pathophysiology: Starling's Equation

$$Q = K(P_{cap} - P_{int}) - C(\pi_{cap} - \pi_{int})$$

P_{cap}=capillary hydrostatic pressure, P_{int}=Lung interstitial capillary pressure (alveolar pressure), K&C=coefficients of capillary walls, π_{cap}=capillary oncotic pressure, π_{int}=interstitial oncotic pressure.

In cardiogenic pulmonary edema elevation of P_{cap} is determining factor.

Sources of Left Ventricular Failure:

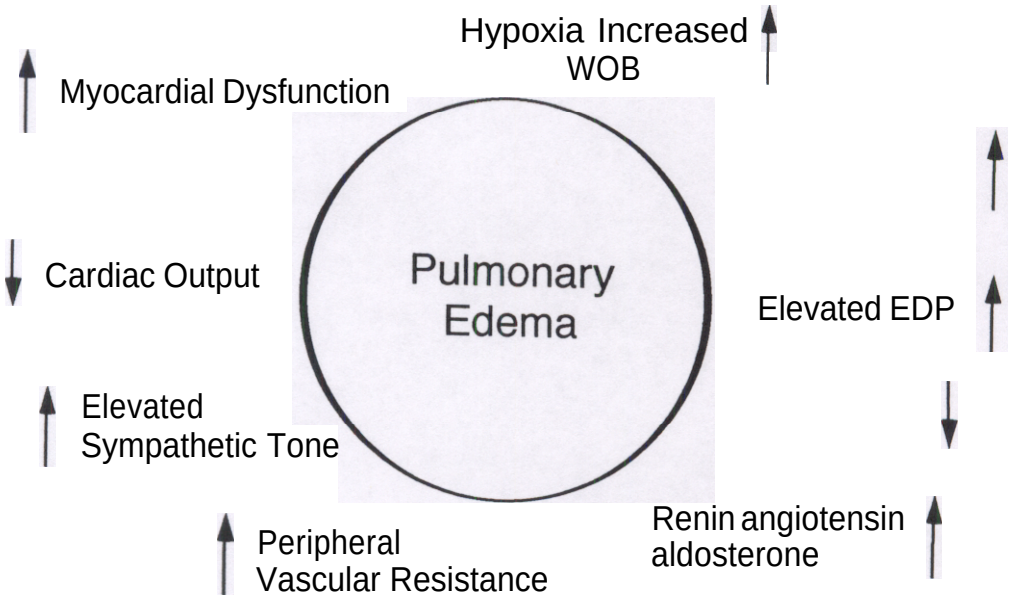
1. Excessive Pulmonary Venous Return (Pre-Load)
2. Myocardial Dysfunction
   a. Systolic
   b. Diastolic
      Contractility function of diastolic volume
3. **Increased** Peripheral Vascular Resistance (After-Load)
4. Disorder of rate/rhythm

Most patients with acute pulmonary edema are not acutely fluid overloaded and their sudden decompensation results from a combination of ventricular dysfunction **and elevated peripheral vascular resistance.**

Clinical Manifestations:

Reflect impaired gas exchange, decreased lung compliance, & hypoxia.

Tachycardia	Tachypnea	Anxiety
Rales	Rhonchi	Wheezes
Diaphoresis	Pallor	Mottled
+/-Edema	Gallop (S3,S4)	JVD
Sputum (Frothy)	Sputum (Blood tinged)	HJR



Management: Simultaneous approach to all sources of Left Ventricular Failure.

Lower "P_{cap}" in Starling's Equation

Raise "Pint" in Starling's Equation

1. Decrease venous return to Left Ventricle
    a. Decrease peripheral venous return to lungs
        1. Decrease inflow of blood into lungs
        2. Lower pulmonary capillary wedge pressure

2. Increase Left Ventricular Emptying
    a. Decrease peripheral vascular resistance
    b. Relax stiff left ventricle
    c. Assist systolic contractility

Management Approaches:

Supplemental Oxygen

100% non-rebreather mask

May decrease cardiac output?

Traditional Management: Morphine and Diuretics

Absolutely no indication for the use of either of these agents in the initial management of Pulmonary Edema!

Morphine:

a. Actions

Vasodilator

Histamine effect

Transient

b. Adverse Reactions

Respiratory depressant

Significant patient problems with use in Pulmonary Edema:

Subjective deterioration 38%

Objective deterioration 46%

Adverse effects 17%

Diuretics:

a. Actions

1. Hemodynamic (Peripheral)

Elevates

Mean arterial pressure

Heart rate

Left ventricular pressure

Systemic vascular resistance

Enhance renin-angiotensin system

2. Hemodynamic (Pulmonary)

Elevates Pulmonary Capillary Wedge Pressure

Decreases stroke volume index

Some newer diuretics may have less adverse hemodynamic effects (Torsemide)

3. Renal

a. Decrease sodium transport

Diuresis

Decreased intravascular volume

b. No renal blood flow in hyperadrenergic state

Fight or flight response

c. Up to 50% Pulmonary Edema patients euvolemic

Comparison studies in patients with nitrates vs diuretics no advantage to diuretics. In dialysis patients, improved outcome in chronic renal failure patients who receive no diuretics compared to those with intact kidneys who receive furosemide.

