Heart Failure (I)

Ronald Zolty, MD, PhD

Case Presentation

- 42 year old African American man, who recently moved from Kansas
- PMH: HTN diag 3 years ago
- Diagnosed in May 07 with severe CHF

  Echocardiogram
  - LVEDD 6.6cm, LVEDS 5.6
  - Severe global hypokinesis, LVEF 30%
  - Moderate right ventricular hypokinesis
  - Severe MR, mod-severe TR

Case Presentation

- Coronary Angiogram: Clean cors
- LV Angiogram: LVEF 27%, MR3+
- PSH: None
- FMH: unremarkable
- Allergy: NKDA

Case Presentation

- MEDS:
  - Cozaar 100 mg QD
  - Atenolol 50 mg QD
  - Spironolactone 50mg QD
  - Lasix 80 mg BID
  - Amlodipine 10 mg QD

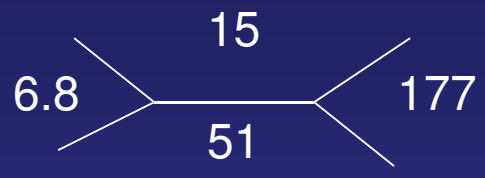
Case Presentation

PE

- BP 95/60 HR 88, RR 20, Anicteric, Afebrile Acyanotic
- JVP ~ 6-8cm
- Chest: Clear, no crackles, no rales, no wheezing
- CV: RRR, PMI displaced
- Abd: Soft, 0 tenderness, 0 HSM
- Legs: No edema

Case Presentation

LAB



LFTs: Normal

UA: WNL

EKG: Normal Sinus Regular Rhythm, LBBB QRS 150 msec

CXR: Cardiomegaly, no infiltrates

Case Presentation

- Adequate Rx ? (6 mistakes)
- What is the next step ?
- Is Pt a candidate for Mitral Valve Repair ?
- Does Pt need Transplant evaluation ?

Heart failure

- Heart Failure an Important Health Problem
- Pathophysiology
  - Diastolic LV Dysfunction
  - Systolic LV Dysfunction
- Clinical Presentation in Patients with HF
- Assessment of Heart Failure
- Hemodynamics

Heart Failure Definition

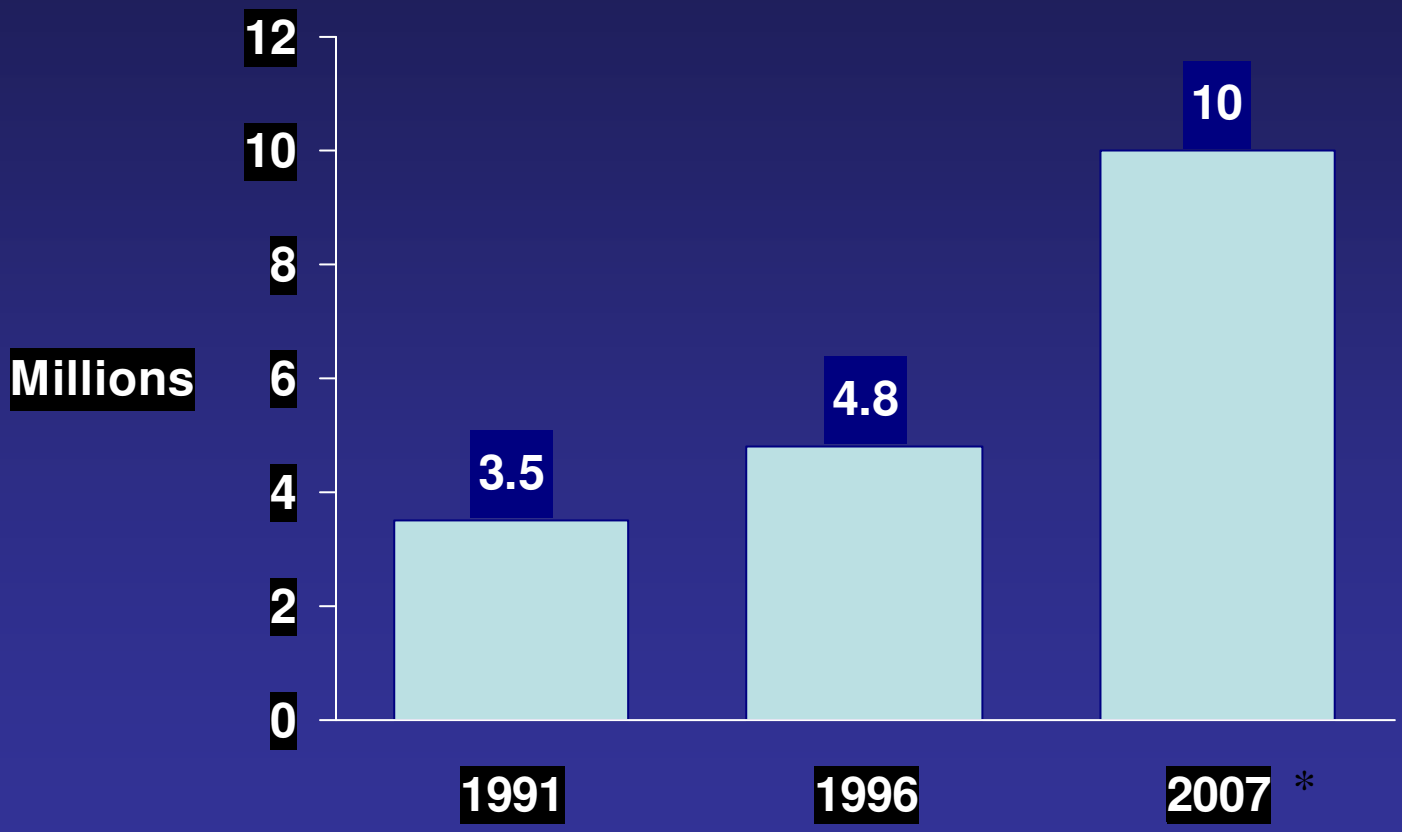
- Clinical Syndrome characterized by:
  - Impaired left ventricular performance (systolic, diastolic or both)
  - Reduced functional capacity
  - Neurohormonal dysregulation
  - Fluid retention
  - Impaired quality of life
  - Increased mortality

Heart Failure: an Important Health Problem

- Approximately 10 million Americans have HF (~1.5% of the US population)
- Incidence of HF has markedly increased over last three decades (250,000 in 1970, currently 550,000 new cases annually).
- Is associated with aging: incidence and prevalence increase in elderly population (Incidence of 10/1000>65 years of age).
- Hospital discharges 1,000,000 (2001).
- Single largest expense for Medicare.
- Five-year mortality rate as high as 50% .

AHA 2001, Heart and Stroke Statistical Update

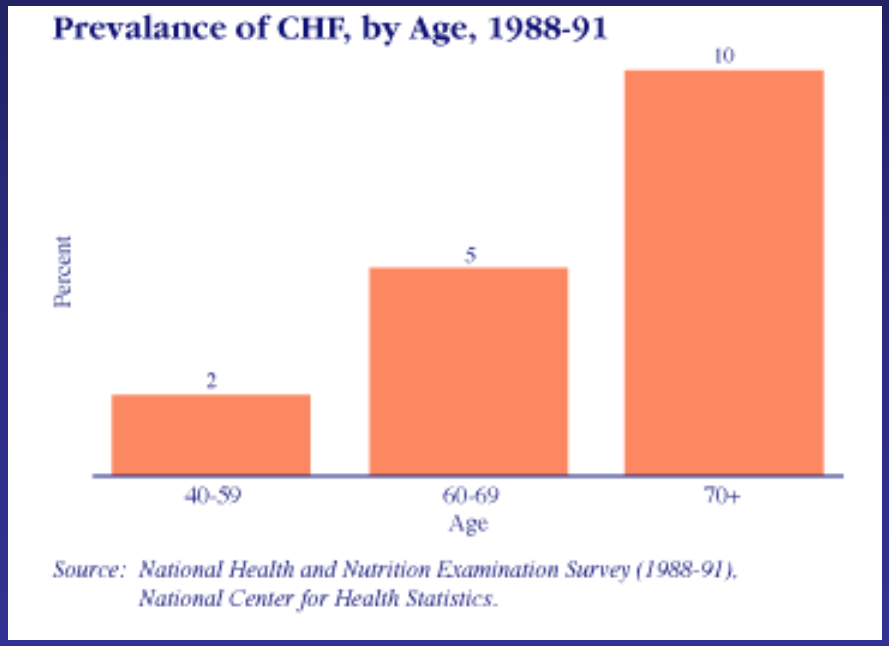
Increasing Prevalence of HF



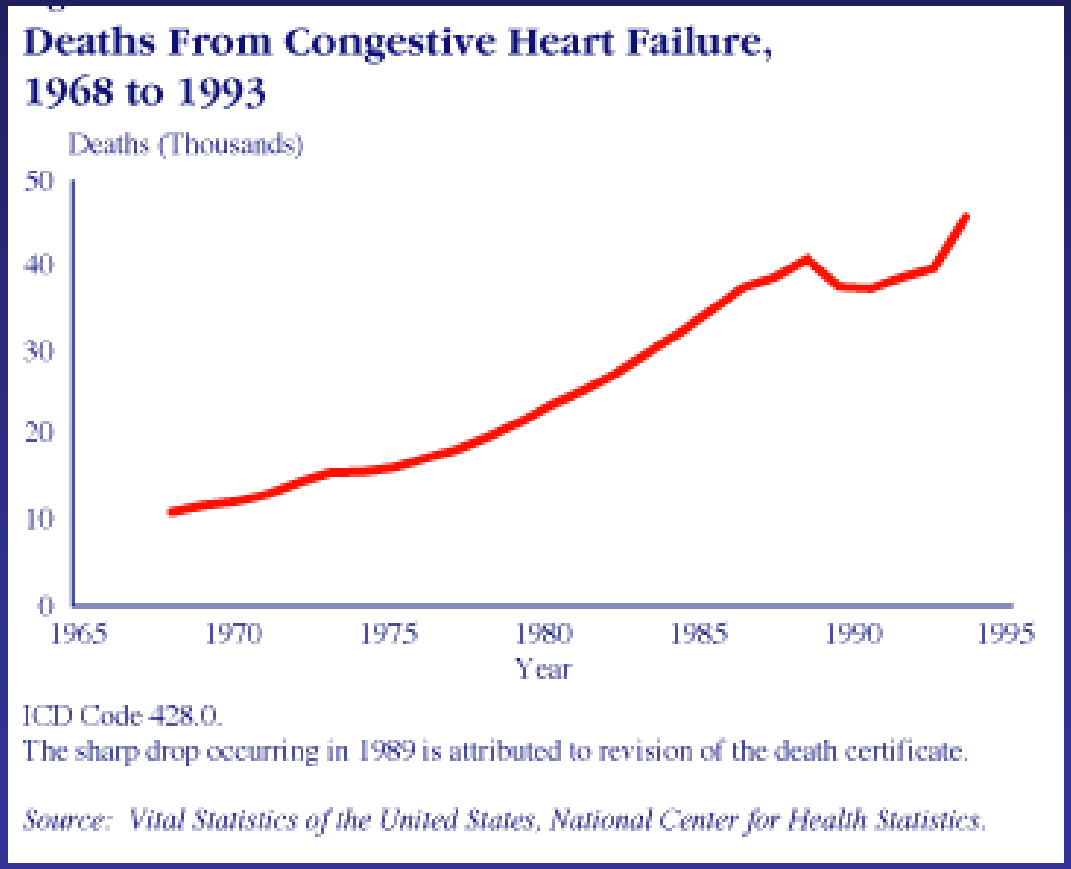
American Heart Association. 2008 Heart and Stroke Statistical Update.1999.

*Rich MW, Nease RF. *Arch Intern Med*. 1999;159:1690-1700.

