



Chapter 81 – Heart Failure

Episode Overview:

1. Define:
 - a. Cardiac index
 - b. Preload
 - c. Afterload
2. Describe:
 - a. How compliance changes the relationship between end diastolic pressures and volume
 - b. the Frank-Starling relationship
 - c. Pousseils Law and LaPlaces Law
3. List 3 CV and 4 Neurohormonal physiologic compensatory mechanisms in CHF
4. List the 5 most common disease processes resulting in HF and briefly describe the contribution of each
5. Describe the different classifications of heart failure:
 - a. Acute vs. Chronic HF
 - b. Systolic vs. Diastolic dysfunction
 - c. Right vs. Left sided HF
 - d. High-output vs. Low-output HF
6. Describe the NYHA function HF Classes and the Killip Classification
7. List 10 common precipitants of acute HF
8. List 6 historical predictors of acute HF and 6 clinical features of acute HF
9. List 5 CXR and 5 ECG findings of HF
10. What is the role of BNP in HF?
11. Describe the primary management goals in acute HF
12. Describe the mechanism of action of NIPPV in HF. Who needs to be intubated? When is it contraindicated?
13. Describe the pharmacologic treatment strategy for:
 - a. Acute pulmonary edema + adequate perfusion
 - b. Acute pulmonary edema + hypotension
14. How do nitrates work in acute pulmonary edema? What is the dose?
15. List 10 treatment options for chronic HF

Rosen's in Perspective

- Heart failure (HF): a “debilitating cardiac syndrome - with -
 - Dyspnea, exercise intolerance
 - Chronic fatigue
 - Huge morbidity and mortality
 - Progressive disease, may begin long before symptoms are evident
- HF definition:
 - Pathophysiologic state where the heart is incapable to pump adequate blood supply to meet the metabolic demands of the body OR requires elevated filling pressures to meet these demands
- We'll discuss more of the pathophys. In question 3.
- Huge costs - “40 billion dollars per year in the USA”
 - 50% mortality after 5 yrs from time of symptoms
 - Death from sudden malignant dysrhythmia or progressive slow decline
 - Mortality predicted by overall cardiovascular-endocrine-renal health

1) Define:

a. Cardiac index

A measurement that relates cardiac output from the LV to the body surface area.

“The CI is a useful marker of how well the heart is functioning as a pump by directly correlating the volume of blood pumped by the heart with an individual's body surface area.”
- Wikipedia

- Normal 2.5-4 L/min/m²
- Determined by:
 - CRAP
 - Contractility
 - Rate - Heart Rate
 - Afterload
 - Preload

b. Preload

- The amount of force stretching the myofibrils before the heart's contraction
 - Factors:
 - Venous return to the chamber
 - Compliance characteristics of the heart muscle (ischemia, hypertrophy)
- We want this to be optimal - as per the Frank-Starling relationship
- The exact value is patient and time specific - we want it to be in the Goldilocks zone
 - If it gets too high though you can tip them into pulmonary edema at their max filling pressure

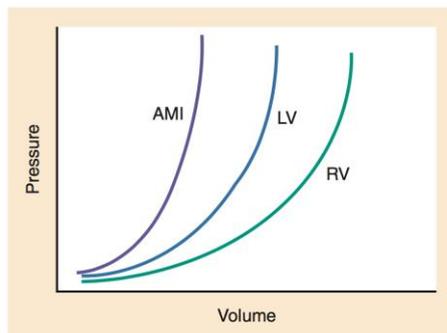


Figure 81-2. The end-diastolic pressure of the chamber is determined by the filling volume and the compliance characteristics of the chamber. The right ventricle (RV) is more compliant than the more muscular left ventricle (LV), which becomes stiffer still under conditions of ischemia or acute myocardial infarction (AMI).

