CARDIAC FAILURE

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Learning Objectives

- By the end of this lecture, you will be able to:
  - Define Heart failure (Congestive heart failure)
  - Effects of cardiac failure
  - Differentiate between right sided & left sided heart failure.

Cardiac Failure

- Inability of the **Cardiac output** to keep pace with the body's demands for supplies and removal of wastes.

Cardiac Failure

- **Results from any structural or functional abnormality that impairs the ability of the ventricle to eject blood (Systolic Heart Failure) or to fill with blood (Diastolic Heart Failure).**

Cardiac Failure

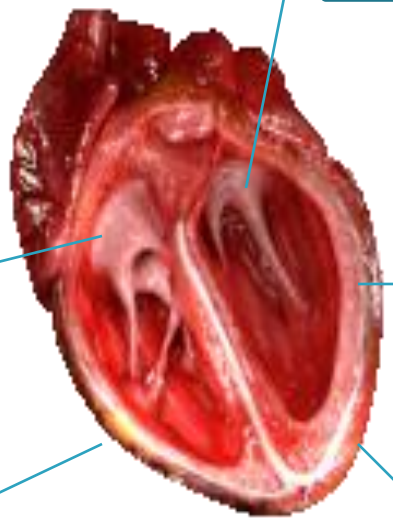
- Any heart condition that reduces the ability of the heart to pump enough blood to meet the body's needs.

- **Causes……..**

Causes

Reduced contractility of the myocardium b/c of diminished coronary blood flow

Damaged heart valves

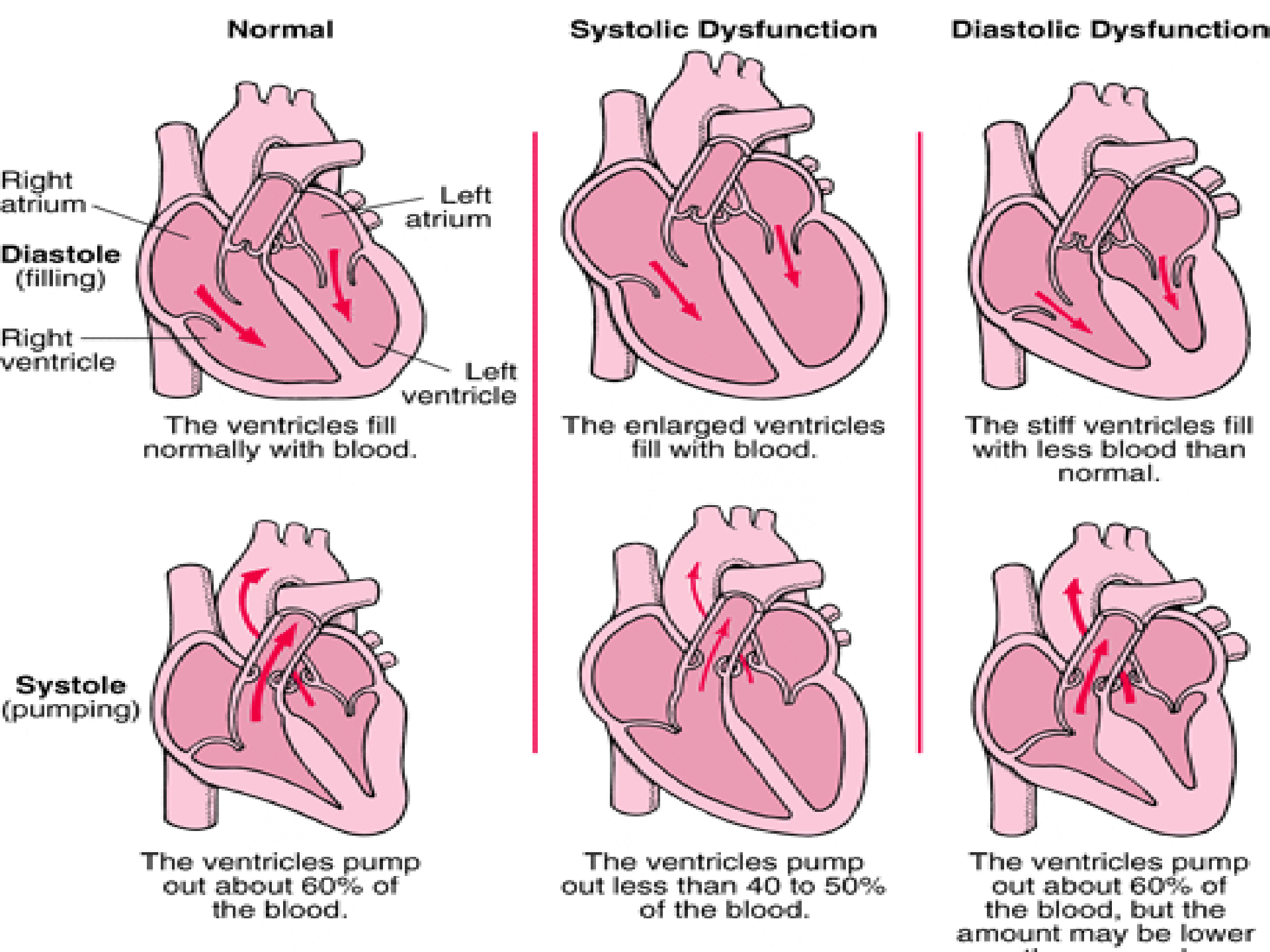


Any other disease

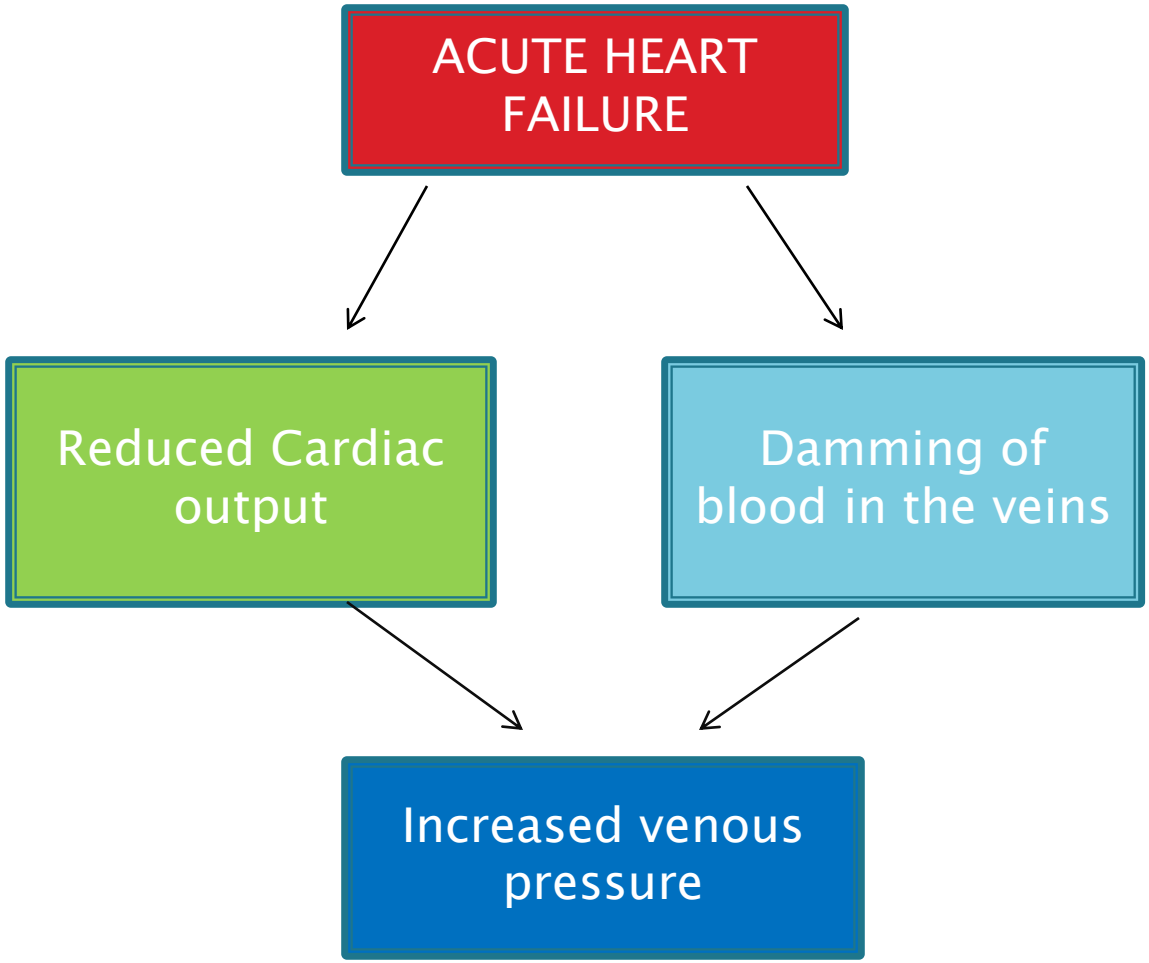
Cardiac Tamponade

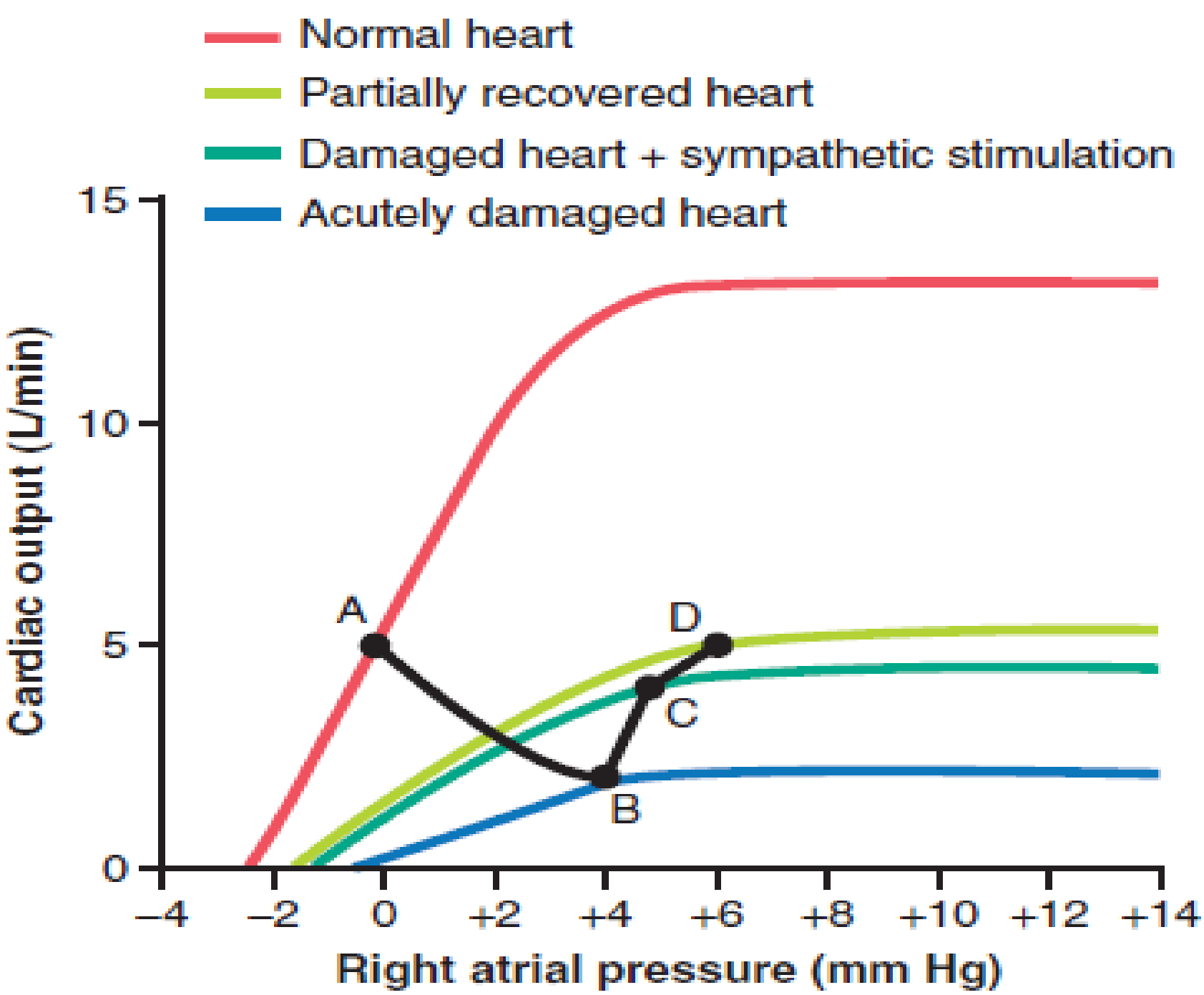
Primary Cardiac muscle disease

Vitamin B1 deficiency


Normal
Systolic Dysfunction
Diastolic Dysfunction
Right atrium
Left atrium
Diastole (filling)
Right ventricle
Left ventricle
The ventricles fill normally with blood.
The enlarged ventricles fill with blood.
The stiff ventricles fill with less blood than normal.
Systole (pumping)
The ventricles pump out about 60% of the blood.
The ventricles pump out less than 40 to 50% of the blood.
The ventricles pump out about 60% of the blood, but the amount may be lower

Acute Effects of Moderate Cardiac Failure




Normal heart
Partially recovered heart
Damaged heart + sympathetic stimulation
Acutely damaged heart
Cardiac output (L/min)
15
10
5
0
A
B
C
D
−4
−2
0
+2
+4
+6
+8
+10
+12
+14
Right atrial pressure (mm Hg)

