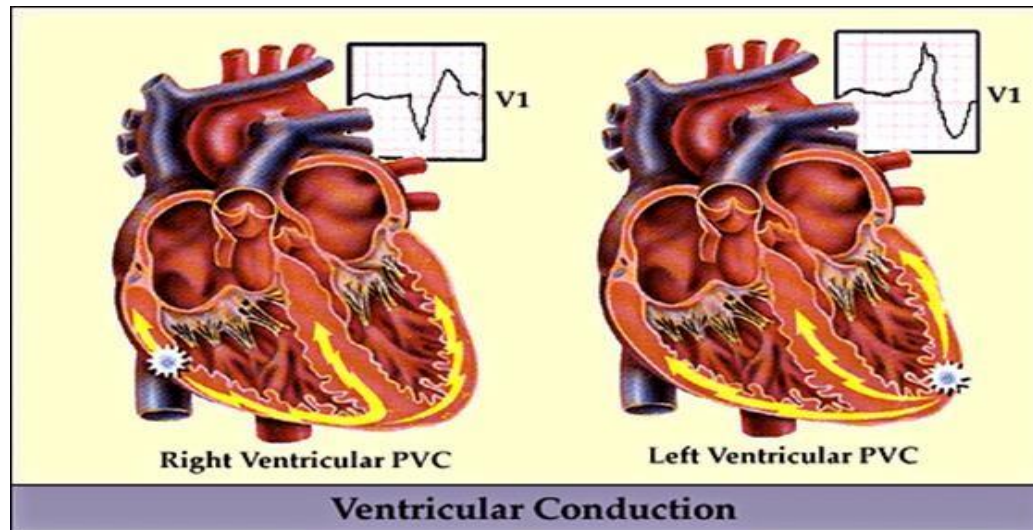


Phases of cardiac cycle, heart sounds.

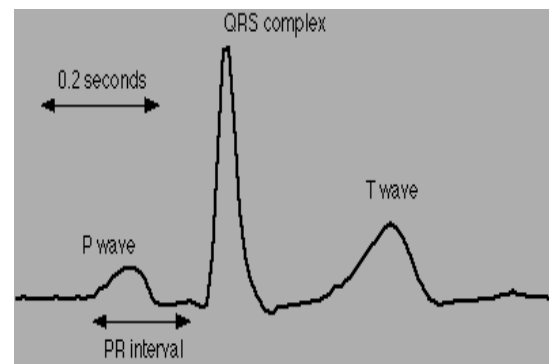


Prof. Zaporozhets
T.Viber +380972420098

Recap from Tuesday

Reading an ECG

The clinician looks for:



1. Voltage Calibration – very large QRS may indicate blockage
2. Heart Rhythm – every beat has a P, followed by QRS
3. Heart Rate – beats / minute
4. Intervals

PR Normal = 0.12-0.2 sec. Decrease = pre-excitation

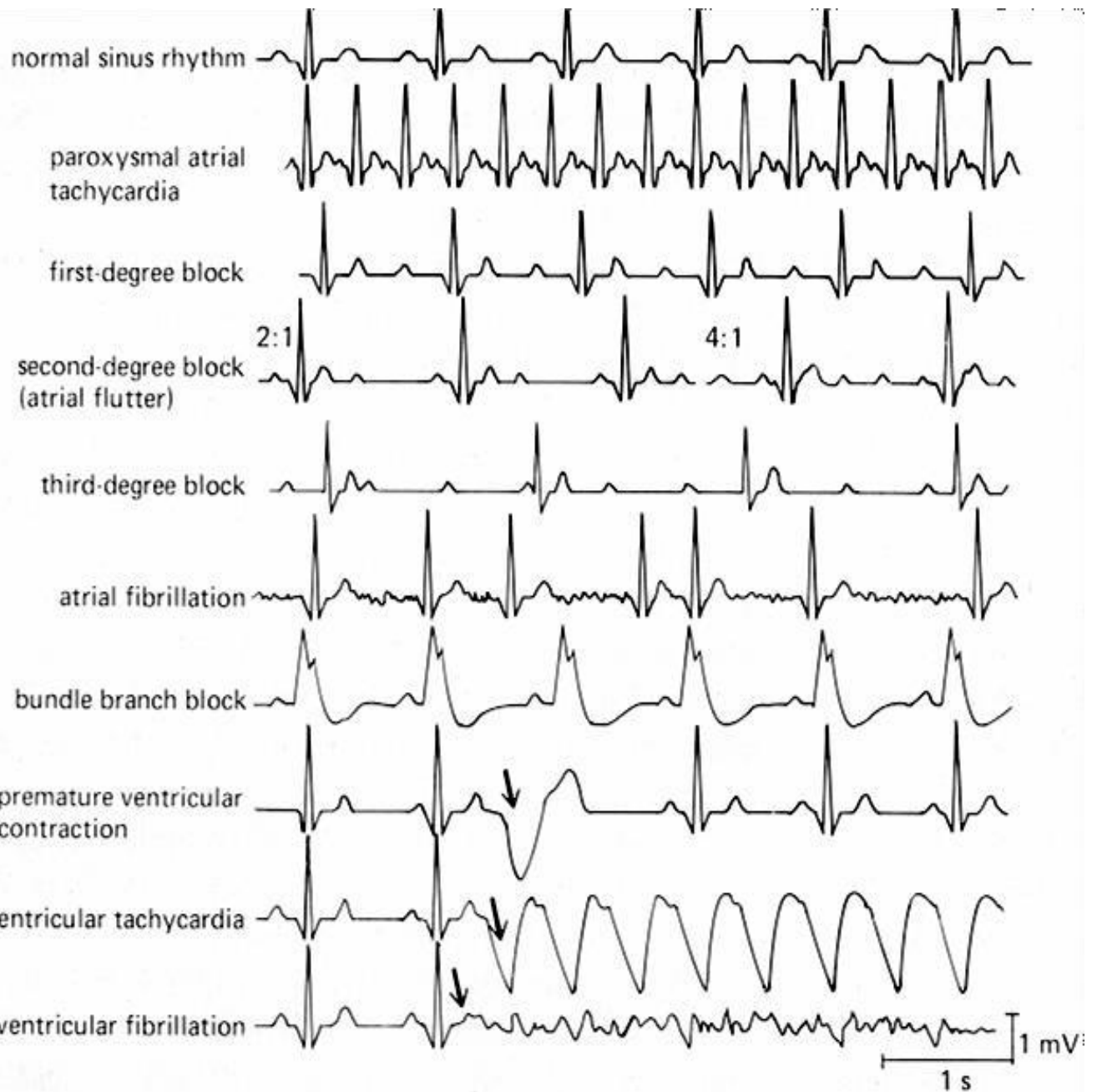
Increase = 1st degree AV block

QRS Normal \leq 0.10 sec. Increase = Bundle block, toxic drugs...

QT Normal \leq 0.44 sec. Decrease = tachycardia

Increase = hypocalcemia, MI, toxic drugs..

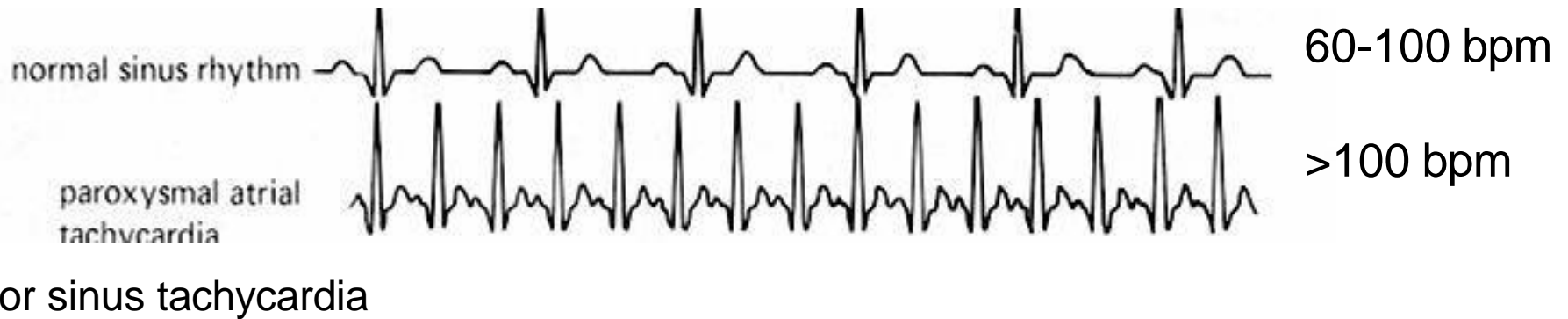
An ECG CANNOT tell about: Blood flow, Valve function, Contraction



ECG pattern summary
Follows p. 47 in Handout

Normal ECG Patterns

Normal rhythm, Normal P-R interval



A-V Node Blocks

Normal or slowed rhythm, Variable PR interval



Symptoms

- usually asymptomatic
- heart rate can be slow

Treatment

If severe bradycardia occurs, then **medications** to improve conduction, or a **pacemaker** may be used.

Bundle Branch block

Enlarged or prolonged QRS rhythm, Abnormal T wave

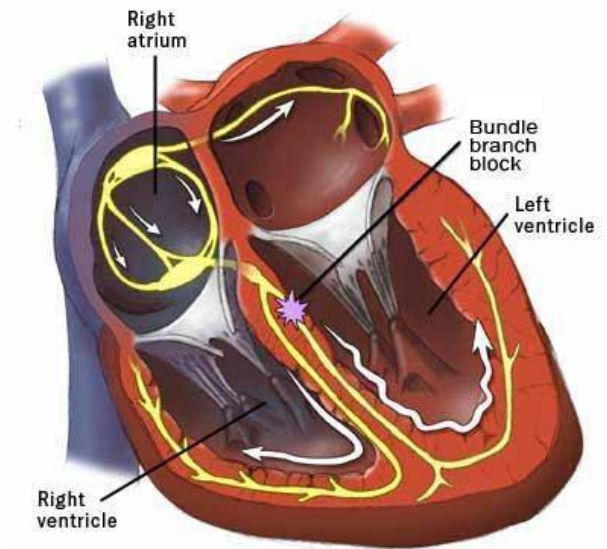


Symptoms

- usually asymptomatic
- heart rate in normal range

Treatment

A ventricular pacemaker may be used if abnormal rhythms or bradycardia occur.



Atrial Fibrillation

y

Rapid oscillating baseline, no defined P wave, Irregular QRS rhythm



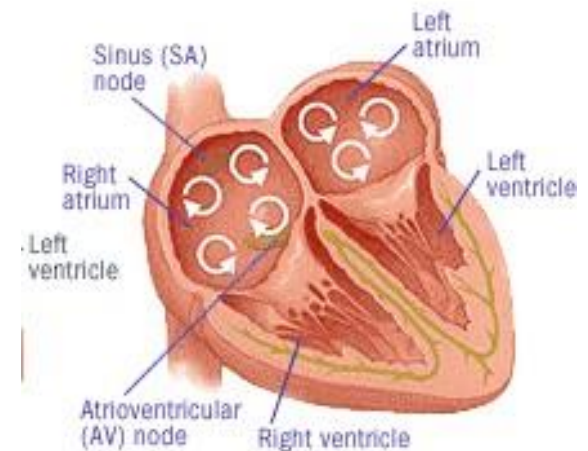
Symptoms

- irregular heart rate
- weakness, lightheadedness
- shortness of breath
- can lead to other complications