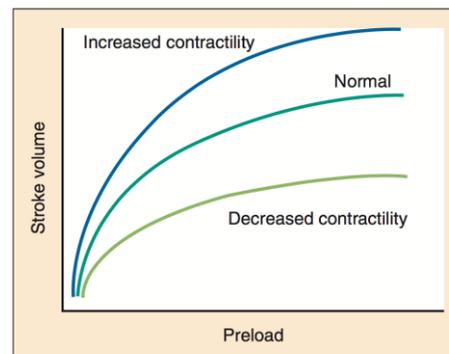


Figure 81-3. Increased preload improves stroke volume irrespective of the contractile state of the ventricle. At any level of contractility, an optimal preload is reached beyond which further increases in chamber pressure may result in increased risk of pulmonary edema, with minimal incremental increase in stroke volume.



c. Afterload

- “The pressure against which the heart must pump to eject blood”
 - $BP = CO \times SVR$
- Determined by the total peripheral resistance and the cardiac chamber size

2) Describe:

a) How compliance changes the relationship between end diastolic pressures and volume

- We discussed this briefly above when we reviewed preload. As a recap, for those of us (me!) who have simple minds and don't get all this flow-physiology stuff...
 - It's covered in Fig 81-2 (above)
- When the compliance of a ventricle decreases (e.g. due to an antero-septal MI) - they need a higher LV filling pressure to produce close to the same stroke volume
- The problem is that, as the pressure rises to maintain cardiac output (a good thing)- the LV end-diastolic pressure also increases. This can result in a bad thing....pulmonary edema

Bottom line:

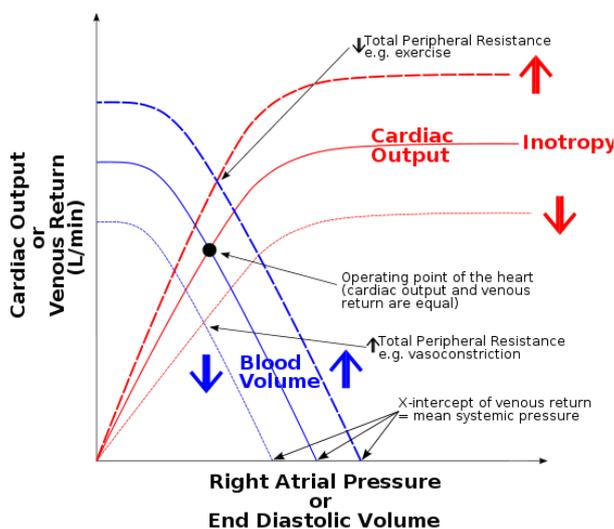
More compliance = Less pressure to achieve the same volume (to a point)

Less compliance = More pressure to achieve the same volume (to a point)

b) The Frank-Starling relationship

A failing heart may be able to temporarily compensate to meet the systemic demands (despite a reduced ejection fraction) by adjusting other factors that contribute to stroke volume.

“The law states that the stroke volume of the heart increases in response to an increase in the volume of blood in the ventricles, before contraction (the end diastolic volume), when all other factors remain constant.” - Wikipedia



By Huckfinne - Own work, Public Domain,
<https://commons.wikimedia.org/w/index.php?curid=9489077>

c) Pousseils Law and LaPlaces Law

- Poiseuilles law:
 - Flow is directly proportional to the fourth power of the vessel radius
 - Bigger is better
- Laplace's law:
 - The larger the ventricular cavity the more mural tension (myocardial work) is needed during contraction
 - Dilated cardiomyopathy = lots of work for the heart because the sarcomeres are stretched too much!
 - This is why failing hearts are ****extremely afterload sensitive****
 - **Afterload reduction converts pressure work into flow work**
- **Big idea:** Maintain preload; Decrease afterload = Happy heart

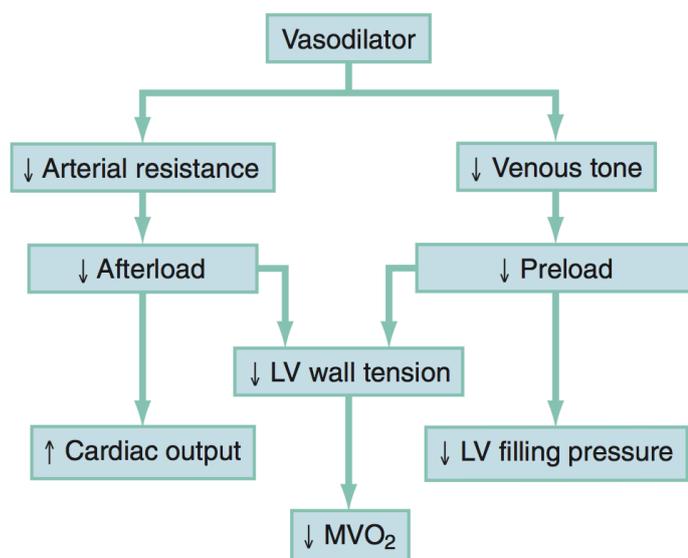


Figure 81-4. Arterial vasodilators decrease resistance to flow and result in increased carbon dioxide. Venodilators decrease venous return to the heart and relieve pulmonary congestion. Balanced agents (e.g., nitrates) do both. Decreased mural tension reduces myocardial oxygen demand (MVO_2) and may relieve ischemia. *LV*, left ventricular.

