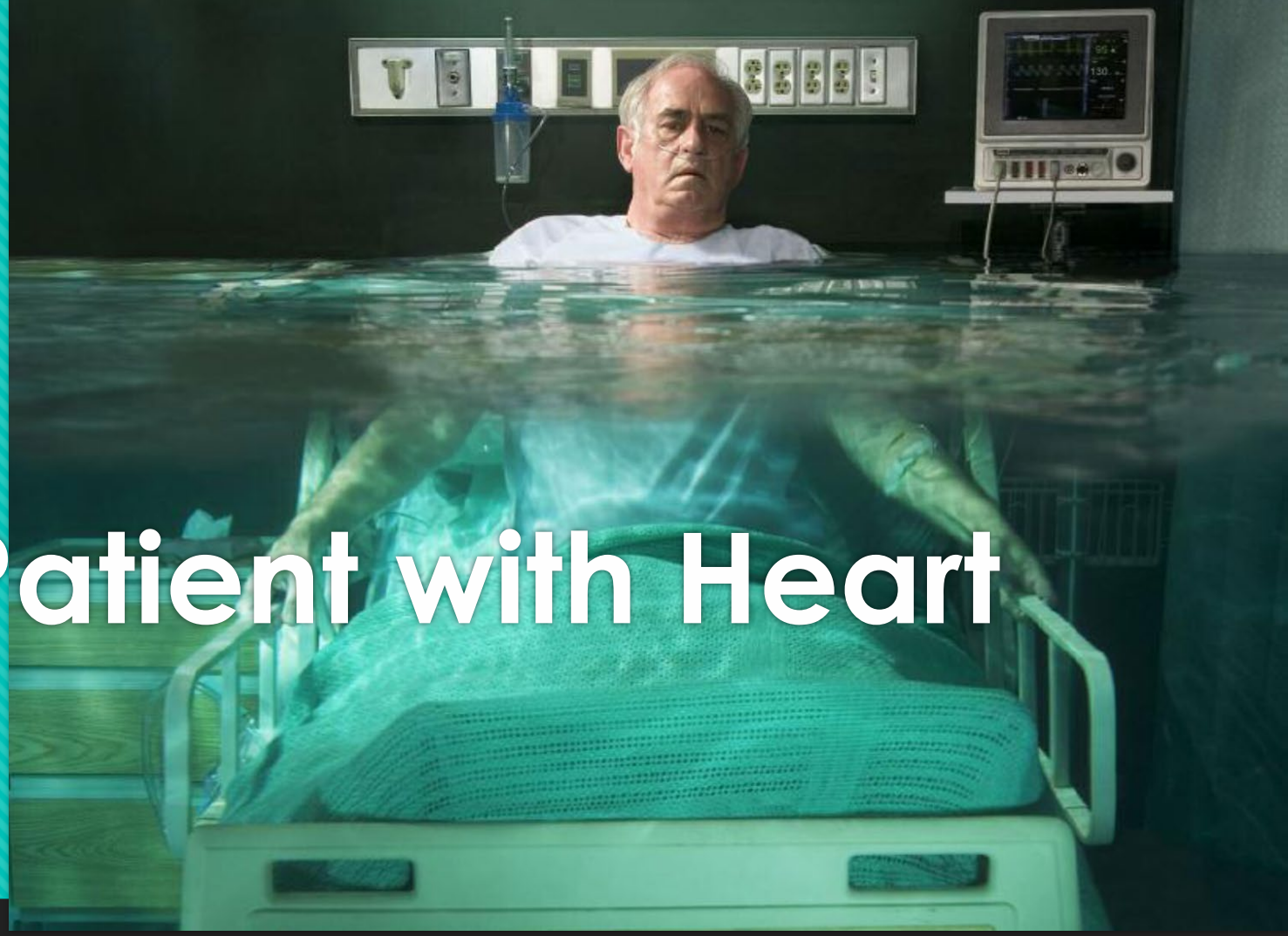
Care of the Patient with Heart Failure



Curtis Townsend RN MHA

Director, Emergency Services

Southwest General

At completion of this educational session, the participant will be able to:

Learning Objectives:

After attending this lecture, participants should be able to:

- Correlate selected cardiovascular structures with functional elements of the cardiovascular system
- Discuss the definitions, etiology, pathophysiology, and clinical manifestations of HF
- Compare and contrast the American College of Cardiology Foundation (ACCF), American Heart Association (AHA), and the New York Heart Association (NYHA) classes of HF
- Examine specific SWG ICD-10 DRGs related to HF
- Integrate the nursing process and collaborative treatment/management of HF
- Evaluate current innovations in treatment strategies for patients with HF

Why I am here…

Nursing advocacy is a necessary part of providing good patient care

84% of new medications are ordered as a result of nurse advocacy behaviors.

*Davidson, Critical Care Medicine 2000; 28:A103

Heart Failure:
Natural History and Prevalence

Why we need to talk about HF

- CHF is the first-listed diagnosis in 875,000 hospitalizations, and the most common diagnosis in hospital patients age 65 years and older.
- In that age group, *one-fifth* of all hospitalizations have a primary or secondary diagnosis of heart failure.
- More than half of those who develop CHF die within 5 years of diagnosis.
- Heart failure contributes to approximately 287,000 deaths a year.
- 80 percent of the 3,241 hospitals CMS evaluated this year will face penalties for readmissions

https://www.emoryhealthcare.org/heart-vascular/wellness/heart-failure-statistics.htm

https://www.advisory.com/daily-briefing/2017/08/07/hospital-penalties

Five year survival

Cancer rates—CHF is worse than…

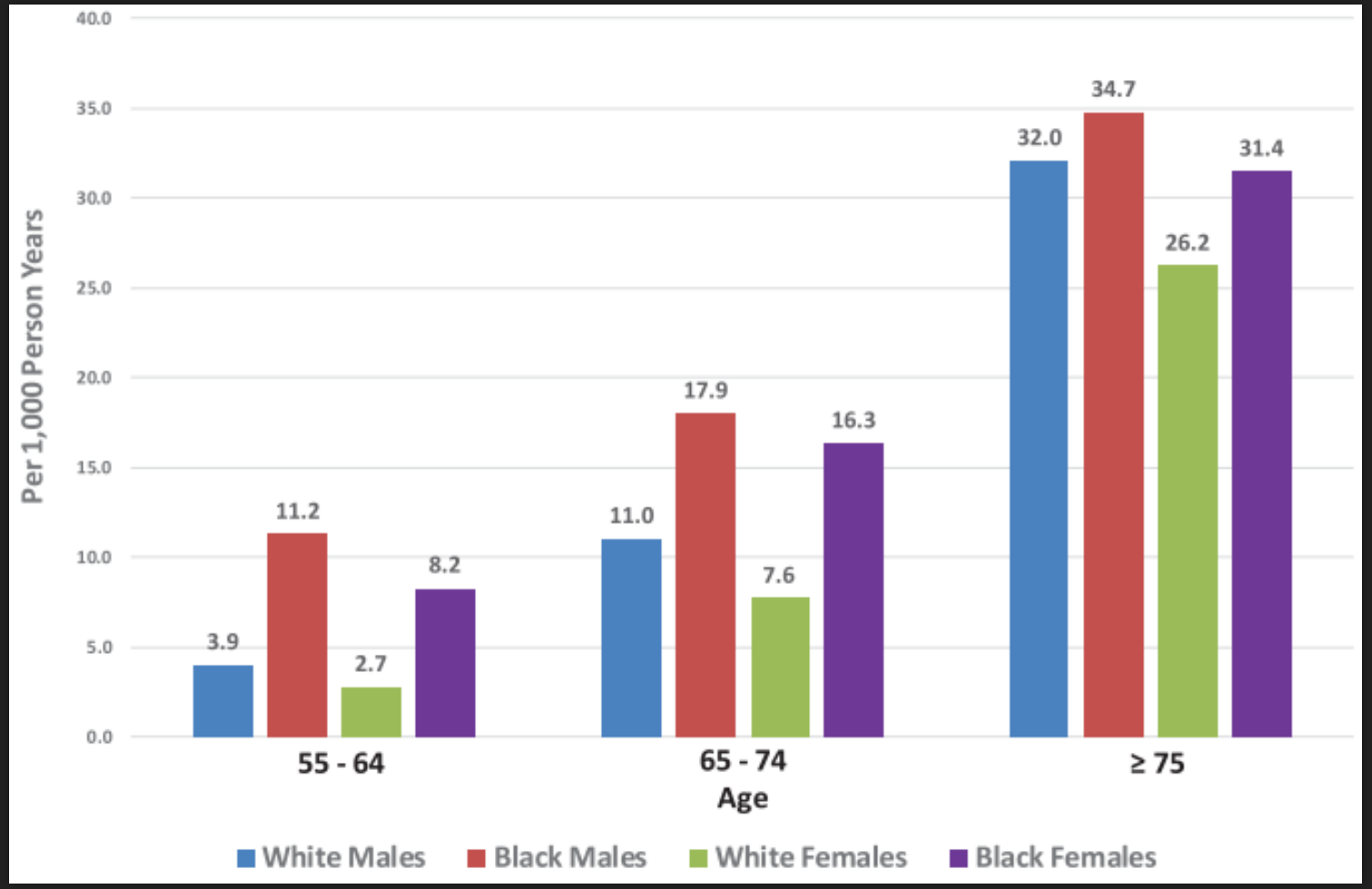
Colo-rectal	64%
Larynx	62%
Breast	90%
Cervical	66%
Prostate	98%
Ovarian	52%
Testicular	96%
Hodgkins & Non-Hodg.	85%/57%
Thyroid	98%
Melanoma	89%
Oral	65%

5-year survival rates

SEER Statistics, National Cancer Institute, 2015

https://seer.cancer.gov/csr/1975_2015/results_merged/topic_survival.pdf#search=5%20year%20survival%20rates%20for%20cancer

HF patients-who are they?