Treatments

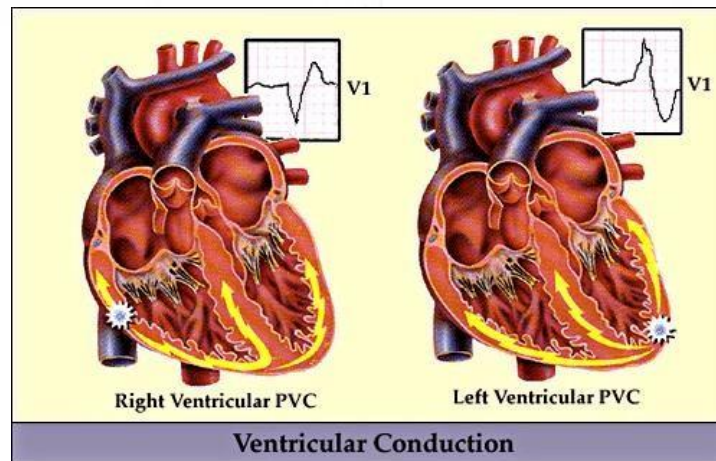
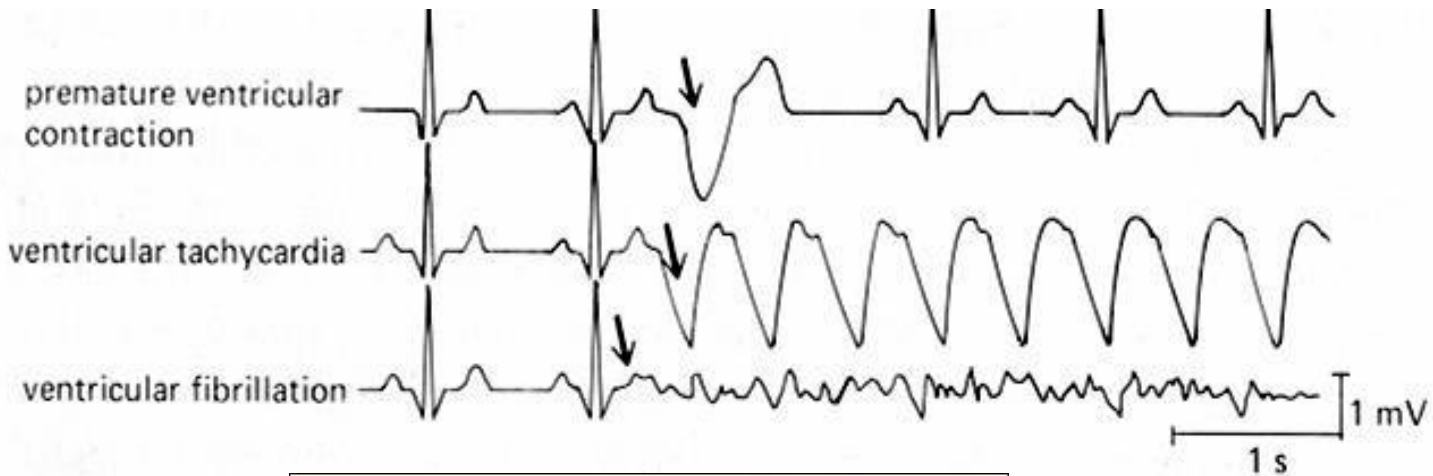
- cardioversion (restore rhythm)
- slowing heart rate (drugs)
- clot prevention (drugs)
- surgical intervention

AFib (atrial fibrillation)



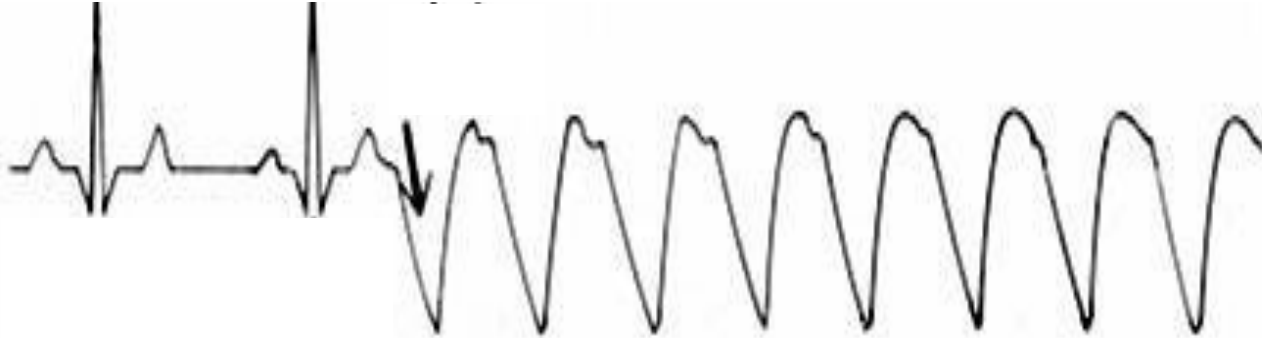
Ventricular arrhythmias

Irregular, absent, or exaggerated QRS rhythms



Ventricular tachycardia

Exaggerated, rapid QRS rhythm

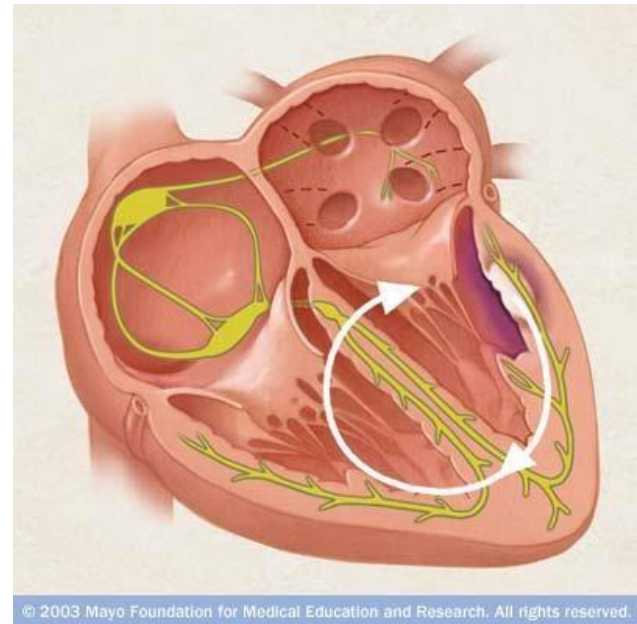


Symptoms

- unsustained <30 sec
 - palpations
 - weakness
- sustained: MEDICAL EMERGENCY
 - palpations
 - dizziness
 - fainting

Treatments

- cardioversion



Ventricular fibrillation

Exaggerated, rapid QRS rhythm

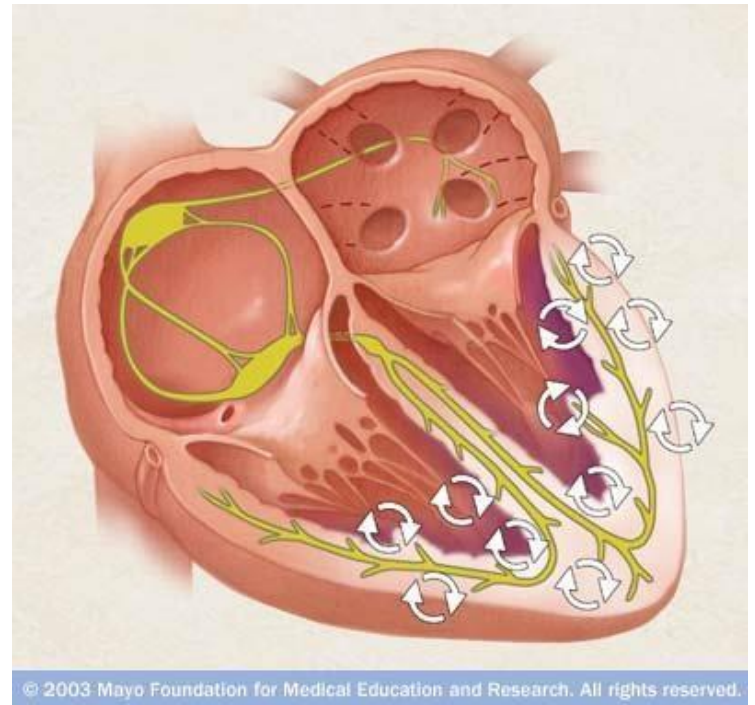


Symptoms

- loss of consciousness

Treatment:

- cardioversion



The cardiac cycle

- A. Introduction
- B. Illustration and analysis of 5 phases of the cardiac cycle
- C. Heart sounds and Abnormalities
- D. The ECG and the cardiac cycle
- E. Cardiac cycle chart

What is the “Cardiac Cycle”?

The organized, recurring sequence of atrial and ventricular depolarization, contraction, and blood flow

- **Diastole:** Ventricular relaxation and blood filling
- **Systole:** Ventricular contraction and blood ejection

Time (Diastole) > Time (Systole)

T_C = cardiac cycle length (seconds)

$$T_C = T_{\text{syst}} + T_{\text{dias}}$$

$$T_C = \mathbf{60/HR} \quad (\text{if } HR = \text{Heart Rate})$$

If $HR = 80$ bpm, then
 $T = 0.75$ sec

And if $T_{\text{syst}} = 0.25$ sec,
then $T_{\text{dias}} = 0.5$ sec

Electrical activity triggers a coordinated wave of contraction to pump blood through the heart.

General Concepts of the Cardiac Cycle

- a. The events of the ECG precede contraction of the myocardium.
- b. Contraction and relaxation of the myocardium cause large changes in pressure.
- c. Pressure changes drive fluid flow and the opening and closing of the heart valves.



ΔP

B. Phases of the Cardiac Cycle

- **Diastole** = Ventricular Relaxation (filling)
- **Systole** = Ventricular Contraction (ejection)

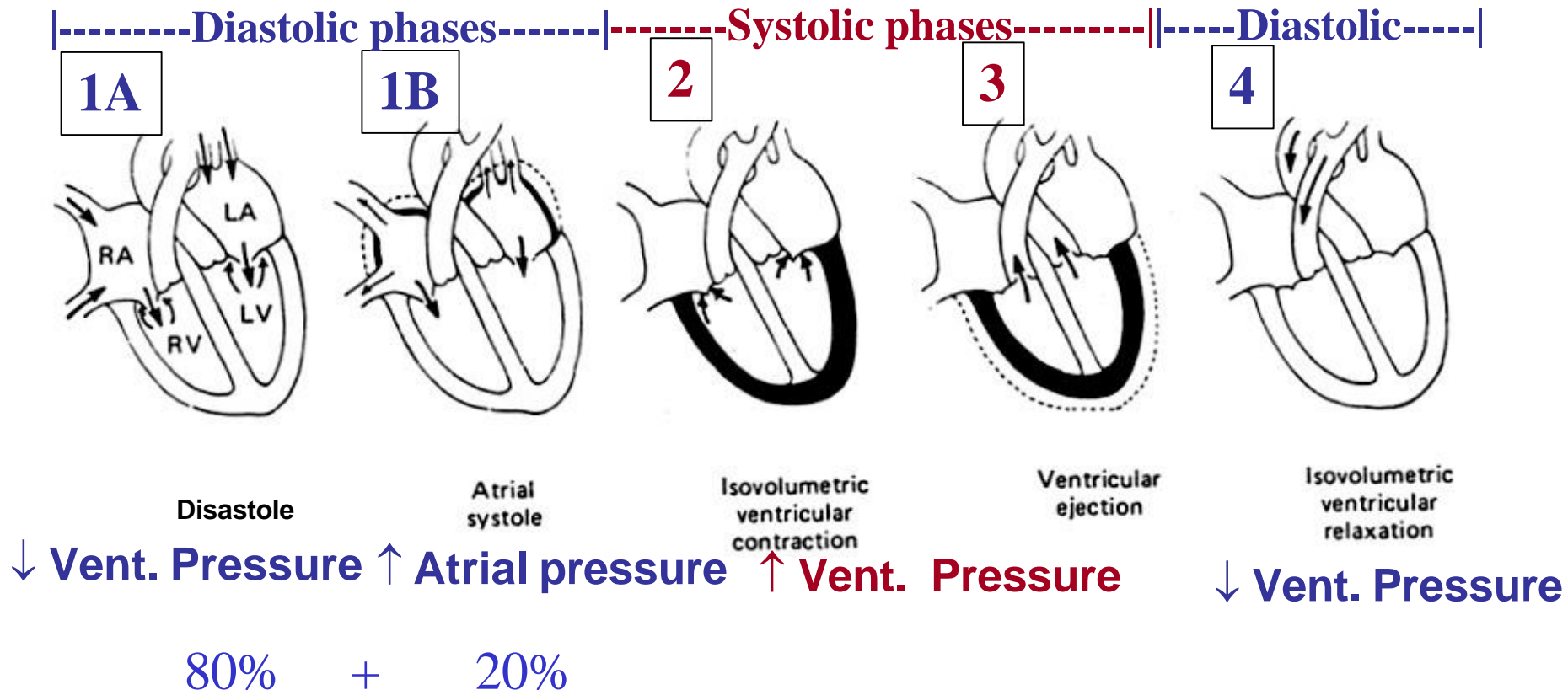
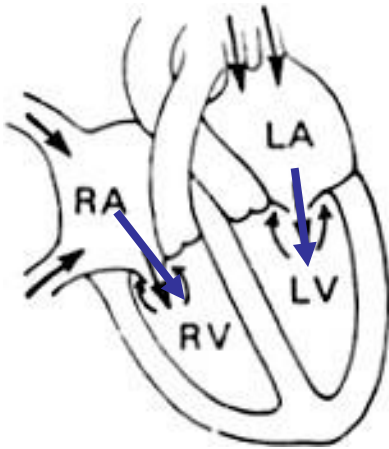


Fig. 44

Diastolic phases:

1A. Diastole - Ventricular Filling



Ventricles are relaxed:

$$P_{\text{vent}} < P_{\text{atria}}$$

A-V valves OPEN

What happens to blood flow?