Let's take a moment and recap all this physiology-ICU-nerd-brainy stuff.....I keep it straight by trying to optimise CRAP - we want all these things to be in the goldilocks zone....

- Contractility - Happy heart muscle
 - Stop the bad drugs
 - Make sure calcium is optimized
 - Give the heart oxygen
 - Decrease excessive catecholamines / acidotic states
- Heart rate
 - Optimal CO depends on: HR and SV
 - HR increases to a MAX of 150-160 improve cardiac output
 - Beyond that you sacrifice diastolic filling time and coronary perfusion and the ability for atrial contraction



- We want the patient in a SINUS rhythm....if they aren't use the right drugs or electricity to reset the heart rhythm
- Afterload
 - Ideally we want a drug to decrease afterload so the heart doesn't work as hard!
- Preload
 - Optimize that preload with either increasing the volume or decreasing it!

3) List 3 CV and 4 Neurohormonal physiologic compensatory mechanisms in CHF

1076 PART III ♦ Medicine and Surgery / Section Three • Cardiac System

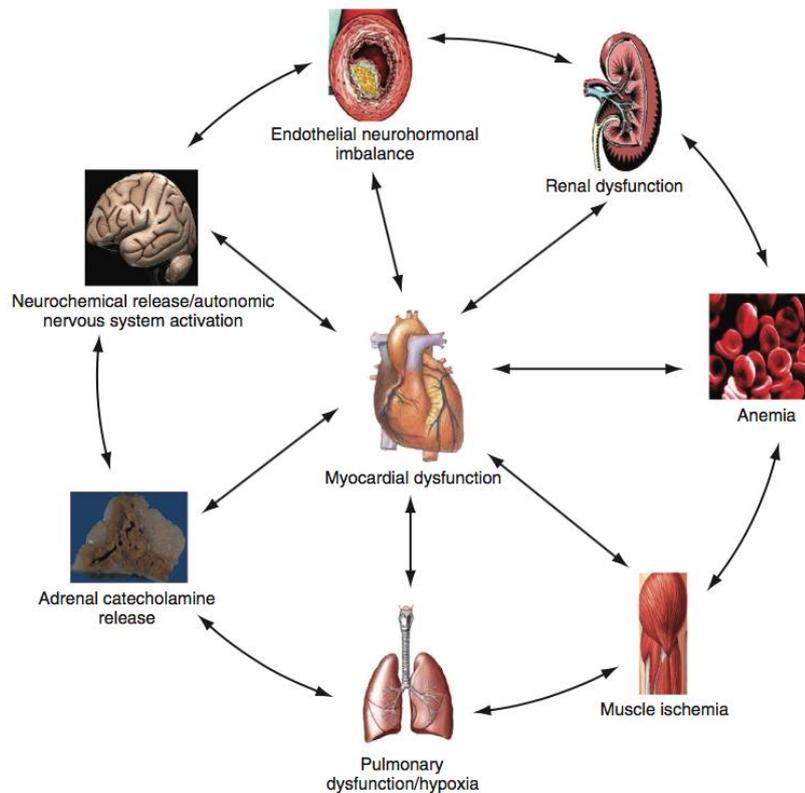


Figure 81-1. The neurohormonal model of heart failure describes a complex interdependence among many organ systems in which a functional disturbance in any component causes complex compensatory changes in the others that are eventually maladaptive. Correction of organ system dysfunction by medications and other interventions may result in correction of these perturbations.