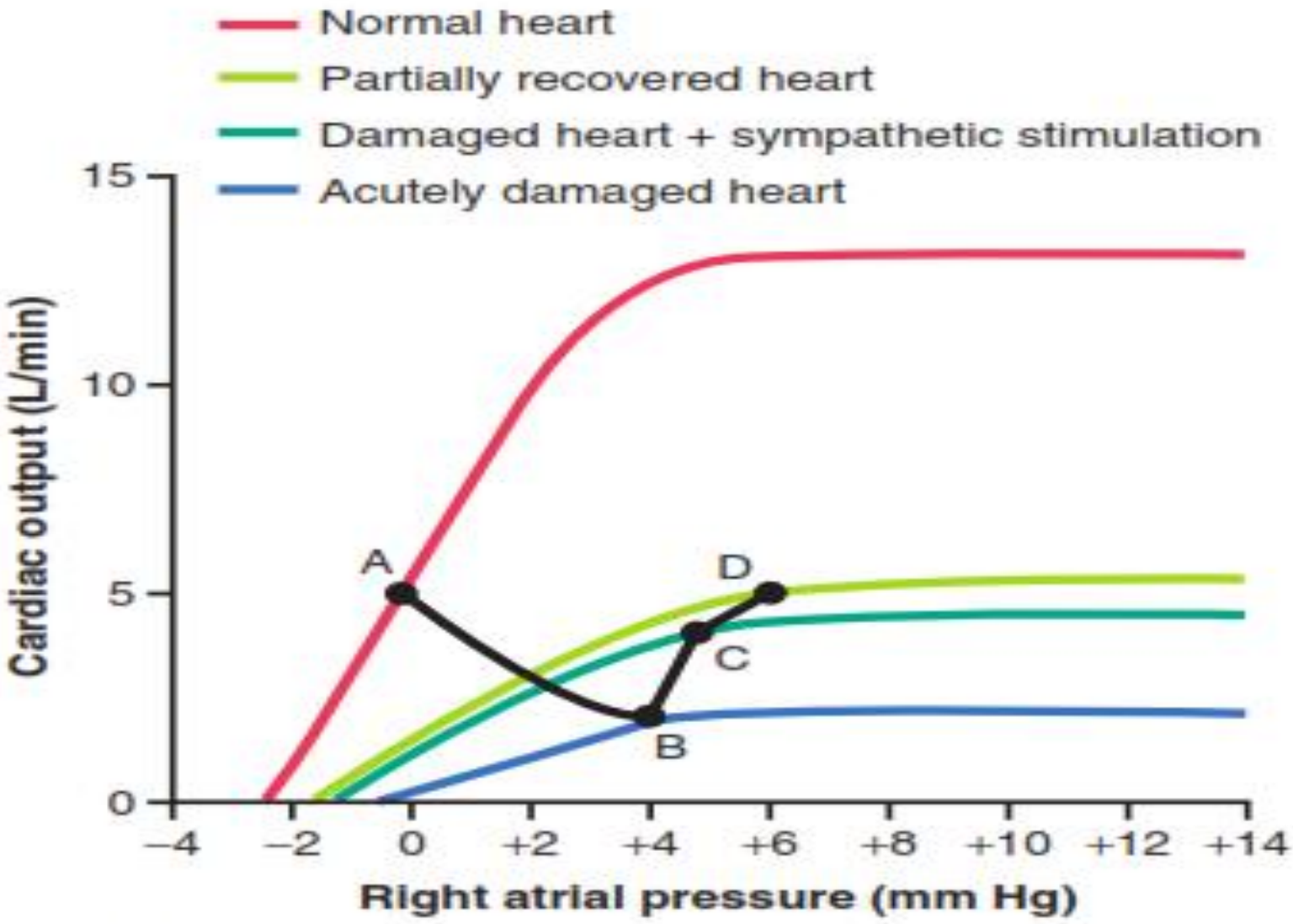
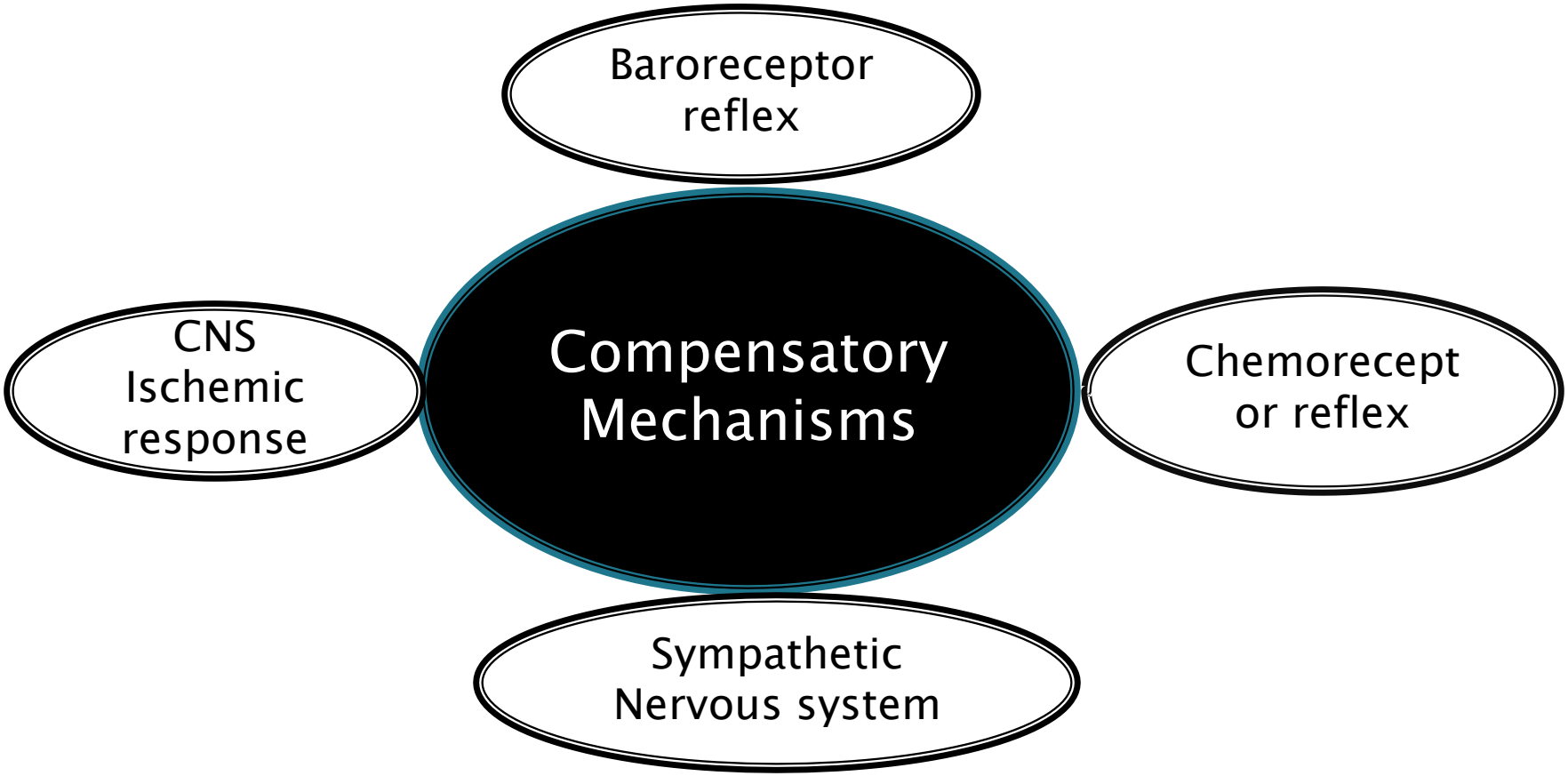


Figure 22-1. Progressive changes in the cardiac output curve after acute myocardial infarction. Both the cardiac output and right atrial pressure change progressively from point *A* to point *D* (illustrated by the *black line*) over a period of seconds, minutes, days, and weeks.

Compensation for Cardiac failure by Sympathetic Nervous Reflexes



- SNS become strongly activated within a few seconds.
- PNS signals to the heart become inhibited at the same time.
- SNS also increases Venous return.

Compensation for acute cardiac failure

- With fall of CO different circulatory reflexes are activated, like baroreceptor reflex , chemoreceptor reflexes and CNS ischemic reflex . Strong sympathetic stimulation has two major effects on circulation

1. Effect on heart
2. Effect on circulation

The sympathetic reflexes become maximally developed in about 30 seconds.

EFFECT ON HEART

- Strongly stimulated by sympathetic stimulation, in this way partially compensating for the nonfunctional muscle.
- Thus, *the heart becomes a stronger pump* as a result of sympathetic stimulation

EFFECT ON CIRCULATION

- Sympathetic stimulation also increases venous return because it increases the tone of most of the blood vessels of the circulation, especially the veins, *raising the mean systemic filling* pressure to 12 to 14 mm Hg, almost 100 percent above normal
- the damaged heart becomes primed with more inflowing blood than usual, and the right atrial pressure rises still further, which helps the heart to pump still larger quantities of blood.

COMPENSATION FOR CHRONIC C.F

After the first few minutes of an acute heart attack, a prolonged semi-chronic state begins, characterized mainly by two events:

1. Retention of fluid by the kidneys and
2. Varying degrees of recovery of the heart itself over a period of weeks to months

Renal Retention of Fluid

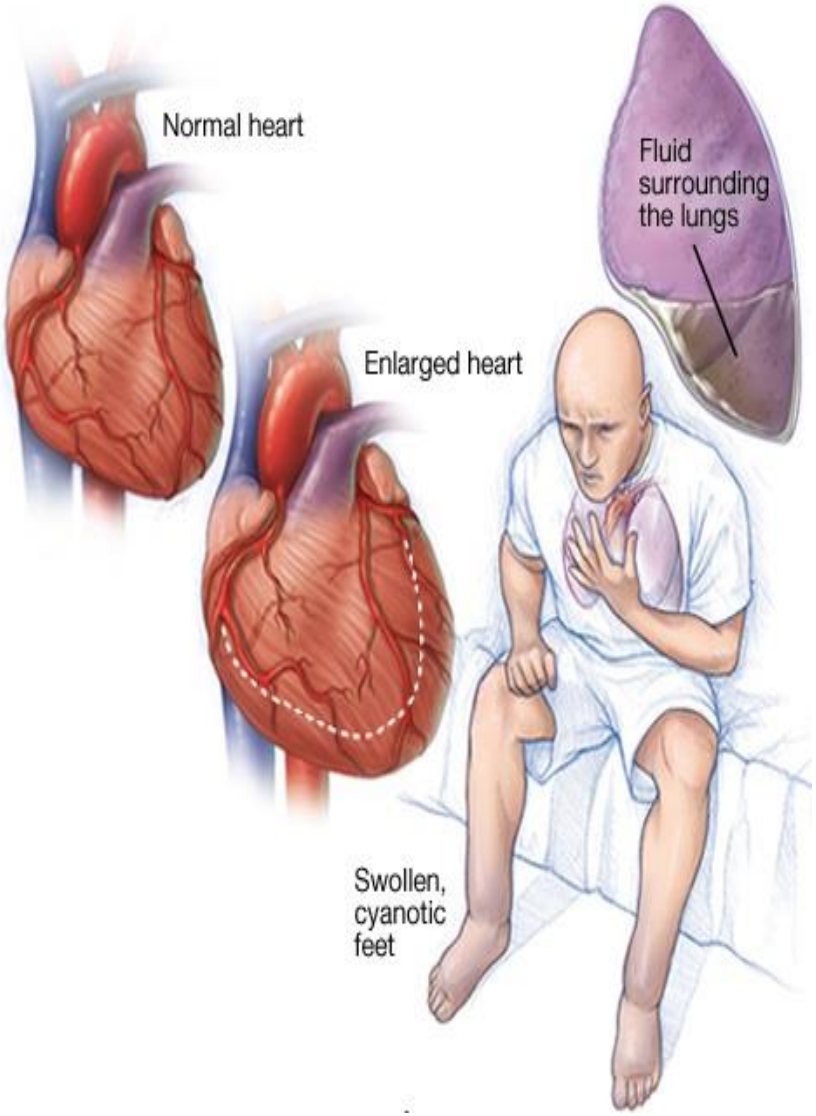
When the cardiac output falls to 50 to 60 % of normal, three effects occur

1. Glomerular filtration is reduced
2. Angiotensin 2 is released
3. ADH is secreted

All these effects help in increasing the reabsorption of Na and water by nephrons, so blood volume increase followed by increase venous return so as a result, CO increases

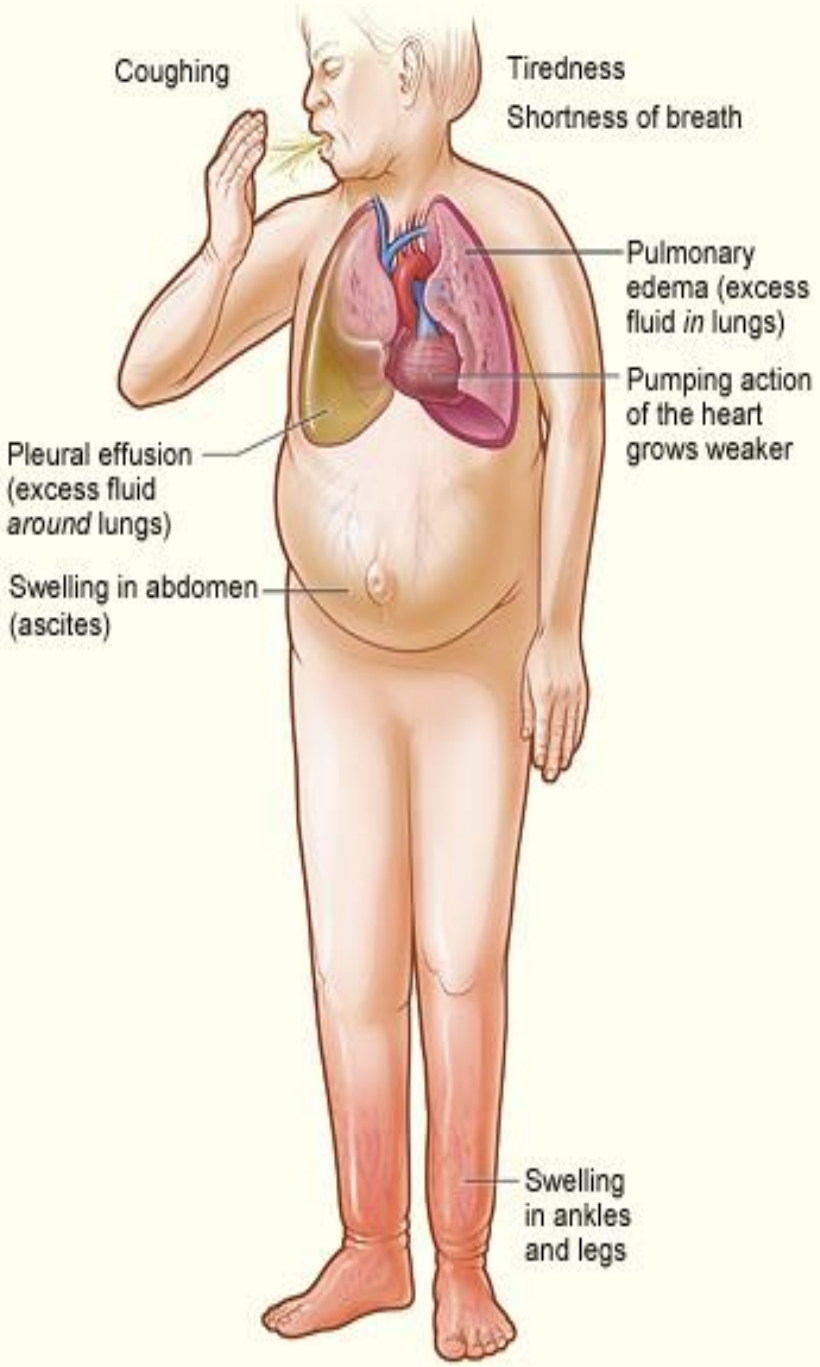
Detrimental Effects of excess fluid retention in severe cardiac failure.

- Increasing the workload.
- Overstretching of the heart.
- Filtration of fluids into the lungs.
- Development of extensive edema.



Chronic Stage of Failure

- Fluid Retention
  - Moderate Fluid Retention
    - Beneficial
  - Excess Fluid Retention
    - Detrimental Effects
      - overstretching of heart
      - pulmonary edema
      - edema



Recovery of heart

- Usually, varying degrees of recovery of the heart occurs itself over a period of weeks to months after cardiac failure.
- New collateral blood vessels.
- Undamaged portion hypertrophies.
- Recovery?
- Recovery occurs during the first few days and weeks.
- Most recovery in 5-7 weeks, although mild degree of additional recovery may continue for months.

Compensated Heart Failure

- When normal CO is restored by acute and chronic compensatory mechanisms after cardiac failure this is called Compensated Heart Failure.
- Aggravated by:

Decompensated CF

- If heart is severely damaged no amount of compensation can help this weakened heart to pump normally. So, the kidneys continue to retain fluid resulting in edema and eventually leads to death.
- This is called Decompensated CF