Causes of Heart Failure

- **Coronary heart disease** – relative risk 8.1; overall PAR 62 percent, 68 percent in men and 56 percent in women.
- **Cigarette smoking** – relative risk 1.6, PAR 17 percent.
- **Hypertension** – relative risk 1.4, PAR 10 percent.
- **Obesity** – relative risk 1.3, PAR 8 percent;
- **Diabetes** – relative risk 1.9, PAR 3 percent.
- **Valvular Heart Disease-** relative risk, 1.5, PAR 2 percent

PAR=population attributable risk

https://www.uptodate.com/contents/epidemiology-and-causes-of-heart-failure

Most Common Comorbid Conditions

- Cardiovascular / Cerebrovascular: CHF – Acute (or Acute on Chronic); Systolic or Diastolic Cor Pulmonale, Acute CVA / Stroke / Cerebral Infarct or Hemorrhage Endocarditis / Myocarditis, Acute MI, Acute Pulmonary
- Embolism Respiratory & Infectious Disease: Aspiration Bronchitis, Aspiration Pneumonia HIV Disease Peritonitis Pneumonia, Including Viral Respiratory Failure, Acute Respiratory Insufficiency, Acute Postoperative Sepsis, Severe Sepsis, Septic Schock

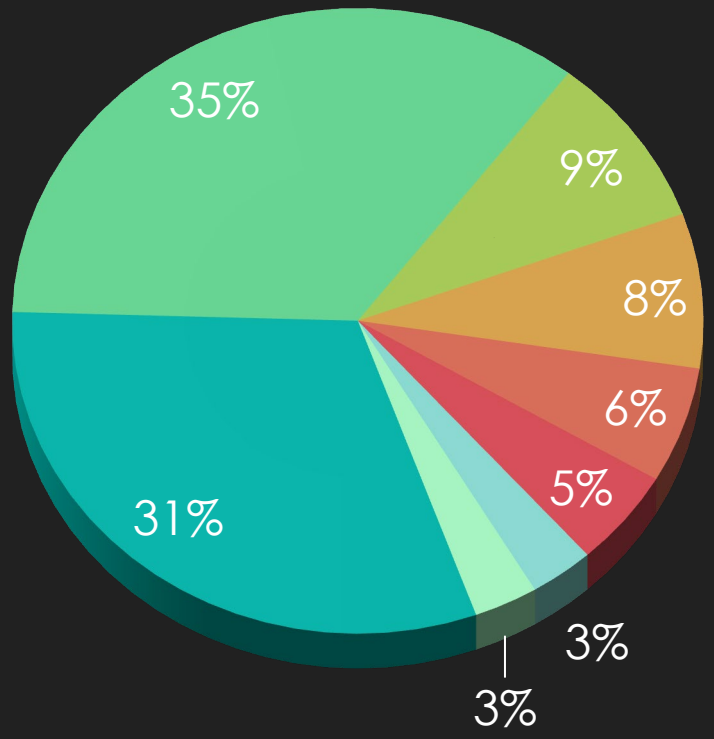
…and it's not getting better

- The prevalence of HF continues to rise over time

with the aging of the population. An estimated 6.5 million American adults ≥20 years of age had HF between 2011 and 2014 compared with an estimated 5.7 million between 2009 and 2012.

- Projections show that the prevalence of HF will increase 46% from 2012 to 2030, resulting in >8 million people ≥18 years of age with HF

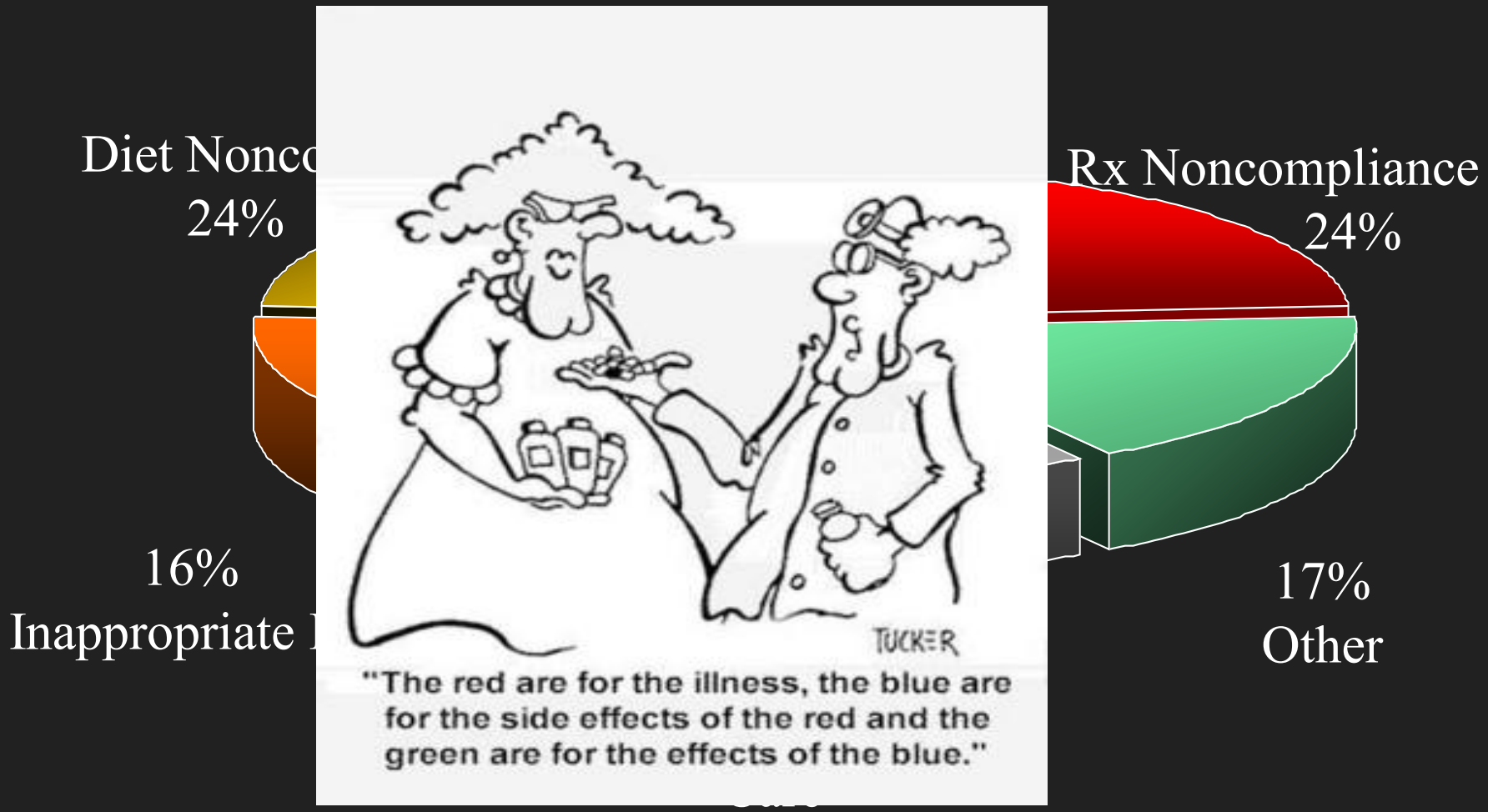
Distribution of Hospitalization Costs for HF



- ICU bed
- Non-ICU bed
- Pharmacy
- Laboratory
- Supplies
- Other Therapy
- Radiology
- Other

Cardiology Roundtable 1998

Causes of Hospital Readmission for Congestive Heart Failure



Vinson J Am Geriatr Soc 1990;38:1290-5

DRGs for HF

- DRG 291 HEART FAILURE & SHOCK W MCC
- DRG 292 HEART FAILURE & SHOCK W CC
- DRG 293 HEART FAILURE & SHOCK W/O CC/MCC

MCC- Major Complications / Comorbid Conditions

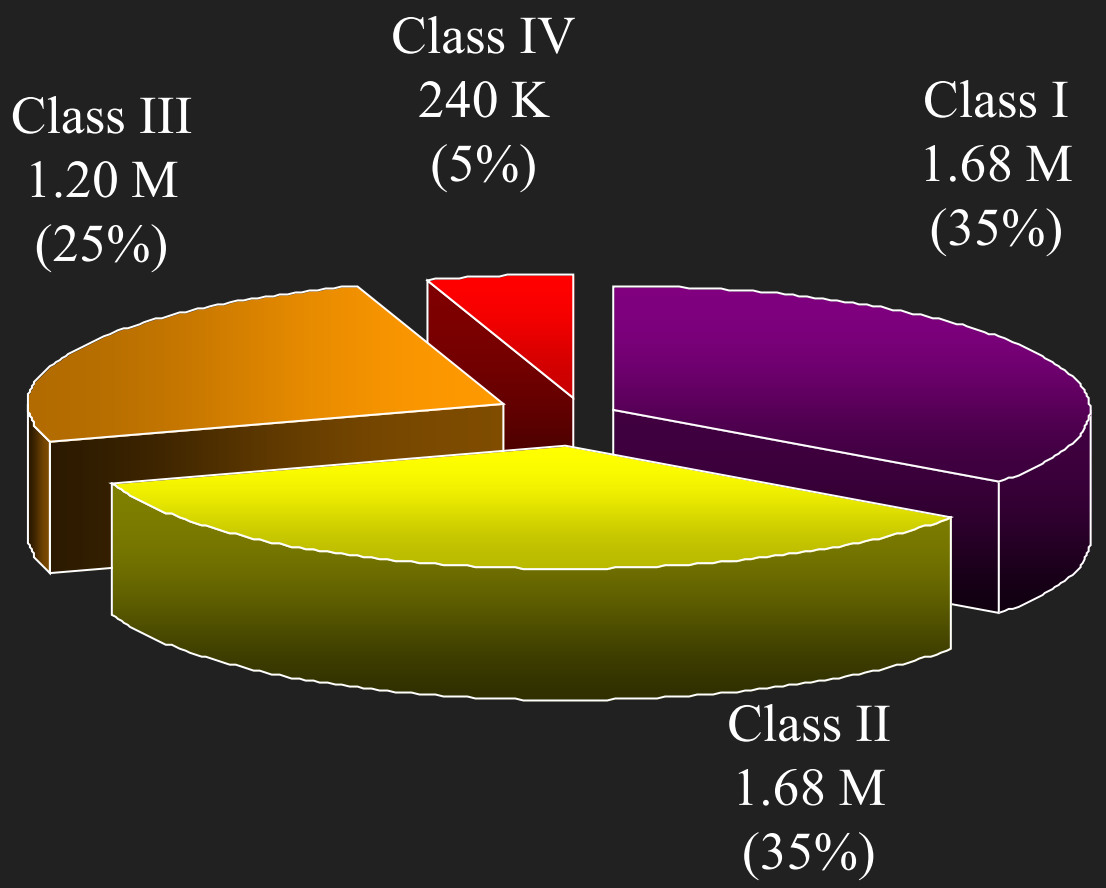
https://www.cms.gov/icd10manual/fullcode_cms/P0136.html

Comparing Stages of HF

Comparison of ACCF/AHA Stages of Heart Failure and NYHA Functional Classifications

ACCF/AHA Stages of HF		NYHA Functional Classification	
A	At high risk for HF but without structural heart disease or symptoms of HF	None	
B	Structural heart disease but without signs or symptoms of HF	I	No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF.
C	Structural heart disease with prior or current symptoms of HF	I	No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF.
		II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in symptoms of HF.
		III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes symptoms of HF.
		IV	Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest.
D	Refractory HF requiring specialized interventions	IV	Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest.