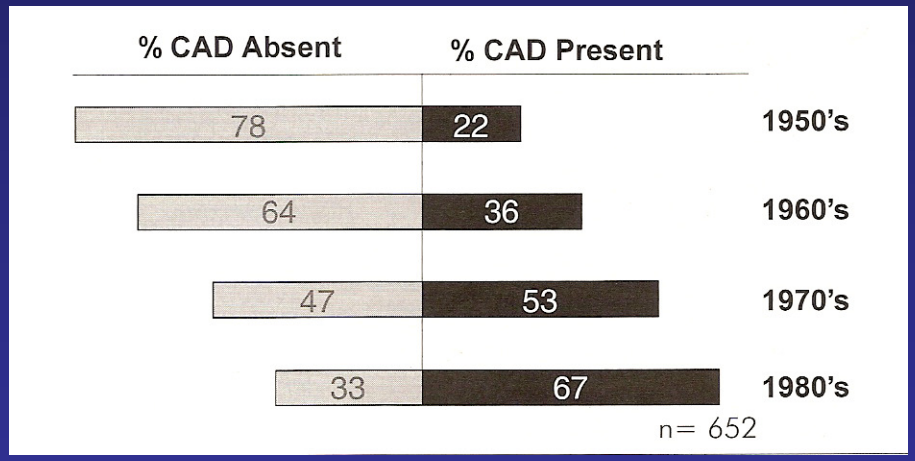
Heart Failure: an Important Health Problem



Congestive Heart Failure: an Important Health Problem



Increase in Coronary Artery Disease as Etiology of Heart Failure Over Four Decades



Ho KK et al Circulation. 1993; 88: 107-115

Relation Between Severity of Heart Failure and One-year Mortality

NYHA Functional Class	Annual Mortality (%)	Sudden Death (%)
II	5-15	50-80
III	20-50	30-50
IV	30-70	5-30

NEW YORK HEART ASSOCIATION (NYHA) FUNCTIONAL CLASS

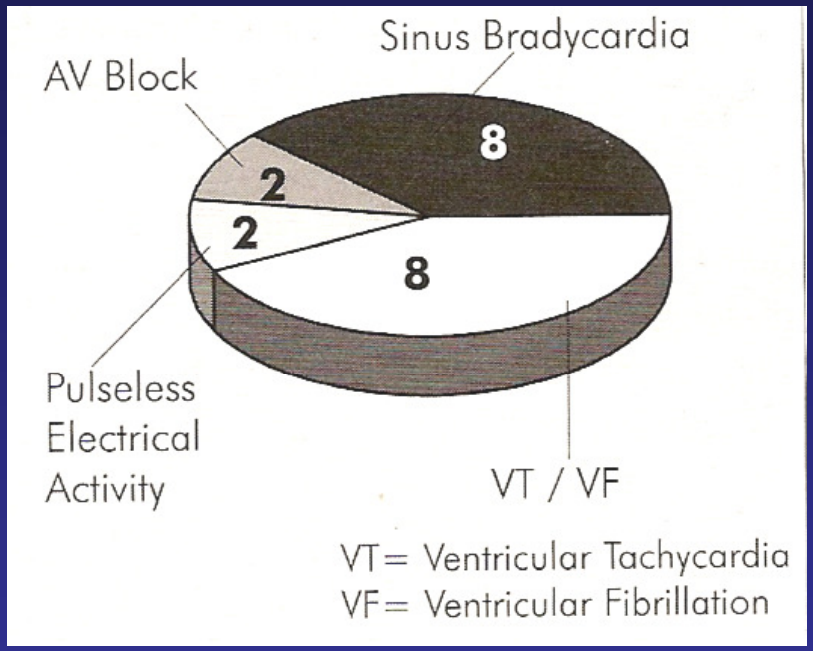
Class I: No limitation of functional activity

Class II: Slight limitatation of activity. Dyspnea and fatigue with moderate physical activity

Class III: Marked limitation of activity. Dyspnea with minimal activity

Class IV: Severe limitation of activity. Symptoms are preset even at rest

Etiology of Sudden Death



Heart rhythm recorded at onset of sudden death in 20 patients with HF awaiting heart transplant

Luu et *al*. Circulation, 1989; 80:1675

Heart Failure in Men *vs.* Women

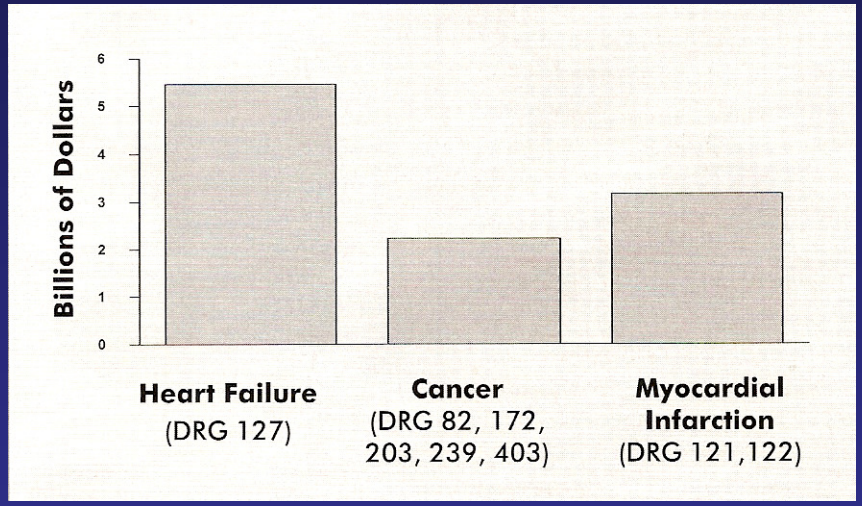
	MEN	**WOMEN**
Annual Incidence (Age >45)	7.2/1000	4.7/1000
Median Survival (excluding <90 day mortality	3.2 years	5.4 years

Total HF Costs (Calculated)

Health Care Financing Administration. Medicare Provider Analysis and Review (MEDPAR) Inpatient Hospital Fiscal Year 1997

Estimate calculated at 4.93% of National Health Expenditures

The Economic Cost of Heart Failure



Comparison of Health Care Financing Administration expenditures of HF compared with cancer and MI according to Medicare Program

O'Connell JB et *al*. *J Heart Lung Transplant*. 1994; 13: S107-S111

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Heart Failure due to Cardiomyopathy

Cardiomyopathy is a disease of the heart muscle

Primary

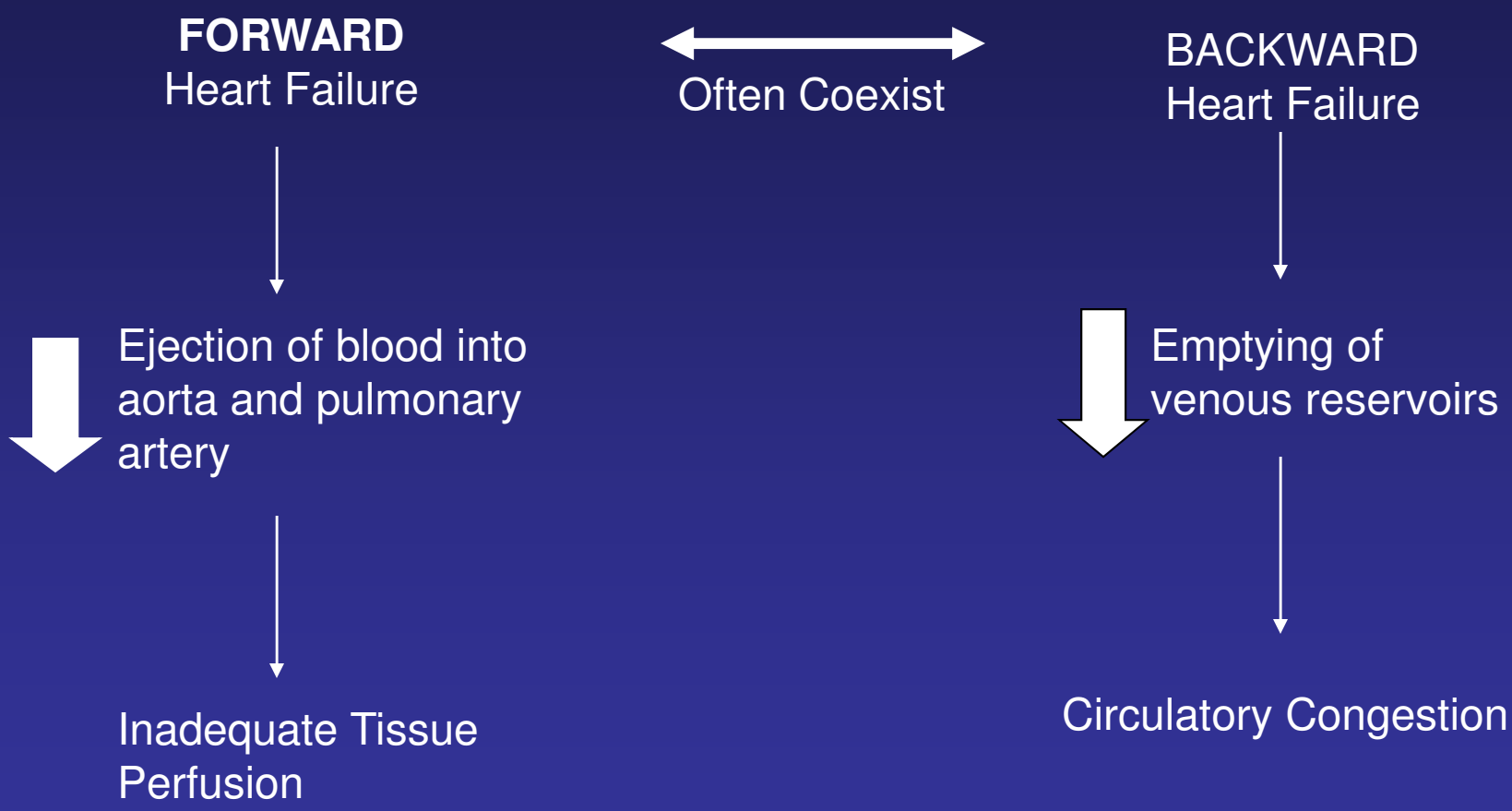
- Dilated
- Hypertrophic
- Restrictive
- Arrhythmogenic
- Unclassified

Secondary

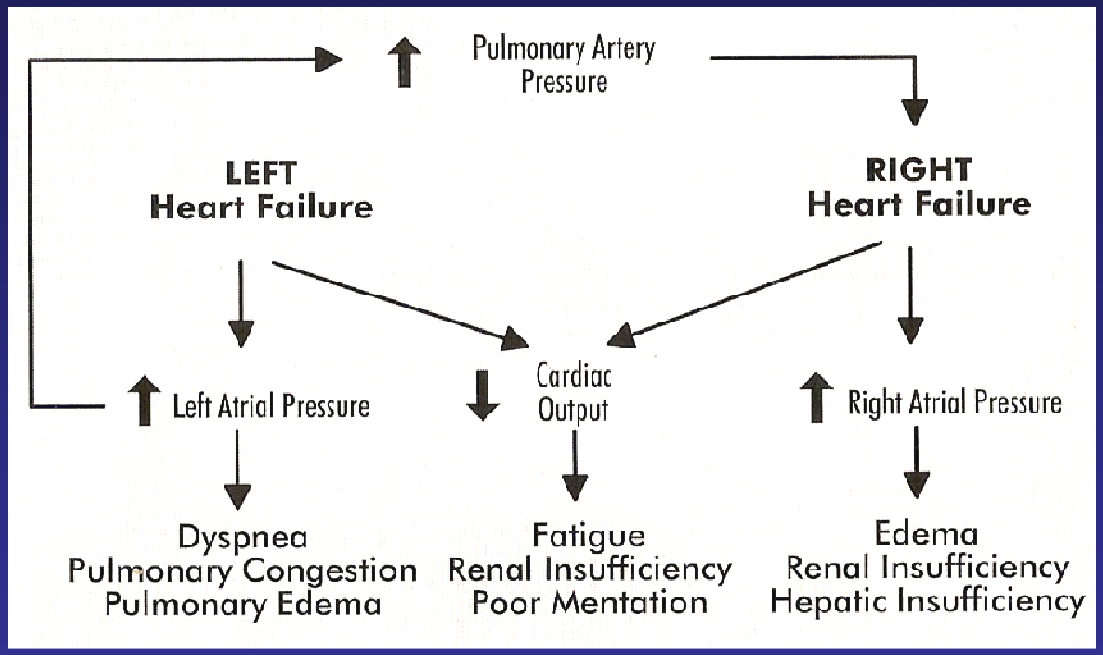
- Ischemic
- Valvular
- HTN
- Others

Report of the 1995 WHO/InternationalSociety and Federation of Cardiology Task Force on the definition and classification of CM. Circ 1996

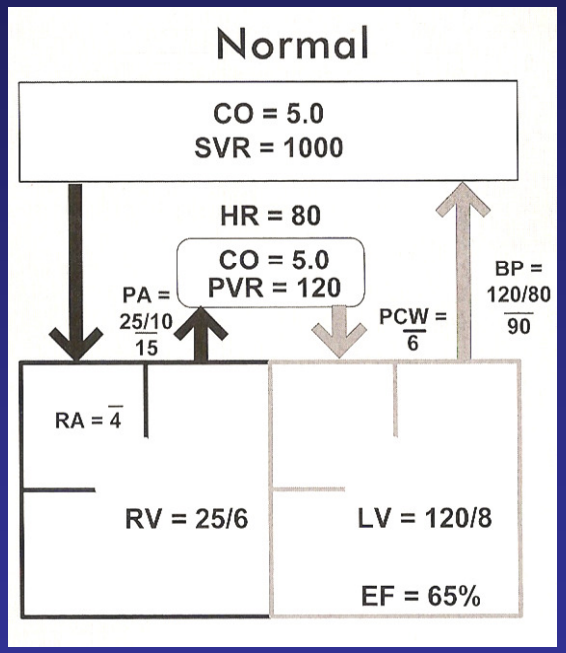
Heart Failure= Forward and/or Backward Heart Failure