INTO Ventricles

1B. Atrial systole



Ventricles are relaxed:

$$P_{\text{atria}} ; P_{\text{ventricles}} < P_{\text{atria}}$$

A-V valves remain OPEN

What happens to blood flow?

INTO Ventricles

Fig. 45a, b

Systolic phases:



2. Isovolumetric Ventricular Contraction
Ventricular Myocardium Contracts
3. Ventricular ejection
Massive Ventricular Contraction

$P_{\text{ventricles}}$ \uparrow
when $P_{\text{vent}} = P_{\text{atria}}$,

A-V Valves CLOSE

$P_{\text{ventricles}} > P_{\text{aorta}}$

Aortic and Pulmonary Valves OPEN

What happens to blood flow?

INTO Aorta and Pulmonary A.

Fig. 45 c,d

Diastolic phases:

4. Isovolumetric Ventricular Relaxation

Repolarization of ventricular myocardium



$P_{\text{vent.}}$ ↓↓

when $P_{\text{vent}} = P_{\text{aorta and pulmonary art.}}$

Aortic and Pulmonary Valves CLOSE
[2nd heart sound]

What happens to blood flow?

NO BLOOD FLOW

Fig. 45e

D. The ECG and the Cardiac Cycle

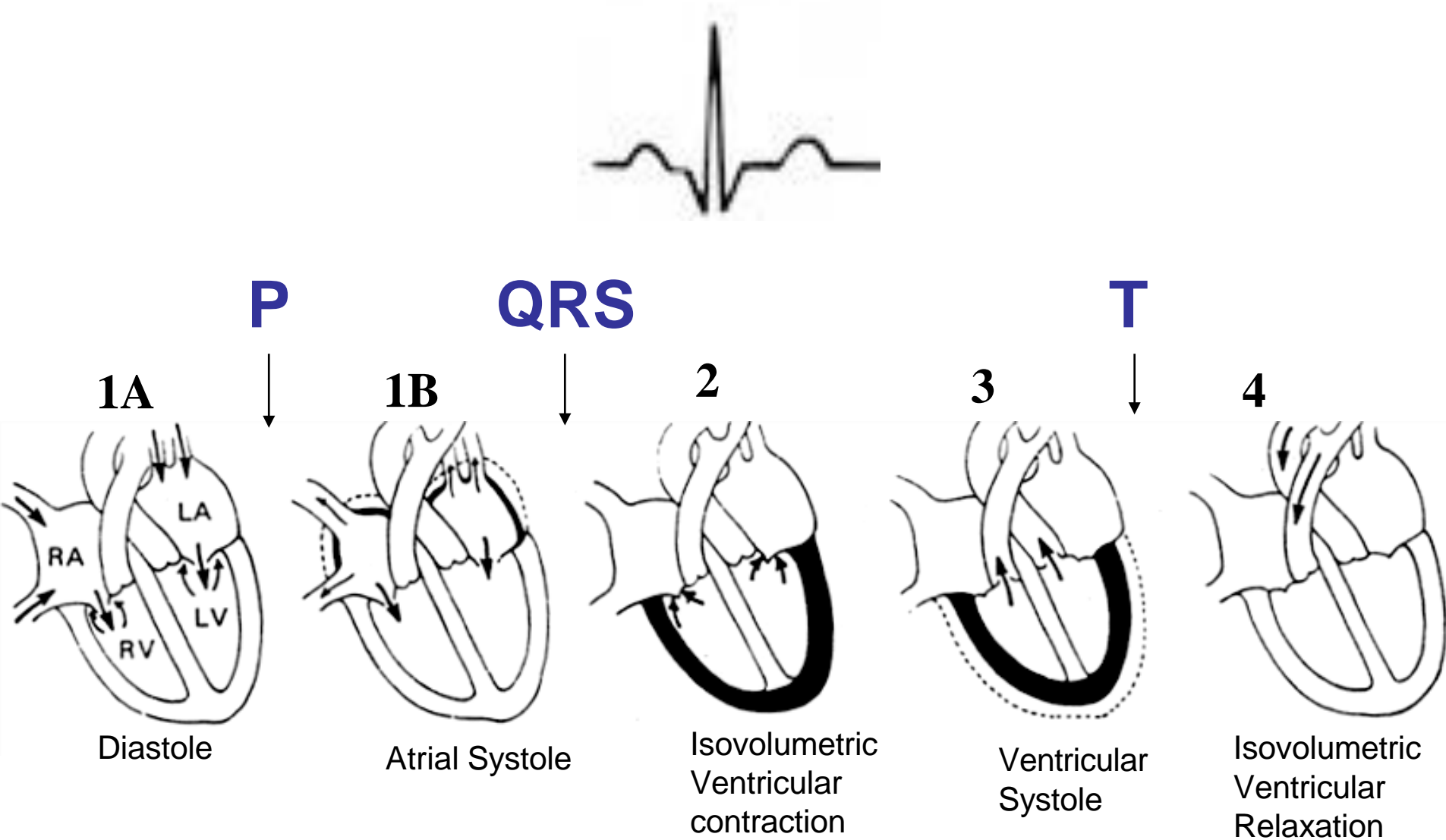
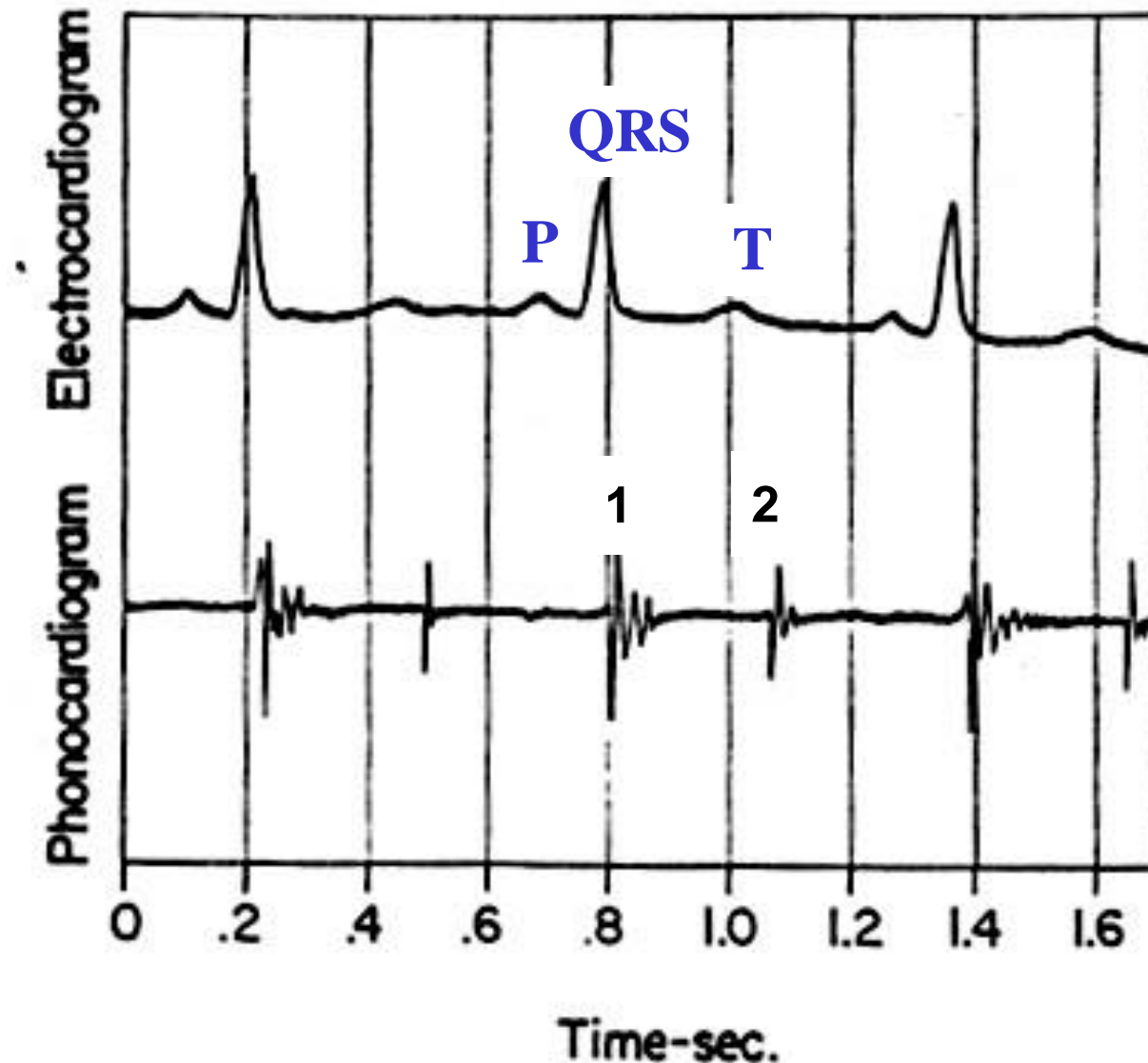


Fig. 45

Heart sounds and the ECG



Normal Heart Sounds: Frontiers in Bioscience

<http://www.bioscience.org/atlas/heart/ekg/normalh.htm>

Fig. 47

From Figure 4-15, p 93; Cardiovascular Physiology Third Edition, RM Berne and MN Levv © 1977 CV Mosby

C. Heart Sounds

Heart sounds correspond to the opening and closing of the valves

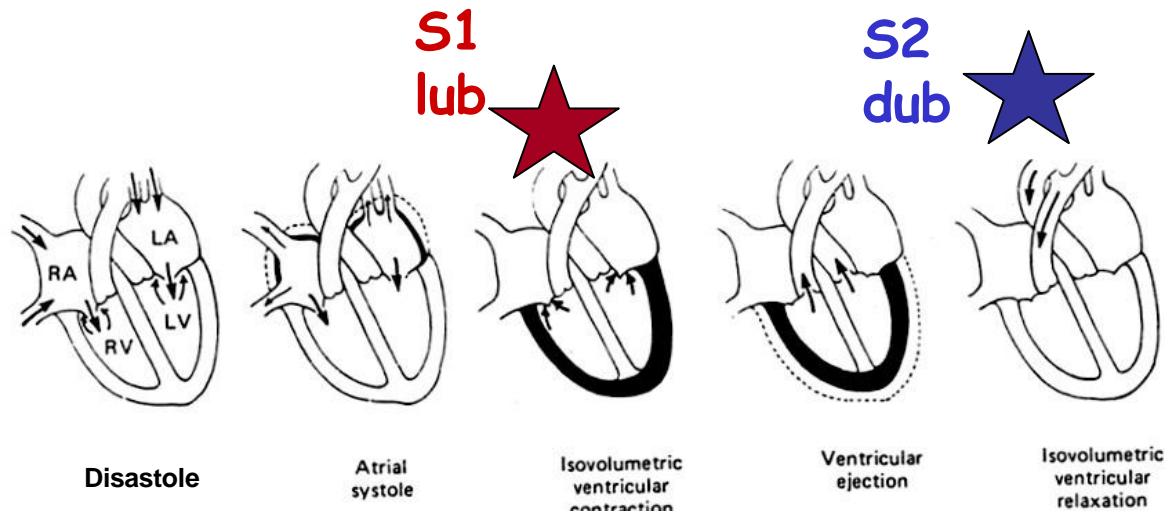


Fig. 46

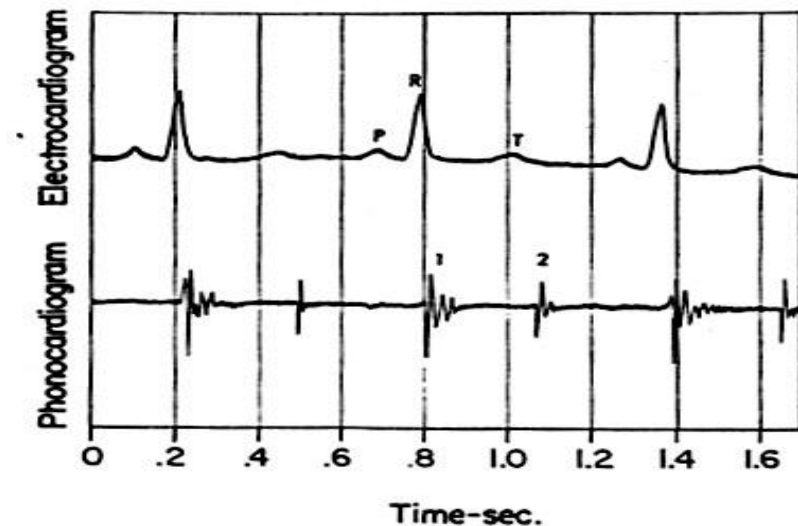


Fig. 47

<http://www.bioscience.org/atlas>



Fig 46. From Figure 29-1, p 466 ; Review of Medical Physiology 13th Edition, WF Ganong © 1987 Lange Medical Publications