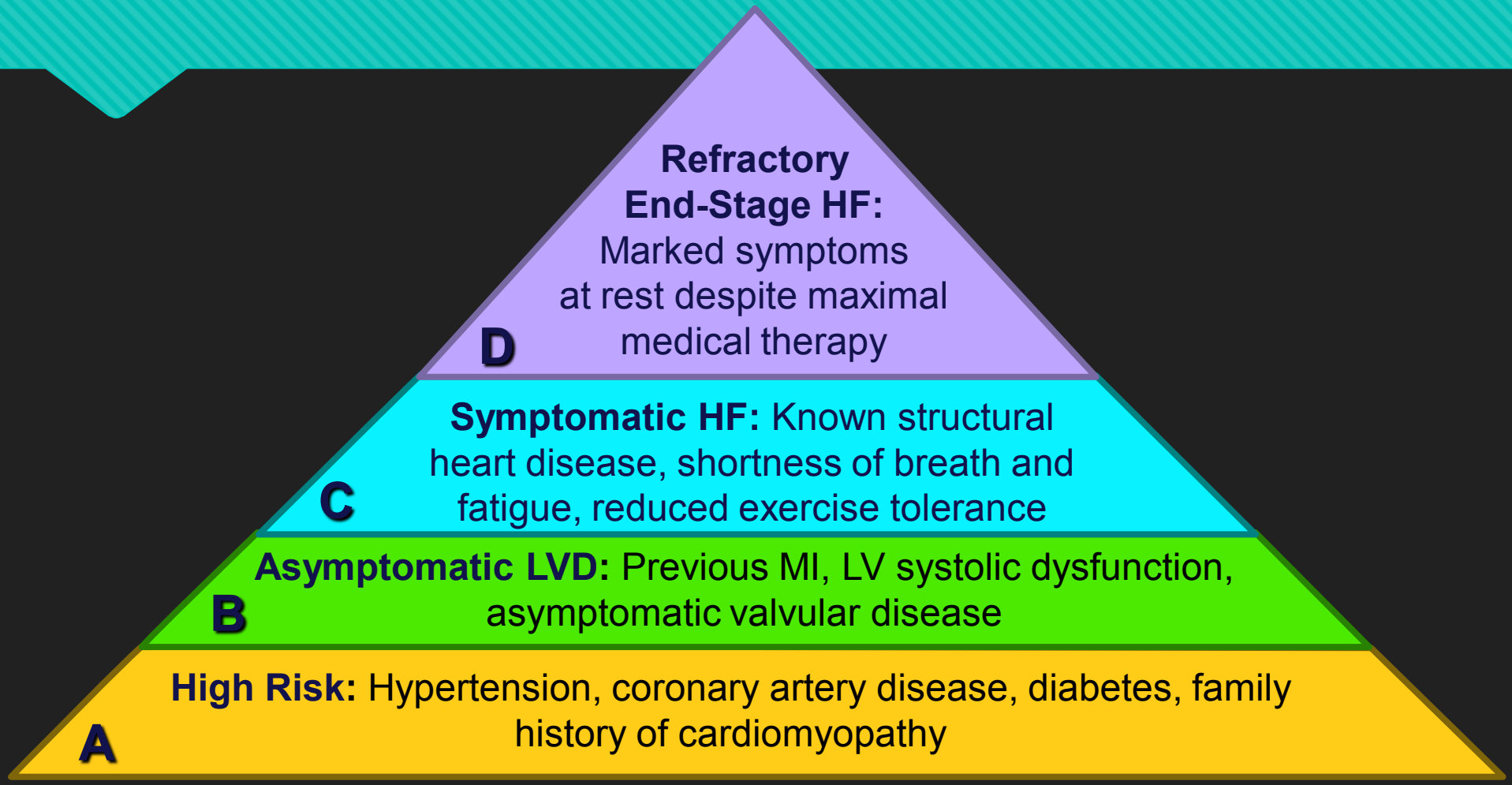
CHF Patient Population by NYHA Class



Class I
No limitations of physical activity
Class II
Slight limitations of physical activity
Class III
Marked limitations of physical activity
Class IV
Inability to carry out physical activities without discomfort and/or symptoms at rest

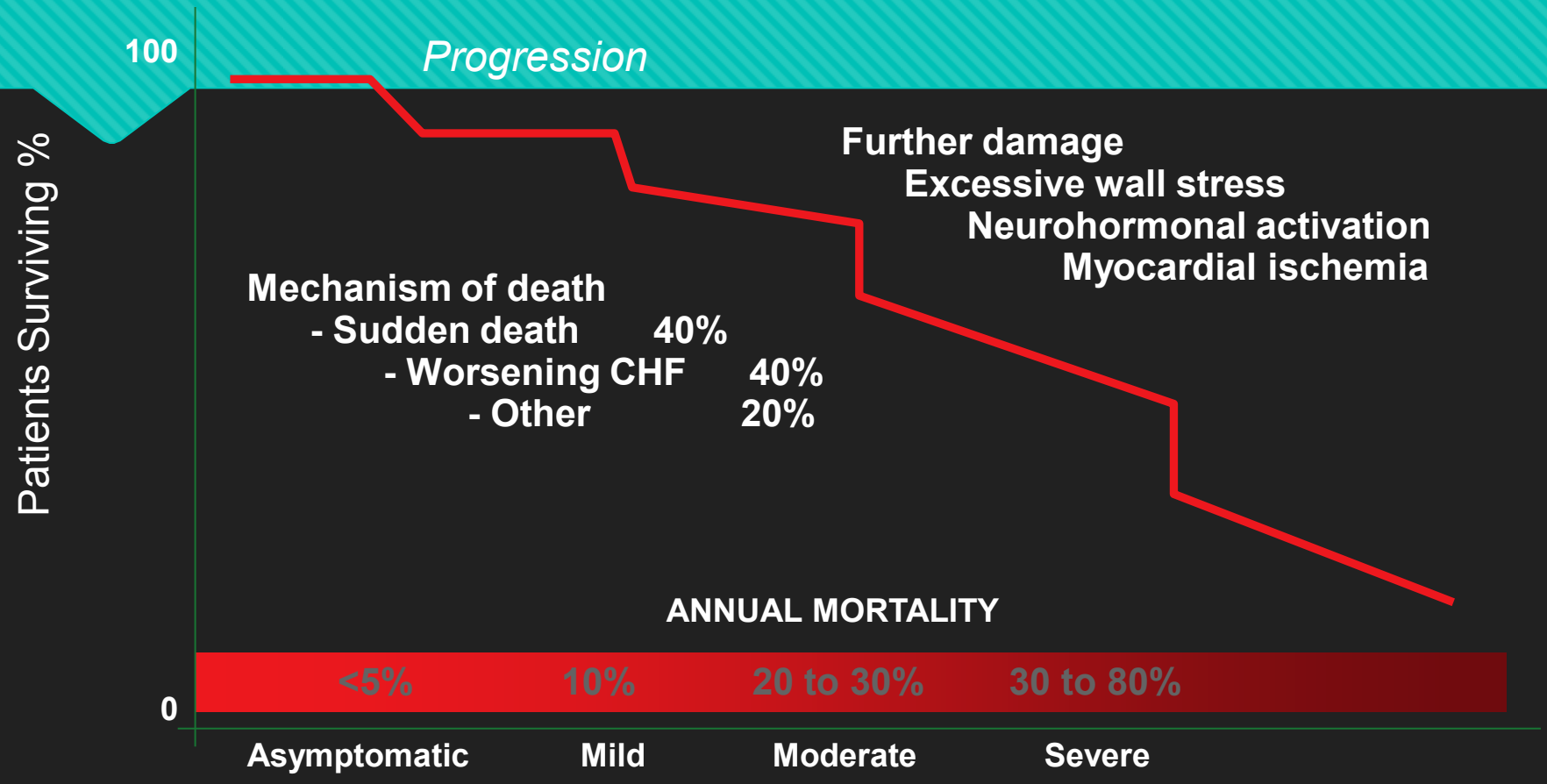
AHA Heart and Stroke Statistical Update 2001

Disease Progression of HF: ACC/AHA HF Stages



Yancy CW, Strong M. *Prim Care Spec Ed.* 2002;6:15

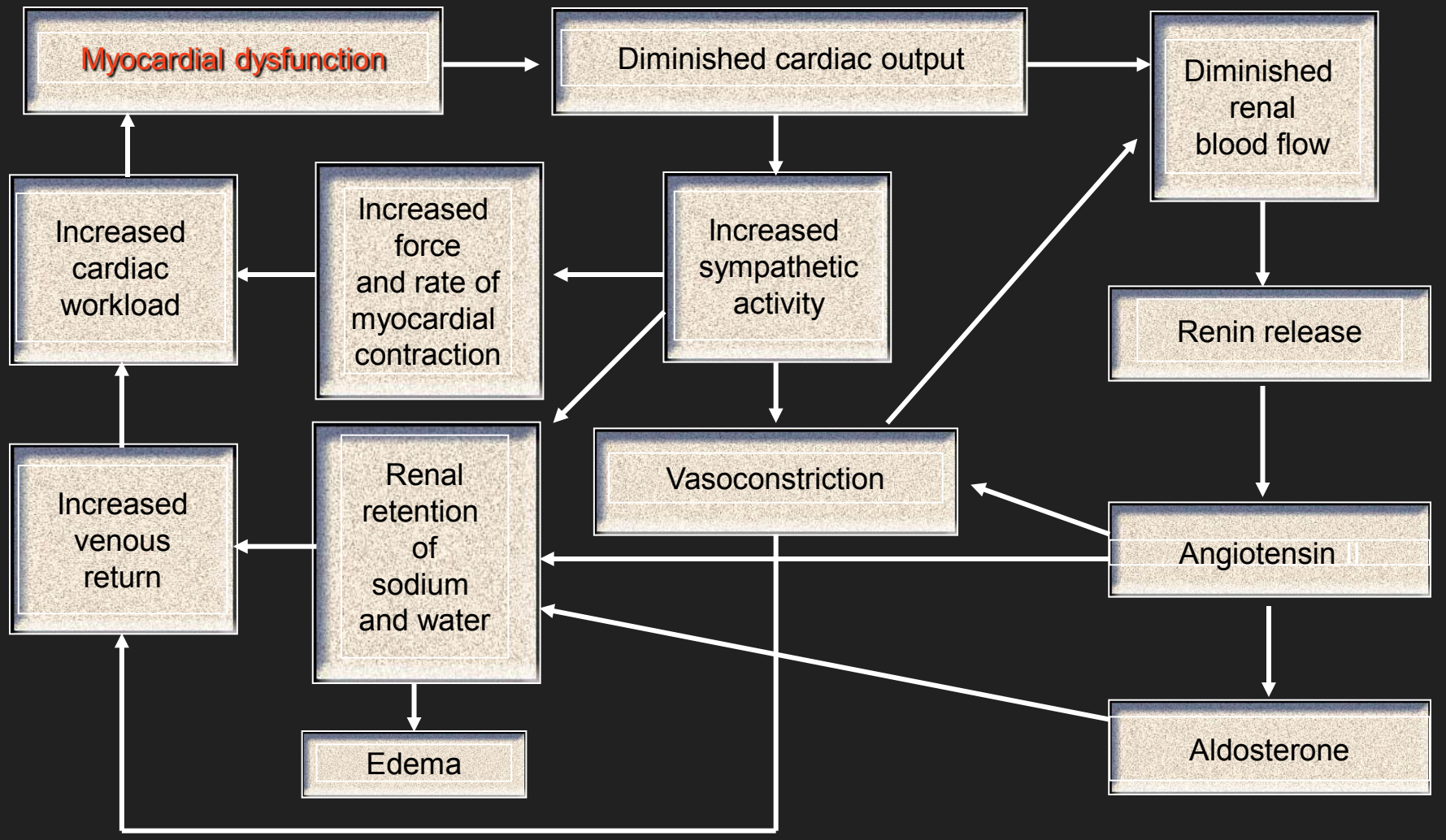
Heart Failure Progression and Mortality



O'Connell JB. *Clin Cardiol*. 2000;23(suppl 3):S6-S10.