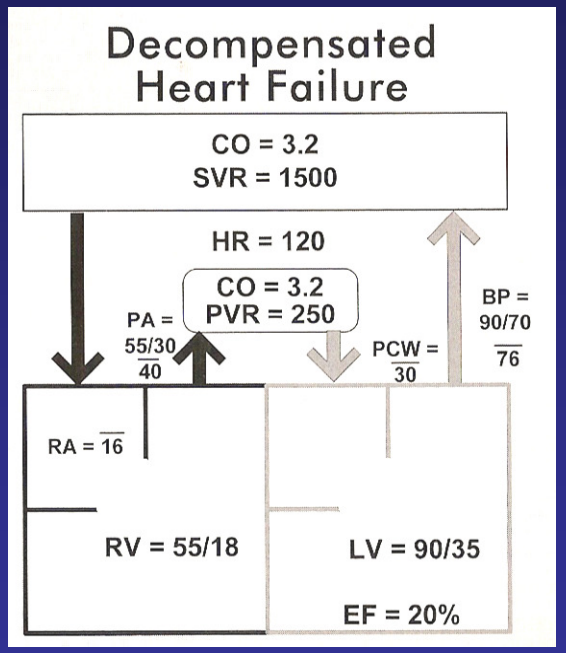


Left *vs.* Right Sided Heart Failure



Normal and Heart Failure Hemodynamics





Useful Definitions

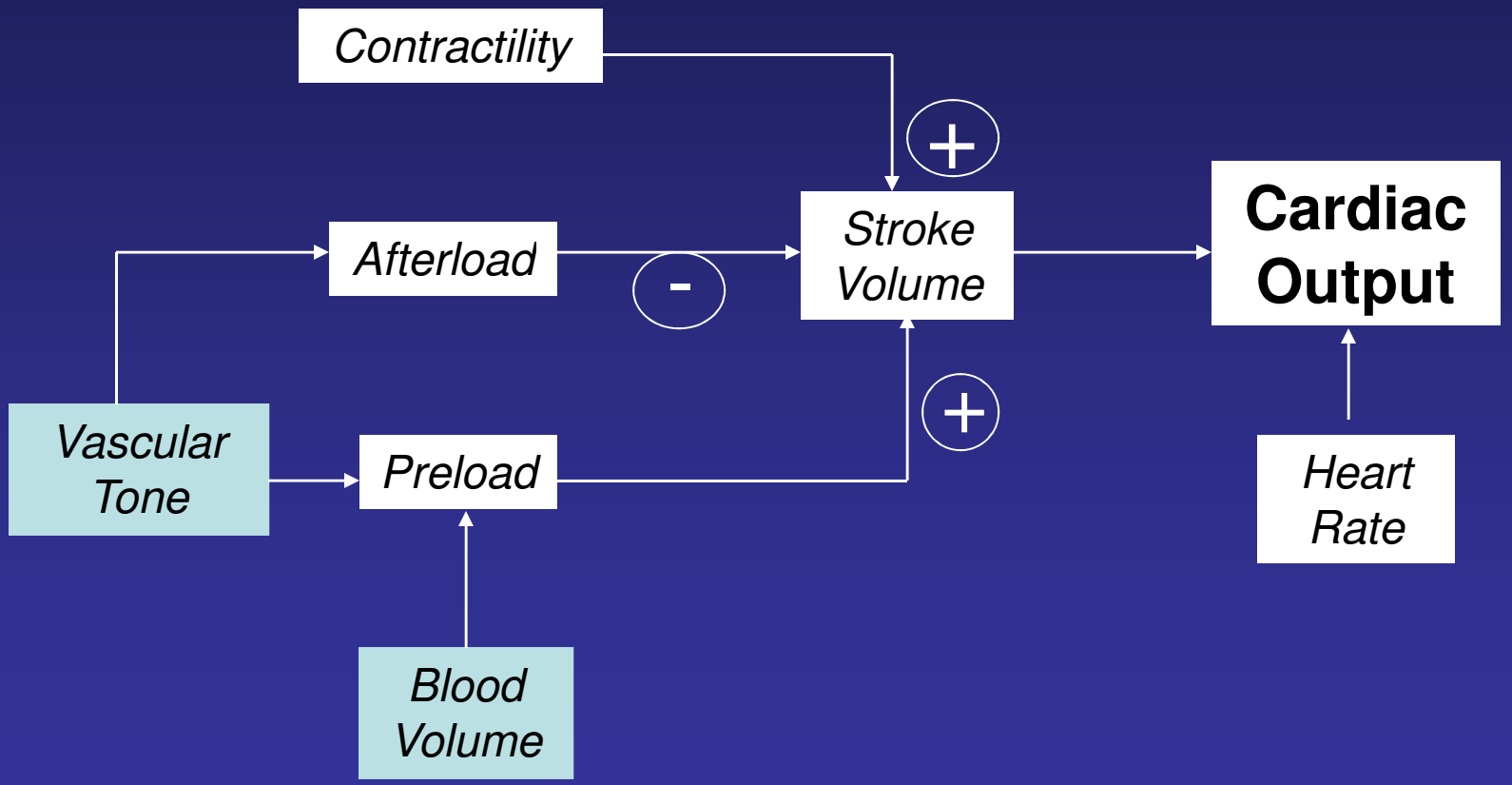
PRELOAD

- Ventricular *end-diastolic* pressure and volume that determine resting sarcomere length just prior to contraction

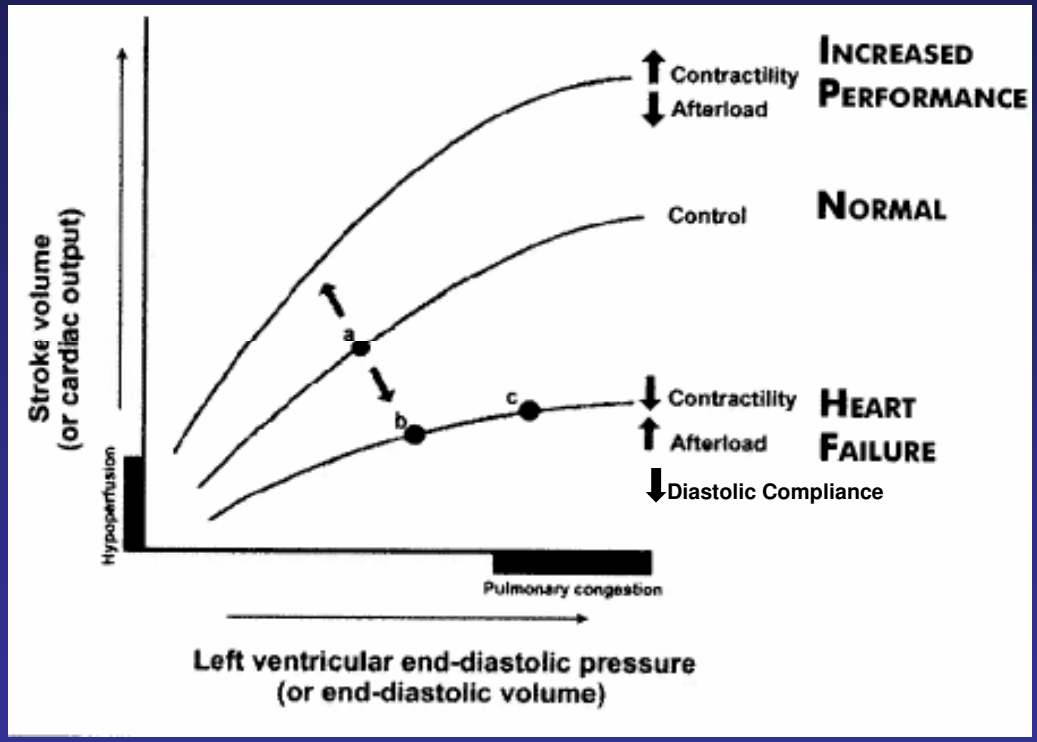
AFTERLOAD

- Pressure and volume *after the onset of contraction* and therefore, the resistance that the ventricle must overcome to eject its contents.

Factors Influencing Cardiac Output



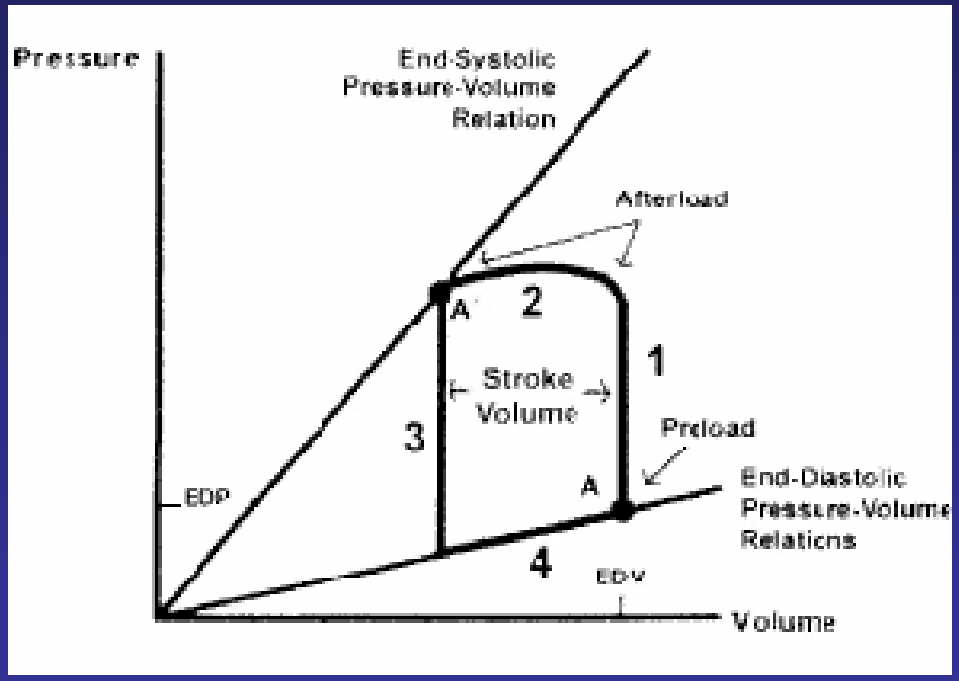
Frank-Starling Curve



Heart Failure results in a downard shift of the curve resulting in hypoperfusion (b), pulmonary congestion (c), or both

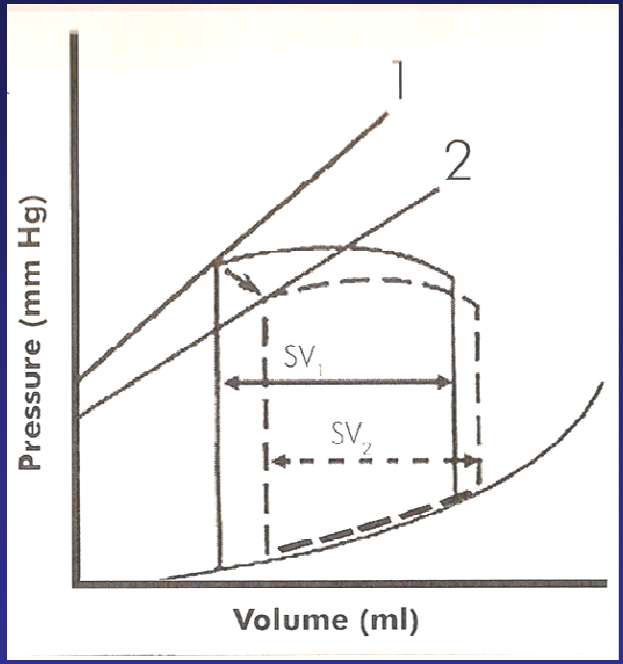
Ventricular Pressure-Volume Relationships

The right and left ventricle are phasic circulatory pumps that convert biochemical energy into mechanical pressure-volume work



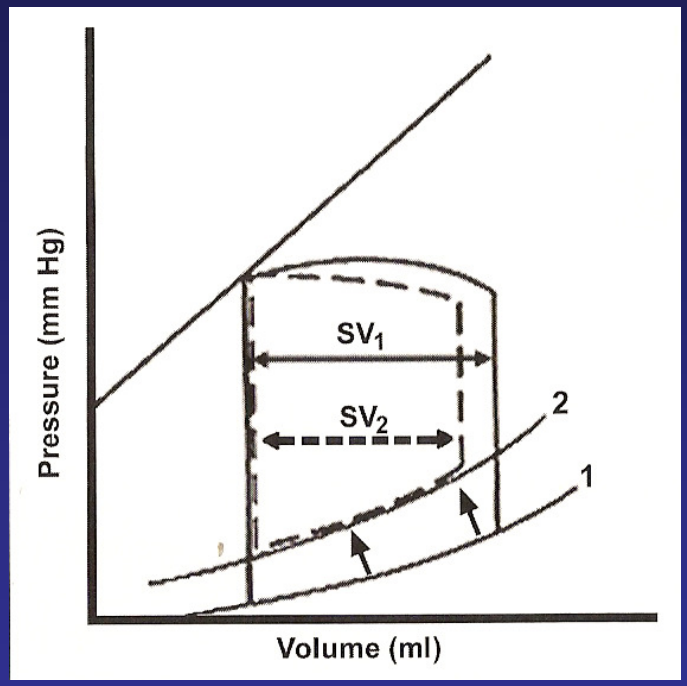
Phases of the cardiac cycle which occur with heart beat (1) Isovolemic systole, (2) Systolic ejection, (3) Isovolemic relaxation, (4) Diastolic filling

Pressure-Volume Loop in Systolic Dysfunction



In LV systolic dysfunction, the end-systolic pressure-volume curve moves from 1 to 2. This leads to a decrease in systolic pressure, stroke volume despite compensatory increase in the operating point on the diastolic pressure-volume curve