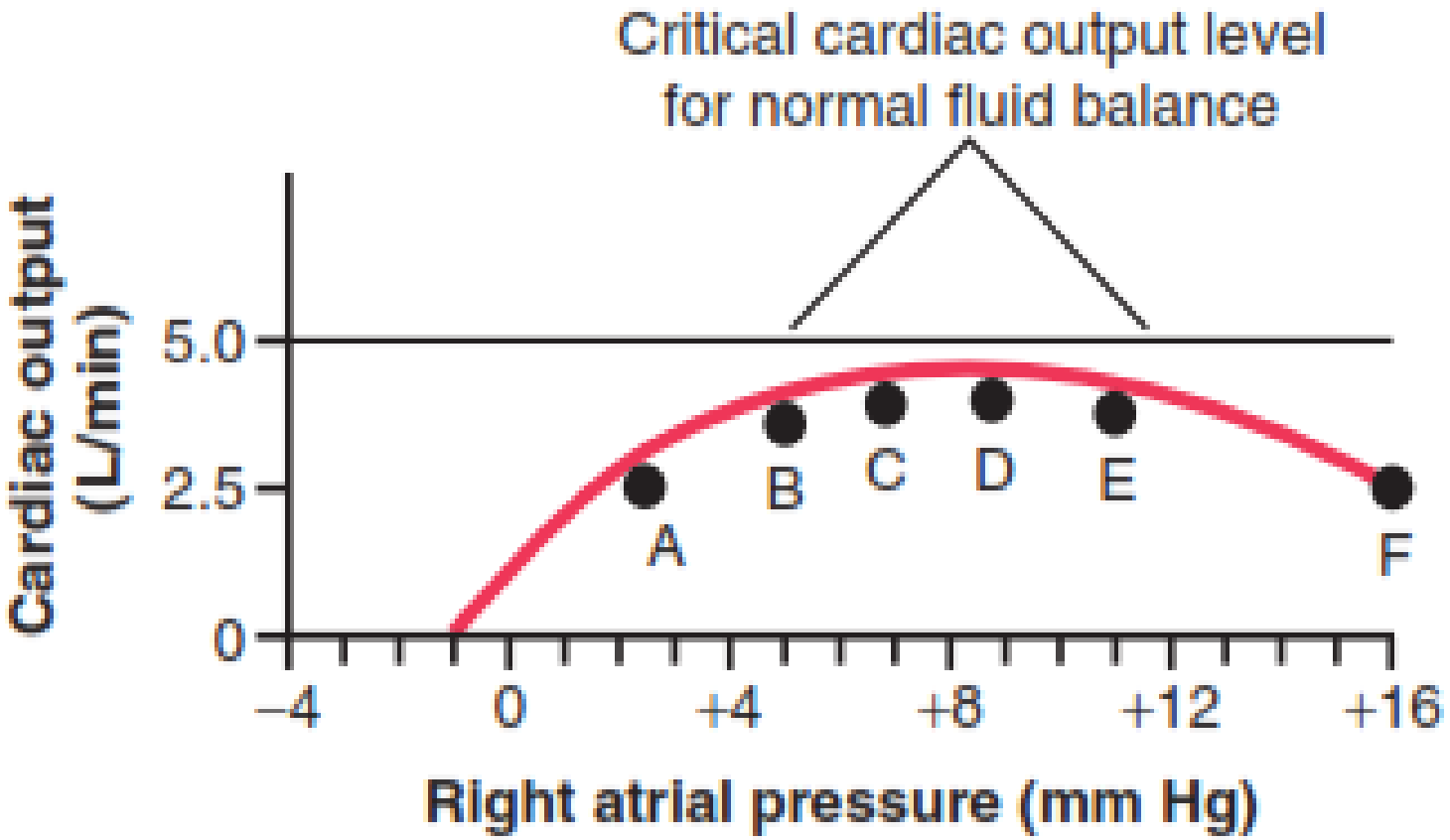
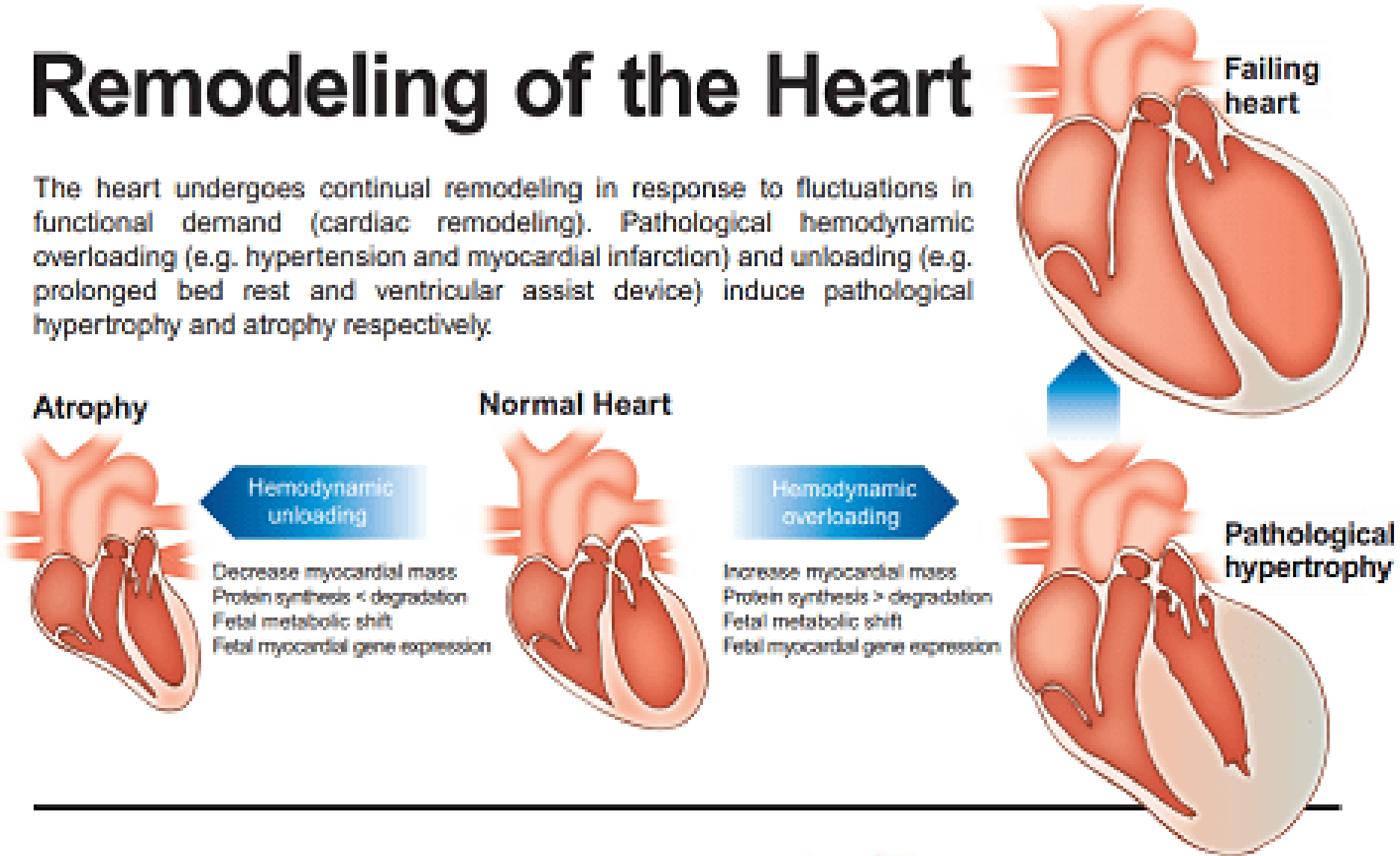


Figure 22-2. Greatly depressed cardiac output that indicates decompensated heart disease. Progressive fluid retention raises the right atrial pressure over a period of days, and the cardiac output progresses from point A to point F, until death occurs.

Recovery of the heart after MI

Remodeling of the Heart

The heart undergoes continual remodeling in response to fluctuations in functional demand (cardiac remodeling). Pathological hemodynamic overloading (e.g. hypertension and myocardial infarction) and unloading (e.g. prolonged bed rest and ventricular assist device) induce pathological hypertrophy and atrophy respectively.



HF and Your Ejection Fraction Explained

RISE ABOVE Heart Failure

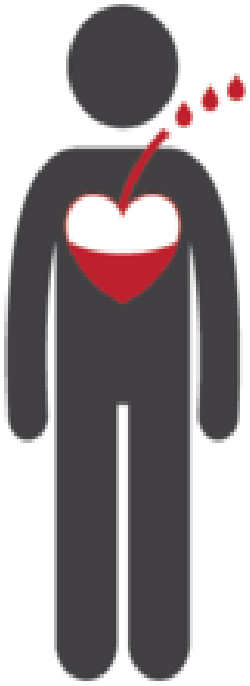
The Ejection Fraction compares the **amount of blood in the heart** to the **amount of blood pumped out.** The fraction or percentage helps describe how well the heart is pumping blood to the body.

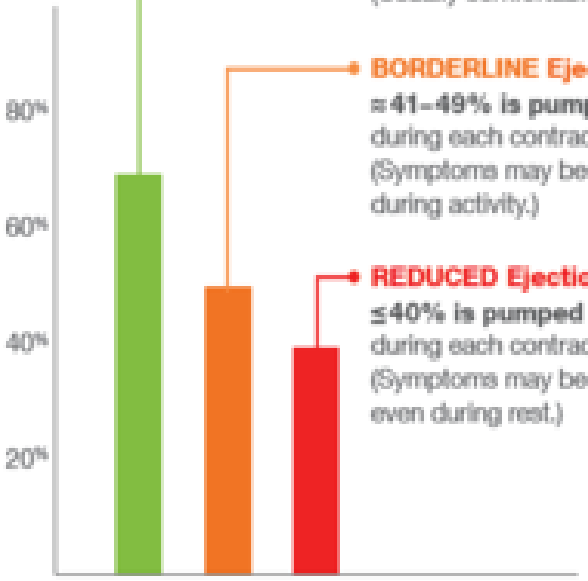
EJECTION FRACTION =

amount of blood **pumped out**

amount of blood **in chamber**

How much blood is pumped out?





NORMAL Ejection Fraction
≈50–70% is pumped out during each contraction
(Usually comfortable during activity)

BORDERLINE Ejection Fraction
≈41–49% is pumped out during each contraction
(Symptoms may become noticeable during activity.)

REDUCED Ejection Fraction
≤40% is pumped out during each contraction
(Symptoms may become noticeable even during rest.)

It is also possible to have a diagnosis of heart failure with a seemingly normal (or *preserved*) ejection fraction of greater than or equal to 50%.

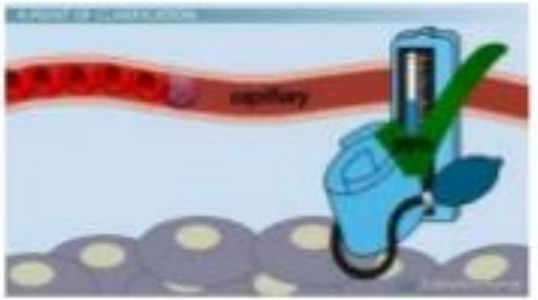
With the proper care and treatment, many patients are able to improve their ejection fraction and live a longer and healthier life. Talk with your healthcare provider about your options.

Compensatory mechanisms in heart failure

(1) Cardiac compensation

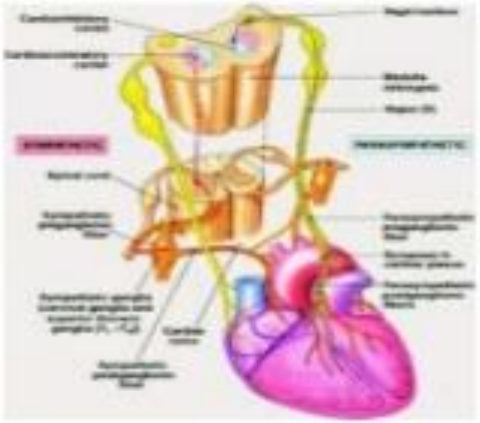
- increased HR and cardiac contractility
- Cardiac dilatation (The Frank-Starling mechanism)
- Myocardial hypertrophy

(2) Systemic compensation



- Increase the blood volume
- Redistribution of blood flow
- Increase of erythrocytes
- Increased ability of tissues to utilize oxygen

(3) neurohormonal compensation



- Sympathetic nervous system
- Renin-angiotensin system
- Atrial natriuretic peptide; endothelin

Treatment of Decompensation

- Decompensation process can be stopped by:

