Educational Session: Winning at Failure
- Modern Management of Cardiogenic Pulmonary Edema

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Winning at Failure

Modern Management of
Cardiogenic Pulmonary Edema

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OBJECTIVES
At the conclusion of this presentation, each participant should be able to...

1. Describe the limitations of morphine and furosemide in the management of cardiogenic pulmonary edema.
2. Identify medications available for rapid preload reduction and afterload reduction.
3. Discuss the use of noninvasive positive pressure ventilation.
I. Pathophysiology

Definitions
- Leakage of fluid from the pulmonary capillaries and venules into the alveolar space as a result of increased hydrostatic pressure.
- Inability of the left ventricle (LV) to effectively handle its pulmonary venous return

Basic causes
- Excessive venous return (preload)
- Excessive systemic vascular resistance (afterload)
- LV dysfunction
  - Systolic dysfunction
  - Disorders of contractility
  - Disorders of rate and rhythm (arrhythmias)
  - Diastolic dysfunction
    - Inadequate ventricular relaxation (ventricular “stiffening”)

A self-perpetuating cycle leads to cardiogenic pulmonary edema:
1. Acute LV systolic dysfunction leads to…
2. Decreased myocardial contractility and cardiac output (CO), leading to…
3. Catecholamine production, leading to…
4. Increased SVR (afterload) and blood pressure, leading to…
5. Increased myocardial wall tension and myocardial oxygen demand, leading to…
6. Myocardial ischemia, leading to…
7. Decreased myocardial contractility and CO (continues the cycle…)
8. Diastolic dysfunction, increased pulmonary artery and capillary hydrostatic pressures
9. Transudation of fluid into pulmonary alveoli and interstitium (pulmonary edema), leading to…
10. Hypoxia, leading to…
11. Myocardial ischemia (continues the cycle…)
12. Anxiety, leading to…
13. Increased catecholamine production (continues the cycle…)
14. Note that as the left side of the heart fails, the right side continues to fill (preload)
15. This produces even greater pulmonary artery and capillary hydrostatic pressures (continues the cycle…)

Many patients (~50%) presenting with cardiogenic pulmonary edema are not fluid-overloaded!
- Vascular dysfunction rather than fluid overload
- Diuretics are often not necessary, or at least over-used
Treatment must be aimed at breaking this cycle!
- Decrease right-sided filling (decrease preload)
- Improve CO and “unload” the left side of the heart (decrease afterload)
  - Results in improved LV diastolic function
- Improve LV systolic function
  - Increase contractility (not usually necessary if steps 1 and 2 are done well)
  - Treat arrhythmias
- By breaking this cycle, fluid is redistributed out of the lungs

II. Goals in Management

ABCs, supplemental oxygen, ECG
Pharmacological treatment — major goals
- Decrease right-sided filling (decrease preload)
- Increase left-sided emptying:
  - Decrease SVR (decrease afterload)
  - Improve LV contractility (inotropic support) — sometimes necessary
    - Inotropes have some adverse effects, therefore avoided when possible

III. Preload Reduction

Traditional treatments
- Morphine
- Furosemide
- Nitrates

**Morphine**
Advantages
- Histamine effect causes some decrease in preload
- Anxiolytic effect may decrease catecholamines
  - Results in a decrease in afterload

Disadvantages
- Limited data (none?) to support the notion of a preload effect
  - At high doses only?
- Respiratory depressant at high doses
- Myocardial depressant at high doses
- Concerns if patient has low blood pressure
  - Myocardial depressant effect
- Histamine-related side-effects may actually increase catecholamines
  - Rash/urticaria
  - Nausea/vomiting
- Evaluated venous tone in the hand and forearm veins in pulmonary edema patients after administration of morphine
  - Venous tone decreased, i.e. produced venodilation in the hand and forearm
  - How does this correlate with preload (PCWP)?? Must look at Swann studies…

Lappas, et al (Anesthesiology, 1975)
- Evaluated filling pressures of the heart and pulmonary circulation in patients with CAD after IV morphine
  - 2 mg/kg IV morphine (5 mg/min infusion)
  - Left and right heart filling pressures increased
  - CI decreased