Fig 47. From Figure 4-15, p 93; Cardiovascular Physiology Third Edition, RM Berne and MN Levy © 1977 CV Mosby

C. Heart Sounds

Abnormal Heart Sounds

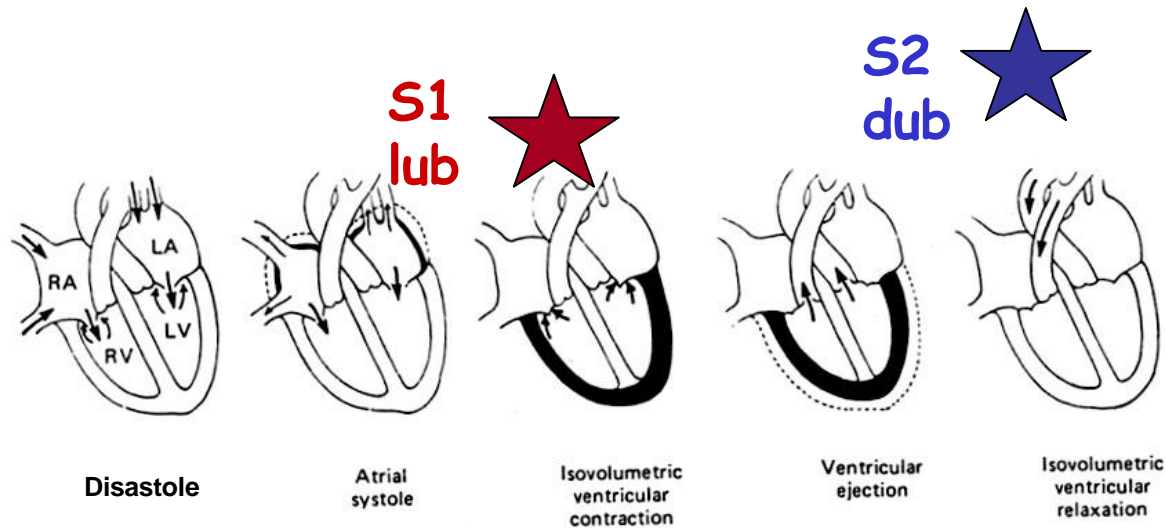


Fig. 46

Stenosis: Partial block when open

Insufficient: Partial leak when closed

<http://www.bioscience.org/atlas> 

Normal Inspiration: S2 splits slightly

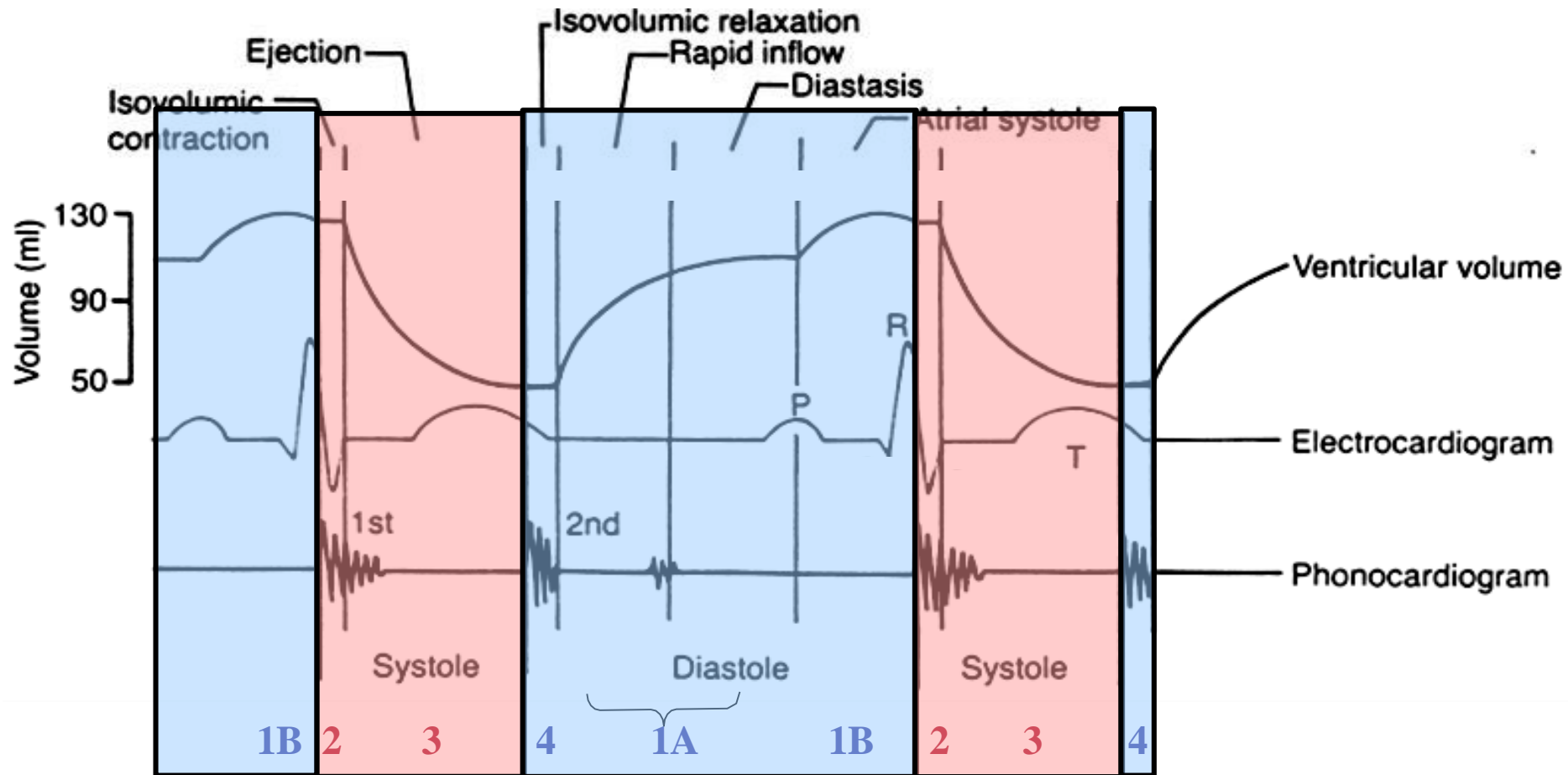
Wide S2 split: Pulmonary valve stenosis

Paradoxical splitting: S2 splits in Expiration, Aortic stenosis

Murmurs: May reflect regurgitation due to insufficient valve

Events of the Cardiac Cycle (Left Heart)

See also Figure 12-20 in Vander, 2004



Events of the Cardiac Cycle (Left Heart)

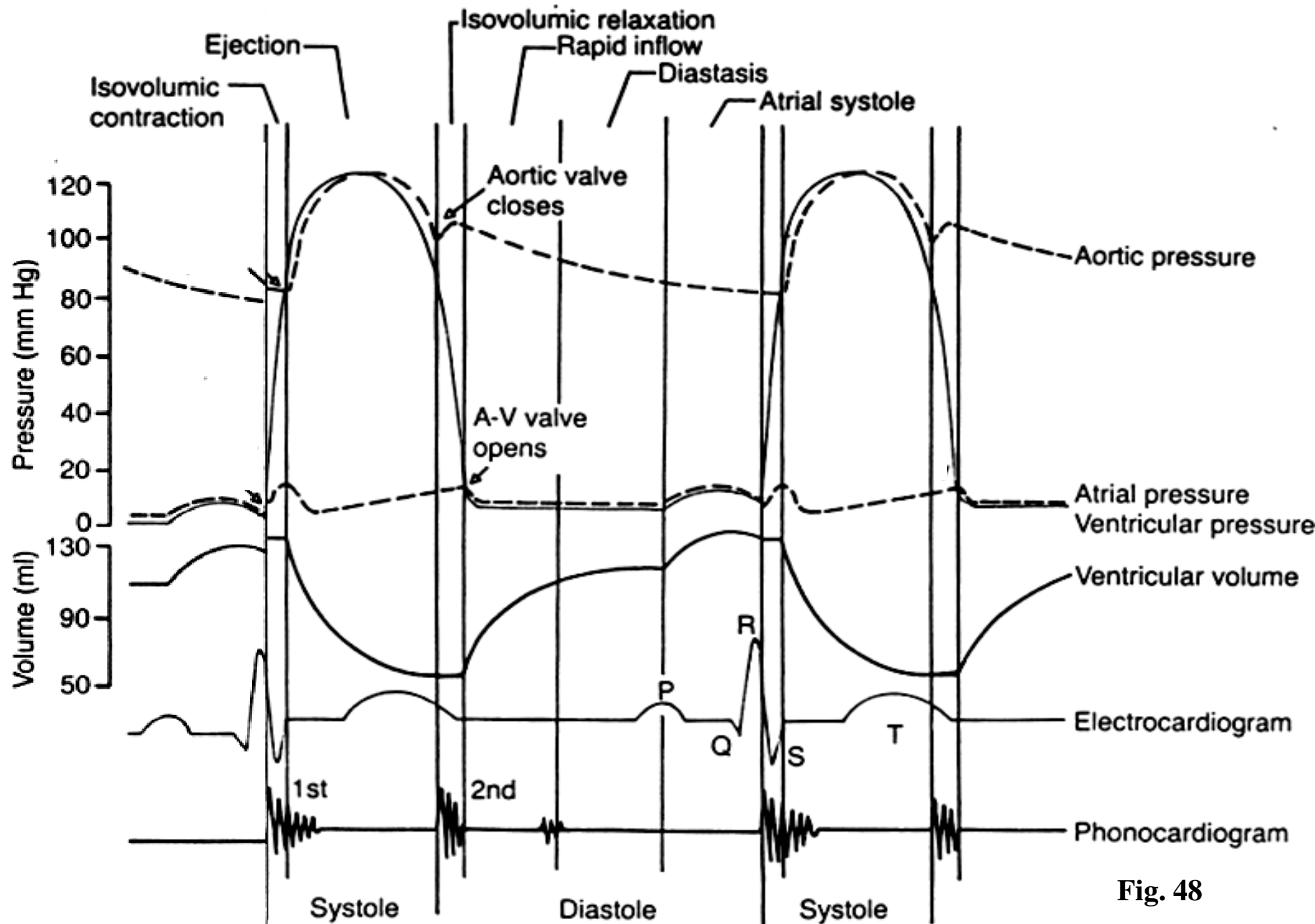
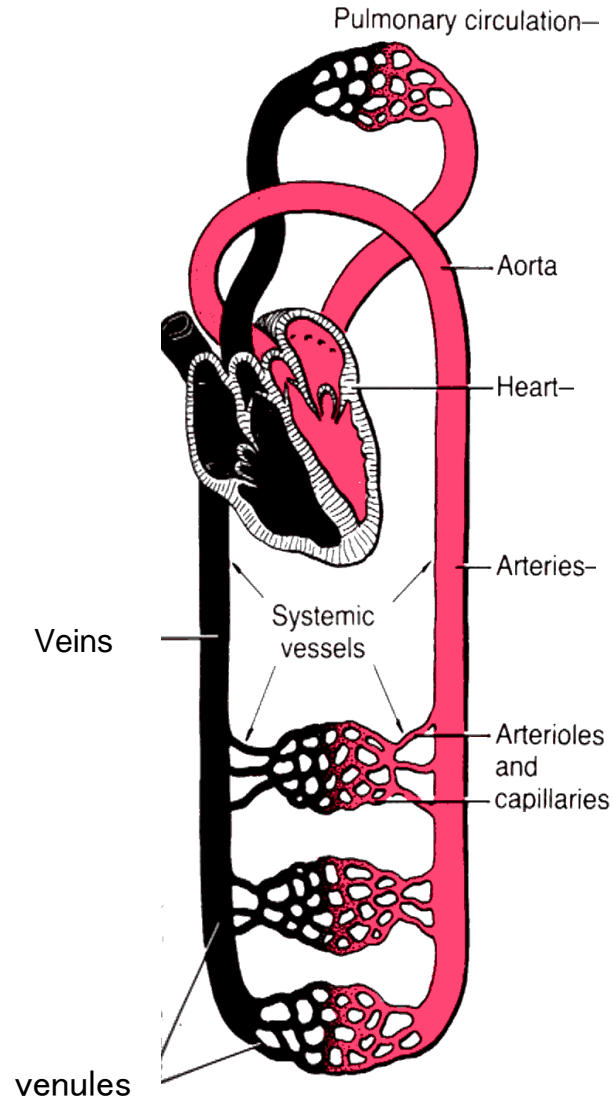