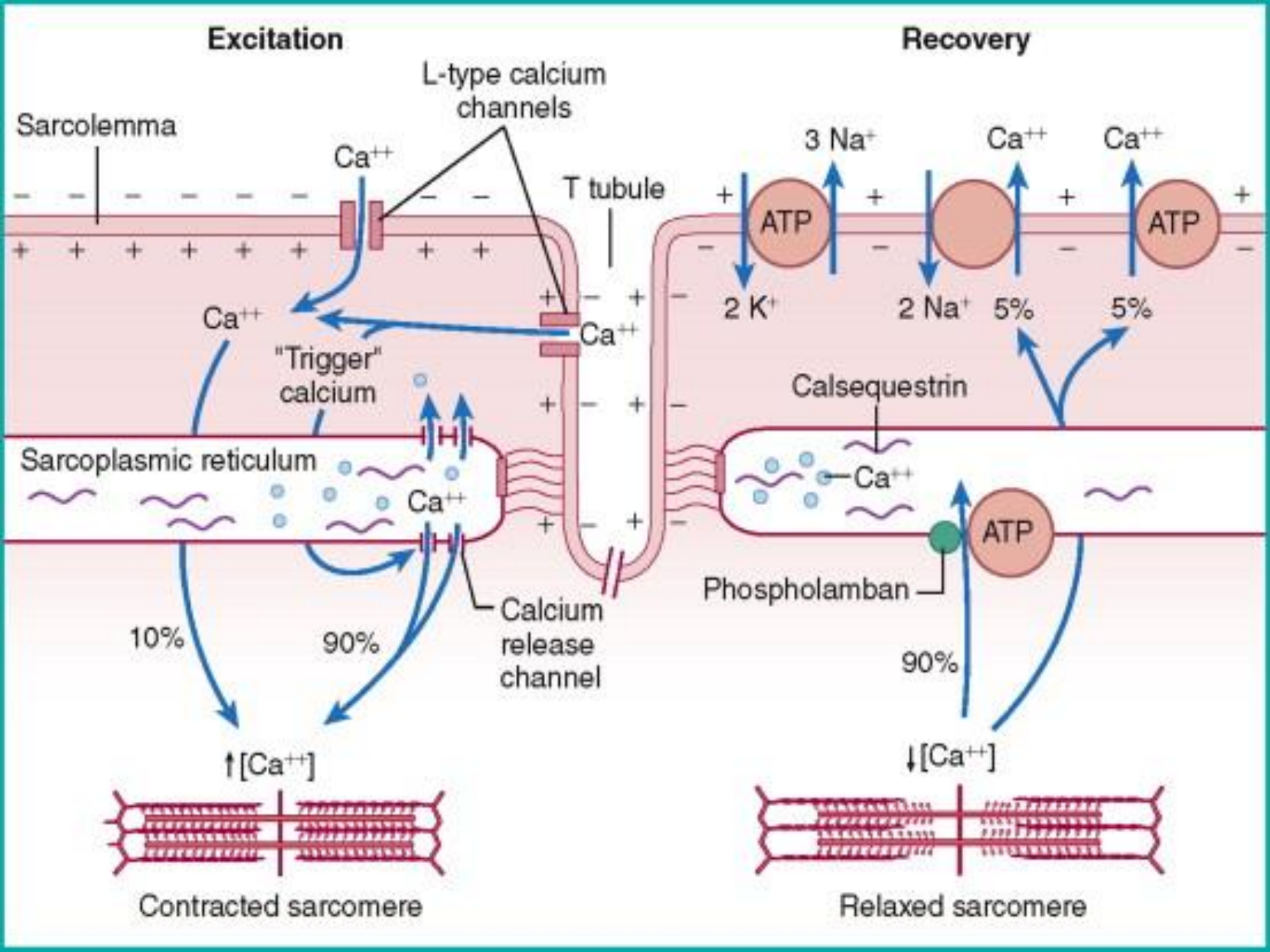
1. Strengthening of the heart

2. Administering diuretic drugs

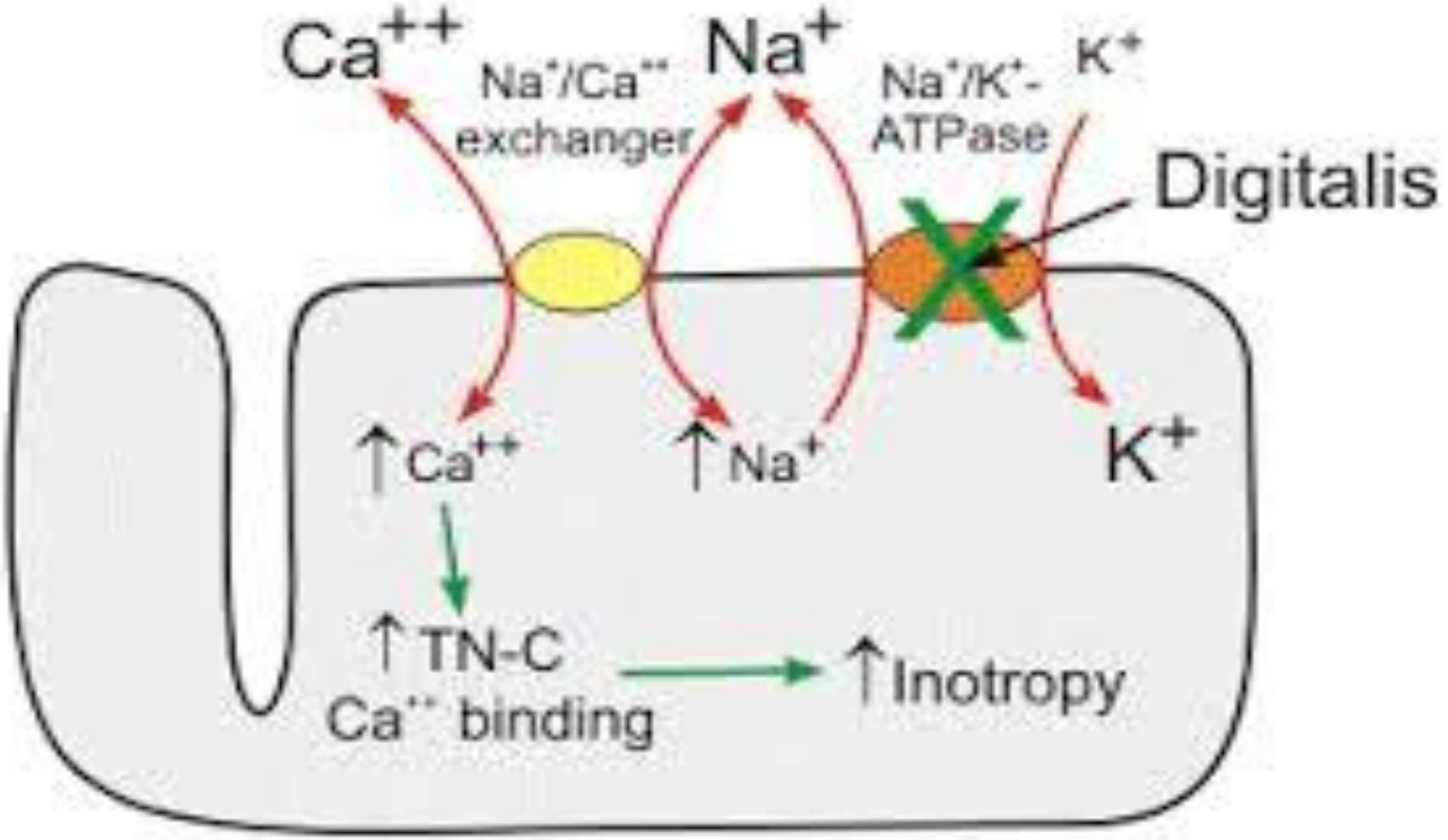
TREATING CONGESTIVE HEART FAILURE

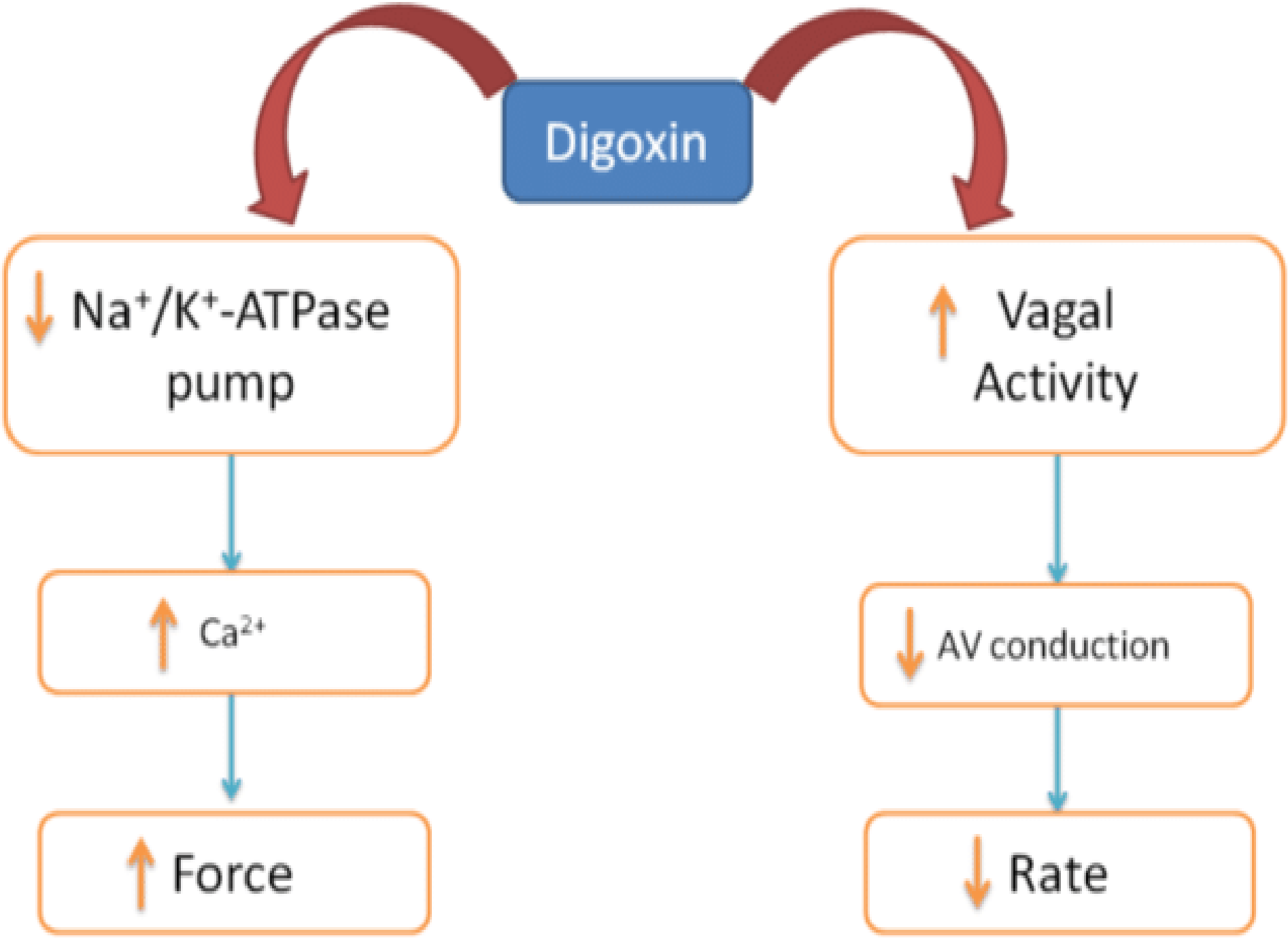
- Upright Position
- Nitrates
- Lasix
- Oxygen
- ACE Inhibitors
- Digoxin

- Fluids (Decrease)
- Afterload (Decrease)
- Sodium Restriction
- Test (Dig Level, ABGs, Potassium Level)


Excitation
Recovery
L-type calcium channels
Sarcolemma
Ca++
T tubule
3 Na+
Ca++
Ca++
ATP
ATP
2 K+
2 Na+
5%
5%
Ca++
Ca++
"Trigger" calcium
Calsequestrin
Sarcoplasmic reticulum
Ca++
Ca++
ATP
Phospholamban
Calcium release channel
10%
90%
90%
↑[Ca++]
↓[Ca++]
Contracted sarcomere
Relaxed sarcomere

MECHANISM OF ACTION OF DIGOXIN




Digoxin
↓ Na+/K+-ATPase pump
↑ Ca2+
↑ Force
↑ Vagal Activity
↓ AV conduction
↓ Rate

Types of Heart failure

- **Unilateral Cardiac Failure:**
  Only one side of the heart is failed it may be:

1.Right HF due to congenital heart disease on right side

2.Left HF due to coronary thrombosis

- **Bilateral Cardiac Failure:**

  Both sides of the heart are not functioning

Unilateral Left heart failure:

When only left side fails --- blood continue to be pumped into lungs --- blood is dammed up in pulmonary circulation --- mean pulmonary filling pressure increase

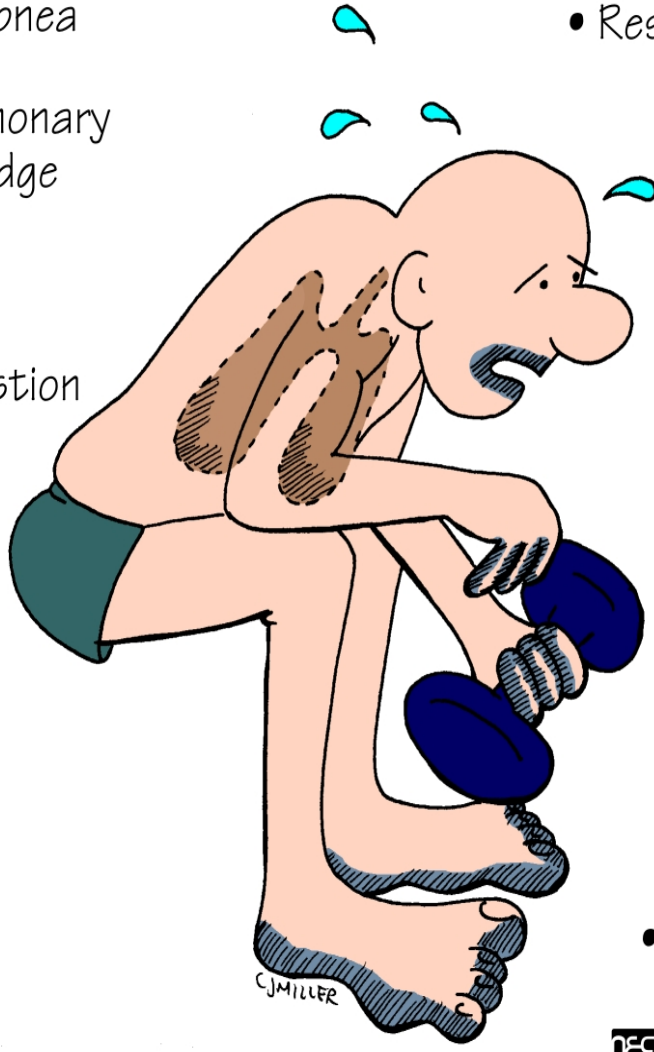
Results:

Pulmonary vessels enlarge --- pulmonary vascular Congestion

Pulmonary capillary pressure increase --- fluid leaks from pulmonary capillary into interstitial space --- pulmonary edema

LEFT SIDED ♥ FAILURE

- Paroxysmal Nocturnal Dyspnea
- Elevated Pulmonary Capillary Wedge Pressure
- Pulmonary Congestion
  - Cough
  - Crackles
  - Wheezes
  - Blood-Tinged Sputum
  - Tachypnea



- Restlessness
- Confusion
- Orthopnea
- Tachycardia
- Exertional Dyspnea
- Fatigue
- Cyanosis

Unilateral Right heart failure:

When only right side fails --- blood is not pumped adequately
from systemic circulation into lungs

Acute Effects:

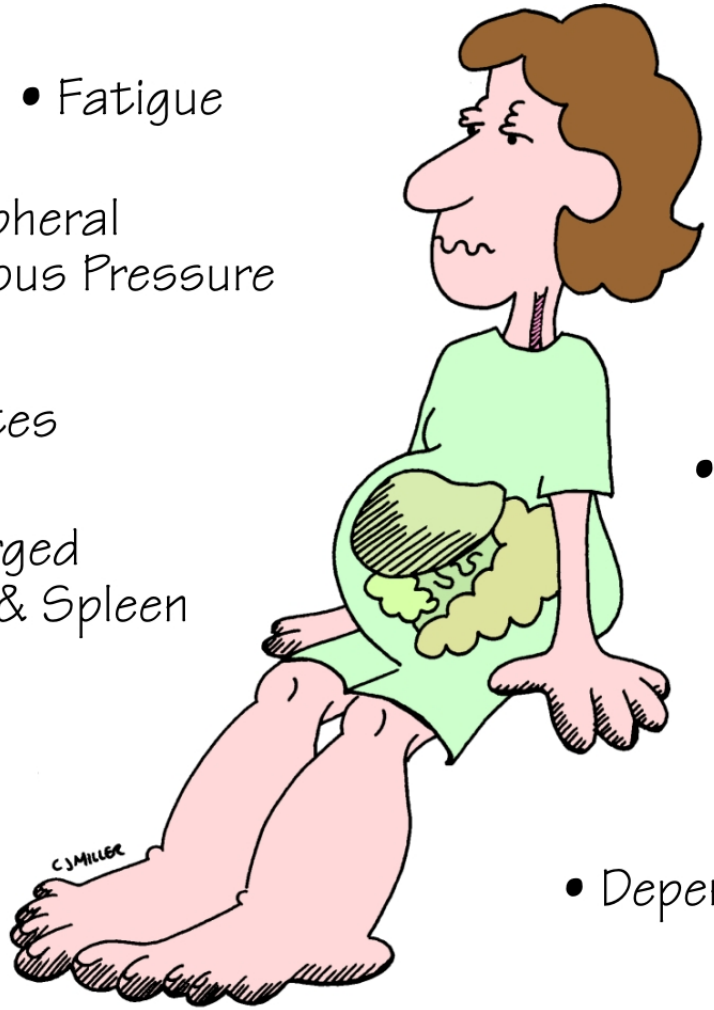
Dec CO
No significant systemic congestion & edema

Chronic Effects:

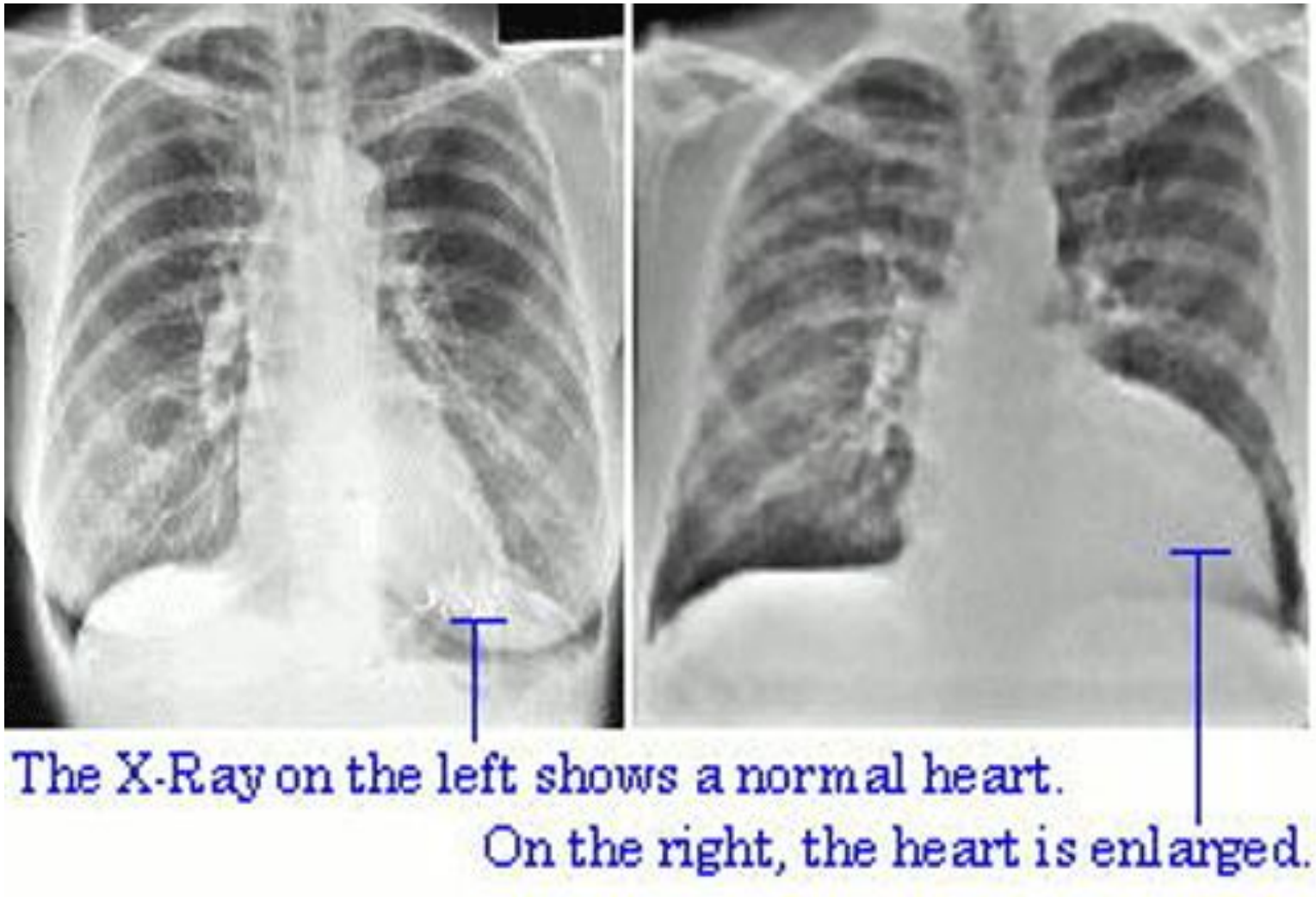
Dec CO --- fluid retention by kidneys
Progressive development of systemic congestion & edema

RIGHT SIDED ♥ FAILURE

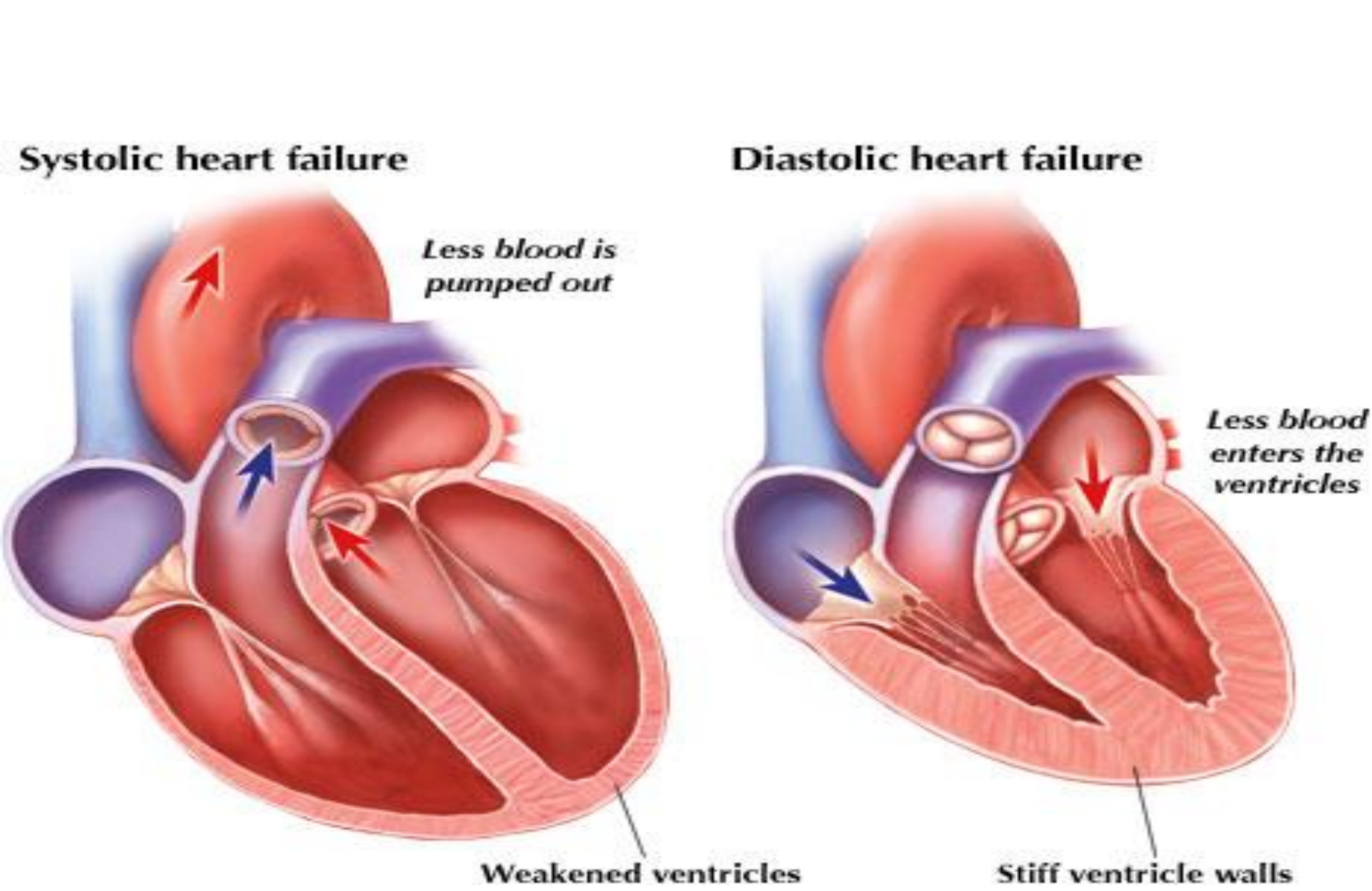
(Cor Pulmonale)