Physiologic Management

Decrease Pulmonary Vascular Congestion:

Nitrates: First line of treatment
- a. Immediate therapy
  - 1. Sub lingual (SL), Transcutaneous (Topical), Intravenous
    - a. No vascular access needed
    - b. 1662 NTG-SL pts. : Adverse Events 0.2%
- b. Actions
  - 1. Increase Venous Capacitance
  - 2. Decrease vena-caval return
    - a. Decrease pulmonary blood flow
    - b. Decrease pulmonary blood pressure
    - c. Decrease LV preload
  - 3. Higher doses may reduce peripheral vascular resistance
- c. Administration
  - a. Sublingual
    - Simultaneous 2- sublingual 1/ 150
  - b. Tanscutaneous
    - 2 inches
    - Not effective if mottled or severely diaphoretic
  - c. Intravenous
    - 1. Infusion-Intermittent bolus
    - 2. 640 APE pts. 1980-1991
      - 1980-1983: Rx: Oxygen-MS-Lasix
      - 1984-1991: Rx: IV-NTG
      - Mortality decreased from 13% to 5.3%

Capoten: First line therapy
- a. Immediate therapy-Sublingual
  - 1. Reduction PCWP in 10 minutes
  - 2. Dyspnea improvement 15 minutes
  - 3. Peak effect 30 minutes
- b. Action
  - 1. Angiotension converting enzyme inhibitor
  - 2. Peripheral vasodilator
  - 3. Decreases peripheral vascular resistance
  - 4. Relax left ventricle

c. Administration
a. 25 mg. sublingual (Capoten)
b. 12.5 mg intravenous (Capoten-England)
c. 1.25-2.5 mg intravenous (Enalapril)

Hamilton: Decreased intubation needs from 25% to 5% SL-Capoten

Schreiber: Urapidil vs NTG 112 APE patients
Decreased Systolic BP: 155 vs 179
Decreased Diastolic BP: 82 vs 97
Increased Oxygen and pH

Annane: Enalaprilat vs Placebo
Decrease PCWP 37% vs 10%
Decrease Diastolic BP: 21% vs 0%
Renal Blood Flow Increase 12% vs 0%

Angiotensin II Recptor Blockade:
Little or no evidence of use in APE. Alternative drug in chronic CHF.

Nitroprusside
a. Requires vascular access
Rapid onset
Titratable
b. Action
1. Direct action on resistance vessels
2. Most effective afterload reducer
c. Administration
Continuous infusion l-5 ug/kg/min

Fenoldopam (Corlopam)
Dl-like dopamine receptor agonist with alpha 2 adrenoceptor activity.
Theoretically effective, scant clinical data.
Effective in hypertension secondary to Renal Artery Stenosis

Effectiveness of Medical Therapy in 181 APE Patients

Variable	KU-LOS	P-value	Regression Analysis ICU Admit Odd-Ratio	P-value	ETI Odds Ratio	P-value
r/oMI	2.71	0.135	2.7	0.064	1.2	0.62
Age	0.28	0.14	0.048	0.08	0.03	
Captopril	0.96	0.026	0.29	0.002	0.16	0.008
NTG	1.9	0.95	1.78	0.23	0.72	0.54
MS	2.17	0.28	3.09	0.002	5.04	<0.001
Loop	2.09	0.266	1.06	0.89	0.9	0.81

Atria1 Natriuretic Factor:

Diuretic and Direct Acting Cardiovascular Agent

1. Decreases Right Atrial Pressures
2. Decreases Systolic Blood Pressure
3. Decreases Pulmonary Blood Pressures

Seta: 22 patients : CHF improvement in all parameters 1 hours following infusion.

Thermal Vasodilation:

Acute pre-load and afterload reduction.

Warm Water Bath (aka Hot Tub) vs Sauna in 34 patients with CHF

HR- Increased 20-25 bpm

CI- increased, SVR- decreased, Diastolic BP-decreased

WWB- Increased PCWP

Sauna- Decreased PCWP

Inotropic Agents

Myocardial Cell receptors

Bind catecholamines

May bind other neurohumoral agents

Stimulate adenyl cyclase

Converts ATP to cyclic Adenosine monophosphate (cAMP)

Activates protein kinase

Opens calcium channel in cell wall

Increase inotrophy

Increase chronotrophy

cAMP lysed by phosphodiesterase to AMP

Cells with chronic failure have downregulated their receptor sites

More diffuse

Less responsive

Catecholamines:
- Dopamine
  - Low doses vasodilator (0.5-2 ug/kg/min)
  - Mod doses inotrope (2-10 ug/kg/min)
  - High doses vasoconstrictor (>10 ug/kg/min)
- Dobutamine
  - Inherent pulmonary vasodilatory properties
  - Positive inotrope
  - Dose 2.5-15 ug/kg/min