Fig. 48

Review of Pulmonary vs. Systemic Comparison



$$F = \Delta P / R$$

Pulmonary

5 L/min
 ~10 mm Hg
 ~2 mm Hg/L min⁻¹

Systemic

5 L/min
 100 mm Hg
 ~20 mm Hg/L min⁻¹

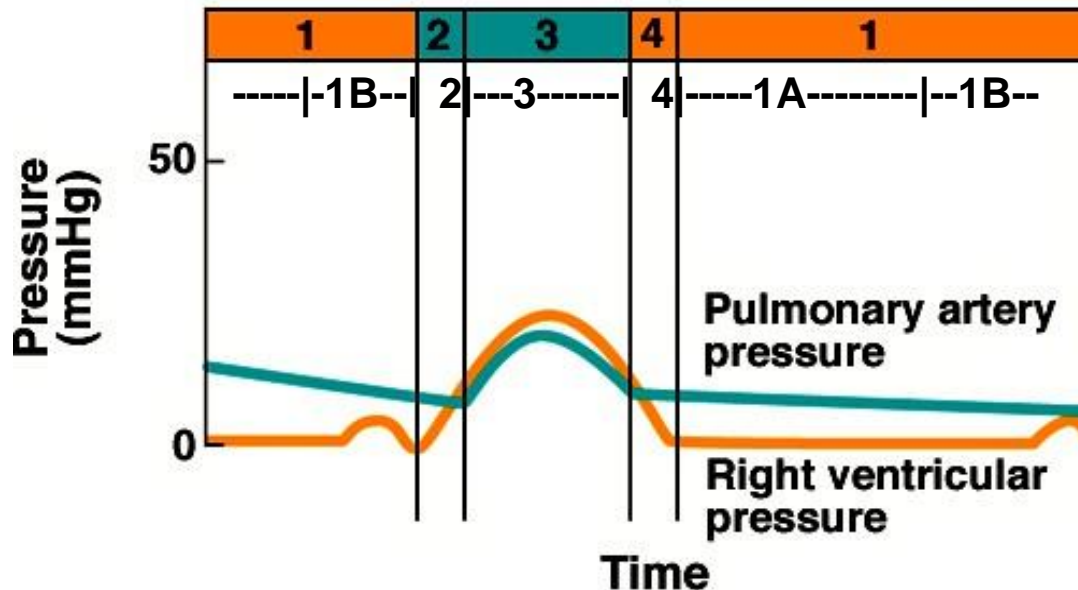
$$R \propto \frac{L\eta}{r^4}$$

The Pulmonary Circulation (**Right Heart**) follows the same sequence, except that the PRESSURE VALUES are LOWER!

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Pressures in right ventricle/pulmonary artery

- 1 = Ventricular filling
- 2 = Isovolumetric ventricular contraction
- 3 = Ventricular ejection
- 4 = Isovolumetric ventricular relaxation



? **Why** is right ventricular pressure so low?

Figure 12-21 from Vander., 2006

Fig. 49

End Diastolic Volume (EDV) and End Systolic Volume (ESV)

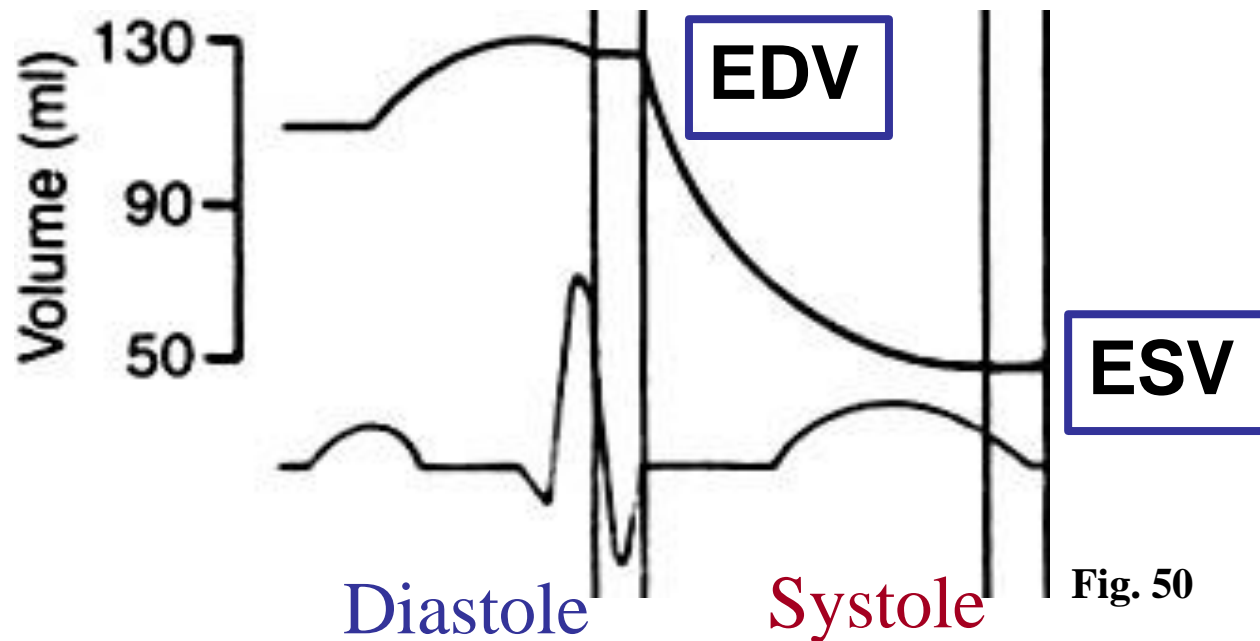


Fig. 50

DEFINITIONS (Fill in values for an average adult male):

Stroke Volume = volume of blood expelled from each ventricle during contraction
= **EDV – ESV** (e.g. 130ml - 50 ml = 70 mls)

Ejection Fraction = fraction of blood volume that is expelled during contraction
= **Stroke volume** / **EDV** (e.g. 70ml / 130ml = ~ 54%)

Cardiac Cycle- Key Points:

1. The cardiac cycle is the recurring sequence of depolarization, contraction and blood flow that results in the pumping of oxygenated blood to the cells and tissues of the body.
2. Blood always flows in response to pressure differences.
3. Systole (ventricular contraction) and Diastole (ventricular relaxation) are the two major phases of the cardiac cycle.
4. Isovolumetric phases occur when the valves of the heart are closed. Closing of the valves causes the 1st and 2nd heart sounds.
5. Pressures in the pulmonary circulation are lower than those in the systemic circulation.

“Enrichment Material for Cardiac Cycle”

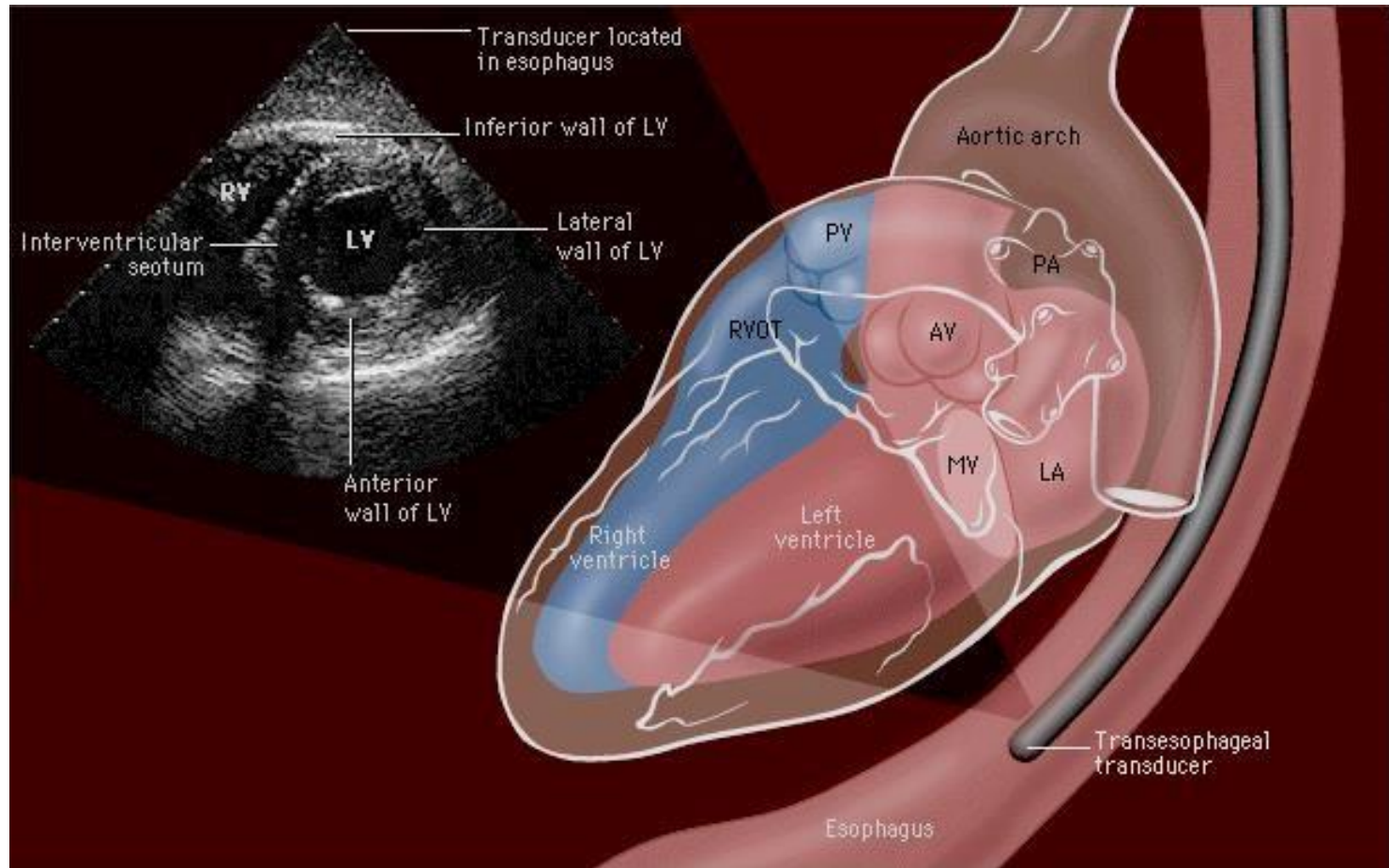
1) Echocardiograms

2) Valve defects

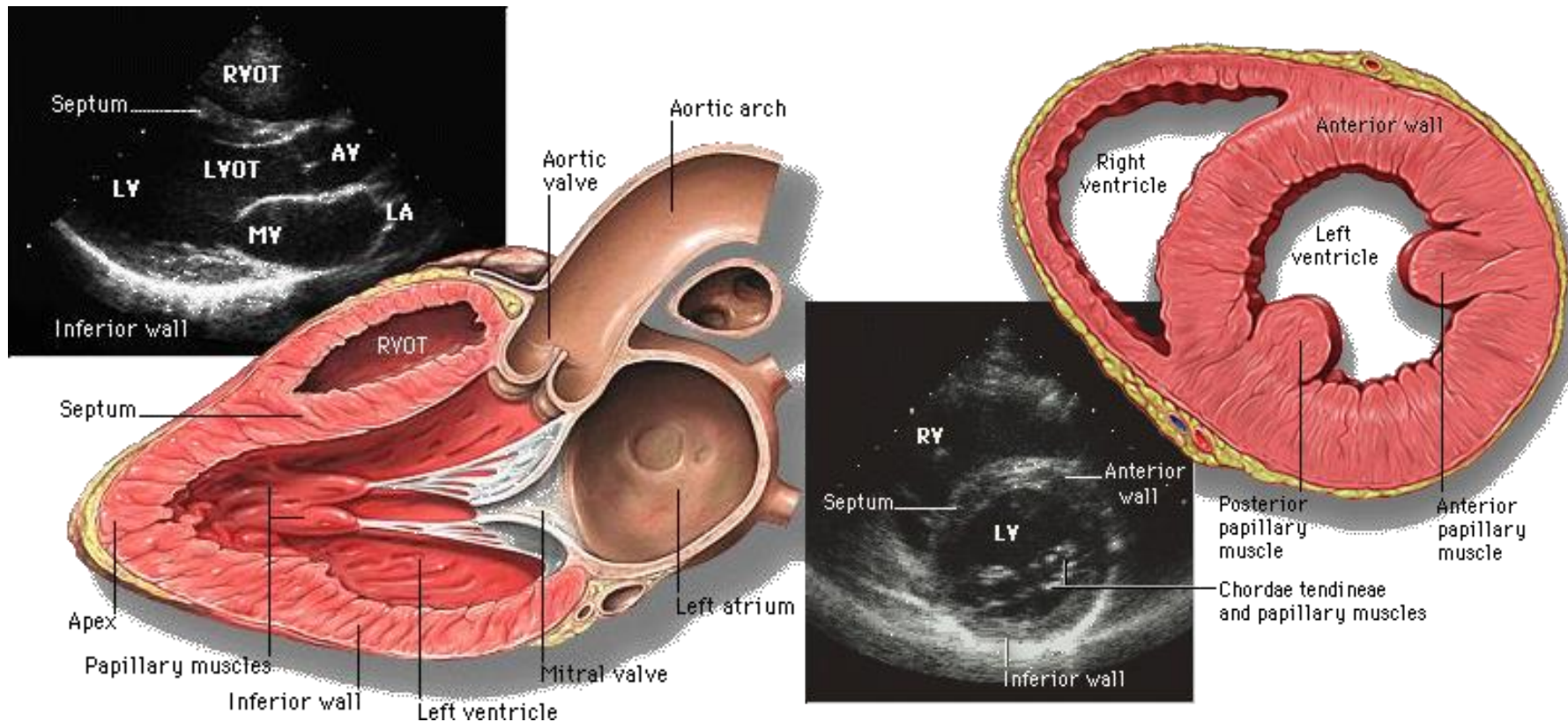
Echocardiography: high frequency (ultrasonic) waves are reflected where there are differences in acoustic impedance