- 0.2 mg/kg IV morphine in AMI patients with severe LV failure (LVF)
- 15 and 45 minutes after injection, BP, HR, and cardiac index (CI) were decreased
- No decrease in preload noted
  - Conclusion — no immediate beneficial hemodynamic effect

- 57 patients with presumed prehospital diagnosis of pulmonary edema
- 38% had subjective deterioration after receiving morphine
- 46% had objective deterioration after receiving morphine
- no patients receiving NTG without morphine had deterioration

- Odds ratios for intubation and ICU admission for pulmonary edema patients
  - Morphine — 5:1

- Based on ADHERE registry of patients admitted with decompensated CHF
- Compared use of morphine vs. no morphine
  - Morphine was an independent predictor of mortality, odds ratio 4.84
  - Was also associated with increased need for mechanical ventilation (5-fold increased risk), increased need for ICU admission, prolonged hospitalization

Morphine for anxiolysis
- Decrease in catecholamines, afterload
- Why not use a benzodiazepine instead?
  - No concerns with rash/urticaria
  - No concerns with nausea/vomiting
  - Less concern with respiratory depression
  - Less concern with hypotension
Summary for morphine
- **Preload reduction**
  - No good evidence to support any immediate reduction in preload centrally
  - Nitrates are more effective, safer
- **Anxiolysis**
  - Side-effect profile favors benzodiazepines

**Furosemide**
Reduce preload by:
- **Removal of total body fluid through renal effect (diuresis)**
  - However, CPE patients often have significantly reduced renal blood flow (RBF; ~20% of normal) due to elevated afterload
  - Furosemide will have a delayed diuretic effect (30-120 minutes)
- **Direct vasoactive effect (venodilation)** supposedly reduces preload within 5-10 minutes
  - Pickkers, et al (Circulation, 1997)
    - Evaluated effect of furosemide on human forearm and hand veins after administration peripherally
    - Result — local administration of furosemide produced dose-dependent venodilation
    - But does this correlate with reductions in preload *centrally* (reduction in right heart filling, reduction in PCWP, etc.)?? Must look at Swann studies…
  - No convincing studies supporting any immediate effect
- More studies actually demonstrate an initial *adverse* hemodynamic effect
    - IV furosemide administered to post-AMI CHF patients
    - Significant reductions in filling pressures occurred *only in patients that had diuresis*
    - IV furosemide produced significant reductions (17%) in CO during the first 90 minutes
    - CO gradually returned to normal after diuresis
    - IV furosemide (1 mg/kg) administered to AMI patients with LVF
    - Initial adverse hemodynamic effects
      - Increase in SBP, DBP, and HR during first 30 minutes
      - Decrease in CO and stroke volume (SV) during initial 90 minutes
    - Parameters returned to baseline over next 60-90 minutes with diuresis
    - Class III and IV CHF patients given IV furosemide
    - Produced early activation of the renin-angiotensin system
- Significant increase in plasma renin, NE, and arginine vasopressin levels
- Produced early adverse hemodynamic effects
- Significant increase in HR, SVR
- Significant decrease in SV
- Gradual return to baseline with diuresis
- Kraus, et al (Chest, 1990)
  - Effects of IV furosemide on PCWP over 1 hour in patients receiving nitrates (for preload reduction) and captopril (for afterload reduction)
  - Furosemide produced increases in PCWP over initial 15 minutes
  - Then decrease PCWP with diuresis
  - If patients were premedicated with nitrates and captopril, furosemide produced an immediate and sustained decrease in PCWP

Summary for furosemide
- Decreases preload through diuresis, but this is a delayed effect
- No consistent data regarding any immediate direct venodilating effect
- Produces an initial activation of the sympathetic nervous system
  - Increased HR, SVR, myocardial oxygen demand; leading to cardiac ischemia
  - Decreased SV, CO, tissue perfusion
- Produces an initial activation of the renin-angiotensin system
- Overall produces adverse hemodynamic effects early-on

Nitroglycerin (NTG)
Advantages
- Rapid, reliable preload reduction
  - Multiple studies comparing NTG vs. morphine or furosemide for preload reduction
    - NTG clearly superior (faster, safer)
- Moderate/high dosages reduce SVR (afterload) as well
  - Maintains or improves SV and CO
- Multiple forms of administration — topical, SL, IV (be aggressive!)
- Short half-life limits any adverse effects
  - Especially important if prehospital misdiagnosis
Caution — hypotension, acute mitral regurgitation (MR), aortic stenosis (AS), pulmonary hypertension, Viagra