Complex interplay between the heart and the rest of the body:

Myocardial dysfunction - pulmonary hypoxia - muscle ischemia (and sarcopenia) - renal dysfunction: All causing compensatory adaptation which becomes MALADAPTIVE

Cardiovasc. Compensation:

- Increased stroke volume
 - Due to an increase in preload (Frank-Starling)
 - This adaptation eventually plateaus, while creating massive myocardial oxygen demand
- Increased cardiac muscle size (hypertrophy +/- fibrosis/apoptosis)
 - Initially helps the heart pump the same CO; but then forms more pathologic



myocyte isoforms leading to more mass than capillary blood flow → fibrosis →
myocyte death

- Increase systemic vascular resistance
 - Takes blood to the brain and heart - but away from skin, muscles, kidneys
 - Forces the heart to do extra work

Neurohormonal changes - counterproductive on a chronic basis

- Renal neurohormonal response
 - RAAS activation - ADH activation
- Neurochemicals and enhanced ANS activation
 - Elevations in vasopressin
 - Inhibition of parasympathetic tone
 - Chronically elevated catecholamines
- Natriuretic peptides
 - From the heart and brain: promote water and sodium excretion, increase vasodilation and inhibit RAAS
- Endothelin
 - Disease in the vascular endothelin response → hypertension

4) List the 5 most common disease processes resulting in HF and briefly describe the contribution of each

1. Coronary artery disease
 - a. #1 cause
 - b. Myocardial necrosis, fibrosis and scarring → dyskinesia
 - c. Aneurysmal dilation of infarcted areas
 - d. Cardiogenic shock -- occurs when 40% of the LV muscle is infarcted
 - e. Chronic coronary insufficiency:
 - i. Diffuse ischemic cardiomyopathy
 1. Worsened in sickle cell disease, diabetes mellitus
 - ii. Ventricular remodelling - dilation, hypertrophy, fibrosis
2. Cardiomyopathy / myocarditis
 - a. Dilated, hypertrophic, restrictive
3. Valvular heart disease
 - a. Mitral or aortic valve regurg/stenosis
4. Pericardial diseases
 - a. Look for the pericardial effusion
5. Pulmonary disease
 - a. COPD - 30% prevalence in people with CHF
 - i. Hypoxia also leads to pulmonary arteriolar vasoconstriction which leads to cor pulmonale
 - b. PE

5) Describe the different classifications of heart failure:

Lots of them here! In the ER - most of these lines are blurred....

a. Acute vs. Chronic HF

- i. We mostly see acutely decompensated HF. e.g. due to a large MI leading to



- acute pulmonary edema
- ii. The person may then bounce in and out of ER with acute worsening of their dilated cardiomyopathy

b. Systolic vs. Diastolic dysfunction

- AHA/ACC definition of systolic dysfunction in HF as a LVEF < 40%
 - Stroke output reduced, less forward flow
- Diastolic dysfunction = the failure of the ventricles to relax, leading to high filling pressure
 - The ventricles can't relax and fill normally
 - Remember that muscle relaxation is an active, energy-requiring process
 - ***these people can have HF with a NORMAL EF!
 - Very commonly asymptomatic, especially in hypertrophic and restrictive cardiomyopathy, hypertension, AS
 - These restrictive changes lead to congestion
 - Higher mortality than systolic dysfunction
 - Very hard to treat....

c. Right vs. Left sided HF

- i. It's a false mantra that "one ventricle can fail independent of the other...."
 - 1. They are in the same circuit and are attached by the Interventricular septum!
 - 2. Catecholamine responses affect both chambers
- ii. This distinction is more helpful in thinking of symptoms of heart failure and their clinical presentations.....e.g..
 - 1. Right HF = hepatosplenomegaly, edema, systemic venous congestion
 - 2. May be helpful in the patient with an acute AMI who presents with an elevated JVP, hypoperfusion and normal lungs.....in that case give IV fluids and norepi (prime the LV!)

d. High-output vs. Low-output HF

- i. High output:
 - 1. Hyperdynamic state with high CO and low arteriovenous O₂ difference.
 - 2. Due to increased preload, sympathetic activity, tachycardia...
- ii. Low-output:
 - 1. Most common type: due to ischemic heart disease, dilated cardiomyopathy, valvular disease, hypertension.
 - 2. Characteristics:
 - a. Systolic dysfunction
 - b. Diastolic dysfunction
 - c. Increased systemic oxygen extraction ratio



6) Describe the NYHA function HF Classes and the Killip Classification

Box 81-2 Classification System for Chronic Heart Failure: New York Heart Association Functional Classes

- I. Asymptomatic on ordinary physical activity
- II. Symptomatic on ordinary physical activity
- III. Symptomatic on less than ordinary physical activity
- IV. Symptomatic at rest

Patients were ranked by Killip class in the following way:

- **Killip class I** includes individuals with no clinical signs of [heart failure](#).
- **Killip class II** includes individuals with rales or crackles in the [lungs](#), an S₃, and elevated jugular venous pressure.
- **Killip class III** describes individuals with frank [acute pulmonary edema](#).
- **Killip class IV** describes individuals in [cardiogenic shock](#) or [hypotension](#) (measured as [systolic blood pressure](#) lower than 90 mmHg), and evidence of peripheral [vasoconstriction](#) ([oliguria](#), [cyanosis](#) or sweating). - From Wikipedia

For a calculator:

https://www.qxmd.com/calculate/calculator_126/killip-class

This is used to predict mortality post-AMI.