Heart Failure: Pathophysiology, Diagnosis, and Hemodynamic Targets

Progression of Heart Failure

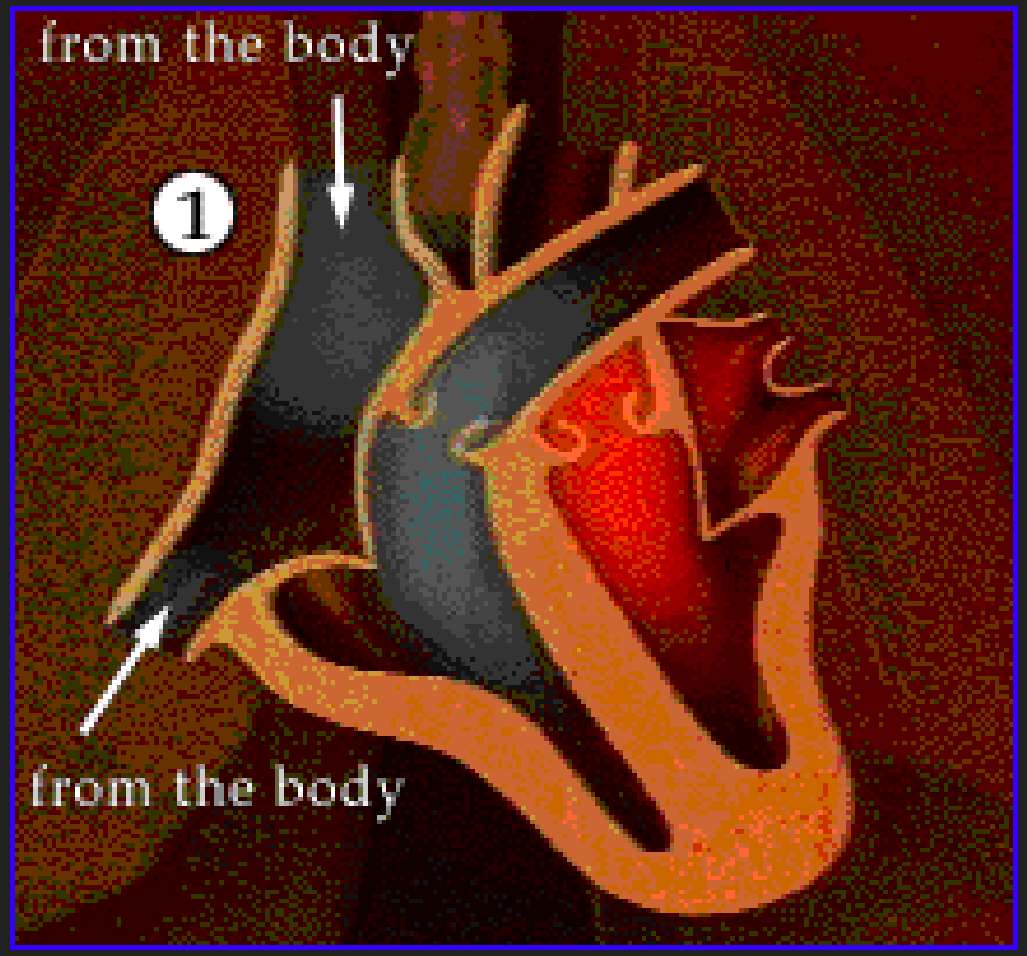


Shah M, Ali V. *Rev Cardiovasc Med.* 2001;2(suppl 2):S2–S6.

What is heart failure?

•The failure of the heart to pump blood adequately.

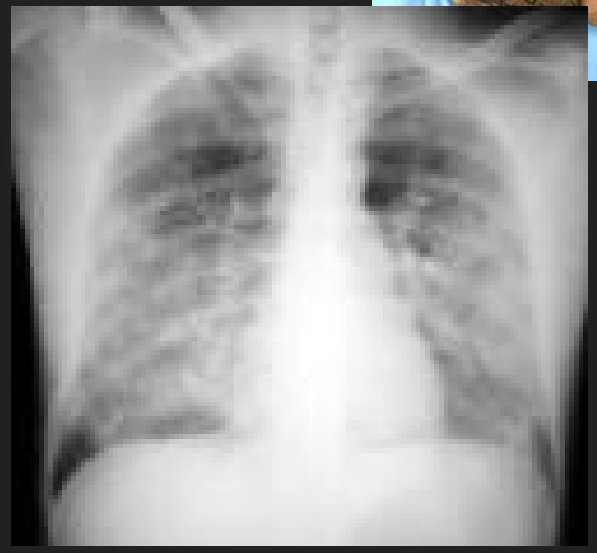
• The heart receives a sufficient volume of blood from the incoming vessels but cannot pump it out adequately.



Right and Left sided HF

- Right sided—the right ventricle cannot pump out enough blood causing fluid to leak out at the capillary level, leading to systemic edema.

- Left sided-left ventricle can't pump out enough blood, causing pulmonary edema.



Primary Causes of Heart Failure

- Coronary heart disease
- High blood pressure over a period of years
- Heart valve dise
- Infections, such
- Diseases of the pericardium
- Myocarditis
- Chemotherapy
- Untreated chronic ta
- Connective tissue disease

- Peripartum cardiomyopathy
- Bradycardia
- oidism
- Pericardial tamponade
- ts
- Radiation
- vere anemia
- ypothyroidism
- Excessive alcoh

Living longer

Surviving heart attacks

obesity

Worsening diets

High blood pressure

How the body compensates…part 1

Activation of the Sympathetic Nervous System

Increase HR, contraction, vasoconstriction (BP)
(causes increased myocardial oxygen demand)

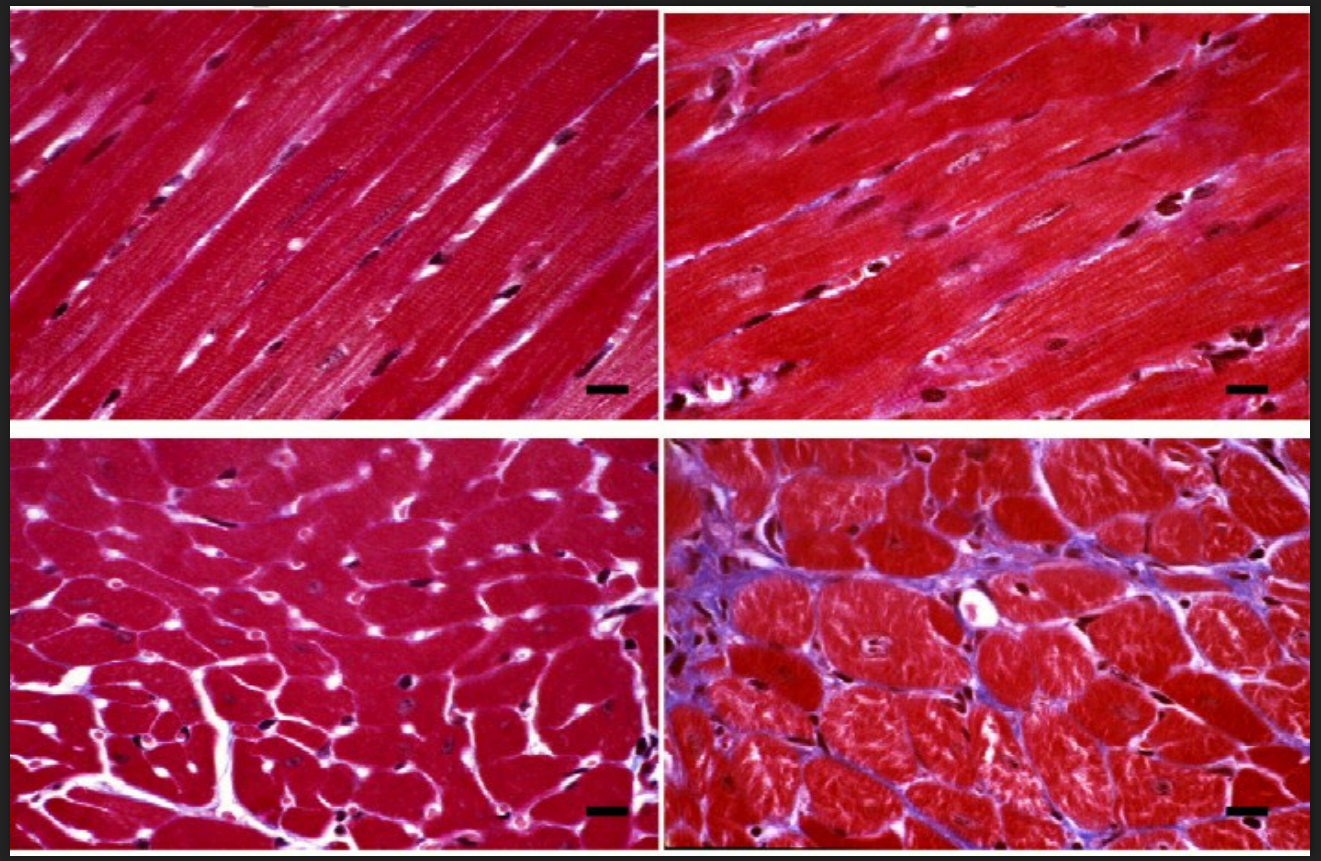
- Long term effects—

  "down regulation"

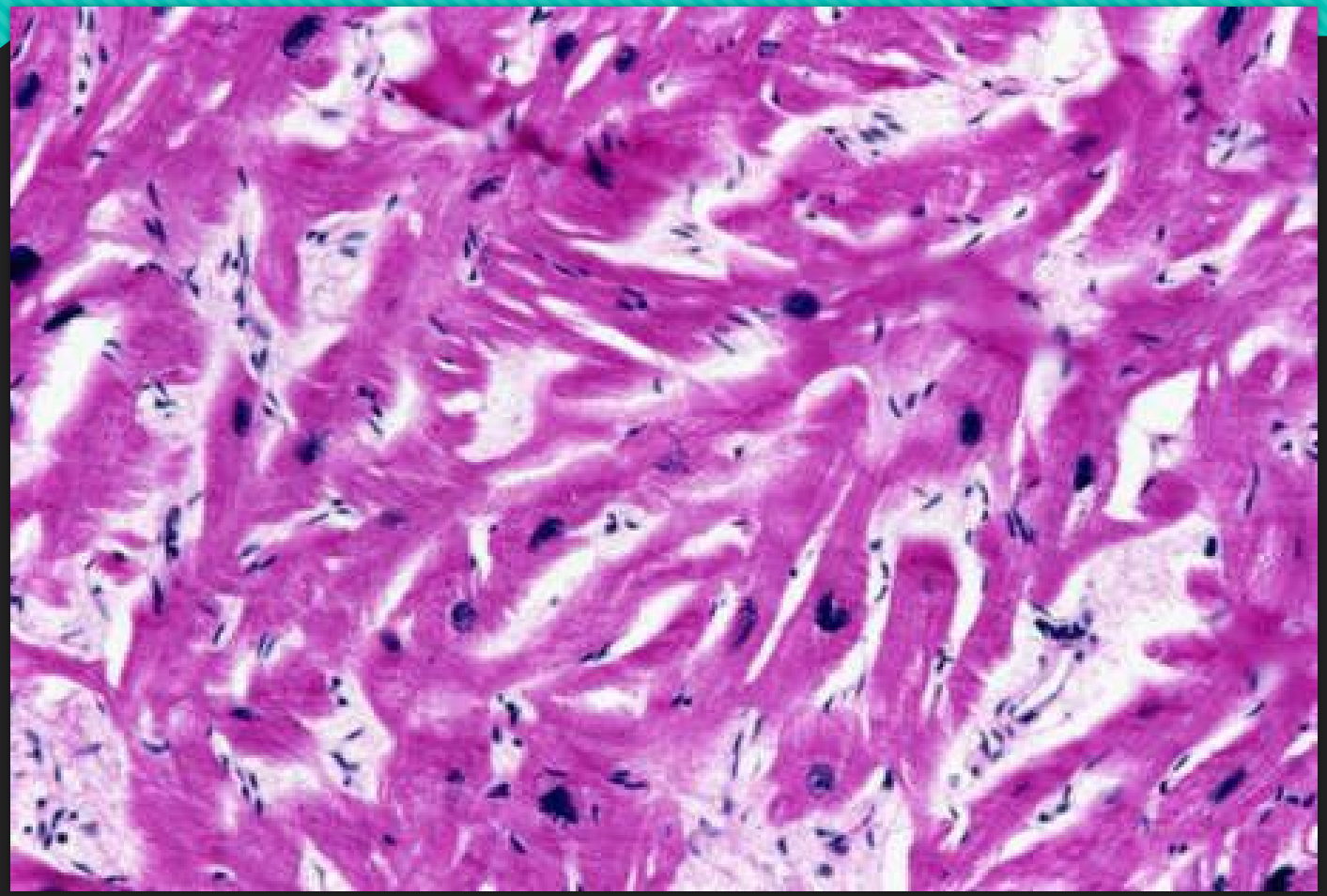
- Chronic- "remodeling"