Bipyridines:
- Phosphodiesterase inhibitors:
  - Positive inotropes
  - Peripheral vasodilators
  - Transient decrease in blood pressure
    - Initial bolus followed by continuous infusion
  - Increased ectopy and arrhythmias
  - Increase lung compliance even prior to cardiac effects
- Agents
  - Amrinone (bolus 0.75 mg/kg, drip 5-10 ug/kg/min)
  - Milrinone (bolus 0.05mg/kg, drip 0.3-0.7 ug/kg/min)
  - Still in clinical trials
    - Vesrinone/ Enoximone

Cardoiogenic Shock
- Secondary to myocardial infarction induced systolic dysfunction
  - Acute coronary artery occlusion
    1. PI-CA
    2. Surgery
    3. Thrombolysis
       - Elevate aortic root pressure prior to thrombolytic

Airway Management

1. Non-Invasive Pressure Support Ventilation (NPSV)

For the purposes of this discussion NPSV will be defined as respiratory support provided to a patient via modification of airway pressures without endotracheal intubation. Included in this general category would be mask Continuous Positive Airway Pressure (CPAP), Intermittent Positive Airway Pressure (IPPB), BiLevel Positive Airway Pressure (BiPAP).

2. Mechanism of Action of NPSV

A. Decreased Work of Breathing (WOB)
- Normal Oxygen consumption (V02): 14%
- Critically ill patients V02 may increase 20%

NPSV decreases WOB
- Increases functional residual capacity
  - Maintains alveolar patency
    - Maybe major advantage
    - No energy expended reopening collapsed alveoli
- Decreases airway resistance
- Improved force-length relationship of chest wall musculature
  - Improved elastic recoil of chest

B. Improved Gas Exchange
- Prolonged Alveolar Patency
  - Extended time for gas/capillary diffusion
    - Improved oxygenation
    - Decrease arterial carbon dioxide tension
  - Improved Fi02
  - Alteration in pulmonary perfusion
    - Redistribution to lower segments
    - Decreased ventilation-perfusion mismatching

C. Increase in Cardiac Output (CO)
- Patients with increased pulmonary capillary wedge pressure
  - CO determined by afterload
- Global increase in intrathoracic pressure
  - Increase in left ventricular (LV) intra-luminal pressure prior to generation of LV wall tension.
    - Decreases LV work
- Pressure gradient between intrathoradc and extrathoracic aorta
  - Equivalent to pharmacologic afterload
- Decrease in right and left ventricular preload

D. Continuous Positive Airway Pressure
- Increases "P_{int}" in Starling's Equation
  - Reverses movement of fluid across alveolar membranes
  - Flow from alveolar space back into capillaries.

$$Q = K(P_{cap} - P_{int}) - C(\pi_{cap} - \pi_{int})$$

3. Equipment

A. Pressure generator
1. Wall oxygen source
2. Full function pressure cycled ventilator
3. BiPAP* System

B. Patient interface:
Full face mask
Nasal Mask
More comfortable
Requires intact dentition

4. Clinical Experience

Non-Invasive Pressure Support in Acute Respiratory Failure

Multiple studies have demonstrated efficacy in APE: Success rates range between 80-95% in avoiding ETI.

Acute Myocardial Infarction with CHF
29 patients Nasal CPAP vs Oxygen
Decreased PCWP 25 to 18
Mortality decreased from 64% to 9%

End Stage Renal Dialysis Patients in Pulmonary Edema
50% of intubated patients extubated to BiPAP in the ED
100% patients primarily managed with BiPAP avoided ETI
All discharged from ED after hemodialysis

5. Characteristics of Successful Emergency Utilization

A. Immediately accessible
Maintained in unit of use
At patient bedside in under 3-5 minutes
Delays in utilization of equipment major deterrent to successful outcome
Stored with tubing in place, no setup time

B. Application by treating personnel
Cross training nurses, physicians and respiratory therapists
No need to recruit specialized personnel from a remote area
Impact time to treatment
Greater tendency to employ device

C. Continuous presence of maintenance personnel
Titration of use of system
Modulation with changes in clinical status
Wean personnel when possible

D. Patient acceptance
Tolerate in a tachypneic state
Rapid improvement in clinical status

E. Immediate availability of personnel capable of performing endotracheal intubation

6. Emergency Department use:

Oxygen bled into anterior mask ports 15 liter/min

Positive pressure is initiated at a lower pressure to allow patient to experience PAP then rapidly titrated to therapeutic pressures.

Therapeutic pressures should be reached within 3-5 minutes of starting NPSV treatment.

CPAP:
Initial pressure: 5-10 cm II20
Titrate to: 15-20 **cm** H2O

BiPAP:
Inspiratory positive airway pressure IPAP
Expiratory positive **airway** pressure EPAP
Initial pressures: IPAP/EPAP 8/3 cm H2O
Titrate to: **15-20 / 12-15 cm** H2O

Management of Pulmonary Edema in the Chronic Renal Dialysis Patient

Patients are truly fluid overloaded

Same principles apply:
Reduce venous return
Reduce peripheral vascular resistance
Easily temporize until dialysis is possible

Management
Requires no vascular access
Nitrates:
NTG sl: 2 simultaneous 1/ 150

Capoten: 25 mg sl
NTG paste: l-2 inches
Non-invassive pressure support ventilation
Interventional dialysis 2-6 hours & Discharge

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