- Fatigue
- ↑ Peripheral Venous Pressure
- Ascites
- Enlarged Liver & Spleen
- May be secondary to chronic pulmonary problems
- Distended Jugular Veins
- Anorexia & Complaints of GI Distress
- Swelling in Hands & Fingers
- Dependent Edema



Cardiomegaly/ventricular remodeling **occurs as heart overworked> changes in size, shape, and function of heart after injury to left ventricle. Injury due to acute myocardial infarction or due to causes that inc. pressure/volume overload as in *Heart failure***


Systolic heart failure
Less blood is pumped out
Weakened ventricles
Diastolic heart failure
Less blood enters the ventricles
Stiff ventricle walls

High CO Failure or Over Loading of Heart:

Inc venous return that can not be pumped by the heart

Caused by:

Dec total peripheral resistance in:

1. Beriberi
2. Thyrotoxicosis
3. Atrerio – venous fistula

Low output Cardiac Failure

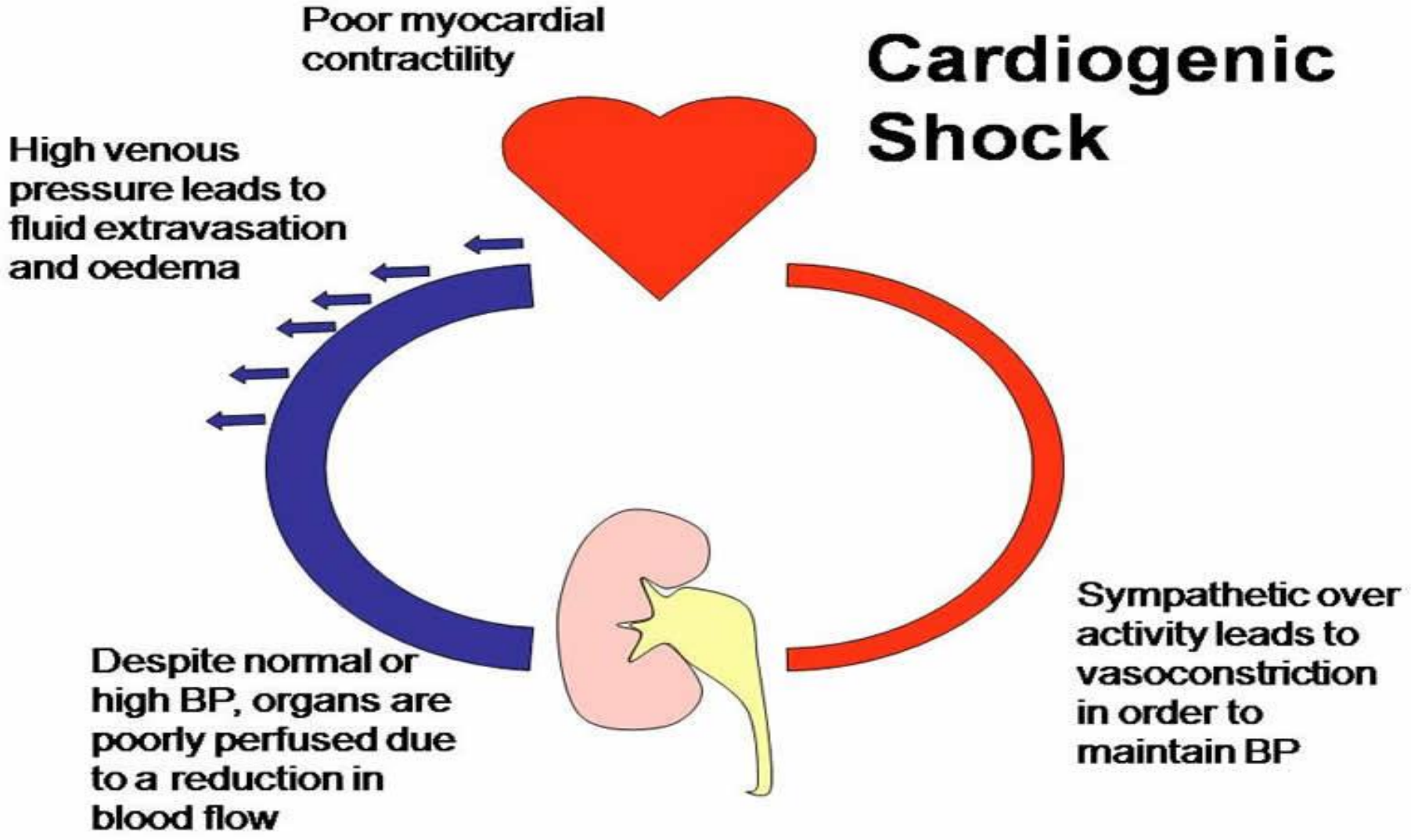
- **Cardiogenic shock** is defined as a systolic blood pressure of **less than 90 mmHg** for **at least 30 minutes**, which is secondary to myocardial dysfunction. It is associated with clinical signs of hypoperfusion, which include:
- decreased urine **output**,
- altered mental status and
- peripheral vasoconstriction

Survival Rate……..

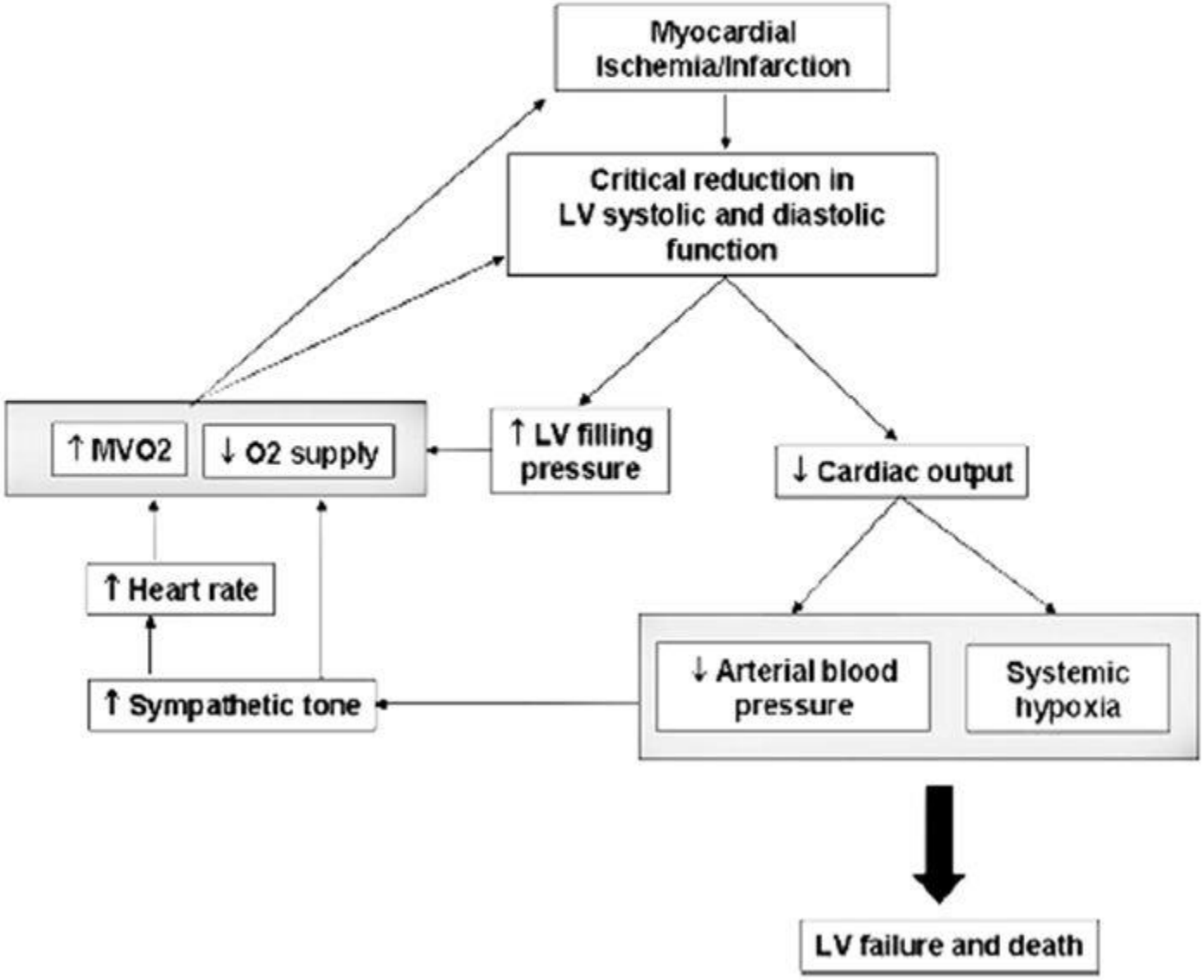
Table 1: The Diagnostic Criteria of Cardiogenic Shock

Cardiogenic Shock
Hypotension:
Systolic blood pressure <90 mmHg for >30 min, or
Vasopressors required to achieve a blood pressure ≥90 mmHg
Elevated Left Ventricular Filling Pressures:
Pulmonary congestion, or
Adequate or elevated filling pressures (wedge pressure >20 mmHg)
Signs of impaired organ perfusion (at least one of the following):
Altered mental status
Cold, clammy skin
Oliguria
Increased serum lactate levels

Pathophysiology of Cardiogenic shock



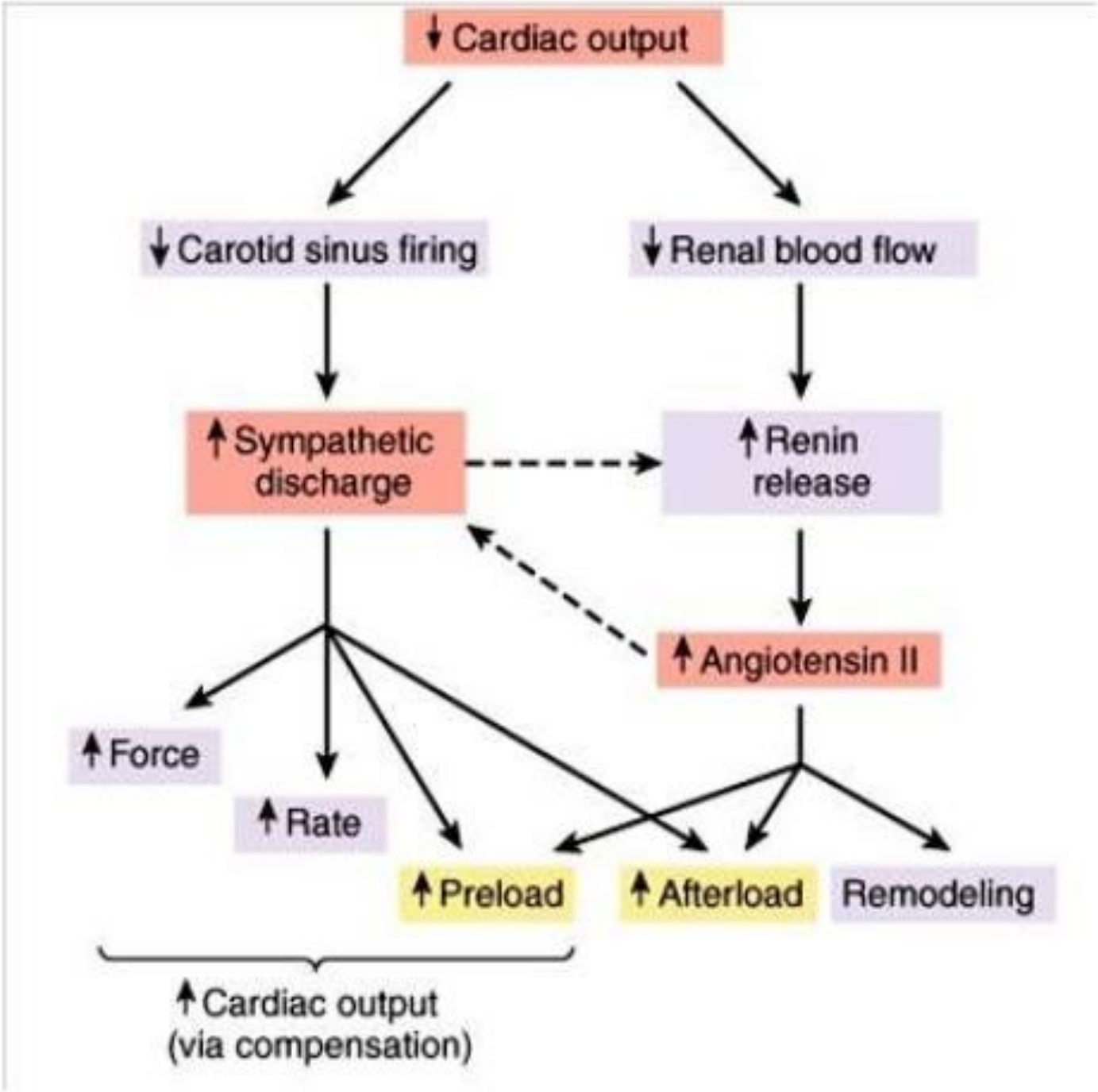
Pathophysiology diagram of cardiogenic shock



Compensation for acute cardiac failure by sympathetic reflexes:

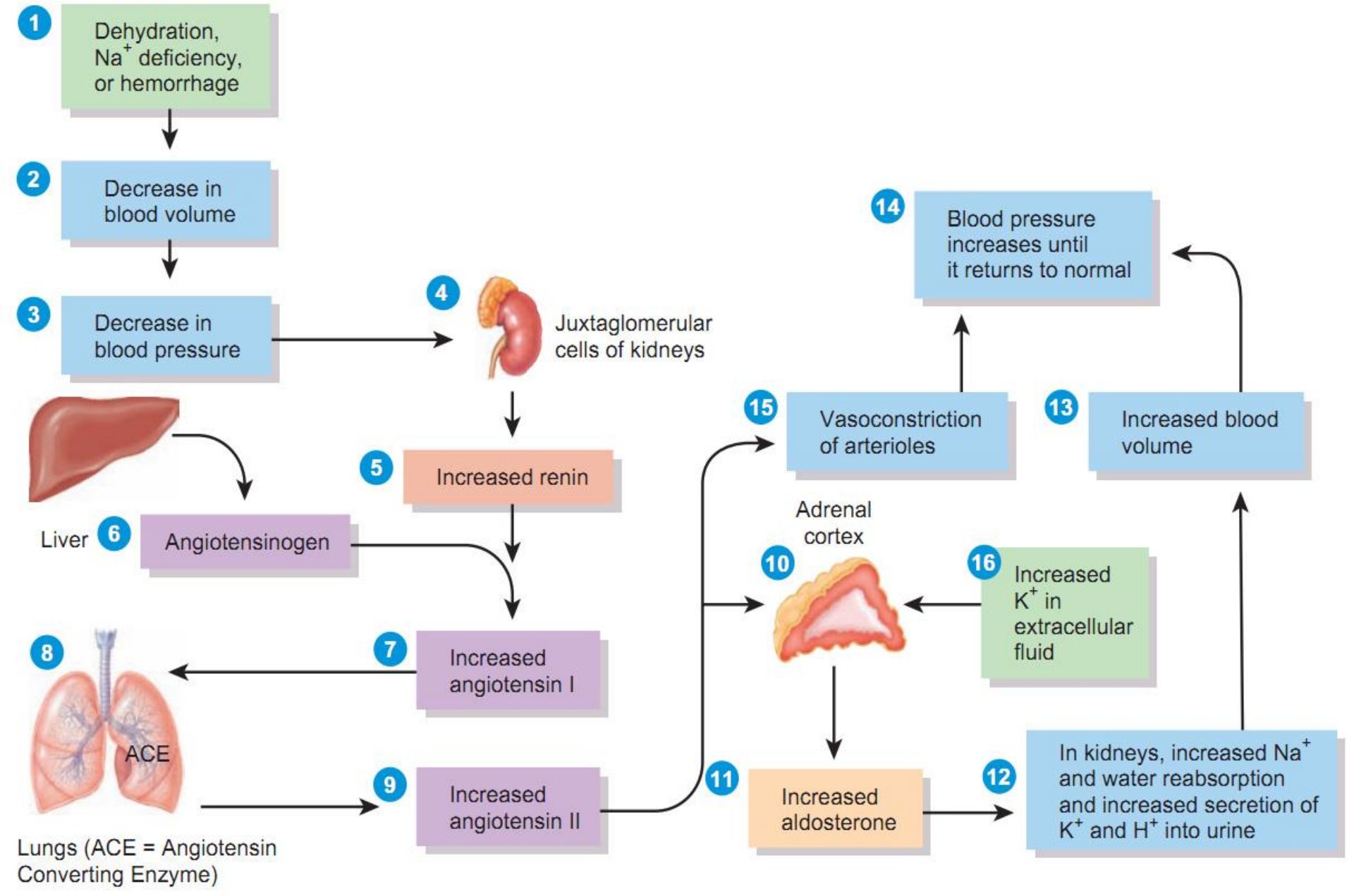
- When heart fails suddenly --- decrease CO-- baroreceptor, chemoreceptor reflexes, CNS ischemic response etc. initiated --- strong sympathetic stimulation causes:

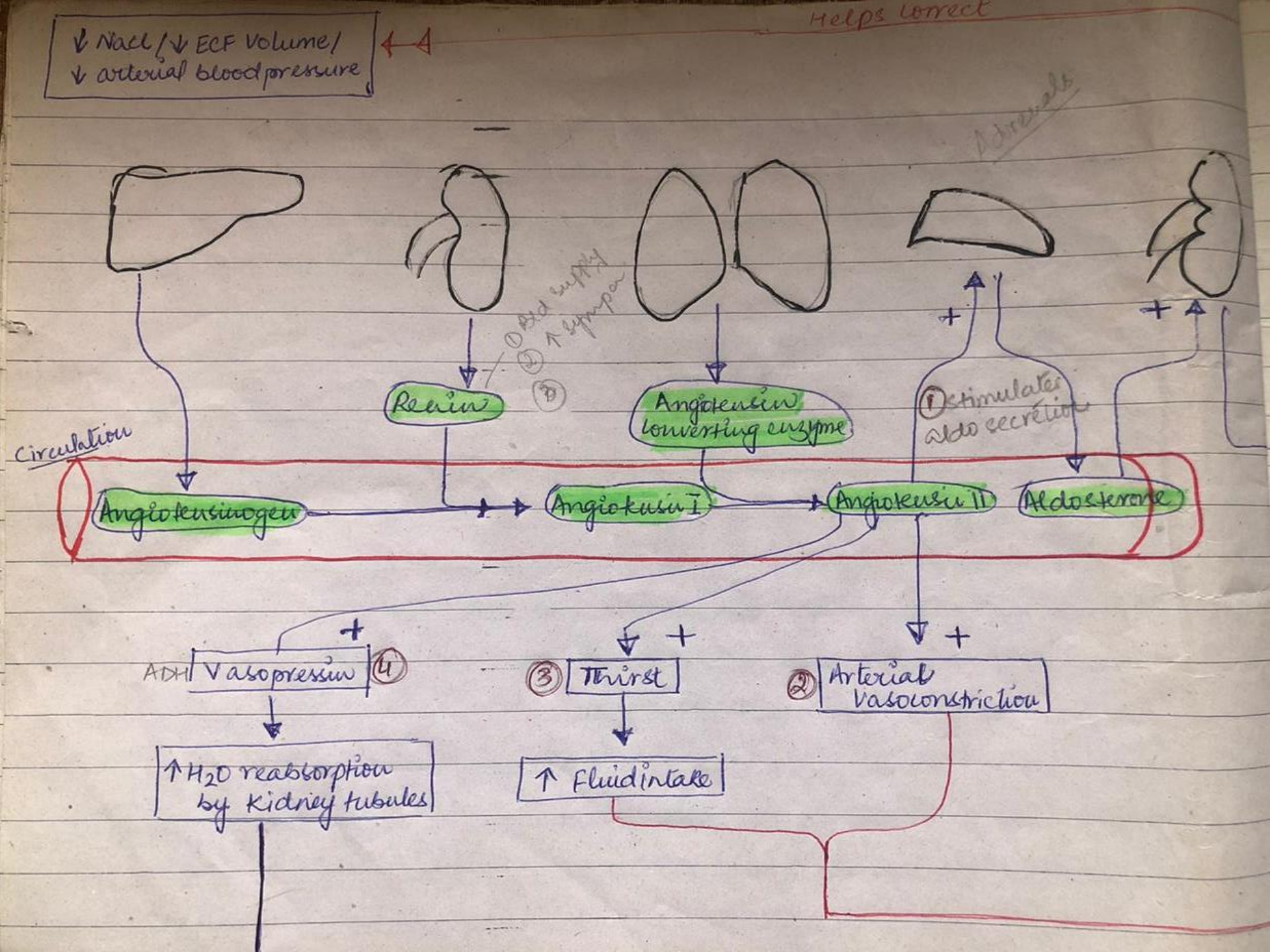
- Inc force of heart contraction
- Inc heart rate
- Inc blood vessel tone
- Inc Mean Systemic Filling Pressure
- Inc venous return
- Inc cardiac output

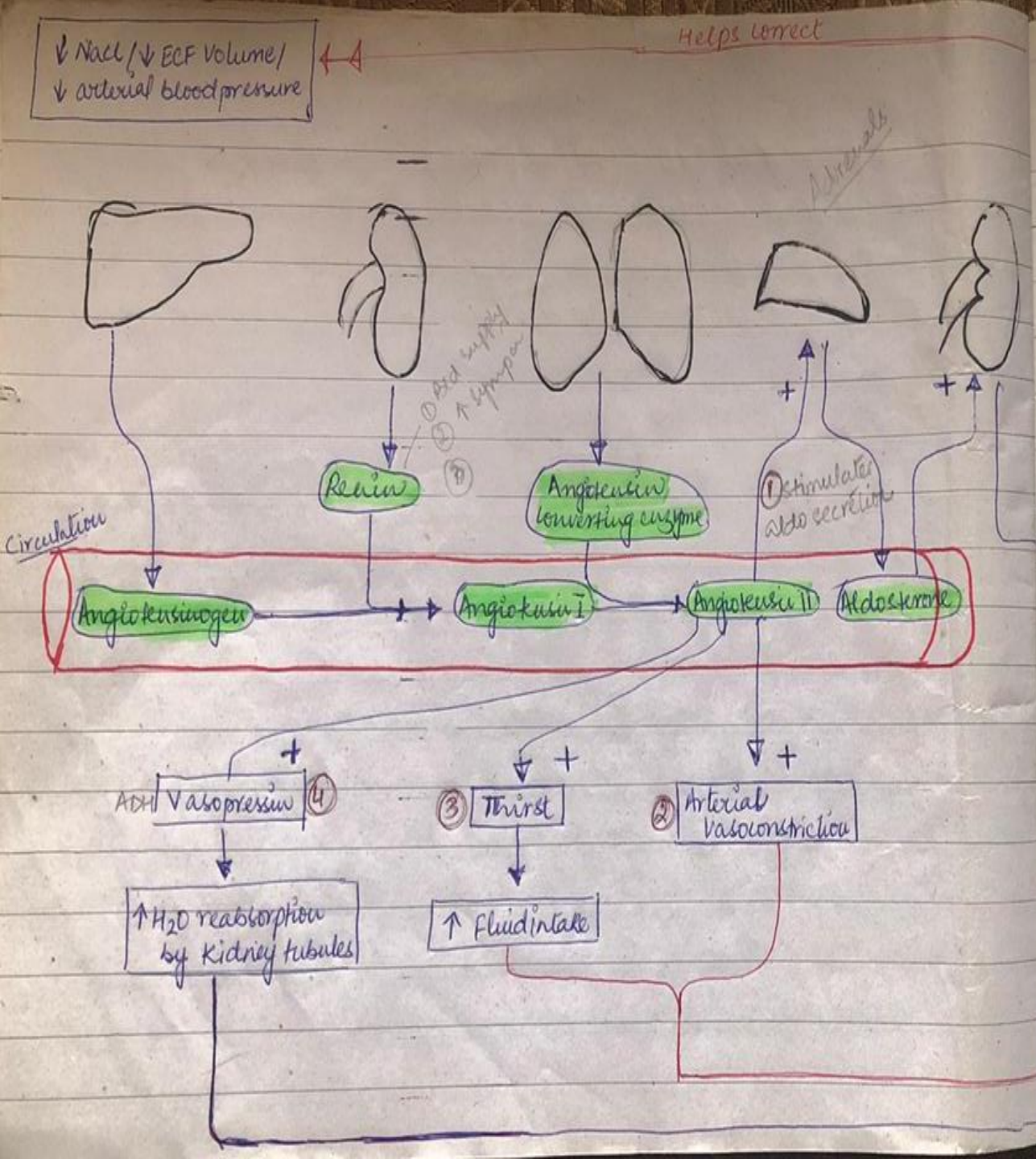

↓ Cardiac output
↓ Carotid sinus firing
↓ Renal blood flow
↑ Sympathetic discharge
↑ Renin release
↑ Angiotensin II
↑ Force
↑ Rate
↑ Preload
↑ Afterload
Remodeling
↑ Cardiac output (via compensation)

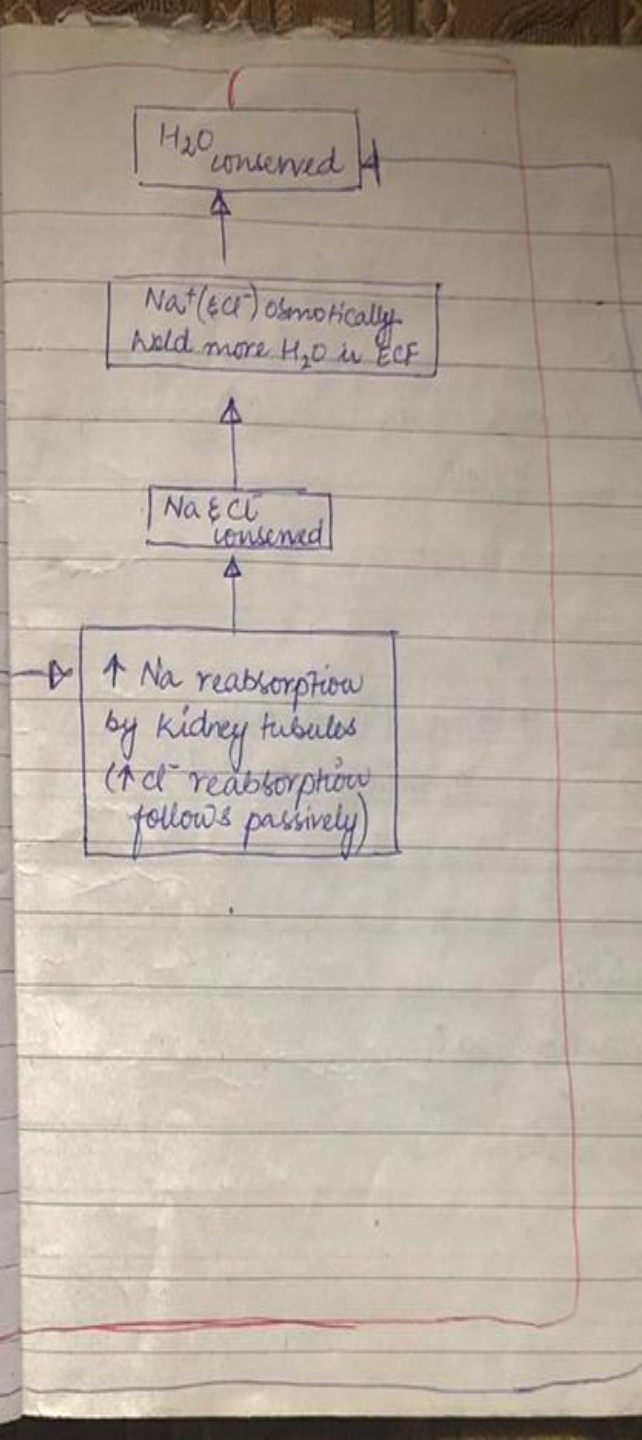
Regulation of aldosterone secretion by the renin–angiotensin–aldosterone (RAA) pathway.

Aldosterone helps regulate blood volume, blood pressure, and levels of Na^+, K^+, and H^+ in the blood.




↓ NaCl / ↓ ECF Volume /
↓ arterial blood pressure
Helps correct
Adrenals
Blood supply
Symp
Renin
Angiotensin converting enzyme
stimulates aldo secretion
Circulation
Angiotensinogen
Angiotensin I
Angiotensin II
Aldosterone
ADH
Vasopressin
Thirst
Arterial Vasoconstriction
↑ H2O reabsorption by Kidney tubules
↑ Fluid intake


↓ NaCl / ↓ ECF volume / ↓ arterial blood pressure
Helps correct
Adrenals
Renin
Angiotensin converting enzyme
Circulation
Angiotensinogen
Angiotensin I
Angiotensin II
Aldosterone
① stimulates aldo secretion
ADH
Vasopressin
④
③ Thirst
② Arterial Vasoconstriction
↑ H2O reabsorption by kidney tubules
↑ Fluid intake


H2O conserved
Na+ (& Cl−) osmotically hold more H2O in ECF
Na & Cl conserved
↑ Na reabsorption by kidney tubules (↑ Cl− reabsorption follows passively)

* Hyperkalemia → ↑ aldosterone secretion

Aldosterone ↑ renal K secretion, restoring serum K^+ to normal.

Angiotensin II, has 4 effects

① stimulates the synthesis and secretion of (adrenal cortex) aldosterone by adrenal

② It increases Na-H exchange in the proximal convuluted tubule. kidney

③ It increases thirst and therefore water intake

④ It causes vasoconstriction of the arterioles.

released as a result of ← low BP and/or low ECF Volume
- as during Hg or
- loss of salt & water
  - by excessive sweating
  - severe diarrhea

Compensated Heart Failure:

When CO is brought to normal by acute & chronic compensatory mechanism after heart failure

Characteristic:

Inc Rt Atrial pressure

Decompensated Heart Failure:

If a heart is severely damaged that sympathetic reflexes & renal fluid retention compensatory mechanisms can not bring CO to normal, so that kidneys continue to retain fluid resulting in edema & death

Characteristics:

Deterioration of myocardium

Peripheral edema in cardiac failure:

Edema in CF is caused by fluid retention by kidneys that increase MSP & capillary pressure

Causes of renal retention of fluid:

1. Dec GFR due to Dec arterial pressure and
   Intense sympathetic constriction of afferent arterioles
2. Activation of renin angiotensin system

3. Aldosterone secretion

4. ADH secretion & increase tubular reabsorption of water

Acute pulmonary edema in Chronic Heart Failure:

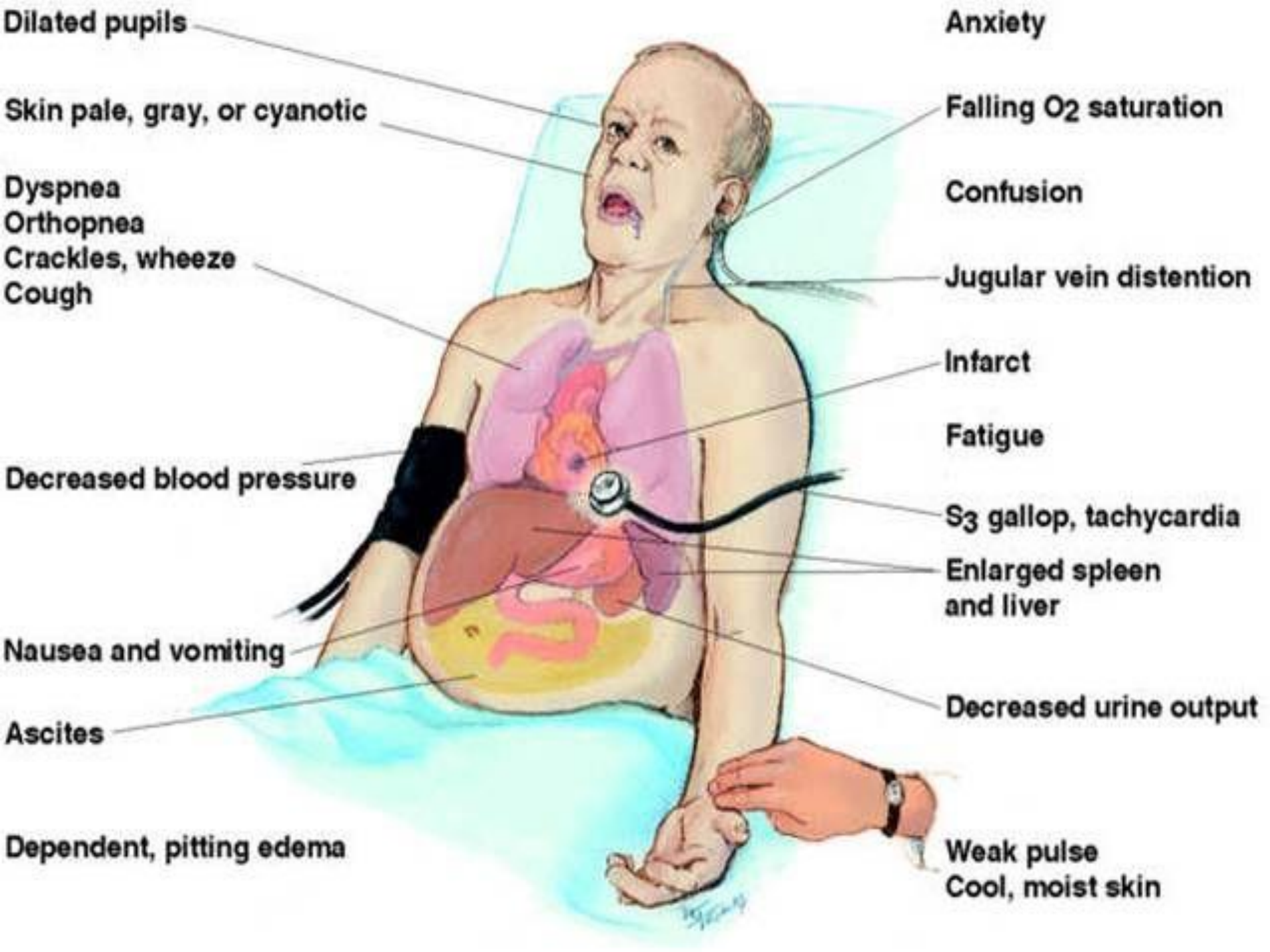
- A temporary overload on already weak left ventricle (as in exercise , emotion etc.)
- Increase venous return
- Blood dam in lungs due to limited pumping capability of left heart
- Increase blood in lungs elevate pulmonary capillary pressure
- Fluid leak into lung tissue & alveoli (pulmonary edema)

Acute Pulmonary edema in Chronic heart failure

- Decrease oxygenation of blood due to fluid in alveoli
- Decrease peripheral resistance due to decrease oxygenation of blood
- Further increase in venous return
- Further damming of blood in lungs & so on

Deterioration of Heart in Chronic Heart Failure:

- Dec blood flow to coronary system
- Subendocardial portion of myocardium dies
- Replaced by fibrous tissue
- Further weakening of heart
- Further decrease in coronary blood flow
- Vicious cycle leads to death


Dilated pupils
Skin pale, gray, or cyanotic
Dyspnea
Orthopnea
Crackles, wheeze
Cough
Decreased blood pressure
Nausea and vomiting
Ascites
Dependent, pitting edema
Anxiety
Falling O_2 saturation
Confusion
Jugular vein distention
Infarct
Fatigue
S_3 gallop, tachycardia
Enlarged spleen and liver
Decreased urine output
Weak pulse
Cool, moist skin

Cardiac Reserve:

Maximum % of CO that can increase above normal is called cardiac reserve

Value:

- Normal Adults 300-400%
- Athletes = 500-600%

Causes:
Factors that prevent the heart from pumping blood satisfactorily

Test:
Calculated by exercise test

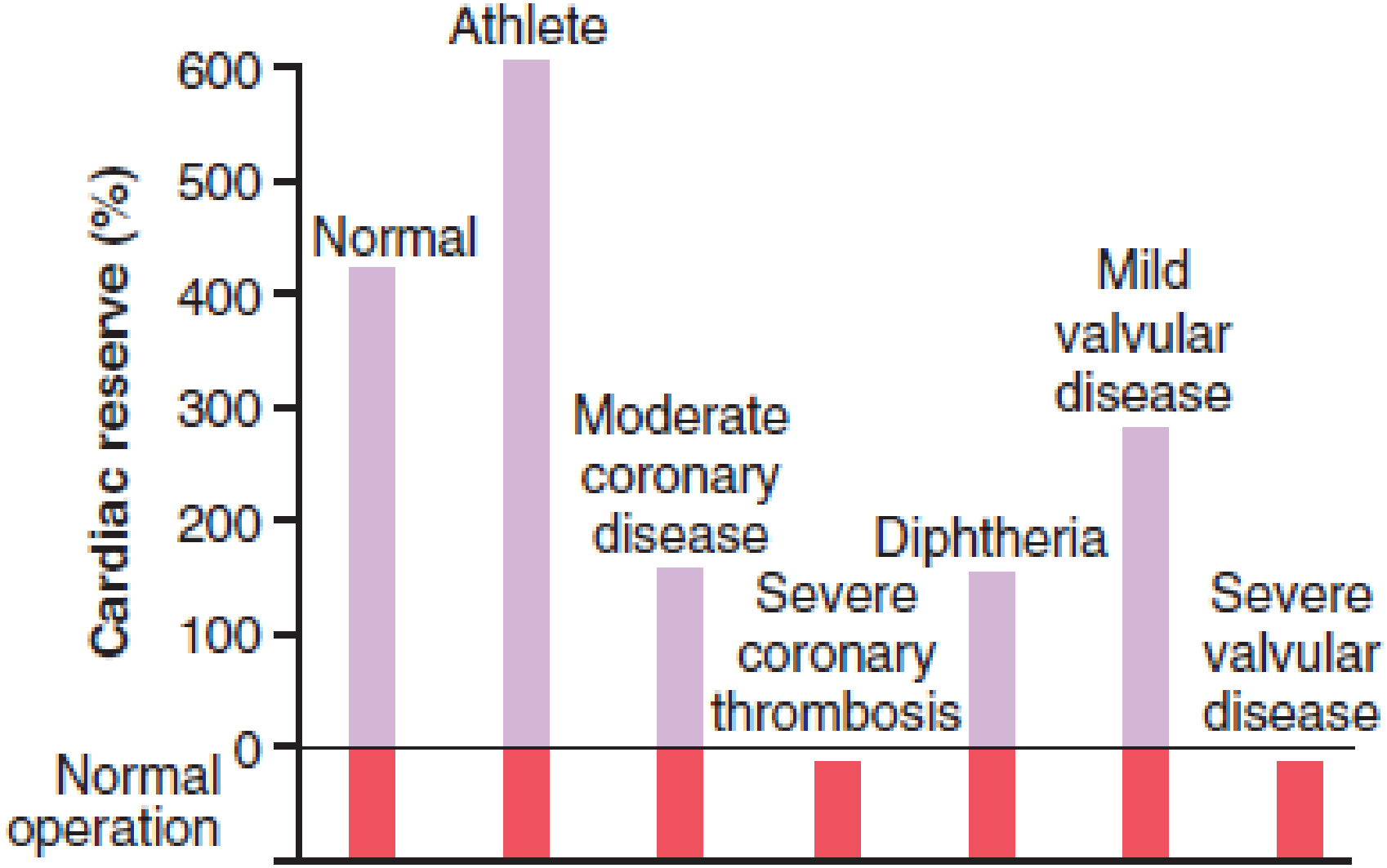
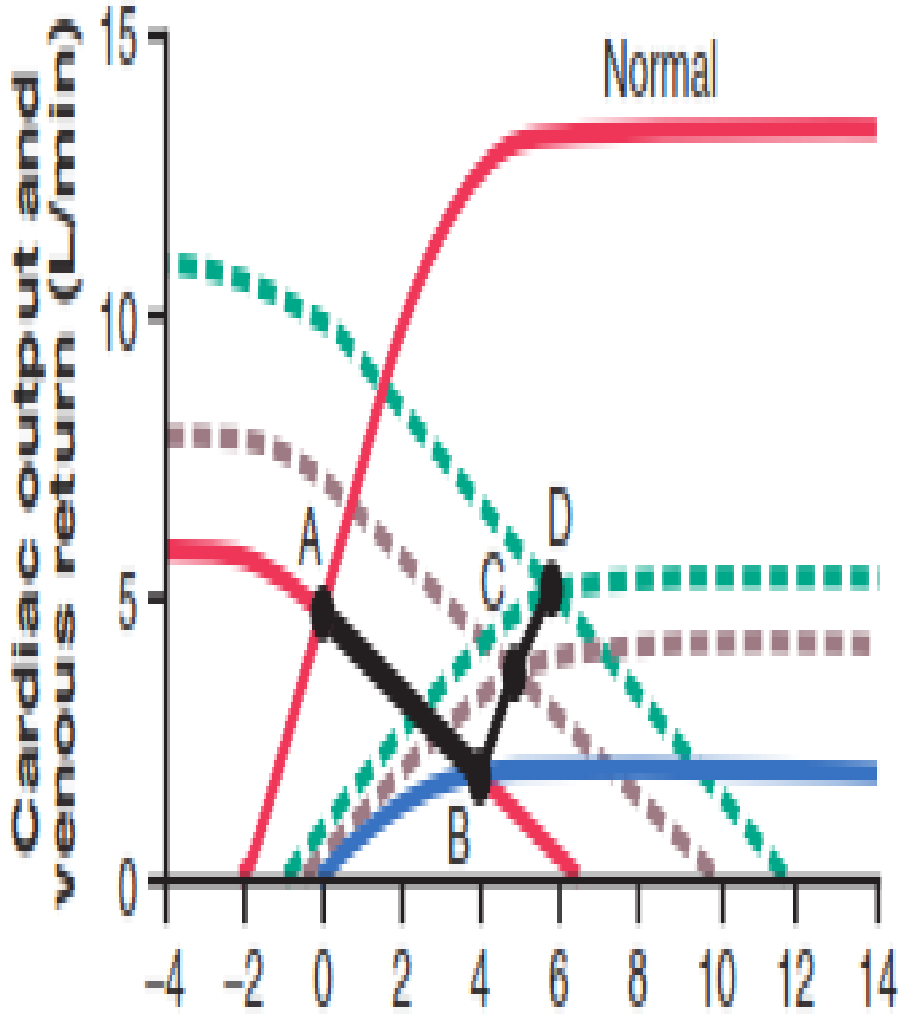


Figure 22-4. Cardiac reserve in different conditions, showing less than zero reserve for two of the conditions.

Progressive changes in CO & Rt atrial pressure

- A. Normal cardiac out put curve, normal venous return.
- B. Acute heart attack
- C. sympathetic stimulation.
- D. During ensuing weeks.



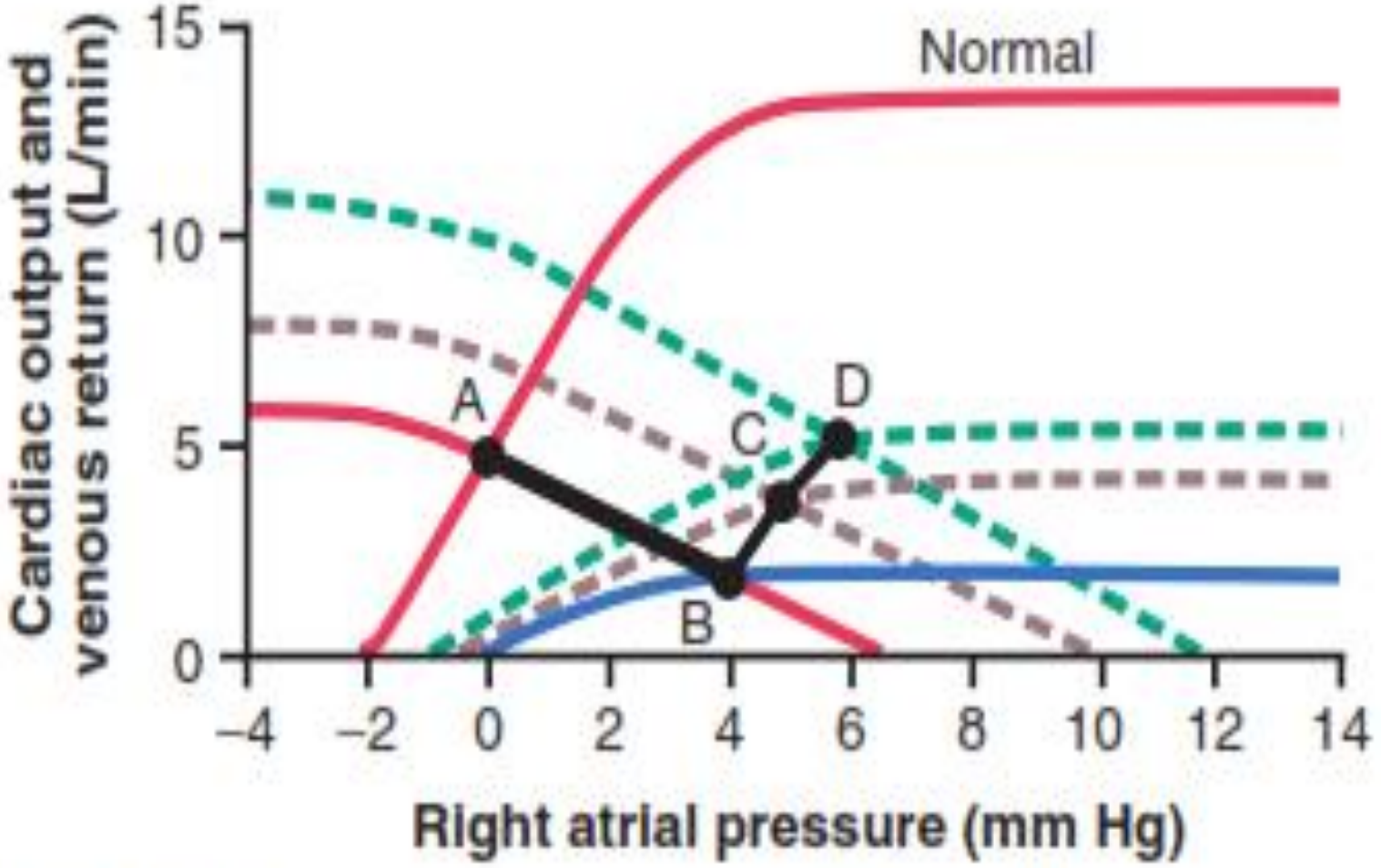


Figure 22-5. Progressive changes in cardiac output and right atrial pressure during different stages of cardiac failure.

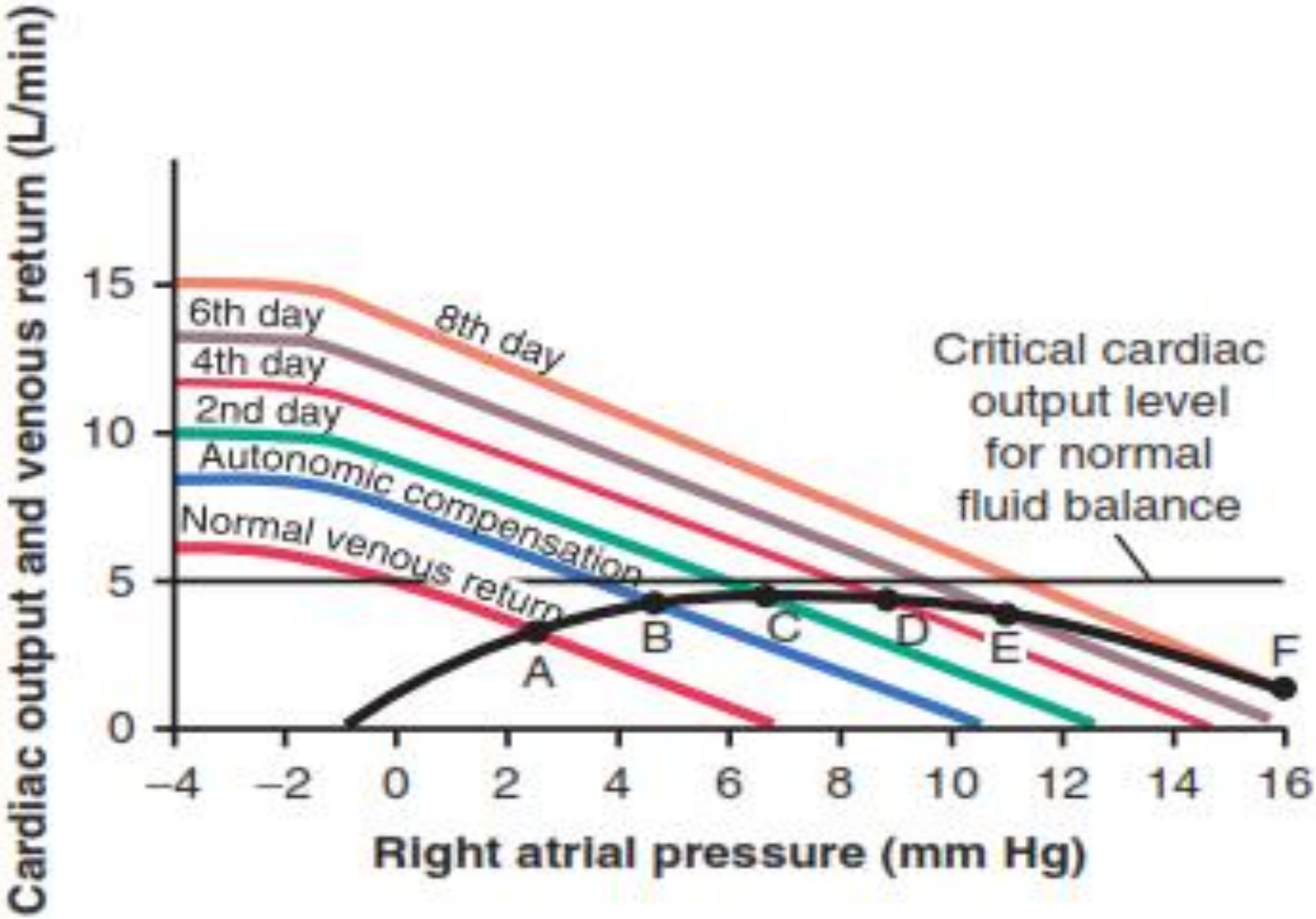


Figure 22-6. Graphical analysis of decompensated heart disease showing progressive shift of the venous return curve to the right as a result of continued fluid retention.