  Beta blockers

Ordinary striated cardiac myocytes



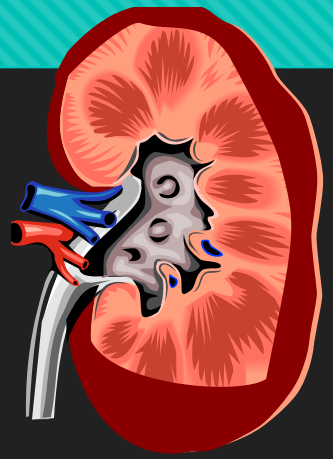
Cardiac myocyte disorganization



How the body compensates…part 2

Decrease blood flow to kidneys activates…

RENIN-ANGIOTENSION-ALDOSTERONE System



Renin--angiotension I-- (ACE)--angiotension II-constricts arteries and veins

A II--Aldosterone-causes kidneys to retain salt and H2O

ACE inhibitors
ARBs

Spironolactone

How the body compensates…part 3

Naturetic peptides are released from atrial and ventricular tissue in response to volume expansion and rising pressure.

- Arterial and venous dilation
- Inhibition of sodium reabsorption

How the body compensates…part 4

Endothelin—released by the vascular endothelium—a potent vasoconstrictor

(especially in the kidney—reduces urine output)

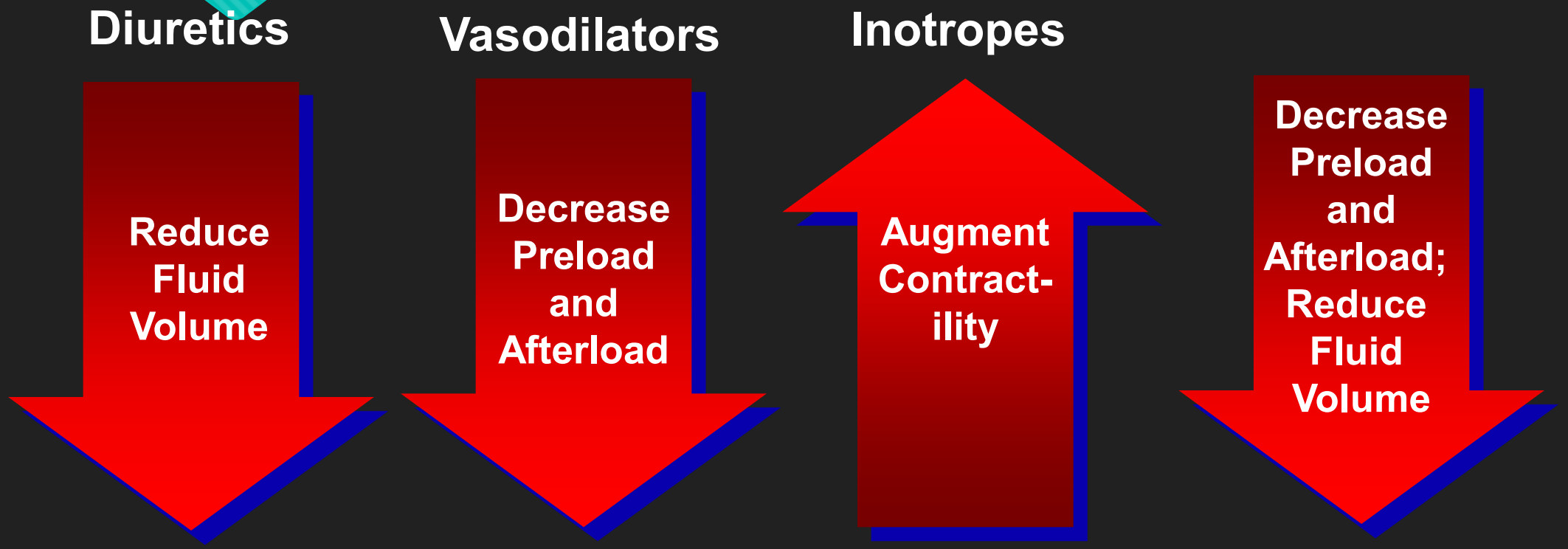
Naturetic peptides-Bnp

Heart Failure Signs and Symptoms are Insensitive and Non-specific

Symptoms Include:

- Dyspnea → Shortness of breath
- Fatigue → Feeling of tiredness
- Peripheral Edema → Swelling of legs and ankles
- Orthopnea → Pulmonary congestion
- Weight gain → Due to fluid retention
- Rales → Abnormal lung sounds

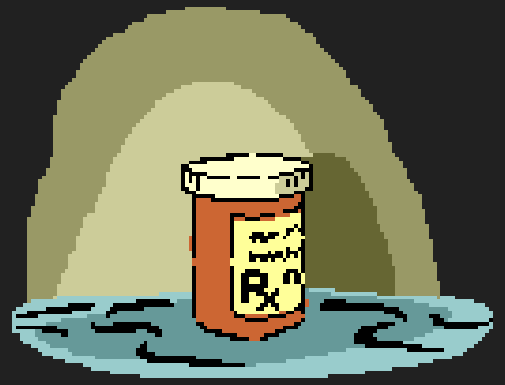
Current Treatments for ADHF



Treatment for the last 20 years

Why we are failing…

- Diuretics (and potassium)
- Vasodilators (especially ACE inhibitors)
- Nitrates
- Digoxin
- Beta blockers
- Inotropes
- Surgery (transplant, valve replacement, etc.)
- Cardiac resynchronous pacing



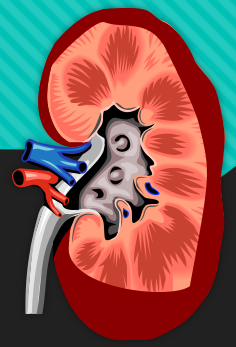
Don’t forget about me…

The kidney mediates much of the abnormal physiology of CHF,

is victimized by CHF,

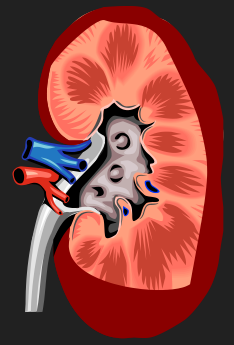
and limits the therapy of CHF

The murderous marriage



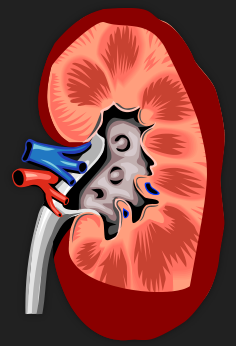
With a decrease in cardiac output, the fight begins—angiotension II

↓cardiac output and ↑ systemic vascular resistance results in ↓renal perfusion



↓renal perfusion associated with ↓GFR and ↓renal excretion of sodium and water

↑intravascular volume and ↑neurohormonal toxins accelerates ventricular remodeling and decreases cardiac output and renal perfusion



Cycle starts again

Diuretic Resistance

- Decreased perfusion of kidney results in decreased delivery of loop diuretics
- Decreased GFR results in decreased filtered load of sodium
- Volume depletion activates plasma renin activity and stimulates SNS
- Increased proximal tubule reabsorbtion of sodium, particularly in setting of elevated AII and elevated catecholamine levels
- Increased distal reabsorbtion of sodium, stimulated by aldosterone
- Associated with tubular hypertrophy: resetting basal rates of sodium reabsorbtion.
- Thiazide diuretics may be used

GFR=glomerular filtration rate, SNS=sympathetic nervous system

Neurohormonal Actions Influencing Diuretic Action

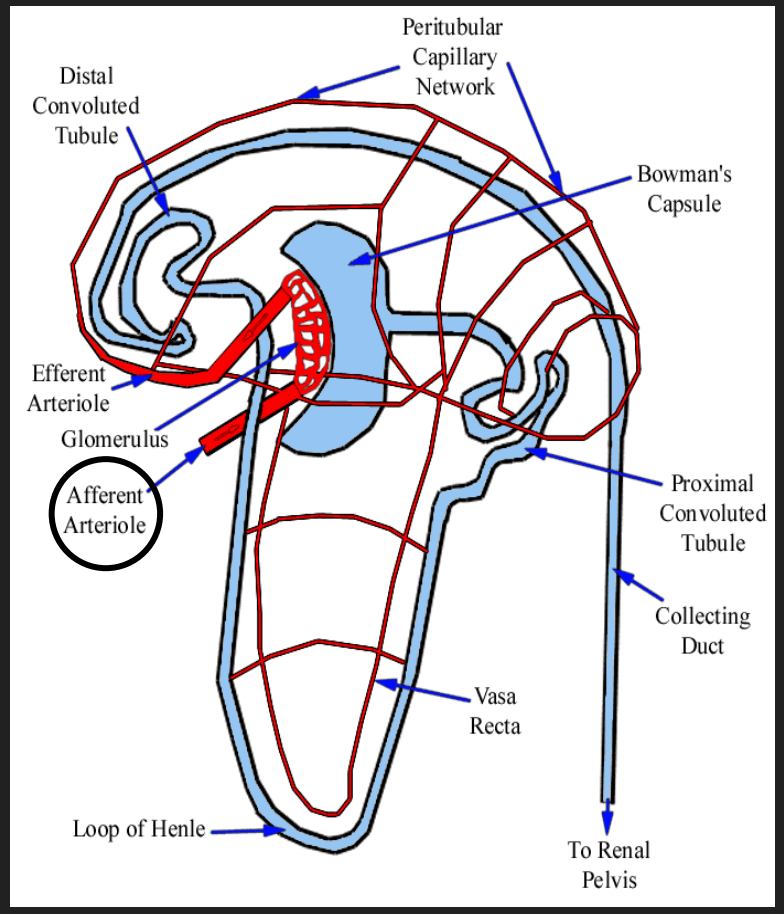
Proximal Tubule
Ang II increases Na reabsorption