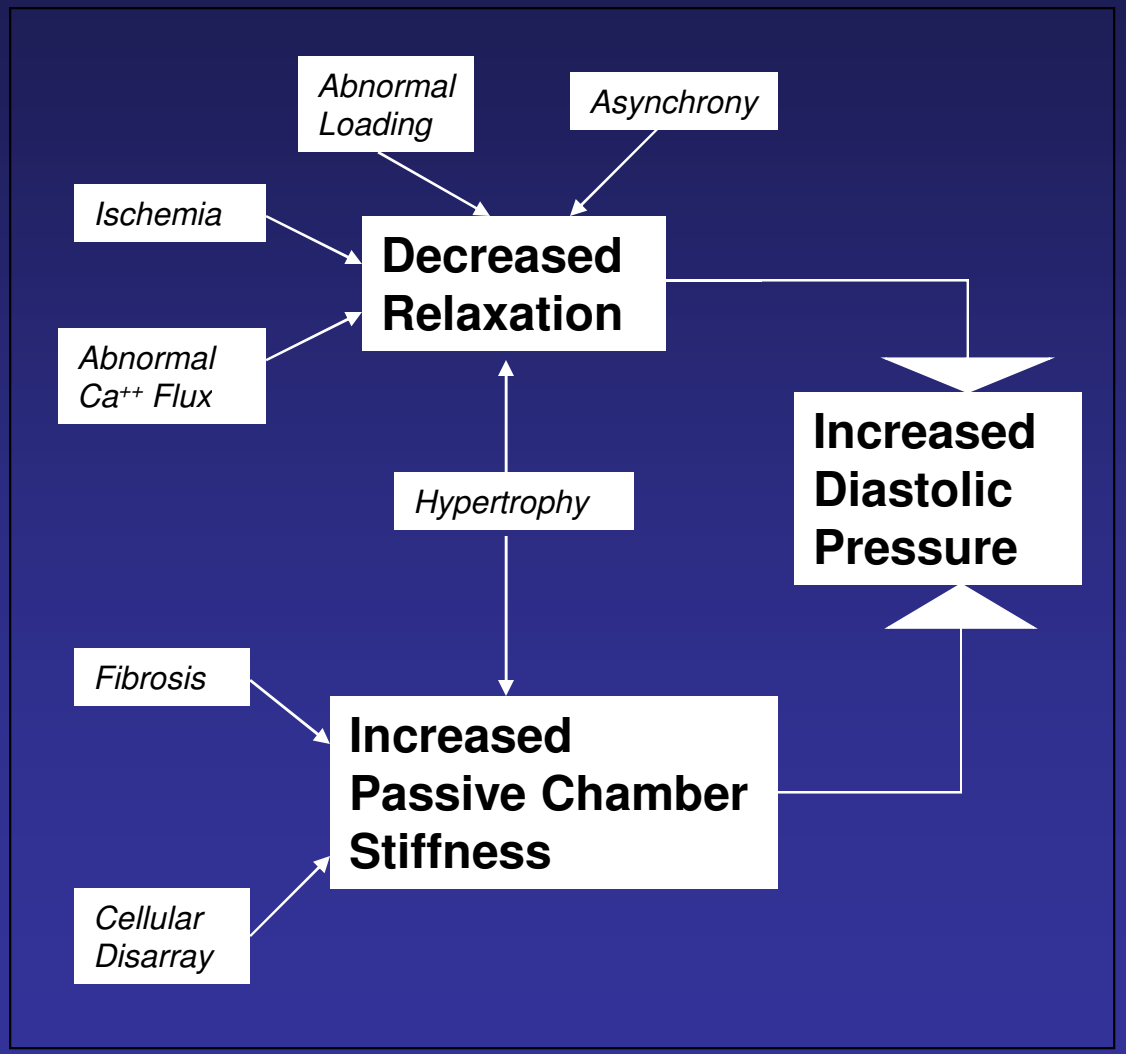
Pressure-Volume Loop in Diastolic Dysfunction



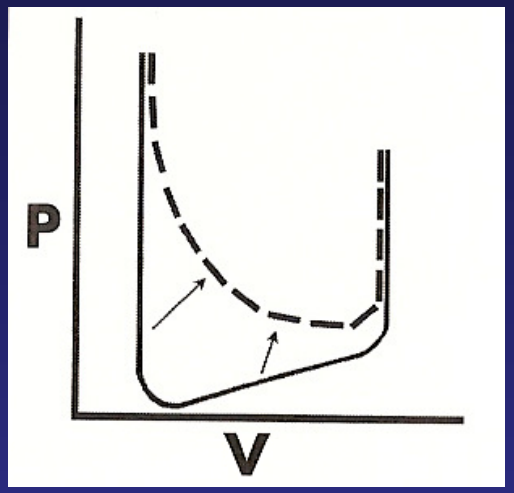
In LV diastolic dysfunction, the diastolic pressure-volume curve shifts from 1 to 2. This leads to an increase in diastolic pressure, a decrease in end-diastolic volume, and a decrease in ventricular stroke volume.

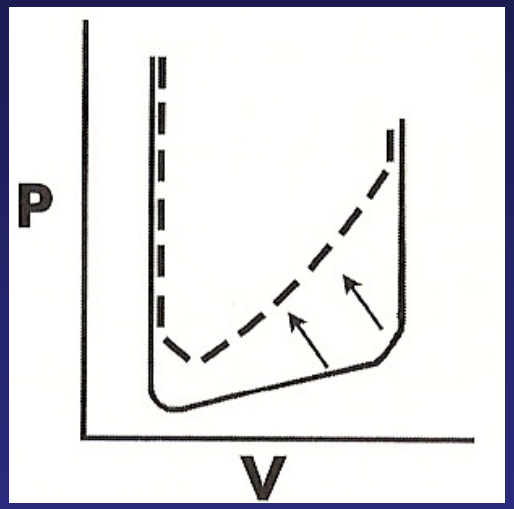
Pathophysiology of LV Diastolic Dysfunction

Decreased left ventricular relaxation, increased passive chamber stiffness, or both will lead to an upward shift in the left ventricular diastolic pressure-volume curve and LV diastolic dysfunction



Pathophysiology of LV Diastolic Dysfunction





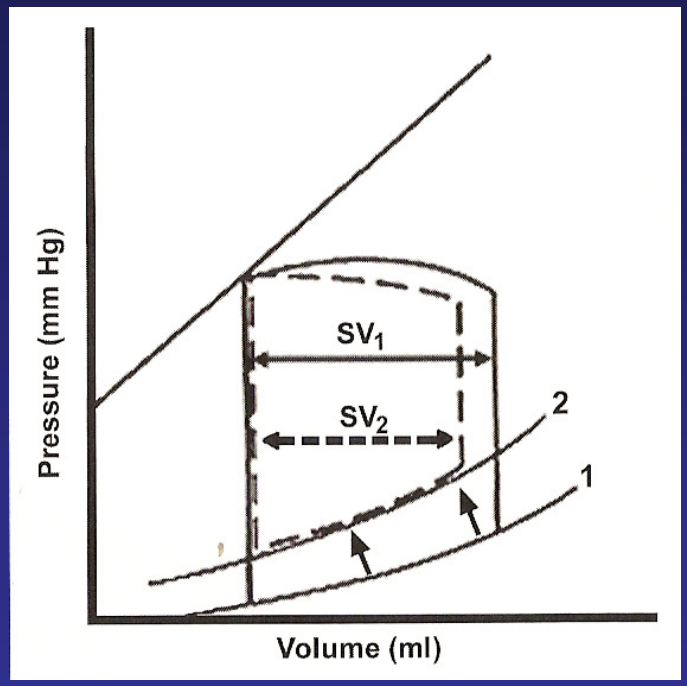
ABORMAL RELAXATION

Ventricular relaxation affects isovolemic relaxation, rapid ventricular filling, and mid-diastolic filling. Abnormal relaxation will result in an increase in LV pressure especially in the first two-thirds of diastole

INCREASED CHAMBER STIFFNESS

Increased chamber stiffness implies a change in the passive stretch properties of the LV muscle. It will predominantly affect the last two thirds of diastole

Pressure-Volume Loop in Diastolic Dysfunction



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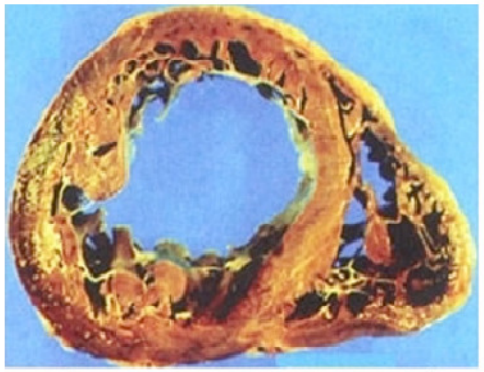
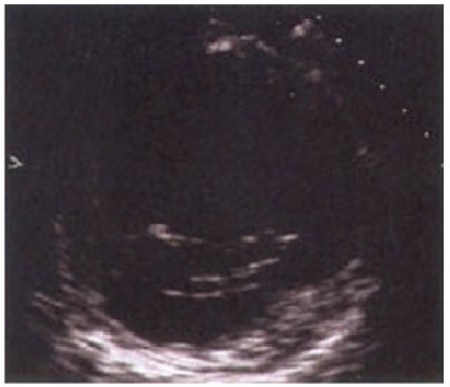
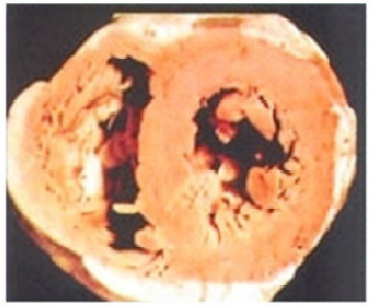
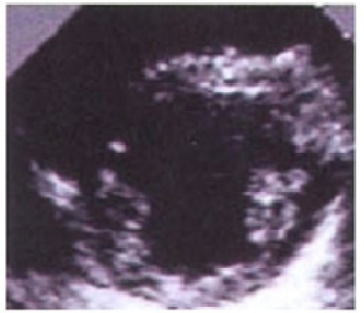