Transesophageal Echocardiography

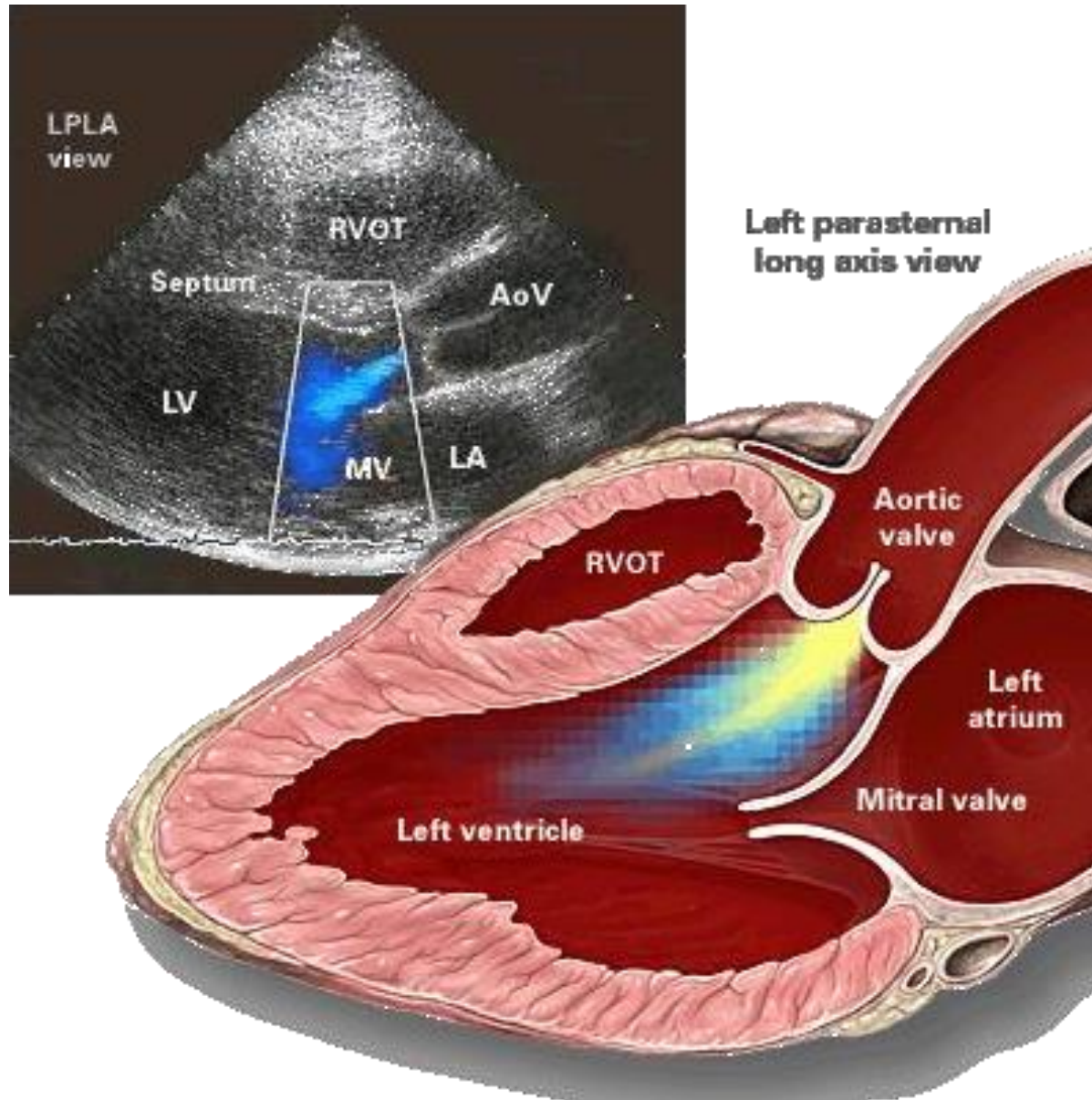


2- dimensional Echocardiography



OT=outflow track

Doppler Imaging reveals direction, velocity and turbulence of blood flow



Movie of echocardiogram of cardiac cycle in normal adult male

http://info.med.yale.edu/intmed/cardio/echo_atlas/views/index.html

“Enrichment Material for Cardiac Cycle”

1) Echocardiograms

2) **Valve defects**

Aortic regurgitation

congenital
aging
disease

Aortic stenosis

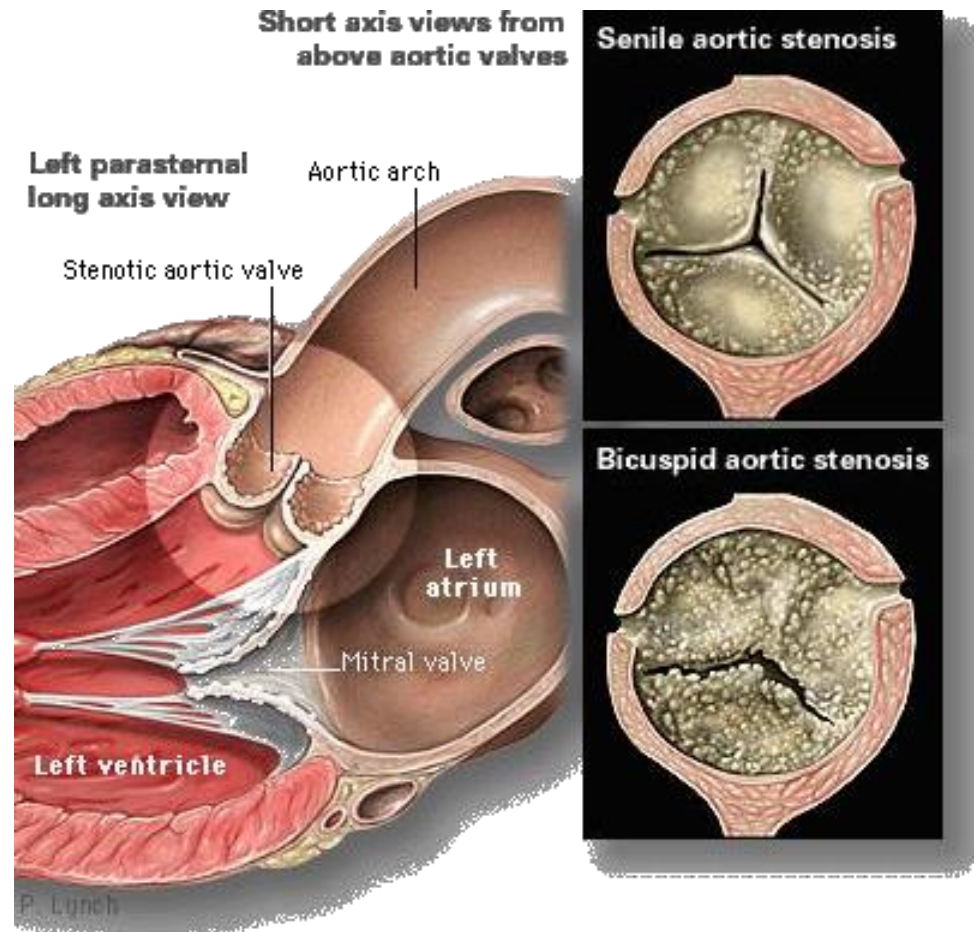
aging
disease



Heart sounds



- 1 A-V valves close
- 2 Aortic and pulmonary valves close



“Enrichment Material for Cardiac Cycle”

1) Echocardiograms

2) **Valve defects**

Mitral stenosis

disease (Rheumatic fever)

congenital

blood clots or tumors



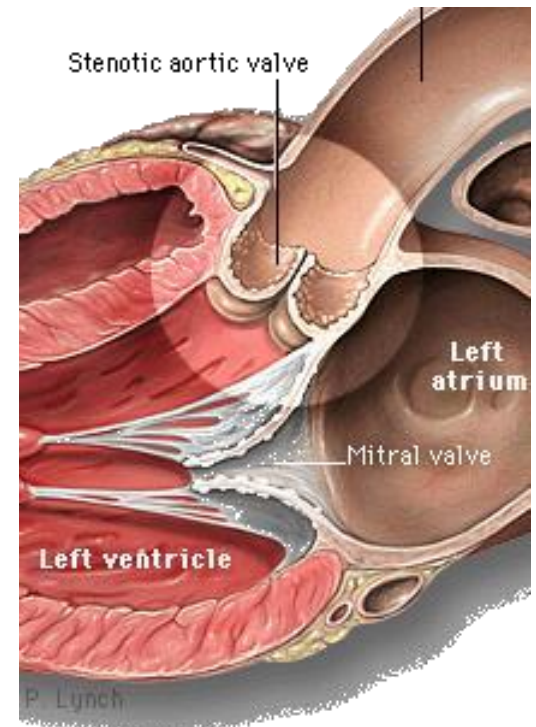
Accentuated first sound



Opening snap

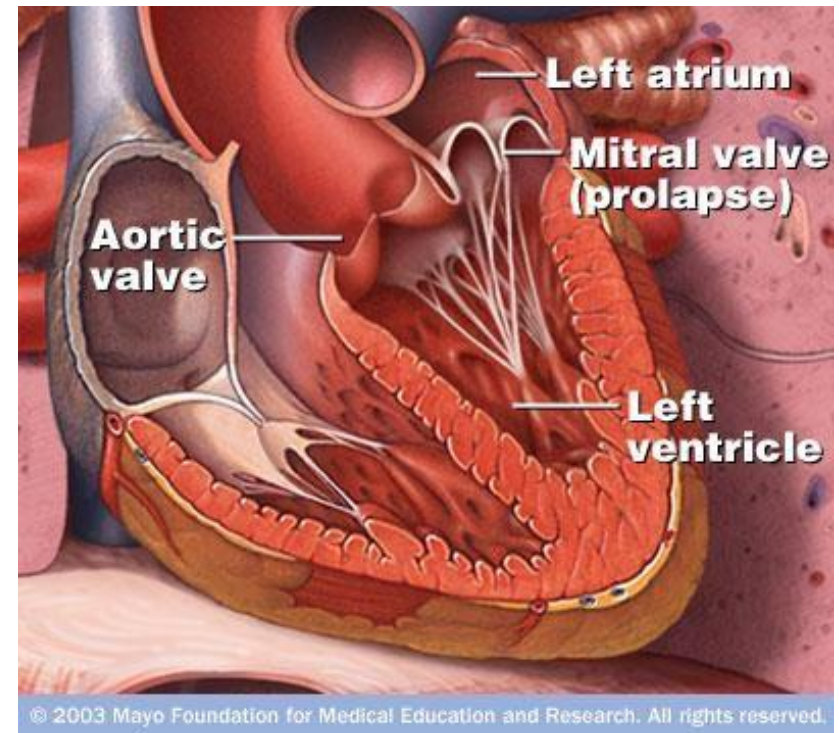


Normal Heart Sounds



“Enrichment Material for Cardiac Cycle”

- 1) Echocardiograms
- 2) **Valve defects**



Mitral valve prolapse

click-murmur syndrome
causes usually unknown



Normal Heart Sounds

The regurgitation of blood increases the likelihood of acquiring bacterial endocarditis. Prophylactic antibiotics are recommended prior to any surgical or dental procedure.