Summary for NTG
- Better than morphine or furosemide for preload reduction
- Safer than morphine or furosemide for preload reduction (especially important in the prehospital setting — see below)
- SL NTG provides rapid and effective initiation of treatment
  - Followed by topical NTG if moderate symptoms
  - Followed by IV NTG if severe symptoms or in extremis
IV. Afterload Reduction

Results in increased CO, restores RBF
- Nitroglycerin
  - SL and moderate/high dose IV
  - Excellent single agent for simultaneous preload and afterload reduction
- Nitroprusside
  - Especially useful for acute MR, severe hypertension
- Hydralazine
- ACE-inhibitors
  - Down-regulate the renin-angiotensin system
  - Decrease adrenergic tone and afterload
  - Improve LV relaxation and CO
  - Treatment of choice for chronic CHF
  - Useful for acute CHF exacerbations also

ACE-inhibitors
- 25 mg SL captopril if BP > 110
- 12.5 mg SL captopril if BP < 110
- Decreased PCWP (preload) noted by 10 minutes
- No change in HR, mean arterial pressure (MAP)
- 12 additional patients with florid pulmonary edema had significant improvement/complete resolution of dyspnea by 15 minutes
  - 8/12 patients — abrupt increase in diuresis without the use of a diuretic (due to improved RBF)

- IV captopril infusion in moderate decompensated CHF or pulmonary edema patients
- Onset of action by 6 minutes
- Decreased SBP, PCWP (preload)
- Increased CO
- No adverse effects

Varriale, et al (Clin Cardiol, 1993)
- Hemodynamic response to 1.25 mg IV enalaprilat in patients with severe CHF + MR
- Increased CO and SV
- Decreased MAP and SVR (afterload)
- Decreased PCWP (preload)
- Decreased the magnitude of MR

- Randomized double-blind controlled study
• 48 patients with pulmonary edema
  • treated with NTG, furosemide, morphine
• 25 mg SL captopril if SBP > 110
• 12.5 mg SL captopril if SBP 90-110
• Clinically significant reduction in “distress scores” by 30 minutes

• Odds ratios for intubation and ICU admission for pulmonary edema patients
• SL captopril — 0.28:1

Other studies
  • PO captopril
• Haude, et al (Int J Cardiol, 1990)
  • SL captopril
  • SL captopril in hemodialysis patients with pulmonary edema
• Tohmo H, et al (Eur Heart J, 1994)
  • IV enalaprilat
• Annane, et al (Circulation, 1996)
  • IV enalaprilat
  • Hemodynamic and subjective improvements can be seen in 6-12 minutes!

• Safety of ED use of SL captopril in NYHA Class 4 patients
  • No increased incidence of hypotension
  • No increased need for vasopressors
  • Decreased ICU length of stay (29 hours vs. 78 hours)

Summary for ACE-inhibitors
• Rapid reduction in afterload and preload
• Rapid reduction in subjective level of distress (decreased anxiety)
• Decreased need for intubation, ICU use
  • Increased bed availability, decreased hospital costs
• Combination with NTG exceeds benefit of either drug alone
• Acceptable alternative to IV NTG in patients with pulmonary edema
  • Works well even as a single agent if patients that can’t tolerate NTG
    • Patients with severe MR, AS
    • Patients taking Viagra

V. Combination Preload and Afterload Reduction
Natriuretic peptides
• Hormone-like substances produced by myocardium
  • Modulate diuresis, natriuresis, vasodilation, venodilation
• Activated and synthesized by the ventricle during heart failure
  • Heart may not be capable of producing adequate concentrations under acute stress (e.g. decompensated CHF)

Nesiritide
• Recombinant form of B-type natriuretic peptide (normally produced in the ventricle)
  • These substances modulate diuresis, natriuresis, vasodilation, and venodilation
• Early studies in decompensated CHF
  • Dose-related decreases in PCWP (preload) and SVR (afterload) as well as increases in CI
  • No increase in HR or arrhythmias
  • Symptomatic improvements