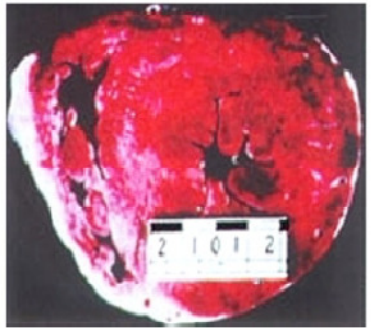
Systolic
Heart Failure



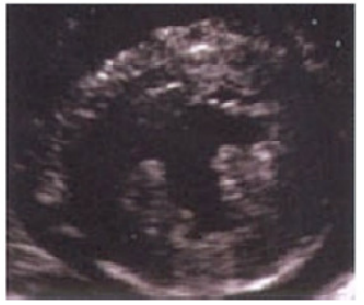


Normal

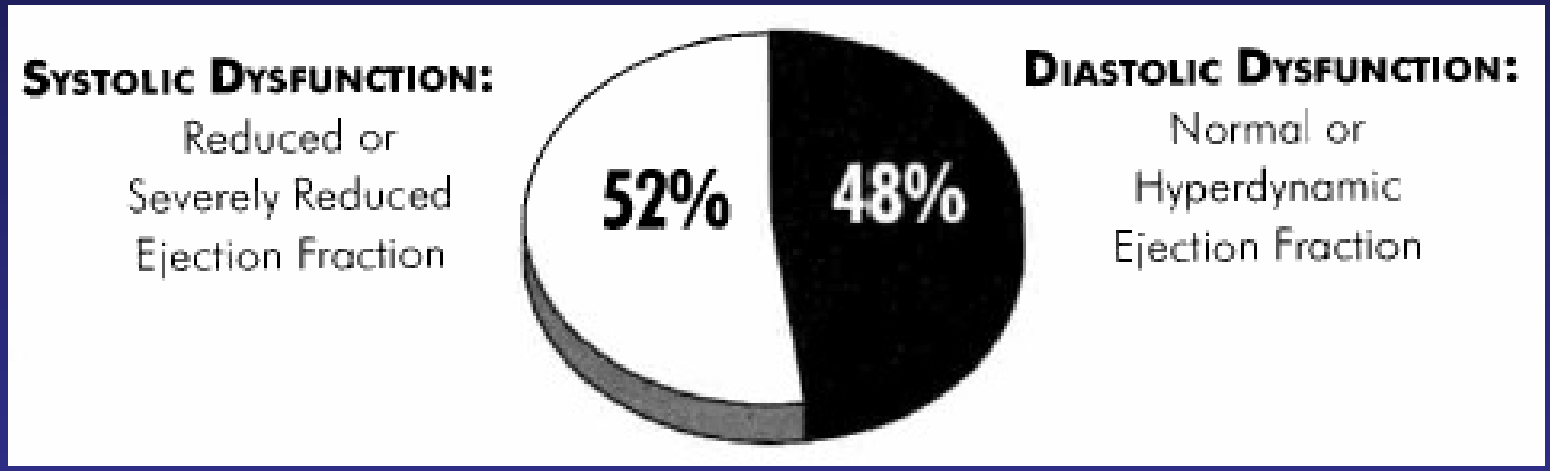




Diastolic
Heart Failure



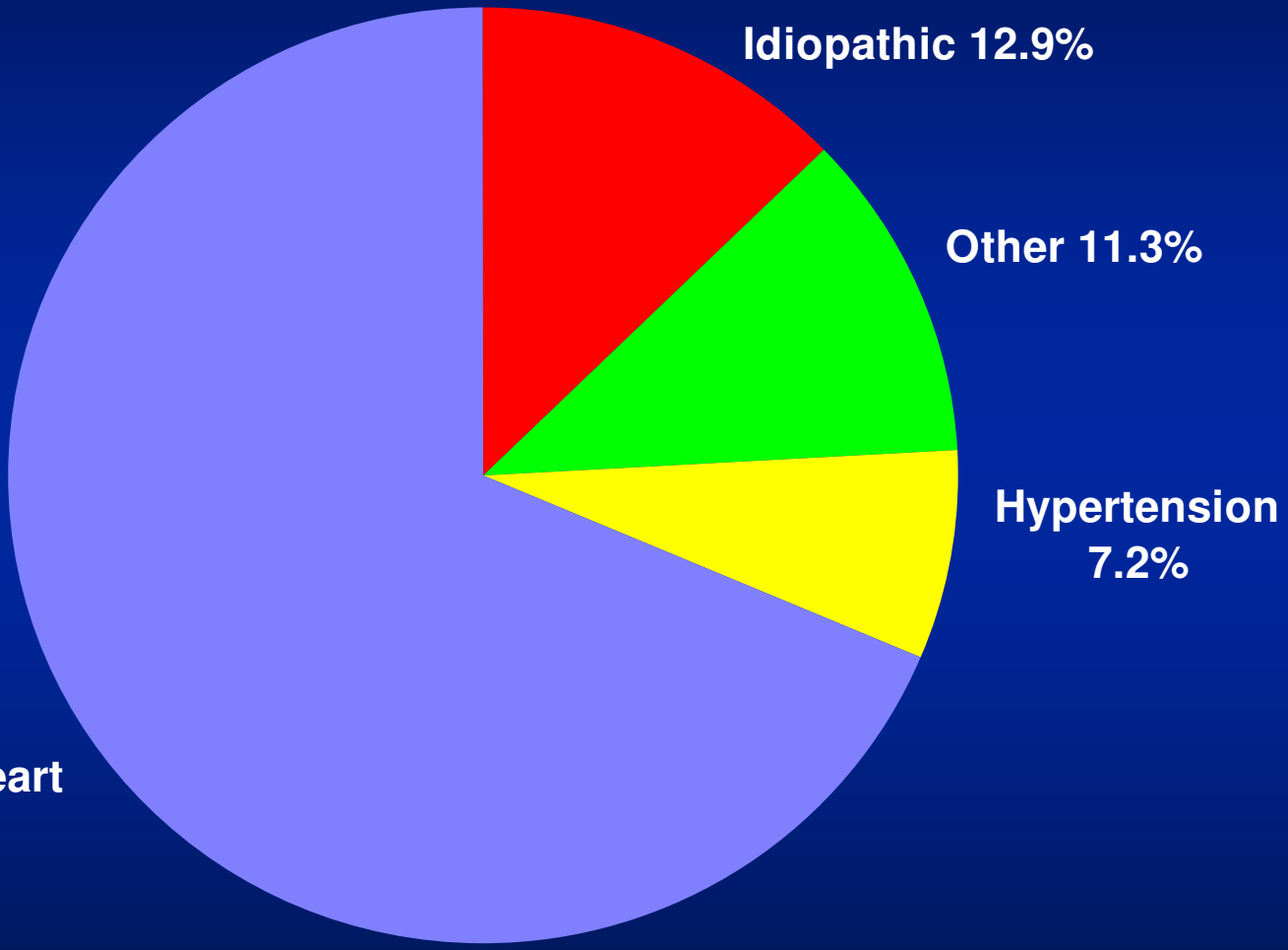
Systolic *vs.* Diastolic LV dysfunction



Of the patients presenting with pulmonary edema, ~ half had preserved LV function by echocardiogram and MUGA

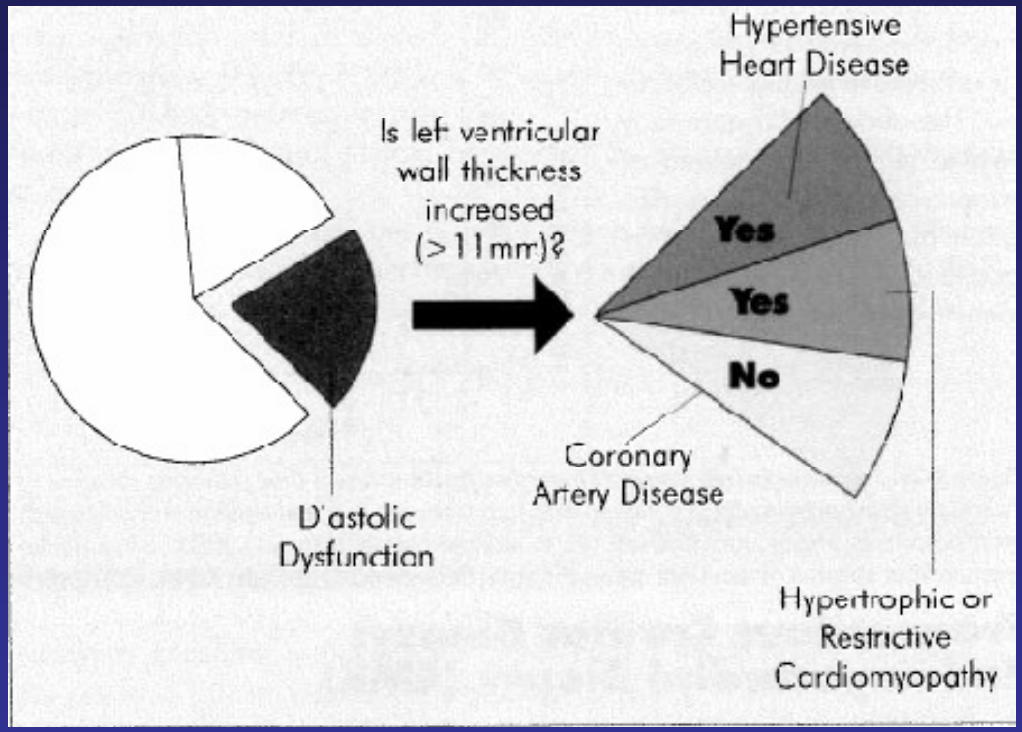
Bier et al Acute cardiogenic pulmonary edema: clinical and noninvasive evaluation. Angiology. 1988; 39:211

Etiology of Heart Failure in the SOLVD Registry



Bourassaet et. al. *J Am Coll Cardiol* 1993: 22:14A-19A

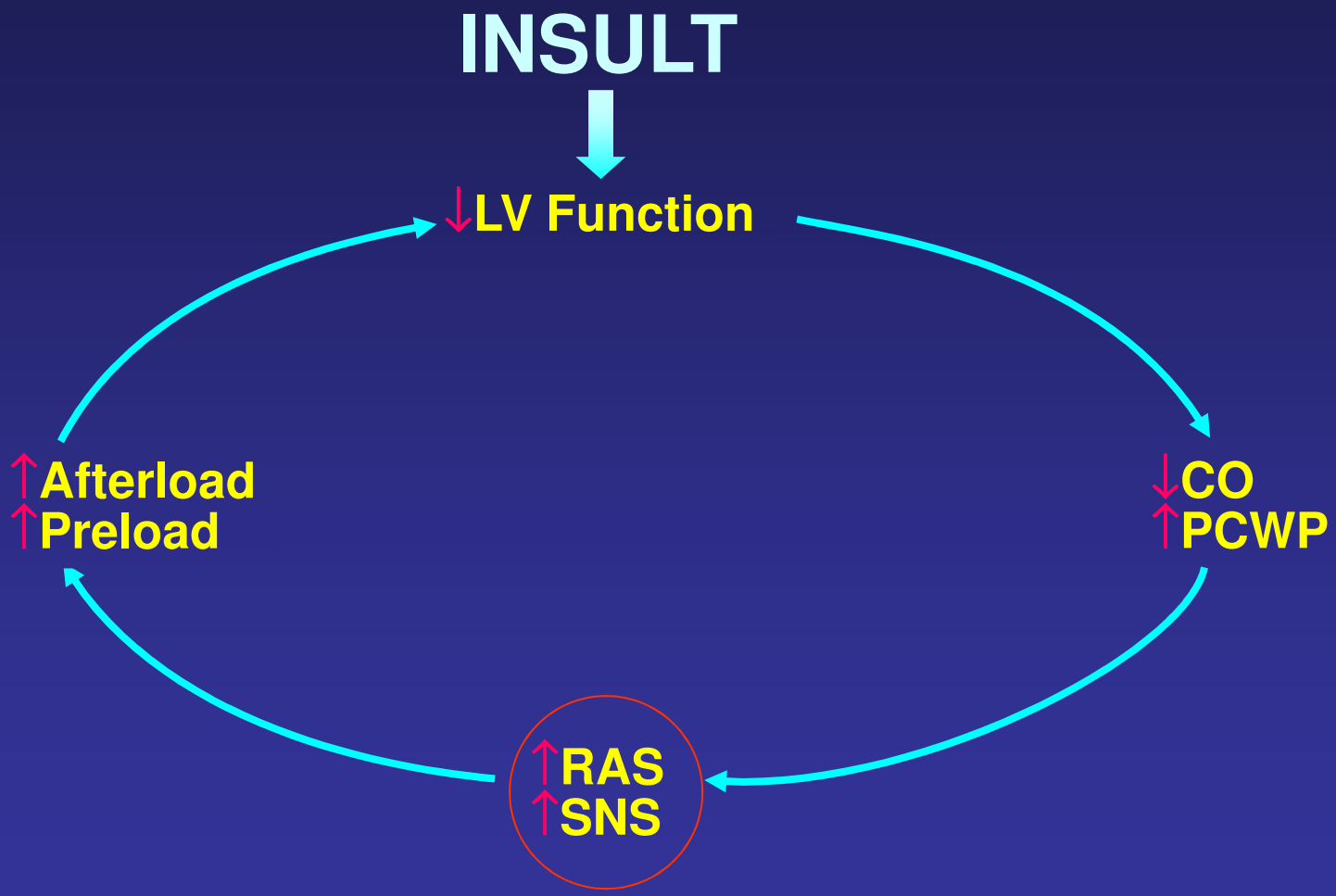
What are the Causes of Diastolic Dysfunction



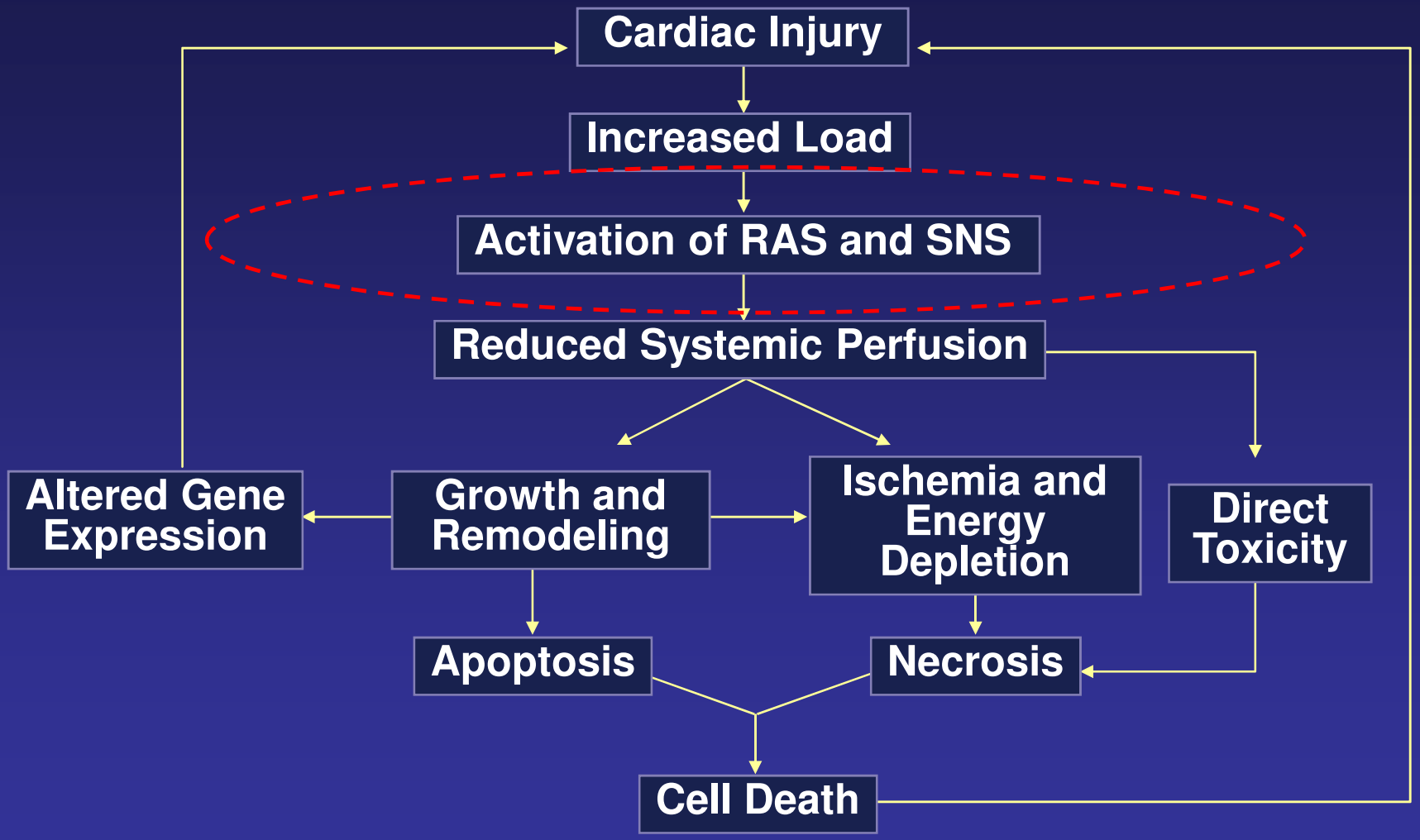
Neurohormonal Activation in LV Systolic Dysfunction

Beyond hemodynamic effect alone, neurohormonal stimulation of the cardiovascular system in HF leads to progressive circulatory dysfunction and subsequent increased morbidity and mortality

Neurohormonal Activation in LV Systolic Dysfunction



Pathophysiology of LV Systolic Dysfunction



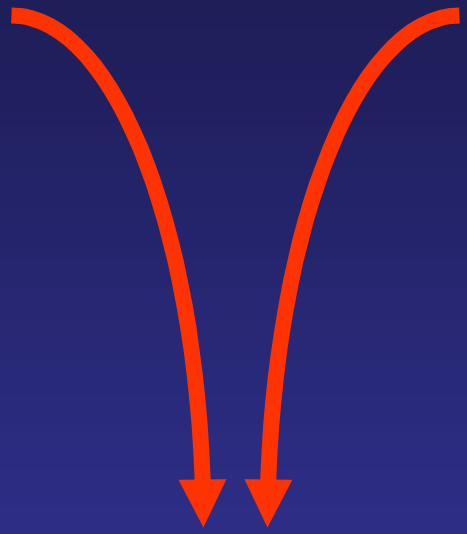
Eichhorn EJ, Bristow MR. *Circulation* 1996;94:2285-2296.