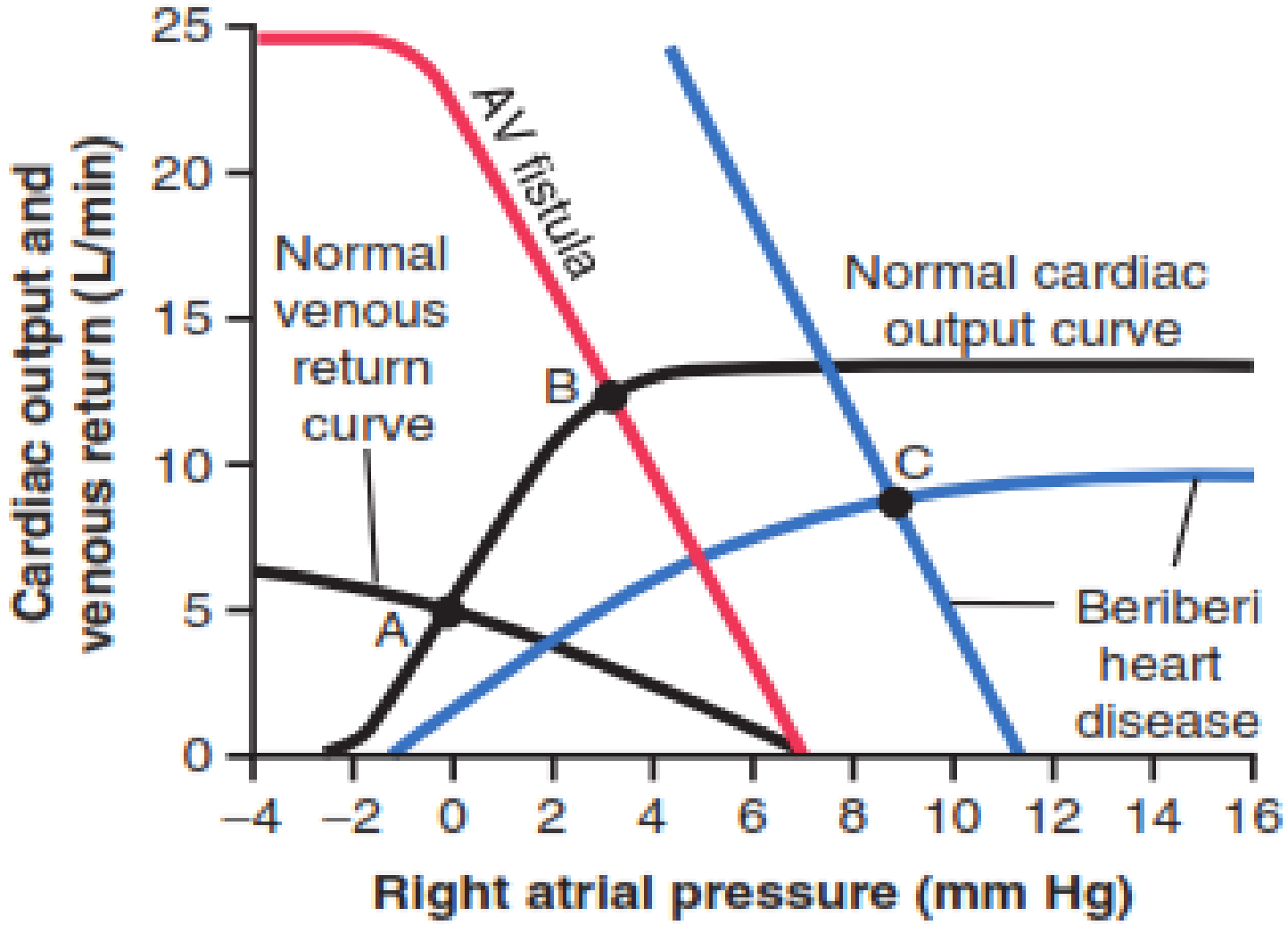
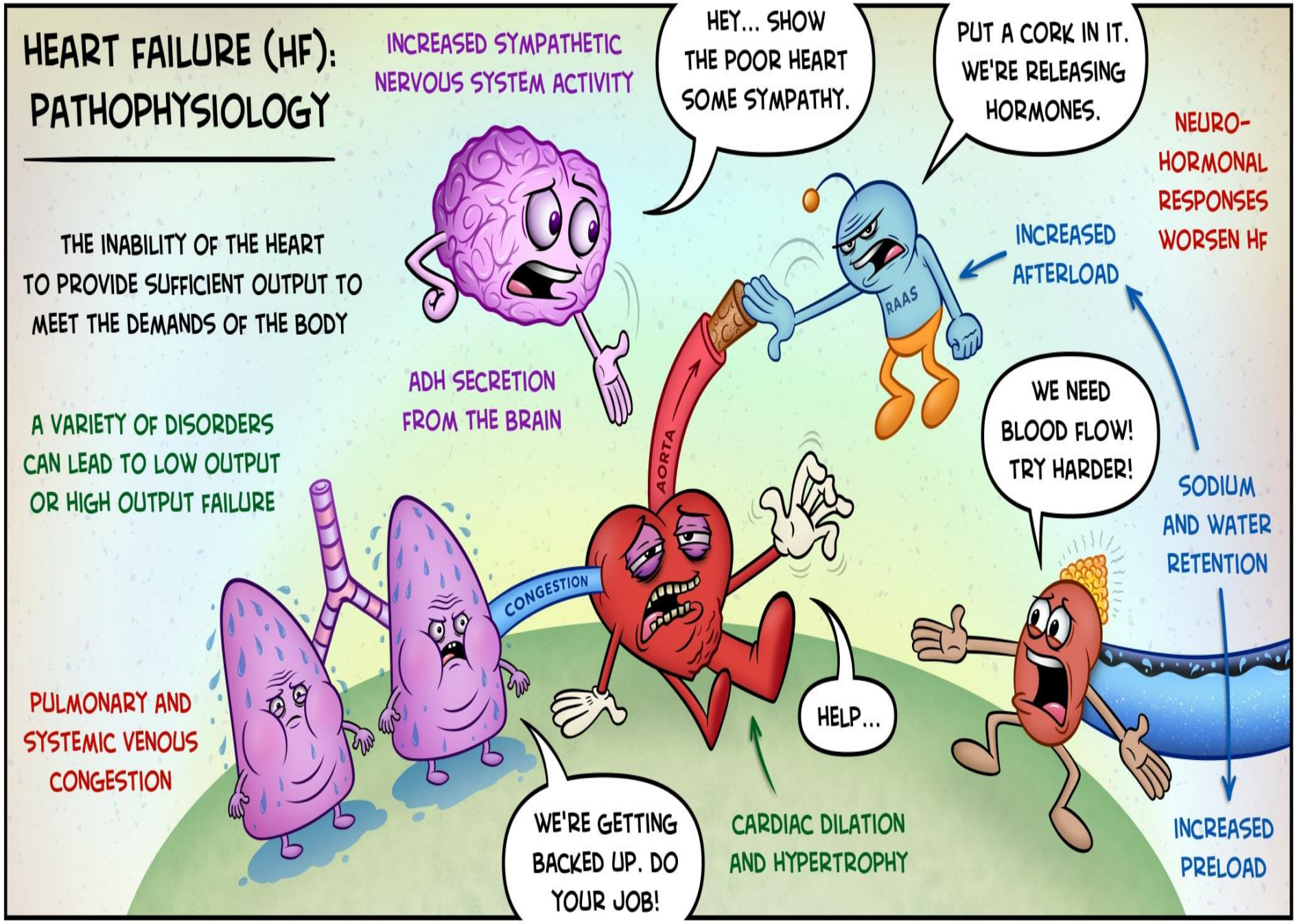


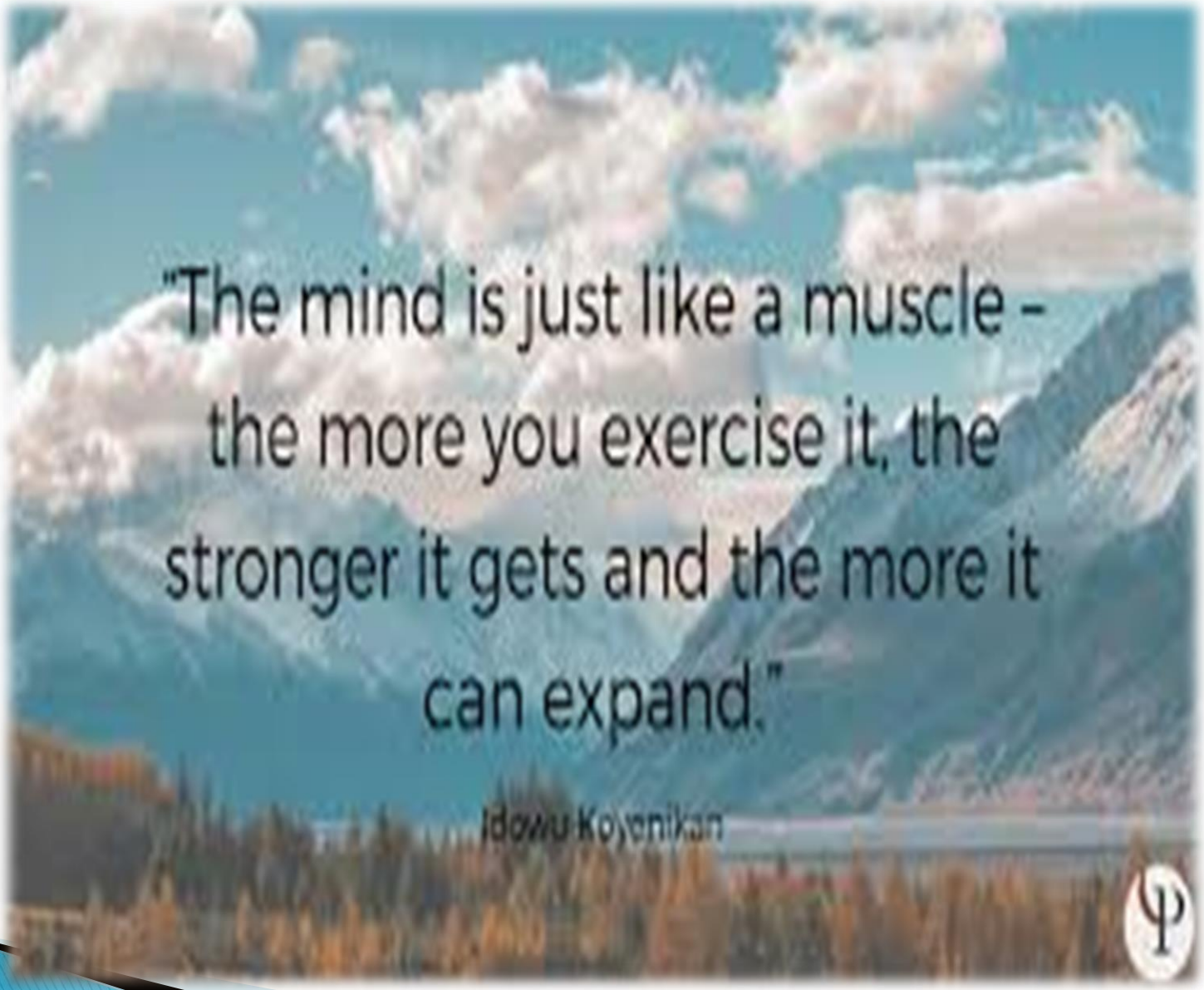
Figure 22-8. Graphical analysis of two types of conditions that can cause high-output cardiac failure: (1) arteriovenous *(AV)* fistula and (2) beriberi heart disease.

MEDICATIONS

- ↓ Fluid load, ↓ Preload, ↓ Afterload
- Improve **ACE inhibitors & Diuretics**
- ↓ Workload of **Digoxin**
  Dobutamine

"Blockers"

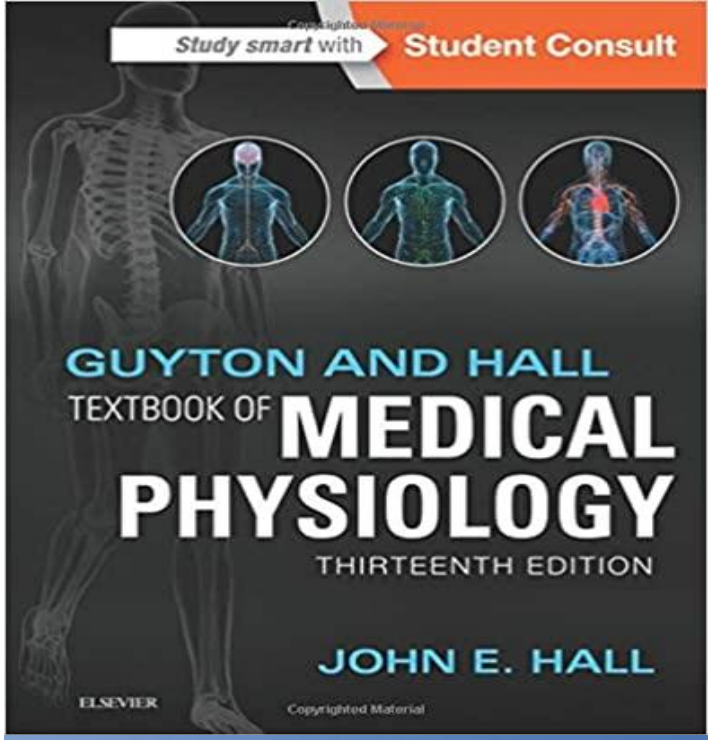


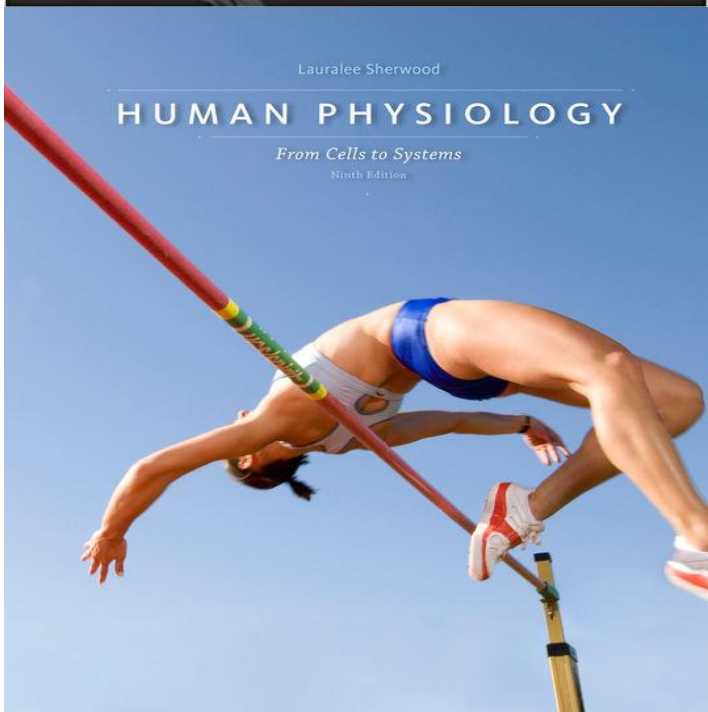

"The mind is just like a muscle –
the more you exercise it, the
stronger it gets and the more it
can expand."

References:

1. Guyton and Hall, Textbook of Medical Physiology, 13th edition.
2. Human Physiology: From Cells to Systems (Lauralee Sherwood) 9th Edition.
3. Google Images/Text

Google





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