Mills, et al (J Am Coll Cardiol, 1999)
• Randomized, double-blind, placebo-controlled study of nesiritide infusion in patients with decompensated CHF; manufacturer-supported
• Significant reductions in PCWP and SVR
• Significant increases in SV and CI
• No effect on HR
• Beneficial effects evident at 1 hour and sustained throughout (24 hour) infusion

• Randomized double-blind placebo-controlled study of nesiritide infusion in patients with decompensated CHF; manufacturer-supported
• Dose-related decrease in PCWP
• Subjective improvements in clinical status
• Most common side-effect was dose-related hypotension (usually asymptomatic)

VMAC Investigators (JAMA, 2002)
• Randomized double-blind placebo-controlled trial of intravenous nesiritide vs. NTG added to “standard treatment” in decompensated CHF; manufacturer-supported
• “Standard treatment” ≠ “optimal treatment”
  • No mention of aggressive use of nitrates, ACE-Is, NIPPV
• Mean baseline PCWP was 28 mm Hg
• Evaluated patients at 3 hours and 24 hours
  • Results at 3 hours
    • Decrease in PCWP by 5.8 (nes.) vs. 3.8 (NTG)
    • No subjective improvement in patients’ status
  • Results at 24 hours
    • Decrease in PCWP by 8.2 (nes.) vs. 6.3 (NTG)
• No difference in dyspnea
• Some improvement in “global clinical status” (never specified what this is!)
  • Admitted this was a non-validated scoring system

• Subsequent analyses…
  • Questions regarding cost-effectiveness
    • 40x more expensive than NTG
    • Duration of hospital stay 2 days longer for nesiritide patients vs. NTG
    • But…decreased readmission rate for nesiritide patients (among survivors)
  • Questions regarding safety
    • Trend towards increased mortality with nesiritide in VMAC trial
      • 19% for nesiritide vs. 13% for NTG at 90 days (p=0.08)
    • Sackner-Bernstein, et al (JAMA, 2005)
      • Meta-analysis of three randomized double-blind trials of patients with acutely decompensated heart failure treated with nesiritide infusions (485 patients received nesiritide, 377 controls)
      • Included FDA data from VMAC and other trials
        • Nesiritide associated with increased mortality at 30 days (7.2% vs. 4.0%)
    • Sackner-Bernstein, et al (Circulation, 2005)
      • Meta-analysis of five randomized trials of nesiritide in acutely decompensated heart failure (1269 total patients)
      • Included FDA data from VMAC and other trials
        • Nesiritide associated with worsening renal function
        • No increase in need for hemodialysis, but…
        • Increase in need for medical intervention for renal function (11.1% vs. 4.2%)
    • Package insert does include information regarding problems with renal function

  • Another quote from package insert: “The VMAC study does not constitute an adequate effectiveness comparison with NTG. In this trial the NTG group provides a rough landmark using a familiar therapy and regimen

• Their conclusion:
  • “When added to standard care in patients hospitalized with acutely decompensated CHF, nesiritide improves hemodynamic function and some self-reported symptoms more effectively than intravenous NTG or placebo.”

• My conclusion:
  • “When added to sub-optimally treated patients hospitalized with acutely decompensated CHF, nesiritide slightly increased mortality, provided no subjective improvement in dyspnea, and barely improved hemodynamic function and some undescribed self-reported symptoms more effectively than sub-therapeutic doses of intravenous NTG or placebo…at a higher cost.”
Cost of nesiritide: $456 per 1.5 mg vial! (approx. enough for 24 hours of treatment)
  (The Medical Letter 11/12/02)

VI. Inotropic Support

Choices
- Catecholamines
  - Dopamine
  - Dobutamine
- Phosphodiesterase inhibitors
  - Amrinone
  - Milrinone
- Intra-aortic balloon pump (bridging device before PTCA/CABG)

Catecholamines
Dopamine
- Low dosages (< 5 mcg/kg/min) — vasodilation
- Moderate dosages (5-10 mcg/kg/min) — inotropic
- High dosages (10-20 mcg/kg/min) — vasoconstriction (increase afterload)
- Moderate and higher dosages are arrhythmogenic

Dobutamine
- Inherent vasodilator properties
- Positive inotrope
- Arrhythmogenic (probably less than dopamine)
- Higher dosages associated with tachycardia