Pathophysiology of LV Systolic Dysfunction

Two Main Protagonists

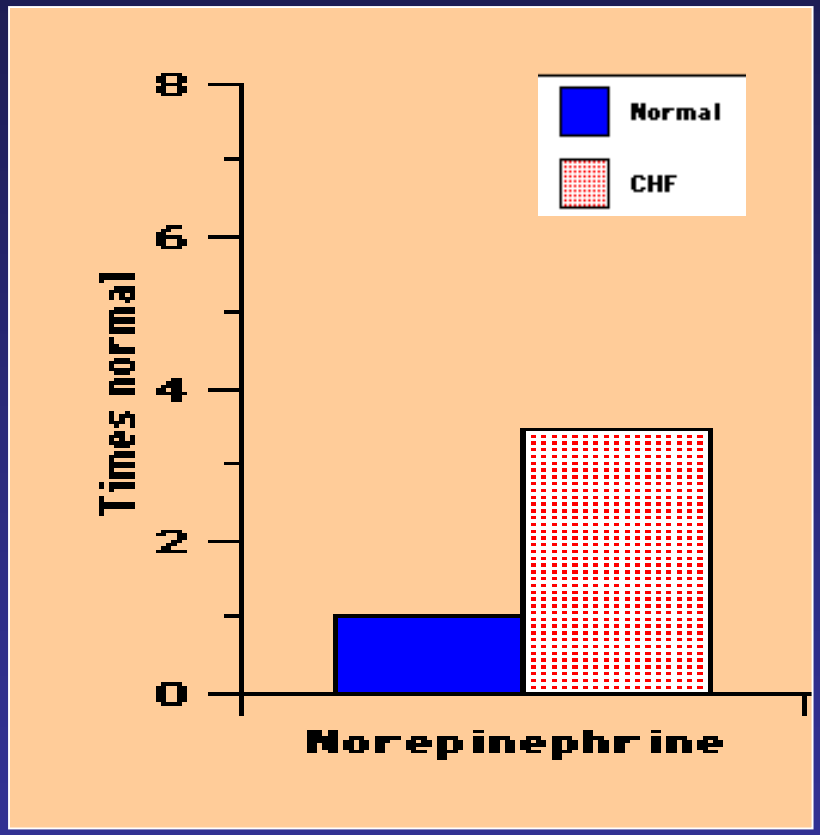
Renin-Angiotensin System (RAS)
Angiotensin II (A II)

Sympathetic Nervous System (SNS)
Norepinephrine (NE)



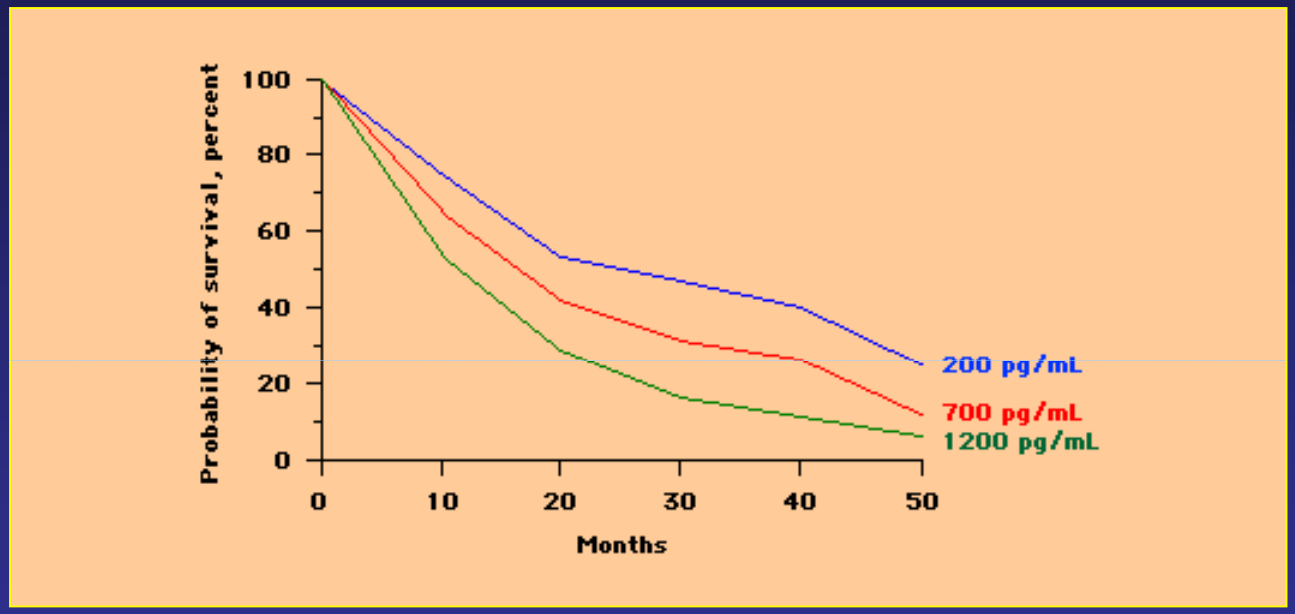
Hypertrophy, apoptosis, ischemia, arrhythmias, vasoconstriction, remodeling, fibrosis

Sympathetic nervous system and Heart Failure



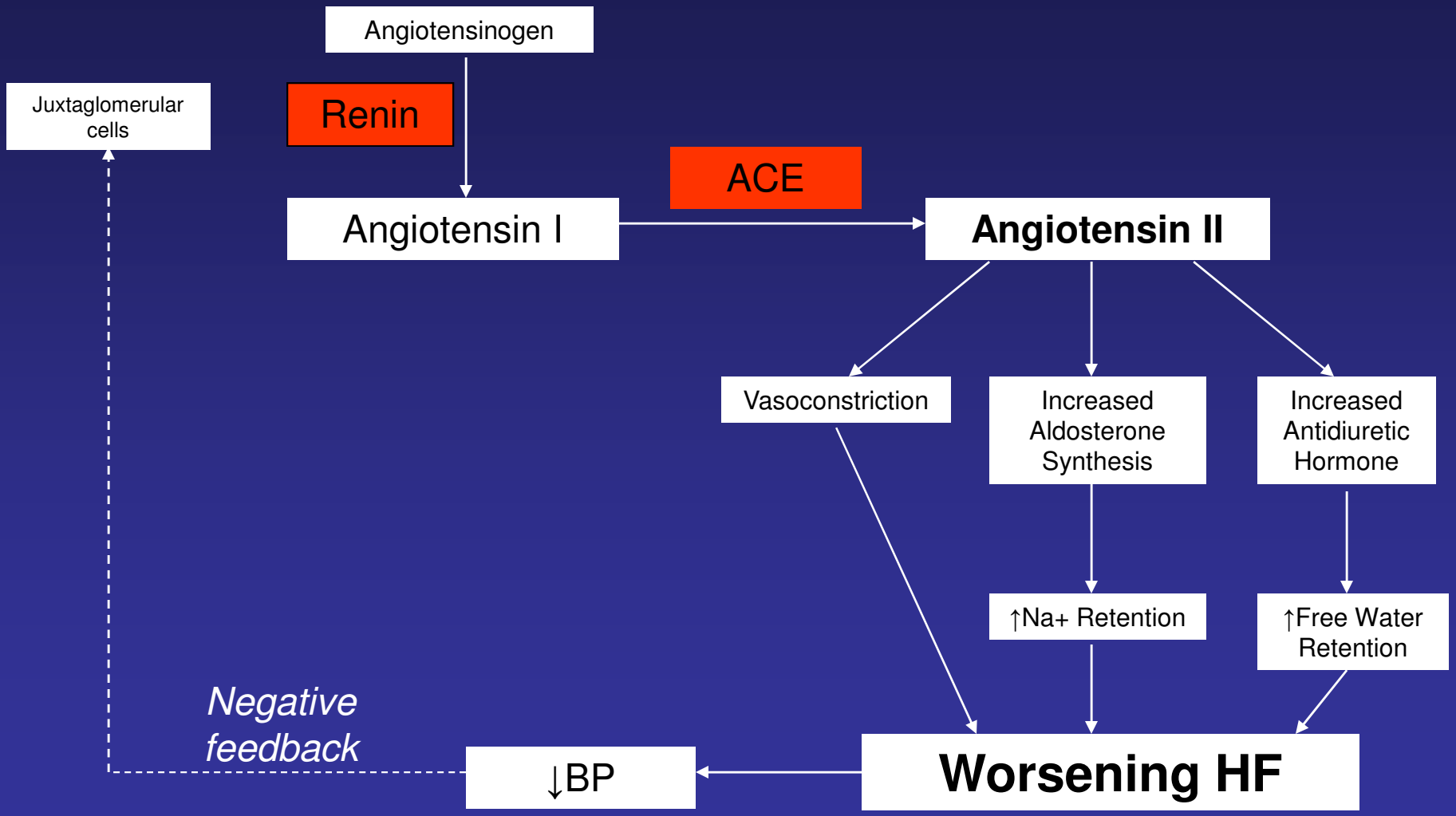
Plasma levels of Norepinephrine in patients with stable CHF treated with digitalis, but no diuretics or vasodilators versus normal controls Francis et *al*, Ann Intern Med; 1984;101:370

Sympathetic nervous system and Heart Failure



Plasma Norepinephrine and survival in CHF Cohn JN et al NEJM 1984; 311:819

Basic pathway of the Renin-Angiotensin-Aldosterone (RAA) System in HF



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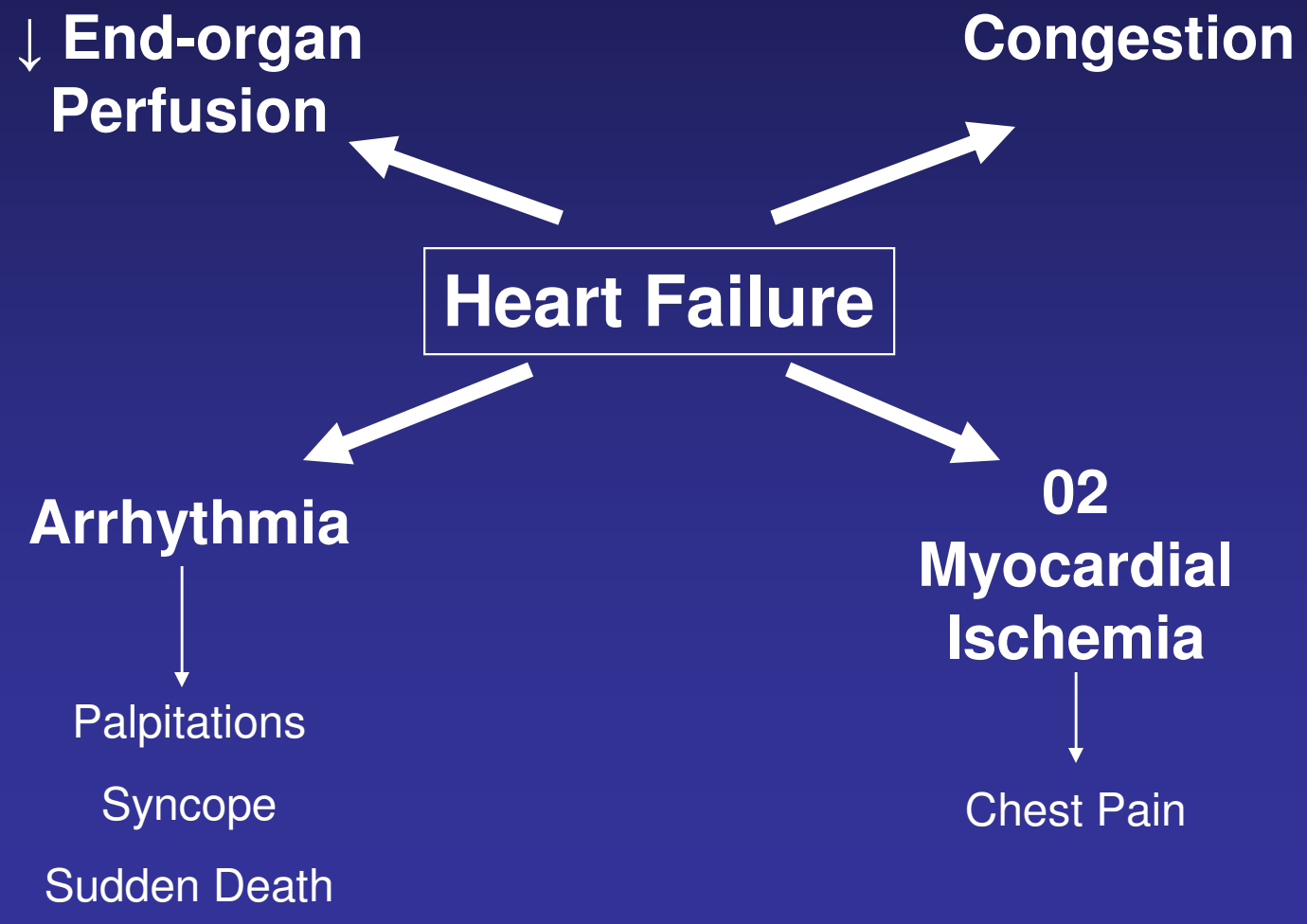
Cardiac Presentations in patients with Heart Failure



Clinical Criteria for Heart Failure

Major Criteria	Minor Criteria
Acute pulmonary edema	Dyspnea on exertion
PND or orthopnea	Night cough
JVD	Tachycardia (>120 beats/min)
Rales	Pleural effusion
S3 gallop	Hepatomegaly
Abdominojugular reflux	Ankle edema
Cardiomegaly on CXR	Vital capacity decrease (1/3 from max)
Increased venous pressure (>16cm H20)	
Weight loss>4.5 kg 5 days into treatment can be classified as a major or minor criterion	

Heart failure, in the Framingham HF study, requires 2 major or 1 major and 2 minor criteria to be present concommitently

Predictive Value of Clinical Findings for Estimating Hemodynamics in Heart Failure

	Capillary Wedge Pressure		
	>22mmHg	≤18mmHg	
Orthopnea +	39	0	39
Orthopnea -	4	7	11
	43	7	50

Sensitivity = 39/43 = **91%**

Specificity = 7/7 = **100%**

PPV = 39/39 = **100%**

NPV = 7/11 = **64%**

Stevenson LW et *al. JAMA* 1989; 261:884-888

Proportional Pulse Pressure and Heart Failure

Proportional Pulse Pressure = (BP systolic – BP diastolic)/BP systolic
Normal ratio is around 0.42.
Value less than 0.25 indicates a low cardiac index and is associated with a poor prognosis in a patient with heart failure.