<http://www.bioscience.org/atlas/heart/sound/sound.htm>

Cardiac Output

- A. Definition of Cardiac Output
- B. Heart rate: Autonomic nervous system regulation
- C. Factors contributing to Stroke Volume
 - 1. Force of Contraction
 - a. End-diastolic fiber length
 - b. Contractility
 - c. Muscle fiber structure
 - 2. Afterload

A. Cardiac Output = Volume of Blood pumped by each ventricle per minute

$$CO = HR \times SV$$

Normal values for an adult male:

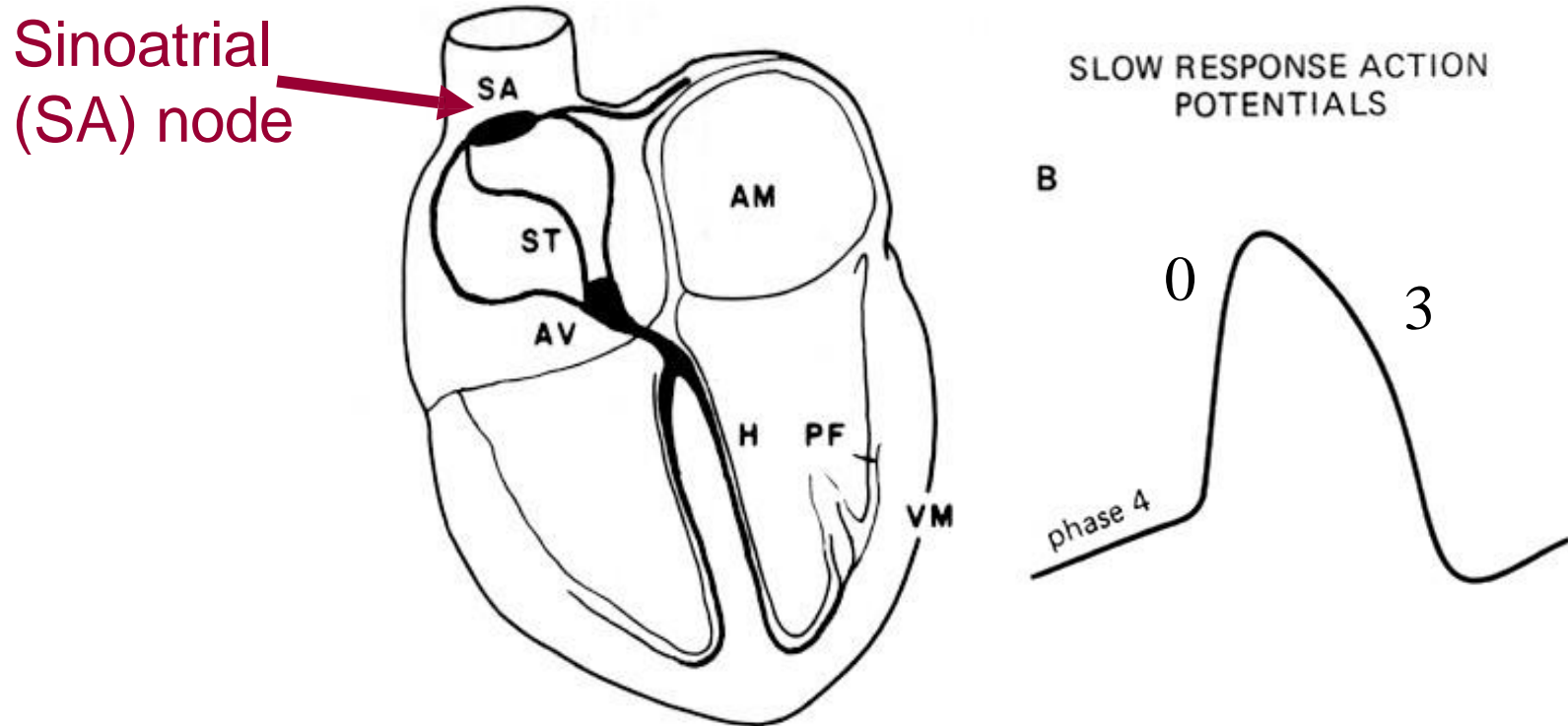
HR- heart rate (beats per minute) = ~70 bpm at rest

SV- Stroke volume (ml per beat) = EDV – ESV = ~70 ml at rest

CO at rest = ~ 5 L / minute

CO during strenuous exercise = 20-35 L / minute

B. Heart Rate is determined **ONLY** by the frequency of pacemaker firing at the **SA node**.



Pacemaker Potential and Heart Rate

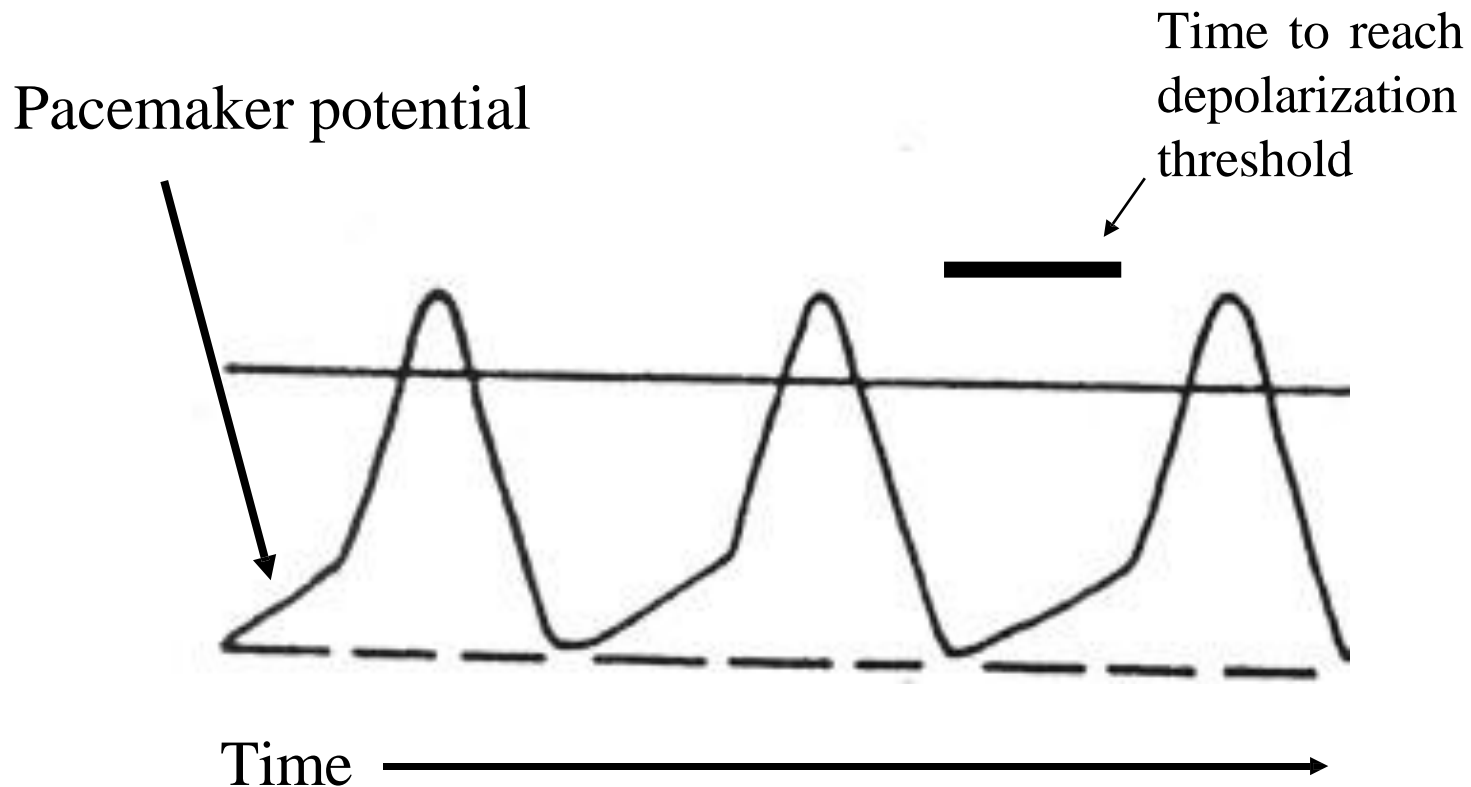


Fig. 52

Effect of Sympathetic and Parasympathetic Stimulation on Pacemaker Potential and Heart Rate

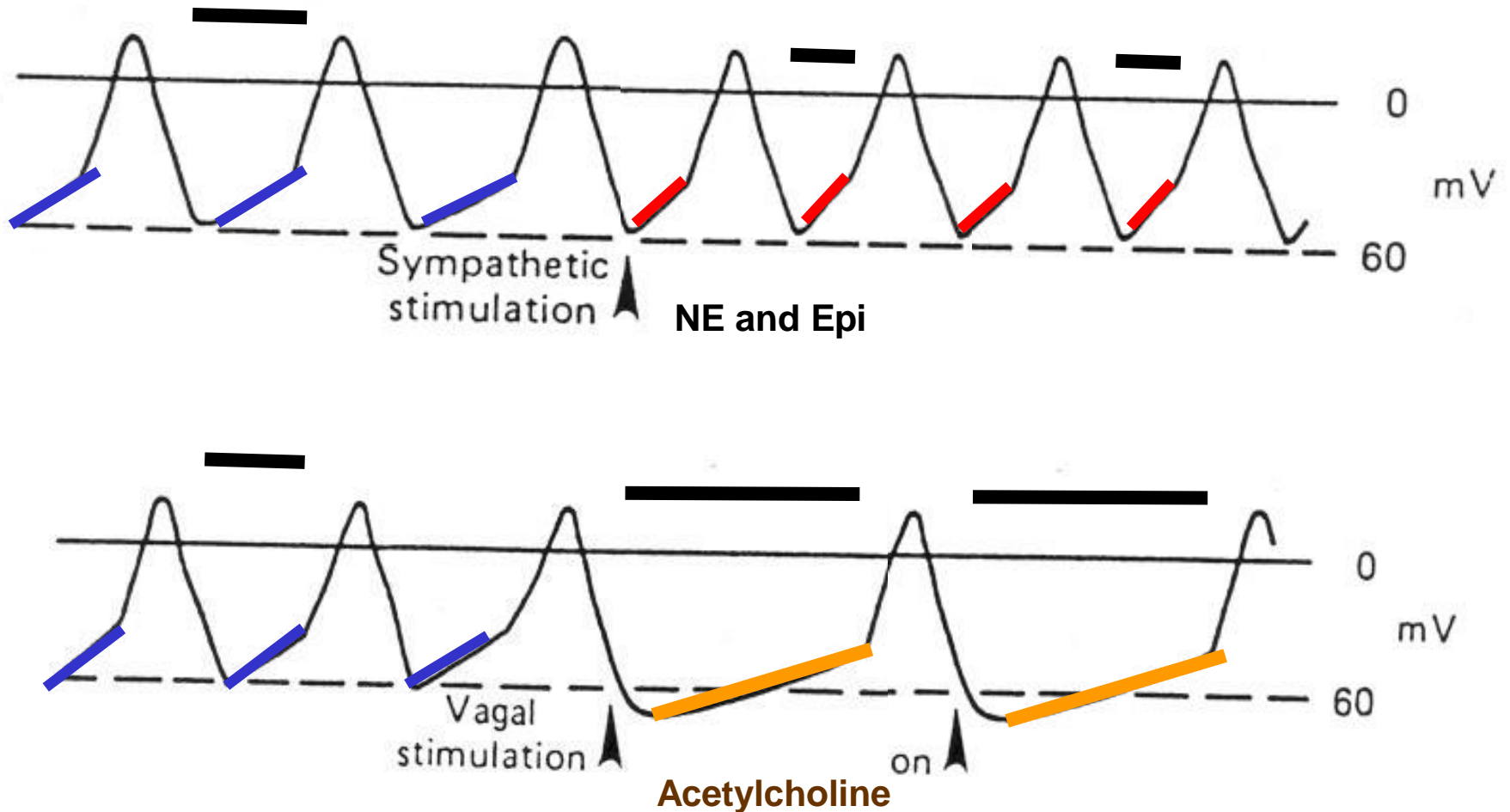
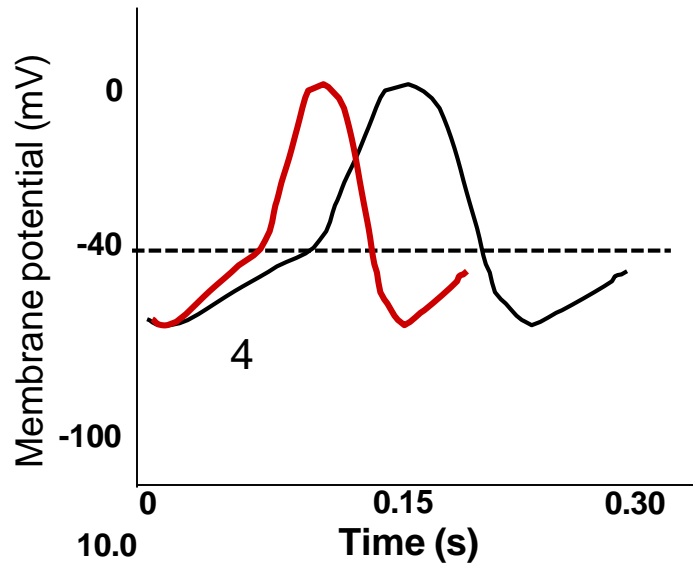


Fig. 52

How do epinephrine and norepinephrine increase heart rate at the SA node?