Glomerulus
Norepinephrine, endothelin, Ang II decrease renal blood flow and GFR

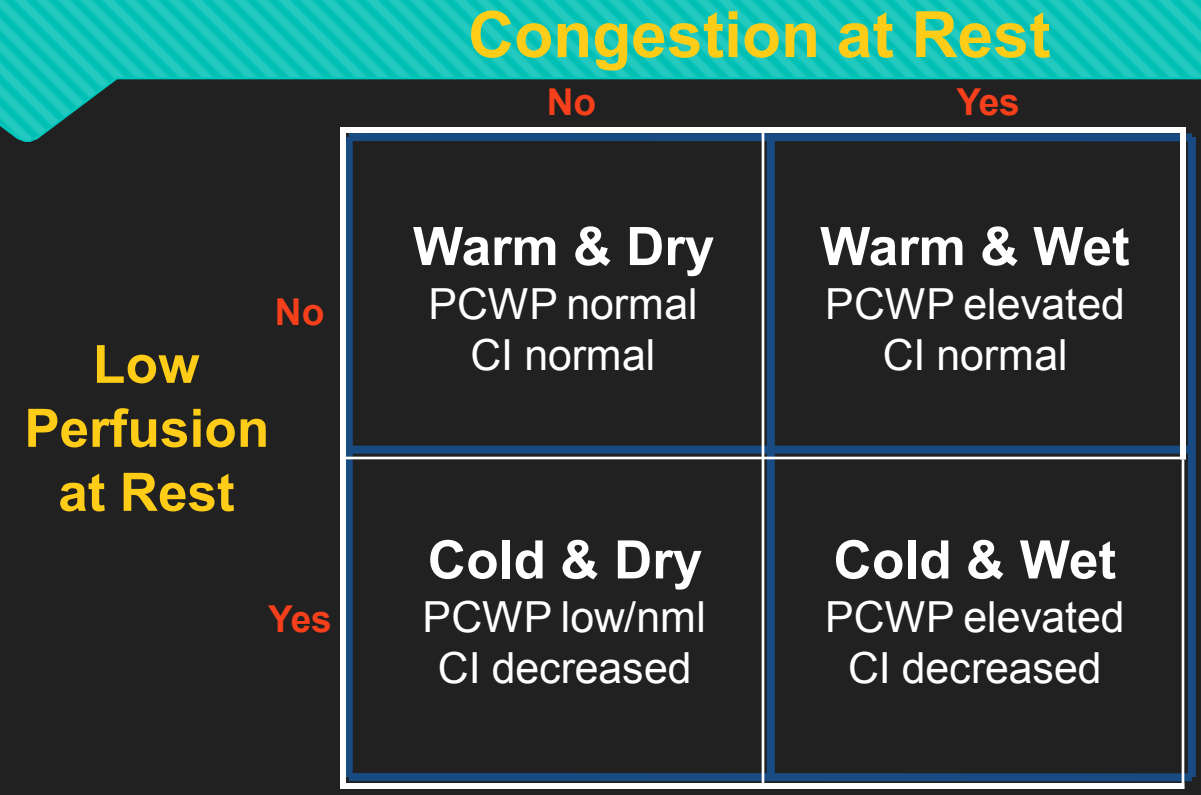
Collecting Duct
Aldosterone increases Na reabsorption

Effects of hBNP on the Kidneys

1. hBNP causes vasodilatation of the afferent arteriole
2. Vasodilatation of afferent arteriole causes an increase in GFR
3. Increase in GFR causes greater renal perfusion allowing more diuretic to get to site of action—therefore, less diuretic is needed
4. hBNP physiologically reverses the negative effects of the RAAS



Rapid Assessment of Hemodynamic Status



Signs/symptoms of congestion

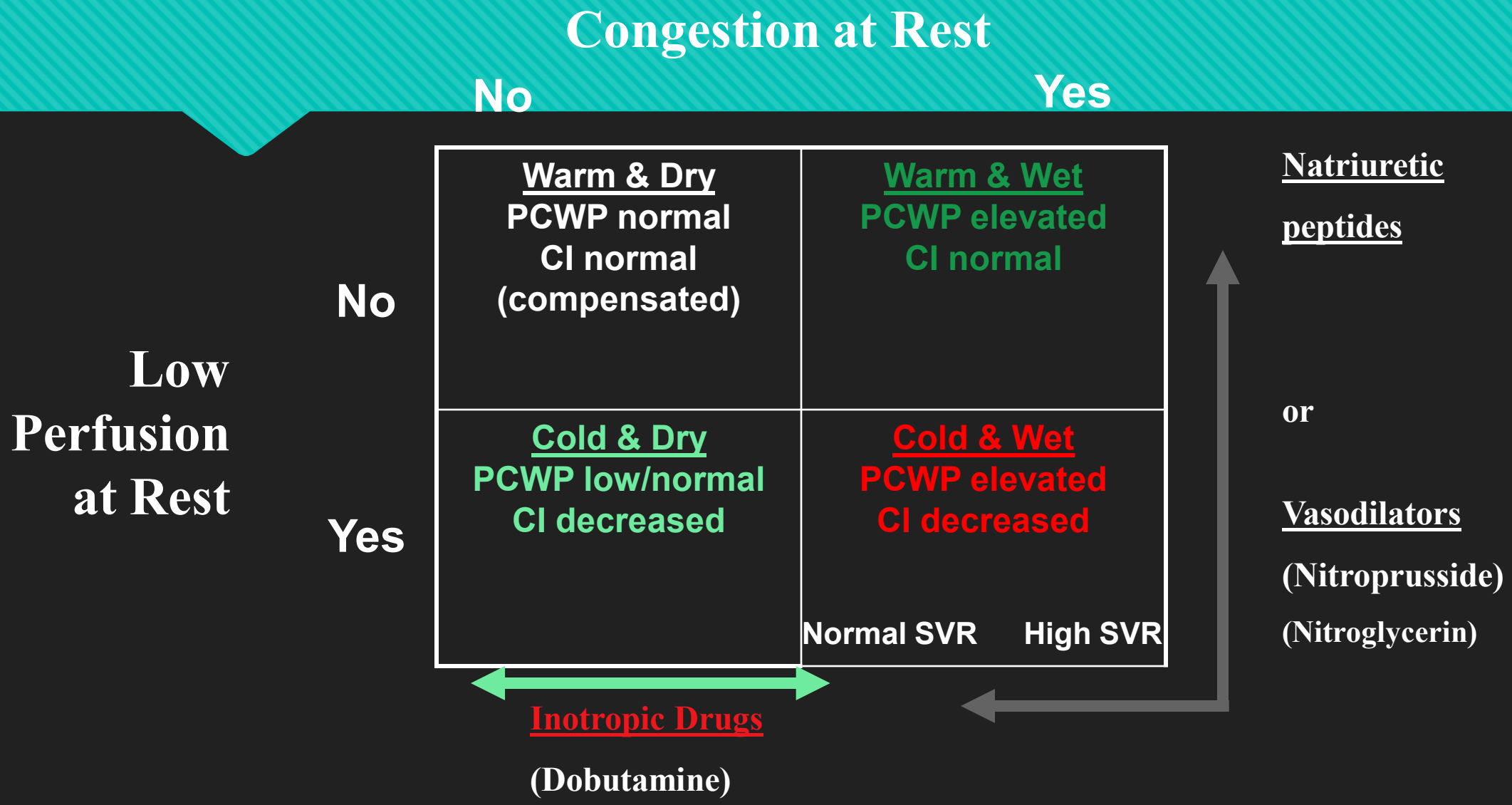
Orthopnea/PND
JV distension
Ascites
Edema
Rales (rare in CHF)

Possible evidence of low perfusion

Narrow pulse pressure
Sleepy/obtunded
Low serum sodium
Cool extremities
Hypotension with ACE inhibitor
Renal dysfunction (one cause)

Fonarow GC. *Rev Cardiovasc Med*. 2001;2(suppl 2):S7-S12.

Patient Selection and Treatment



Ideal agent for decompensated heart failure

- Vasodilatation (venous and arterial)
- Rapidly decreases ventricular filling pressures
- Rapidly decreases symptoms of congestion
- Not chrono-or inotropic (O2 demand)
- No tachyphylaxis
- Provides neurohormonal suppression
- Promotes diuresis/natriuresis

Natriuretic Peptides: Pharmacokinetics and Biologic Activity

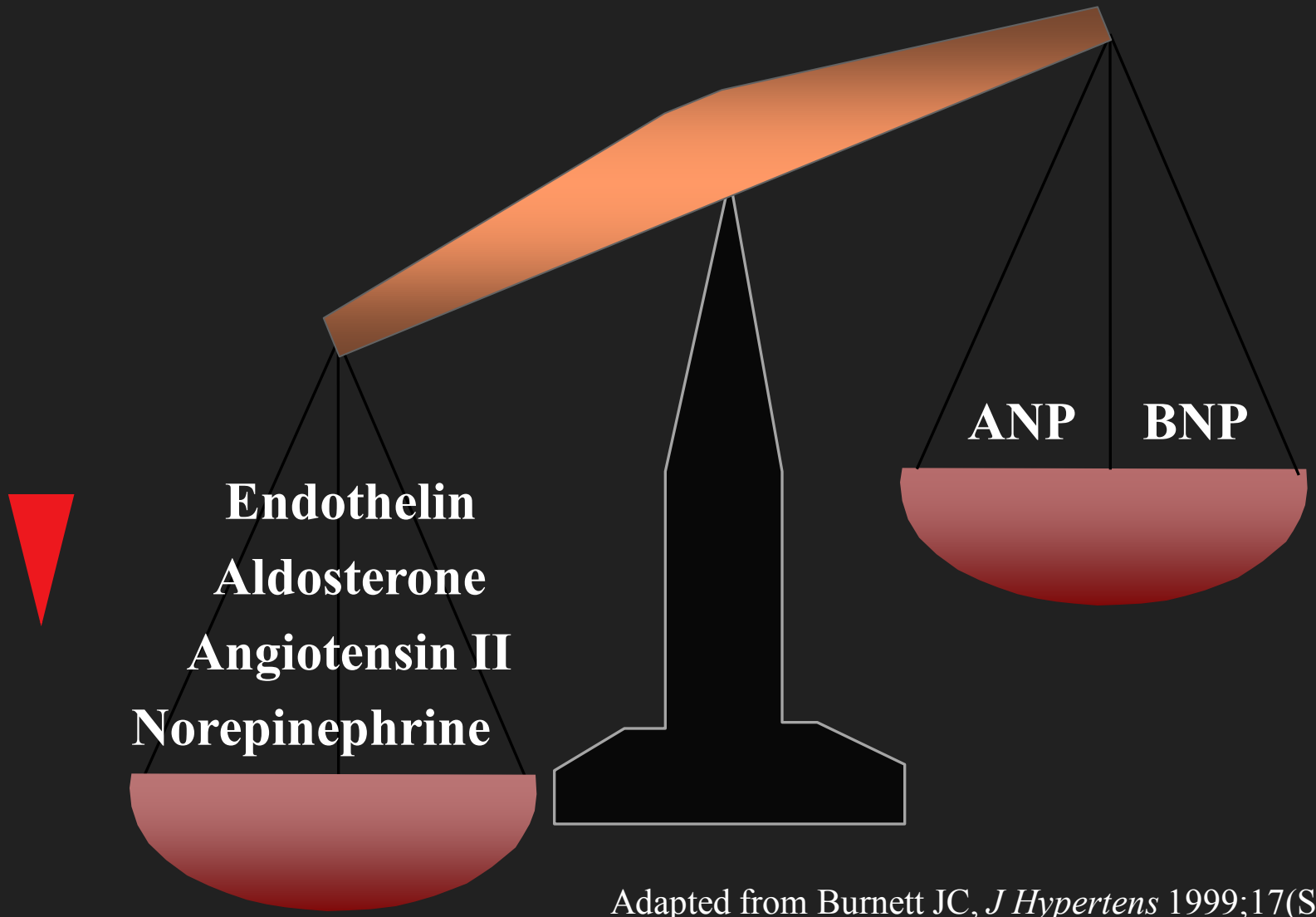
Natriuretic Peptides: Origin and Stimulus of Release