	Cardiac Index (L/min/m^2) ≤2.2	>2.2	
PPP ≤ 25%	29	3	32
PPP > 25%	3	15	18
	32	18	50

Sensitivity = 29/32 = **91%**

Specificity = 15/18 = **83%**

PPV = 29/32 = **91%**

NPV = 15/18 = **83%**

Stevenson LW et *al.*
JAMA 1989; 261:884-888

Use of Jugular Venous Distension (JVD) to Assess Fluid Status

	Capillary Wedge Pressure		
	>18mmHg	≤18mmHg	
+JVD	21	1	22
- JVD	16	14	30
	37	15	52

Sensitivity = 21/37 = **57%**

Specificity = 14/15 = **93%**

PPV = 21/22 = **91%**

NPV = 12/19 = **47%**

Butman et *al. JACC* 1993; 22:968-974

Use of Inducible Abdominojugular Reflux to Increase Sensitivity of Physical Exam in Heart Failure

	Capillary Wedge Pressure		
	>18mmHg	≤18mmHg	
+JVD or +AJR	30	3	33
- JVD and- AJR	7	12	19
	37	15	52

Sensitivity = 30/37 = **81%**

Specificity = 12/15 = **80%**

PPV = 30/33 = **91%**

NPV = 12/19 = **63%**

Butman et *al. JACC* 1993; 22:968-974

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Assessment of Heart Failure

Three-Step Assessment of Heart Failure

1. Does presentation fit the diagnosis of HF
2. Is systolic or diastolic LV function abnormal
3. Are there treatable causes of HF present

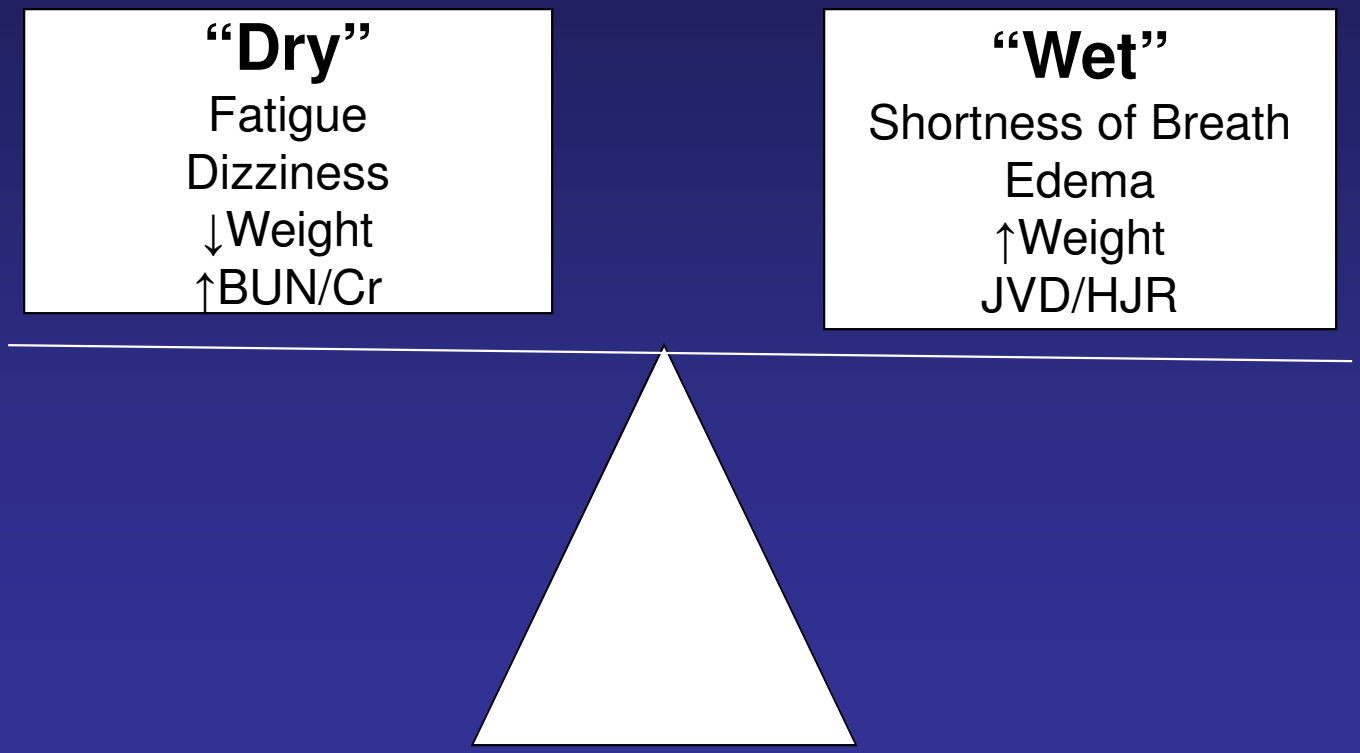
Step1 Basic data

- History and Physical Exam
- Lab: Chem 20, CBC, UA
- ECG
- CXR
- Echocardiogram

Step1 Basic data

- History is the key to the diagnosis of the presence of heart failure
- Cardinal Symptoms in History for Possible HF
  - Shortness of breath
  - Fatigue
  - Edema
  - Chest pain

Physical exam
Is the patient “wet” or “dry”



Heart Failure “Lethal Triad”

- BP systolic < 100
- HR > 100
- $[Na^+]$ < 130

Step1 Laboratory

Chemistry

- Na^+: ↓ *c/w high ADH and angiotensin II levels*
- K^+: *Often ↑ with ARF due to inadequate renal perfusion*
- BUN/Creat: ↑ *c/w with inadequate renal perfusion*
- Bilirubin, SGOT/SGPT, Alk Phosphatase: *may indicate passive congestion*
- Troponin: *to r/o ischemia*
- BNP

Laboratory

CBC

- Anemia: may contribute to heart failure decompensation or high output heart failure is sustained

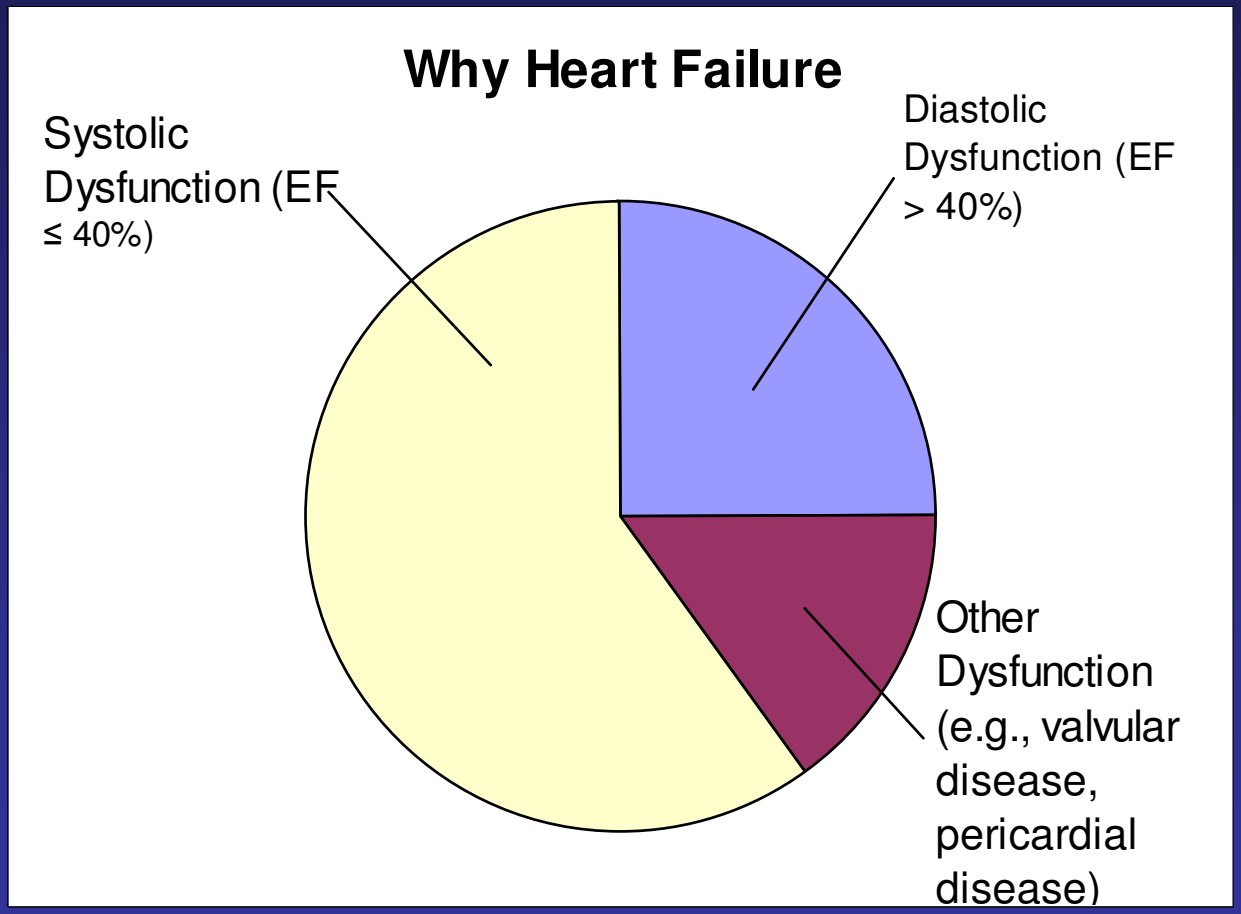
Urinalysis

- Proteinuria: may explain edema due to inadequate oncotic pressures

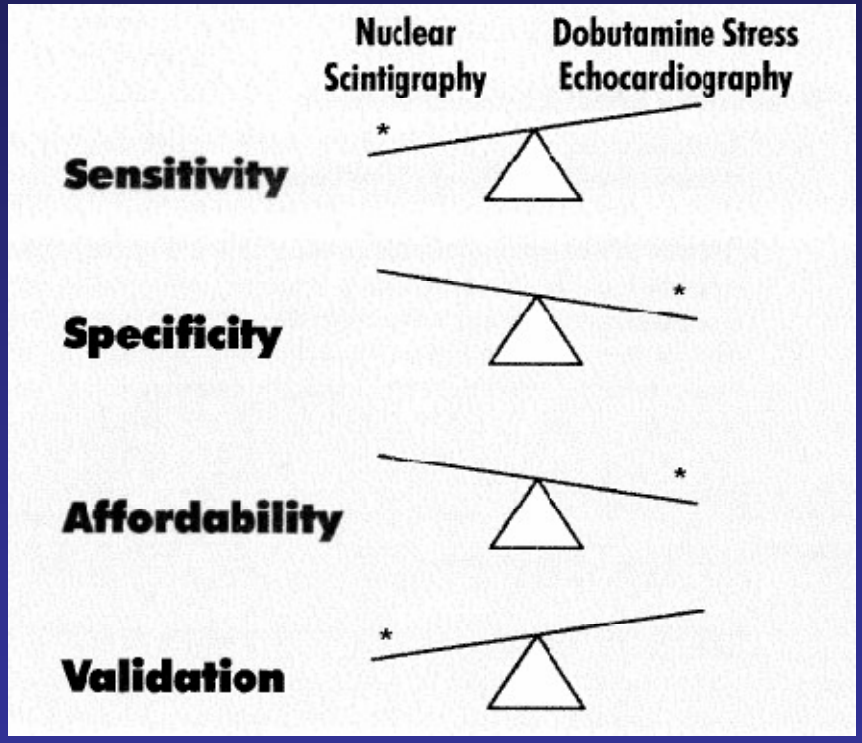
EKG

Findings	Suspected Diagnosis
Acute ST-T changes	Myocardial ischemia
Atrial fibrillation, other tachyarrhythmia	Thyroid disease
Q waves	HF due to reduced LV performance
Low voltage	Pericardial effusion, amyloidosis
LVH	Diastolic dysfunction

Step 2 Echocardiogram



Step 3 Nuclear vs DSE for Detecting Myocardial Ischemia



Brown KA et *al . Am J Cardiol* 1998; 81:1050-1053

Causes of Non-Ischemic Dilated Cardiomyopathy

- Idiopathic
  - Familial
  - Peripartum
- Genetic
  - Dystrophin
- Infectuous
  - Echovirus, Coxsackie, Chagas, Q fever, Typhus
- Granulomatosis disease
  - Sarcoidosis, Wegener's
- Metabolic/endocrine
  - Pheochromocytoma, Hypothyroidism, Hemochromatosis
- Collagen vascular disease
  - LED, Scleroderma
- Neuromuscular disease
  - Duchenne's, Becker's
- Toxins
  - ETOH, Amphetamines, Cocaine, Adriamycine, Cyclophosphamide, 5-FU

Precipitating Factors of Decompensation

- Lack of compliance
  - Diet
  - Drugs
- Uncontrolled hypertension
- Cardiac arrhythmias
- Anemia
- Inadequate/inappropriate therapy (meds, fluid)
- Infection
- Myocardial ischemia
- Endocrine disorders (thyrotoxicosis)

HF and anemia

- Anemia is common in CHF patients
- Treating anemia improves heart function and reduces hospitalizations.
- Silverberg et al reviewed the records of 142 heart failure patients and found that 56% were anemic (hemoglobin less than 12g/dL).
- The worse the CHF, the more likely patient was anemic: from 9% anemic in class I patients to 79% in class 4.