7) List 10 common precipitants of acute HF

Box 81-1 Common Precipitating Causes of Acute Heart Failure

- Sodium and volume excess
 - Systemic hypertension
 - Myocardial infarction or ischemia
 - Systemic infection
 - Dysrhythmias
 - Acute hypoxia or respiratory problems
 - Anemia
 - Pregnancy
 - Thyroid disorders
 - Acute myocarditis
 - Acute valvular dysfunction
 - Pulmonary embolus
 - Excess exertion or trauma
 - Pharmacologic complications
- Cardiac
 - Rate, rhythm, muscle,
 - Pulmonary
 - Pharmacologic
 - Cocaine
 - Corticosteroids, NSAIDs, and vasodilator medications leading to sodium retention and volume expansion
 - ****NSAIDs are BAD for people with CHF!!!
 - Impair renal homeostasis, interfere with ACE & diuretics

- Metabolic / systemic
 - Missed dialysis
 - Anemia
 - Thyroid
 - Sepsis
 - Acute stress states/emotional upset

8) List 6 historical predictors of acute HF and 6 clinical features of acute HF

- Hx
 - Past history of HF
 - PND
 - SOBOE
 - Orthopnea
 - Nocturia
 - Hx of any type of heart disease
- Px
 - Hypertension
 - Diaphoretic
 - Pulmonary crackles
 - Pulmonary wheezes (cardiac asthma - due to peribronchial edema) - which may respond to bronchodilator therapy!!
 - JVD
 - Edema
 - S3

9) List 5 CXR and 5 ECG findings of HF

CXR: From radiopedia: <https://radiopaedia.org/articles/heart-failure-basic>

- Pleural effusions
- Cardiomegaly (enlargement of the cardiac silhouette)
- Kerley B lines (horizontal lines in the periphery of the lower posterior lung fields)
- Upper lobe pulmonary venous congestion (bat wing appearance)
- Interstitial oedema.

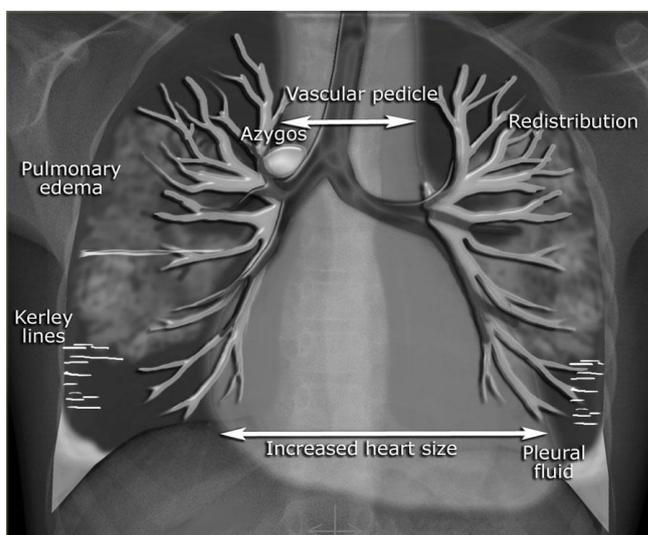


Image from:
<http://www.radiologyassistant.nl>



ECG:

- i. LVH
- ii. LAE
- iii. RAE
- iv. RV Strain: ST depression and T wave inversion in the leads corresponding to the right ventricle, i.e The right precordial leads: V1-3, often extending out to V4. The inferior leads: II, III, aVF, often most pronounced in lead III as this is the most rightward-facing lead
- v. Fascicular blocks and bundle branch blocks

10) What is the role of BNP in HF?

Rosen's describes BNP has having many benefits:

"Natriuretic peptide levels correlate with ventricular function, NYHA classification, and prognosis. Results of large clinical trials confirm that BNP levels are the strongest predictor of outcome in HF compared with other neurohormones and clinical markers. There is often a disconnect between the perceived severity of HF by clinicians and the degree of BNP elevation, yet BNP levels are better predictors of 90-day outcome than physician judgment. High pre-discharge BNP and NT-proBNP levels are strong, independent predictors of death or rehospitalization after decompensated HF." From Rosen's 8th Ed.