Peptide	Primary Origin	Stimulus of Release
ANP	Cardiac atria	Atrial distension
BNP	Ventricular myocardium	Ventricular overload
CNP	Endothelium	Shear stress of endothelium

ANP = Atrial Natriuretic Peptide
BNP = B-type Natriuretic Peptide
CNP = C-type Natriuretic Peptide

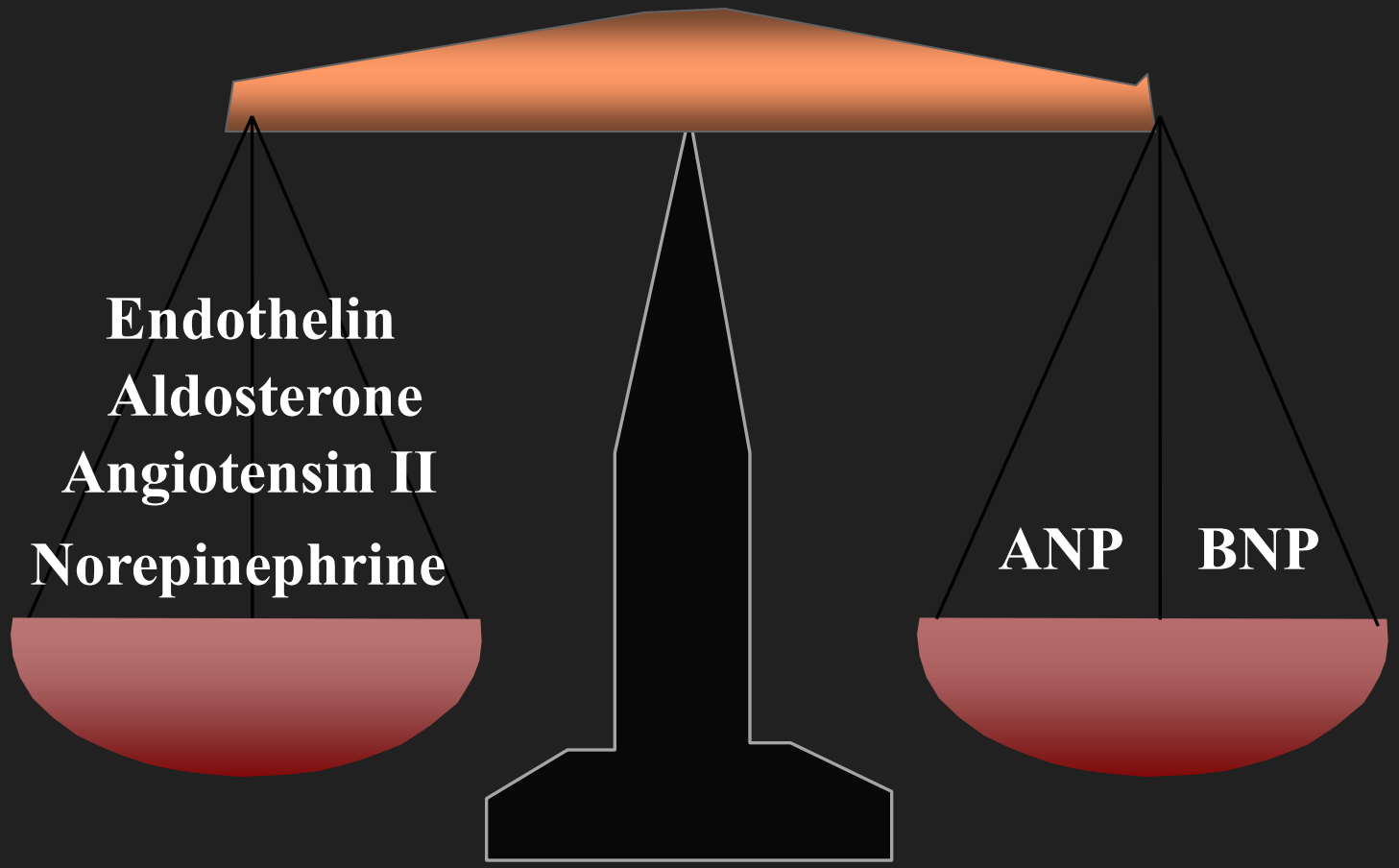
Adapted from Burnett JC, *J Hypertens* 2000;17(Suppl 1):S37-S43

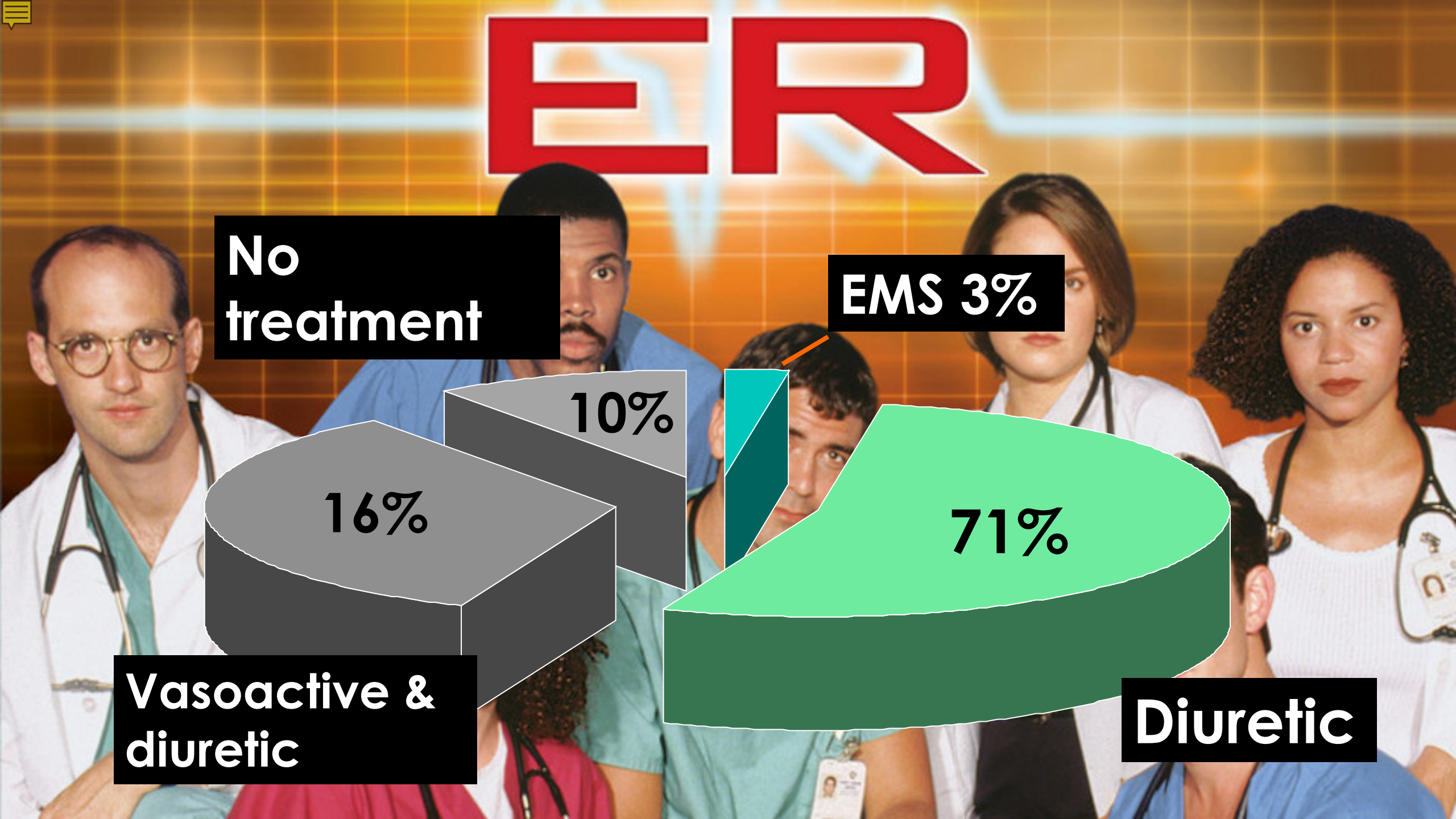
The Natriuretic Peptide System is Overwhelmed in Acute Decompensated Heart Failure



Adapted from Burnett JC, *J Hypertens* 1999;17(Suppl 1):S37-S43

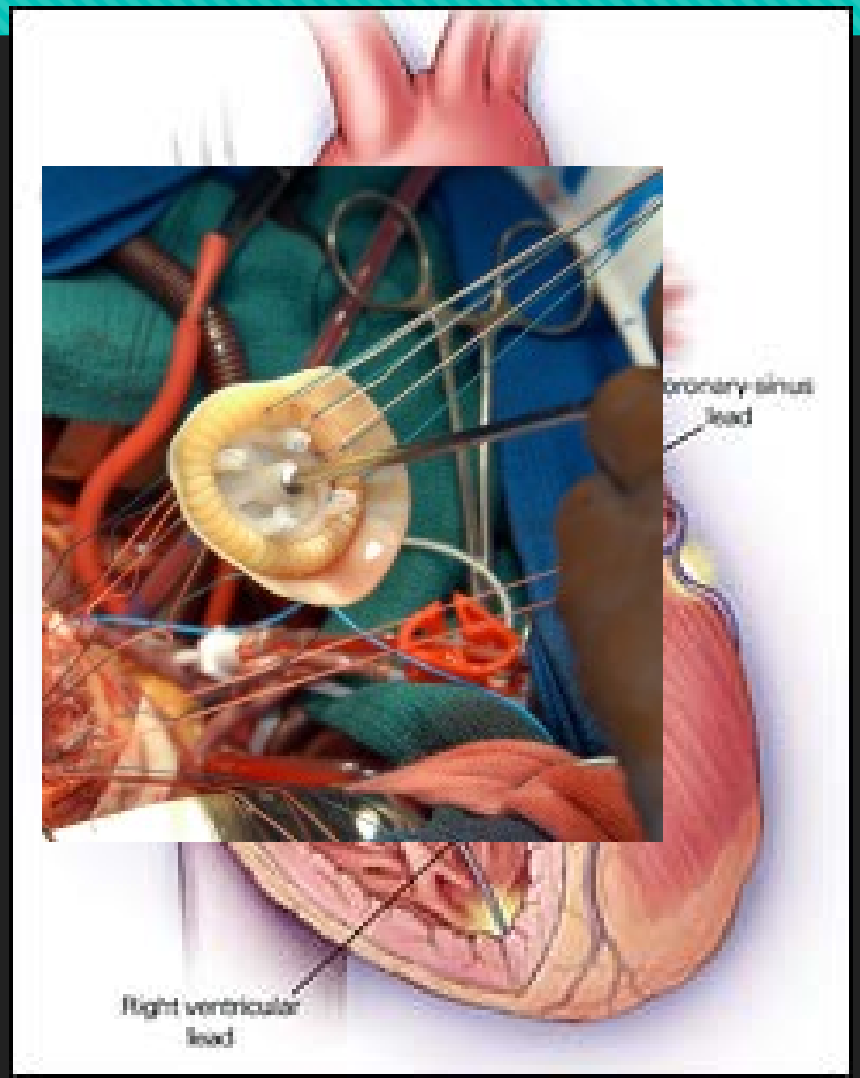
Effects of Endogenous hBNP




ER
No treatment
EMS 3%
10%
16%
71%
Vasoactive & diuretic
Diuretic

Devices for heart failure

Resyncronization therapy
Bi-ventricular pacing
Dor Procedure or SAVER (Surgical Anterior Ventricular Endocardial Restoration)
Dynamic Cardiomyoplasty
Enhanced External Counterpulsation (EECP)