Drawbacks to the catecholamine class
- Tachycardia/arrhythmias, especially at higher dosages
- Increased myocardial oxygen consumption, ischemia
- Patients with severe CHF have very high endogenous circulating levels of plasma catecholamines
  - Tolerance develops rapidly, higher dosages are needed
  - Higher dosages produce adverse effects
  - Causes dopamine/dobutamine to be less effective inotropes

Phosphodiesterase inhibitors
- Work independent of adrenoreceptor activity and plasma catecholamine levels
- No development of tolerance
- Induce inotropic support as well as decreased preload and afterload
- Amrinone — disappointing results
- Milrinone — excellent results
Milrinone
- 7 studies comparing milrinone to dobutamine in patients with severe decompensated CHF (including post-AMI patients)
  - Similar or greater increase in SV/CI
  - Greater reduction in PCWP (preload) and SVR (afterload)
  - No increase in myocardial oxygen consumption
  - Less tachycardia
- Drawbacks — occasional arrhythmias, more expensive, no proven change in mortality

Summary for inotropic support
- Catecholamines (dopamine, dobutamine) may require large dosages in severe CHF
  - Tachycardia, arrhythmias, increased myocardial oxygen consumption
- Milrinone provides preload and afterload reduction in addition to inotropic support
  - Superior to dobutamine for the patient with borderline hypotension

VI. Noninvasive Positive Pressure Ventilation (NPPV)

Physiology
- Maintains positive airway pressure during entire respiratory cycle
  - Maintains patency of stiff fluid-filled alveoli, prevents collapse during exhalation
    - Decreases work of breathing
      - Less energy spent trying to reopen collapsed alveoli
    - Improves oxygen and carbon dioxide exchange
  - Increases intrathoracic pressure
  - Decreases preload and afterload (and increases CO)

Two types
- Continuous positive airway pressure (CPAP)
  - Single airway pressure is maintained throughout all phases of respiratory cycle
- Bilevel positive airway pressure (BiPAP)
  - Allows for separate control in inhalation and exhalation
  - Higher pressures can be applied during inspiration and lower pressures during exhalation
    - Greater patient comfort

CPAP associated with reduced need for endotracheal intubation
- Rasanen, et al (Am J Cardiol, 1985)

CPAP associated with reduced ICU length of stay and hospital costs

BiPAP associated with reduced need for endotracheal intubation

BiPAP associated with reduced ICU length of stay

CPAP vs. BiPAP in acute cardiogenic pulmonary edema patients
  • BiPAP associated with more rapid improvements in VS, but…
  • BiPAP associated with increased rate of myocardial infarction
    • Study was criticized because BiPAP group was sicker, had more chest pain patients
• Levitt (*J Emerg Med*, 2001)
  • BiPAP vs. mask ventilation for cardiogenic pulmonary edema
  • No increase in rate of myocardial infarction for BiPAP group
  • CPAP vs. BiPAP in undifferentiated acute respiratory failure
  • No increase in rate of myocardial infarction or mortality in subgroup analysis of pulmonary edema patients

Most recent meta-analyses of NPPV in patients with cardiogenic pulmonary edema
  • NPPV associated with decrease in mortality, need for endotracheal intubation, ICU and hospital length of stay, hospital costs
  • No significant difference between CPAP vs. BiPAP

• CPAP and BiPAP improve symptoms and acidosis faster than standard oxygen, but no effect on 7-day mortality or 7-day intubation rates

• Problems
  • Very high acuity? → 7-day mortality of patients was ~ 10%, patients presented *acidotic* (pH ~ 7.2) rather than alkalotic
  • 7-day intubation rates ~ 3%; why was intubation rate < mortality rate?
  • How soon was NIV employed? No indication…
  • NIV used for “minimum” 2 hours…why look at 7-day outcome for a therapy that is only being used for “minimum” 2 hours? What about 24-hour outcome instead?

Summary for noninvasive positive pressure ventilation
• Decreased work of breathing
• Improved oxygen and carbon dioxide exchange
• Improved preload, afterload, and CO
• Reduced need for endotracheal intubation
• Reduced ICU length of stay
• Reduced hospital costs
• **Reduced mortality? Maybe…**
  Must be used early to maximize the benefit!