In contrast, the authors of UpToDate have a very different opinion....

According to Uptodate: "The value of serial BNP measurement in guiding management of acute HF has not been established.....As separate issue is that some patients with acute HF do not have elevated BNP or NT proBNP levels; natriuretic peptide levels may be mildly or not significantly elevated if the etiology of the HF is inflow tract obstruction (eg, mitral stenosis)], constrictive pericarditis, or if the HF is very acute (first hour or so)"

In real life, the likelihood of BNP changing ER management is low....but it remains a frequently ordered test, often to help our admitting colleagues rather than our disposition.

11) Describe the primary management goals in acute HF

According to Rosen's 8th ed.

"The approach focuses on (1) determining underlying cardiac pathology, (2) identifying the acute precipitant, and (3) mitigating the acute decompensation. The immediate therapeutic goals are to improve respiratory gas exchange, maintain adequate arterial saturation, and decrease LV diastolic pressure while maintaining adequate cardiac and systemic perfusion. The acute congestive state can be controlled by (1) reducing cardiac workload through decreased preload and afterload, (2) controlling excessive retention of salt and water, and (3) improving cardiac contractility. Patients may have a wide spectrum of symptoms and signs ranging from mild dyspnea on exertion to full-blown cardiogenic shock with hypotension and concomitant respiratory failure."

**Remember.... trying to optimise CRAP - we want all these things to be in the goldilocks zone....



- Contractility - happy heart muscle
 - Stop the bad drugs
 - Make sure calcium is optimized
 - Give the heart oxygen
 - Decrease excessive catecholamines / acidotic states
- Rate
 - Optimal CO depends on: HR and SV
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 - Beyond that you sacrifice diastolic filling time and coronary perfusion and the ability for atrial contraction
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 - Ideally we want a drug to decrease afterload so the heart doesn't work as hard!
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12) Describe the mechanism of action of NIPPV in HF. Who needs to be intubated? When is it contraindicated?

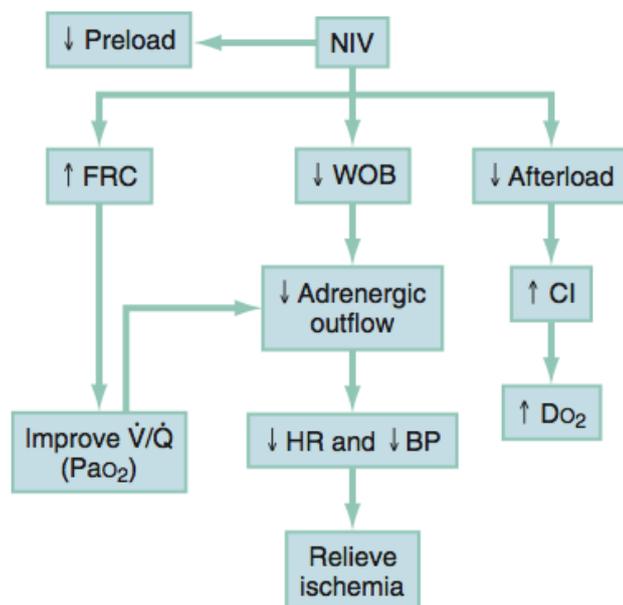


Figure 81-6. Noninvasive ventilation (NIV) techniques recruit collapsed alveoli and increase functional residual capacity (FRC), which improves oxygenation and reduces work of breathing (WOB). These factors tend to reduce sympathetic tone, heart rate (HR), and blood pressure (BP), relieving myocardial ischemia. NIV also acts as an afterload-reducing agent, which tends to directly improve cardiac index (CI), systemic oxygen delivery (DO₂), partial pressure of oxygen in arterial blood (PaO₂), and ventilation-perfusion ratio (V̇/Q̇). Preload is also reduced by NIV.

Check out the podcast for information on who needs intubation and who shouldn't get NIPPV!