Medications

- ACE: A ARB: A The clinical strategy of inhibition of the renin-angiotensin system with ACE inhibitors *(Level of Evidence: A)* (9–14), OR ARBs *(Level of Evidence: A)* (15–18), OR ARNI *(Level of Evidence: B-R)*(19) in conjunction with evidence-based beta blockers (20–22), and aldosterone antagonists in selected patients (23,24), is recommended for patients with chronic HF*r*EF to reduce morbidity and mortality.ARNI: B-R
- ARB: A The use of ARBs to reduce morbidity and mortality is recommended in patients with prior or current symptoms of chronic HF*r*EF who are intolerant to ACE inhibitors because of cough or angioedema (15–18,27,28).
- ACE: A The use of ACE inhibitors is beneficial for patients with prior or current symptoms of chronic HF*r*EF to reduce morbidity and mortality (9–14,25).
- ARNI: B-R In patients with chronic symptomatic HF*r*EF NYHA class II or III who tolerate an ACE inhibitor or ARB, replacement by an ARNI is recommended to further reduce morbidity and mortality(19).
- Level C Diuretics –loop diuretics with evidence of fluid retention

2016 ACC/AHA/HFSA Focused Update on New Pharmacological Therapy for Heart Failure: An Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure

Key Nursing Treatment for HF

- Rapid triage to appropriate environment for safe clinical care: coronary care unit, cardiology ward, general medical ward
- Objective monitoring for change in signs and symptoms suggestive of response to treatment. Promptly identify and address relevant changes in clinical status
- Discharge planning and referral to a multidisciplinary disease management program
- Anxiety of the patient should be addressed by promptly answering questions and providing clear information to the patient and family.
- Ensure effective and consistent communication between the patient and/or family and multi-disciplinary team
- End of life care

ACEs and ARBs

Angiotension II receptor blockers

- Azilsartan (Edarbi)
- Candesartan (Atacand)
- Eprosartan.
- Irbesartan (Avapro)
- Losartan (Cozaar)
- Olmesartan (Benicar)
- Telmisartan (Micardis)
- Valsartan (Diovan)

ACE Inhibitors

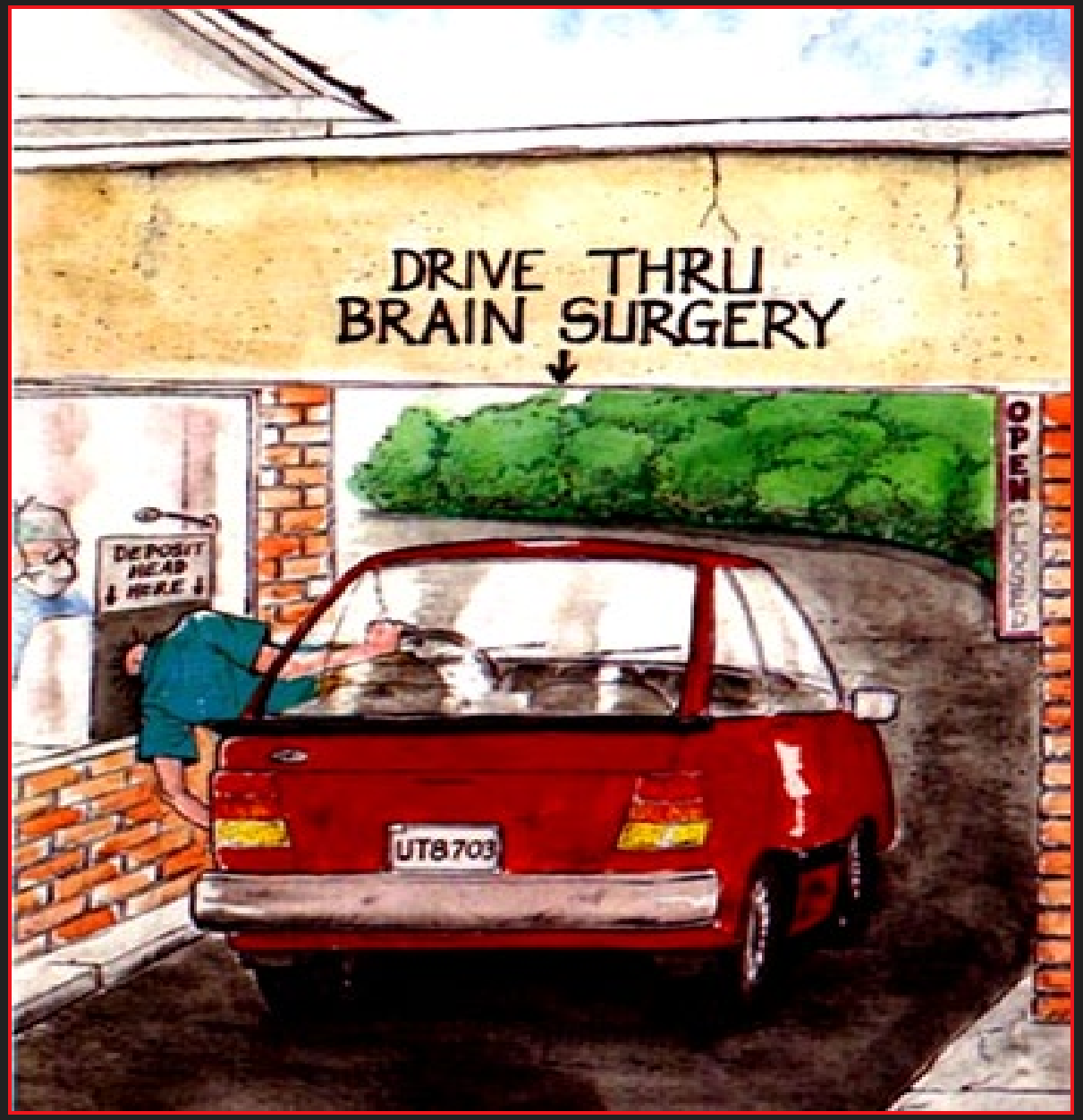
- benazepril (Lotensin, Lotensin Hct),
- **captopril** (Capoten),
- **enalapril** (Vasotec),
- fosinopril (Monopril),
- **lisinopril** (**Prinivil**, Zestril),
- moexipril (Univasc)
- perindopril (Aceon),
- quinapril (Accupril),

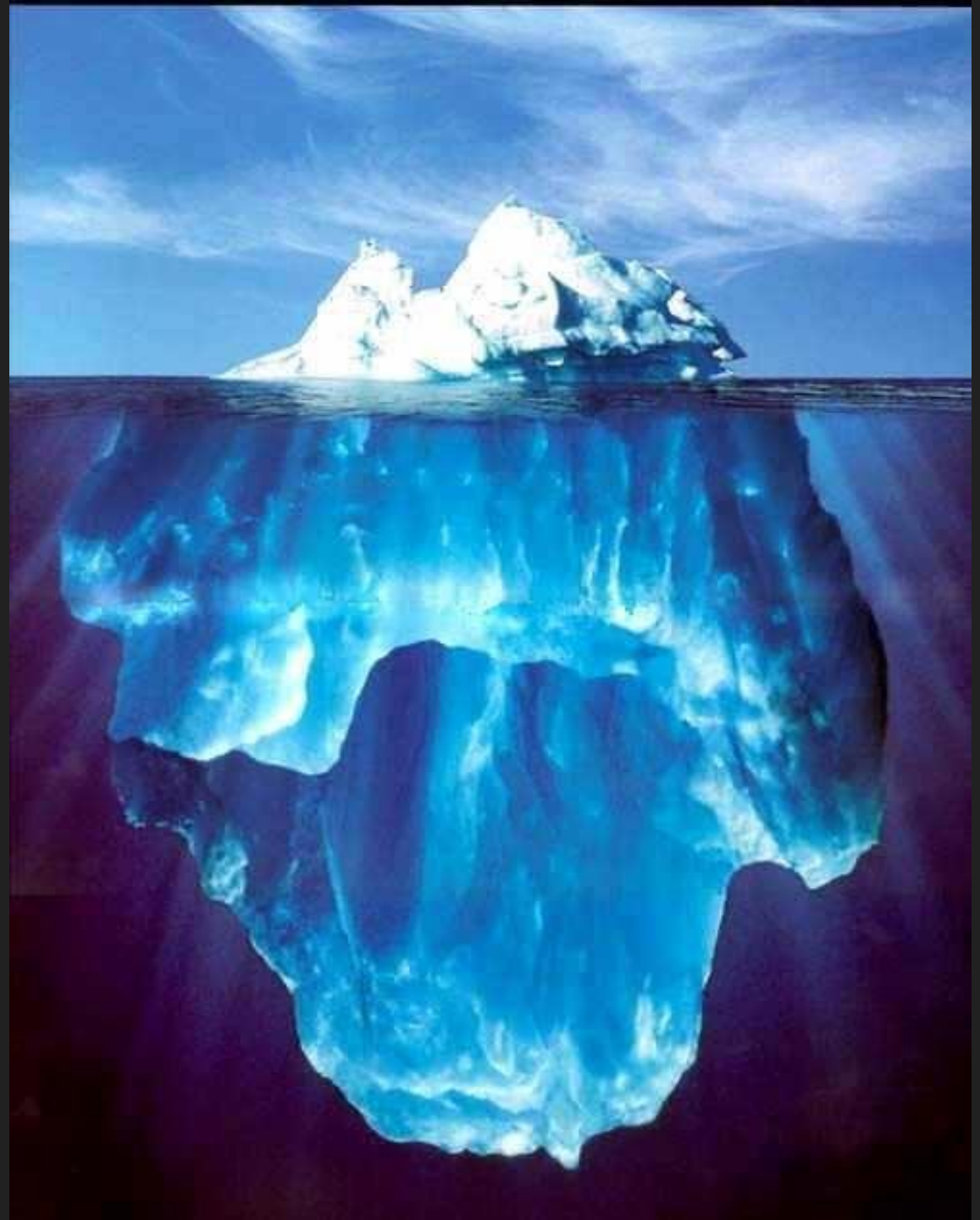
Neprilisyn

Neprilysin –endopeptidase-catalyses the degradation of ANP, BNP and breaksdown Angiotension II

Sacubitril – inhibits nepilysin- increases BNP

Entresto – Sacubitril and Valsartan (ARB)


DRIVE THRU
BRAIN SURGERY
DEPOSIT HEAD HERE
OPEN
UTB703



What’s next?

Questions?