## VII. Prehospital Treatment

Differential diagnosis for the patient with severe dyspnea and hypoxia is vast
• Most common in older adults patients
  • Decompensated heart failure/cardiac pulmonary edema
  • Pneumonia
  • Asthma exacerbation
  • COPD exacerbation
  • Pulmonary embolus
• Clinical assessment is difficult, often unreliable
  • Prehospital limitations — no thermometer, no x-ray

Is empiric treatment safe? With which drugs?
• Hoffman, et al (Chest, 1987)
  • Compared NTG, furosemide, morphine in 57 presumed prehospital pulmonary edema patients
  • Best outcome with NTG
  • Adverse effects noted in patients receiving furosemide
    • > 25% of patients later required fluid repletion, some hypotensive
    • Significant electrolyte abnormalities developed in some
  • 23% of patients were misdiagnosed and didn’t have pulmonary edema, inappropriately treated
    • Worse outcome in patients receiving furosemide and/or morphine
    • Patients that received NTG alone had no adverse effects
• Kosowsky, et al (*Prehosp Emerg Care*, 2001)
  • Evaluated prehospital use of CPAP for 19 presumed pulmonary edema patients
  • 6/19 (32%) were misdiagnosed and didn’t have pulmonary edema, inappropriately treated
    • No adverse effects to using CPAP in these patients
  • Evaluated outcomes in 599 prehospital presumed decompensated CHF patients
  • 18% of patients were misdiagnosed, inappropriately treated for CHF
    • Asthma, COPD, pneumonia, bronchitis
    • Patients receiving NTG alone — 2.2% mortality
    • Patients receiving morphine and/or furosemide (+ NTG) — 21.7% mortality
  • Asthma, COPD, pneumonia, bronchitis patients treated with bronchodilators — 3.8% mortality
• CHF patients that were misdiagnosed and “inappropriately” treated with bronchodilators — no increased mortality

• Jaronik, et al (Prehosp Emerg Care, 2006)
  • Evaluated 144 prehospital presumed decompensated CHF patients given furosemide
  • 42% of patients had a final diagnosis that was not CHF, furosemide considered “inappropriate”
  • 17% of patients diagnosed with sepsis, dehydration, or pneumonia (without CHF), furosemide considered “potentially harmful”
  • Nine patients died, seven of whom received furosemide “inappropriately”

• Authors’ own EMS system now requires online medical control approval for administration of furosemide

• Plaisance, et al (Eur Heart J, 2007)
  • Evaluated prehospital patients with decompensated CHF for early CPAP (within 15 minutes of arrival) vs. late CPAP (after 30-45 minutes)
  • NTG, diuretics, and vasodilators provided as medication-alone group and also in combination with early vs. late CPAP
  • Early CPAP group had best outcome, with or without medications, in terms of $pO_2$, dyspnea scores, tracheal intubation rates, and (trend) mortality rates
  • Takeaway point: early CPAP made a noticeable difference, even when just applied 15 minutes faster, and even when compared alone vs. combination late CPAP plus medications

VIII. Summary

• Treatment should be based concept of fluid redistribution
  • NTG — first-line agent
    • IV NTG is excellent single-agent
  • ACE-inhibitors — second-line agent
    • In addition to or instead of NTG
  • Furosemide — third-line agent
    • After preload and afterload reduction
  • Morphine — no indication!
    • No proven benefit, potential harm
  • Preload reduction — NTG more effective
  • Anxiolysis — benzodiazepines have fewer side effects
  • Nesiritide
    • Still need to prove that it’s useful for patients not responding to “optimal treatment”
    • May be useful for patients that cannot tolerate NTG or ACE-inhibitors
  • Inotropic support
    • Milrinone better than dobutamine from hemodynamic standpoint
    • No difference in mortality amongst the various choices
• Noninvasive positive pressure ventilation
  • Produces more rapid improvement, decrease intubations, decreased length of ICU stay, decreased hospital costs
  • Consider early use

• Prehospital treatment
  • Increased morbidity and mortality if misdiagnosed and treated with morphine and/or furosemide
  • Consider limiting treatment to NTG, bronchodilators, NPPV (if available)

Questions or comments? Please contact me:
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