HF and anemia

- 26 were still anemic and had severe CHF even after 6 months of maximum HF treatment.
- Erythropoietin and IV iron raised hemoglobin *and* EF, even though their heart failure meds were not changed.
- This treatment also reduced hospitalizations by 92%, improved heart class, reduced Lasix doses, and slowed progression of kidney failure.

DrugsThat Can Exacerbate Heart Failure

- Class I antiarrhythmics

  (e.g. procainamide, quinidine, disopyramide, flecainide)
- Calcium channel blocker (except Amlodipine)
- Beta-blockers
- TZD
- Non-steroidal anti-inflammatory drugs

  (e.g., indomethacin, naproxen)
- Alcohol or illicit drugs

  (e.g., cocaine, amphetamines, etc.)

Heart failure

- Heart Failure an Important Health Problem
- Pathophysiology
  - Diastolic LV Dysfunction
  - Systolic LV Dysfunction
- Clinical Presentation in Patients with HF
- Assessment of Heart Failure
- Hemodynamics

Swan-Ganz Catheter

- Clinical estimation of patient's volume status can be difficult….
- Patients with pulmonary edema generally do not require SG catheter.
- SG, may be useful in patients not responding appropriately to therapy or if it is unclear whether pulmonary edema is due to cardiac or non-cardiac causes.
- SG catheter is useful in cardiogenic shock.
- RHC with Swan-Ganz catheter is the GOLD STANDARD METHOD to measure intracardiac pressures, cardiac output, and the hemodynamic response to therapy.
- Unfortunately, it is an invasive procedure that can be associated with significant complications…..

Possible Indications for Pulmonary Artery Balloon Catheter (Swan-Ganz)

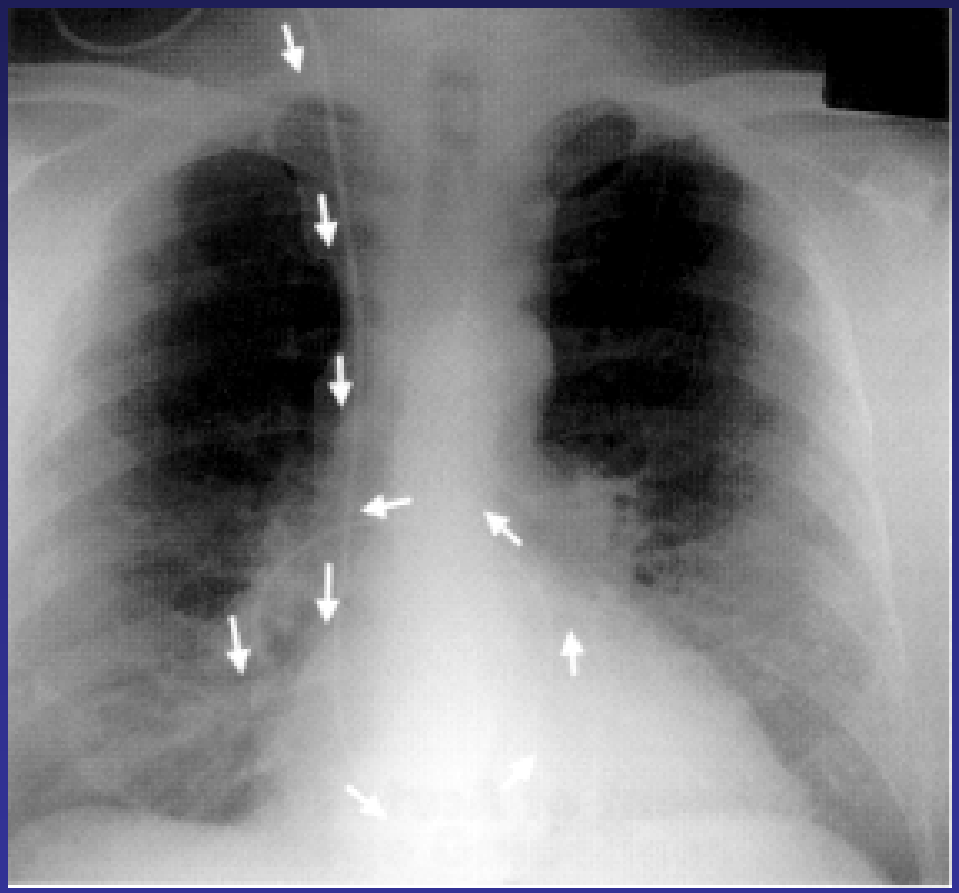
Suspected hypoperfusion

- Life-threatening organ dysfunction
- Refractory hypotension
- Possible need for LVAD

Hemodynamic Profile

- Severe symptoms out of proportion to clinical examination
- Intolerance to diuresis

Swan-Ganz Catheter



Swan-Ganz Catheter

$$SVR = \frac{(\text{mean Arterial Pressure} - CVP) \times 80}{\text{cardiac output}}$$ Dynes/sec/cm-5

$$PVR = \frac{\text{mean PAP} - \text{Wedge}}{\text{cardiac output}}$$ Wood Unit

Trans-pulmonary gradient = mean PAP – Wedge

If Pulmonary hypertension secondary to ↑ filling pressures (Trans-pulm Grad < 15mmHg)

Swan-Ganz Catheter Examples

PA	Mean PA	Wedge	TP Gradient	Consistent with
50/35	40	31	9	Elevated left sided filling pressures
92/37	55	12	43	Intrinsic pulmonary process

Swan-Ganz Catheter Hemodynamics Parameters

CVP (mmHg)	PAP (mmHg)	Wedge (mmHg)	BP (mmHg)	Cardiac Output	Cardiac index	SVR (Dynes/sec/cm^{-5})
3	20/10	9	120/80	5.3	2.6	1100
1	15/3	4	90/60	3.5	1.9	1600
15	50/35	32	78/60	3.2	1.6	1800
16	75/32	12	80/62	3.1	1.6	1500
0-1	25/0-2	6	85/56	7.8	4.3	800
18	30/18	18	88/60	3.1	1.5	1850

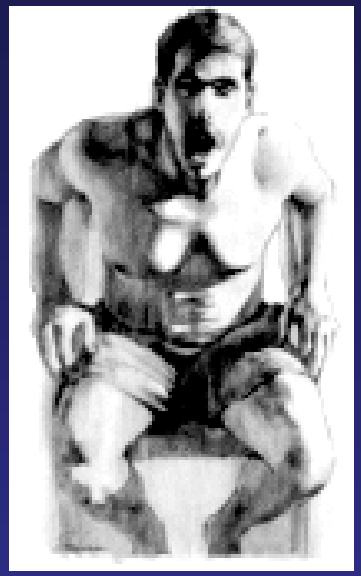
Swan-Ganz Catheter Hemodynamics Parameters

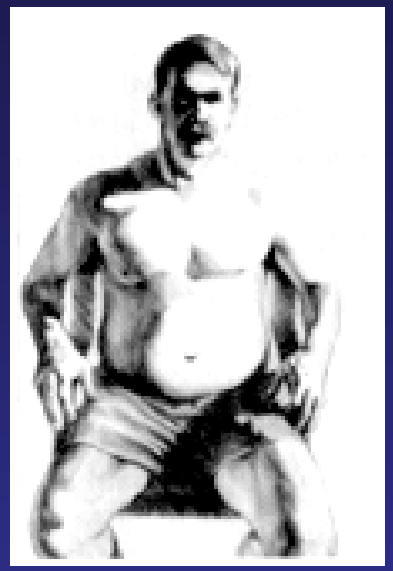
	CVP (mmHg)	PAP (mmHg)	Wedge (mmHg)	BP (mmHg)	Cardiac Output	SVR (Dynes/sec/cm^{-5})
Normal	3	20/10	9	120/80	5.3	1100
Hypo-volemia	1	15/3	4	90/60	3.5	1600
Cardiogenic shock	15	50/35	32	78/60	3.2	1800
Pulmonary Embolism	16	75/32	12	80/62	3.1	1500
Septic shock	0-1	25/0-2	6	85/56	7.8	800
Tamponade	18	30/18	18	88/60	3.1	1850

Swan-Ganz Catheter Hemodynamics Parameters

	CVP (mmHg)	PAP (mmHg)	Wedge (mmHg)	BP (mmHg)	Cardiac index	SVR (Dynes/sec/cm^{-5})
Normal	0-5	25/6-12	6-12	120/80	≥2.5	900-1200
Hypo-volemia	0-1	15/2-6	<6	<90/60	<2	>1500
Cardiogenic shock	>8	50/30	30	<90/60	<2	>1500
Septic shock	0-1	25/0-2	<6	<90/60	>4	<800
Tamponade	12-18	30/18	18	<90/60	<2	>1500

Conclusion





"I can't breathe"

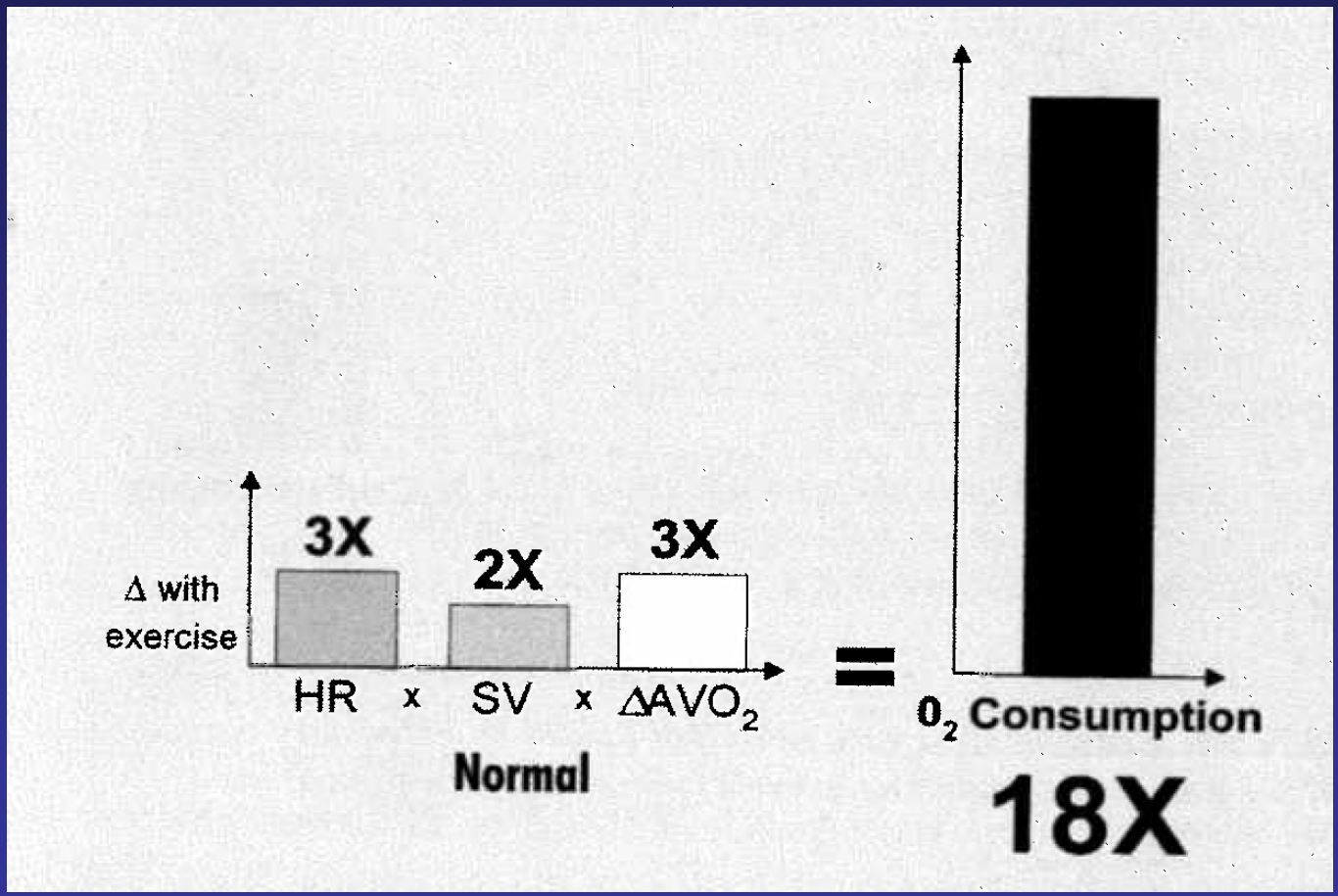
"I can walk 30 minutes without stopping"

Heart Failure Reduces Peak Oxygen Consumption with Exercise

02 consumption = HR x SV x Δ AV02

Δ AV02 = 02 content in the arterial system - 02 content remaining when blood flow converges back to the central venous circulation

Heart Failure Reduces Peak Oxygen Consumption with Exercise



Heart Failure Reduces Peak Oxygen Consumption with Exercise