13) Describe the pharmacologic treatment strategy for:

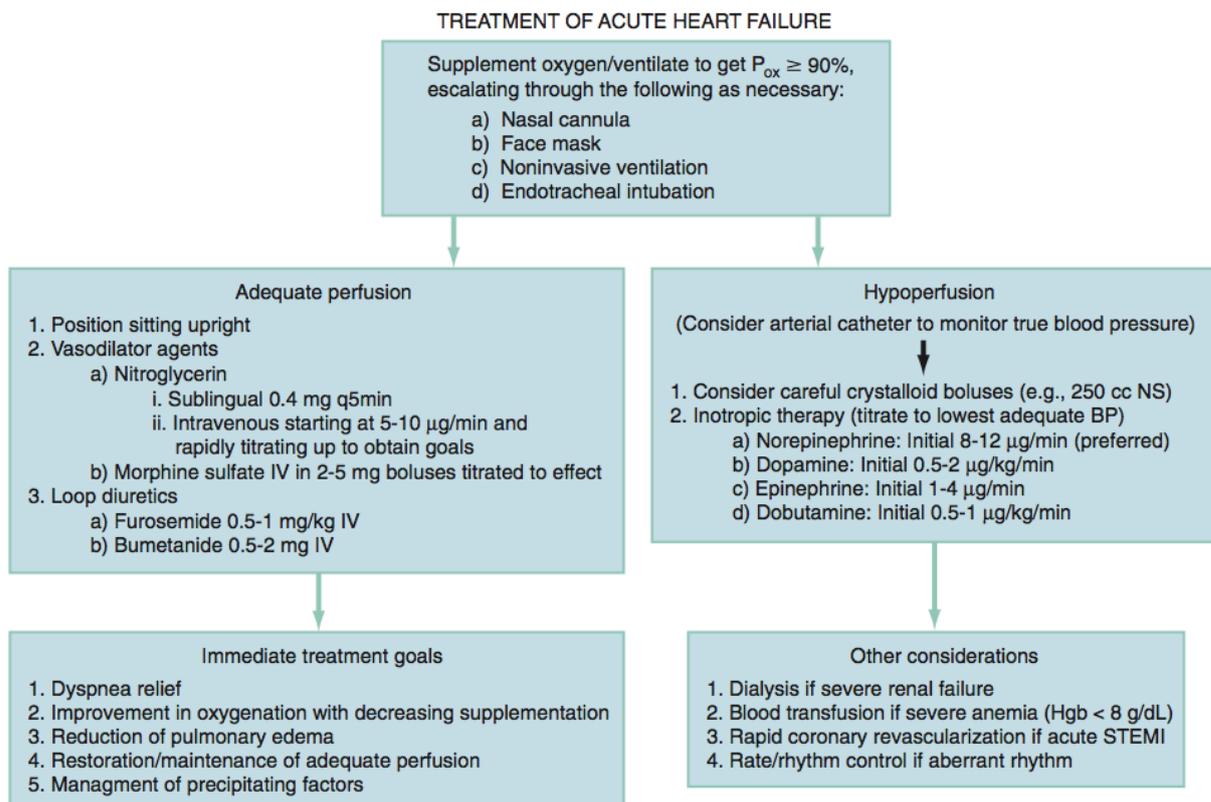


Figure 81-5. A general therapeutic strategy for acute heart failure requires thoughtful application of interventions to achieve a reasonable improvement in early clinical treatment goals. Appropriate interpretation of the perfusion with use of clinical criteria and occasionally arterial pressure monitoring is central to the strategy. *BP*, blood pressure; *Hgb*, hemoglobin; *IV*, intravenously; *NS*, normal saline; *P_{ox}*, pulse oximetry; *STEMI*, ST segment elevation myocardial infarction.

a) Acute pulmonary edema + adequate perfusion

- i. Memory AID: “the patient’s heart is failing and the blood is pooling into a PONND”

(Position, O₂, NIPPV, Nitrates, Diuretics) “PONND”

b) Acute pulmonary edema + hypotension

- i. **POND plus vasopressors / inotropes**

-> Carefully avoid nitrates!

14) How do nitrates work in acute pulmonary edema? What is the dose?

- Vasodilatation and arterial dilatation
- Drops preload reduces afterload, thereby improving filling pressures and dropping SVR

See figure above for dosing strategy



15) List 10 treatment options for chronic HF

1. Cardiovascular exercise and strength training / conditioning program
2. Obesity reduction
3. Healthy diet
4. Beta-blockers
5. ACE Inhibitors
6. ARBs
7. Diuretics
8. Spironolactone
9. Digoxin
10. Cardiac resynchro therapy (biventricular pacemaker)

These medications have been shown to reduce five-year mortality