β_1 receptor activation – faster depolarization

- Increases $P_{Na+(F)}$
- Increases $P_{Ca^{2+}}$

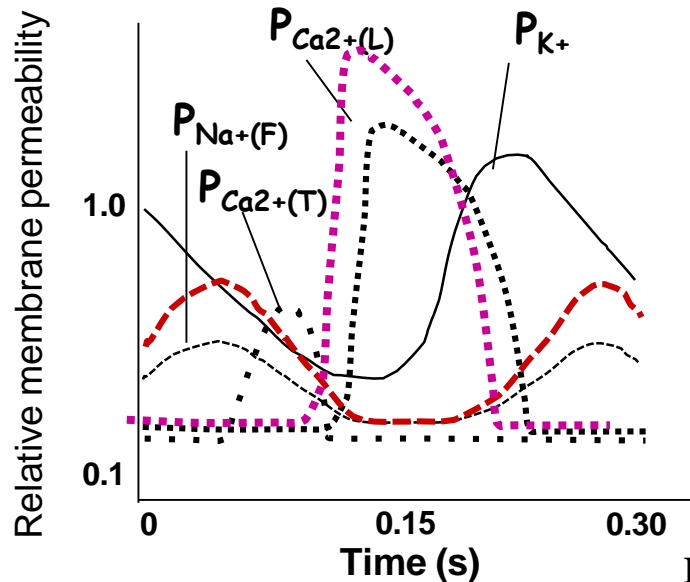
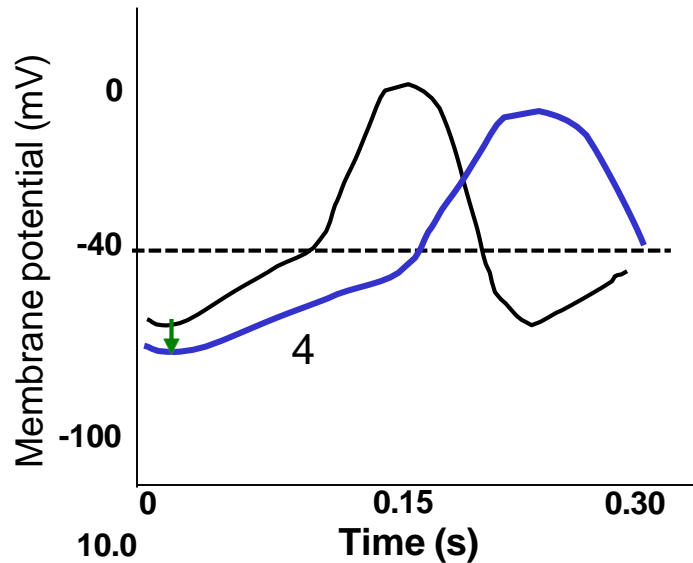


Fig 27

How does acetylcholine decrease heart rate at the SA node?



M_2 receptor activation

- decreases $P_{Na+(F)}$
- decreases membrane potential by increasing P_{K+}

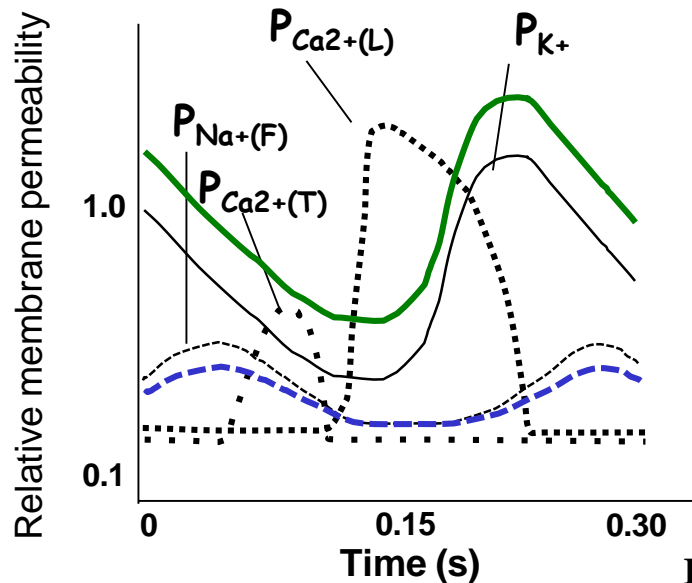
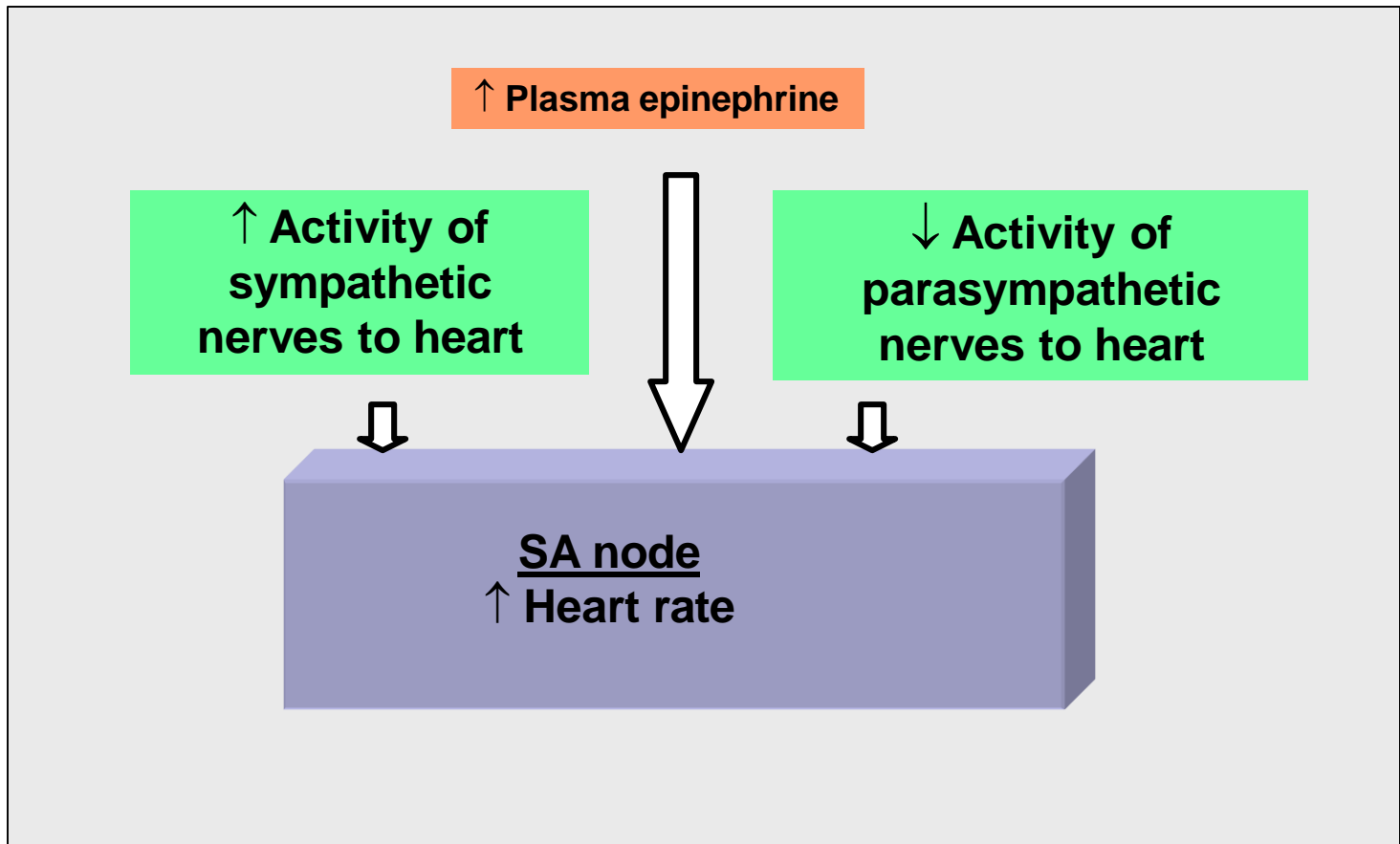


Fig 27

$$\text{CO} = \text{HR} \times \text{SV}$$



Copied from FIGURE 12-23, Vander, Sherman and Luciano's Human Physiology

C. Stroke Volume

Stroke volume = volume of blood expelled from each ventricle during contraction = EDV - ESV

(e.g. 120 ml - 50 ml = 70 ml)

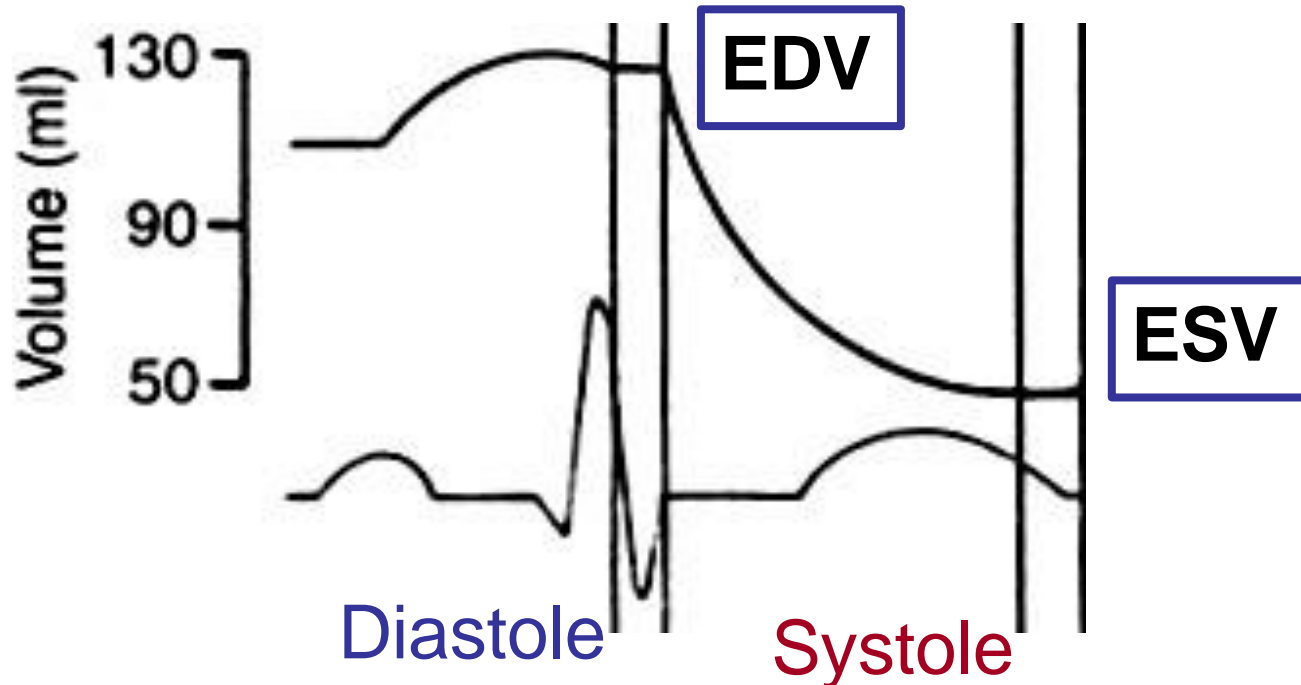
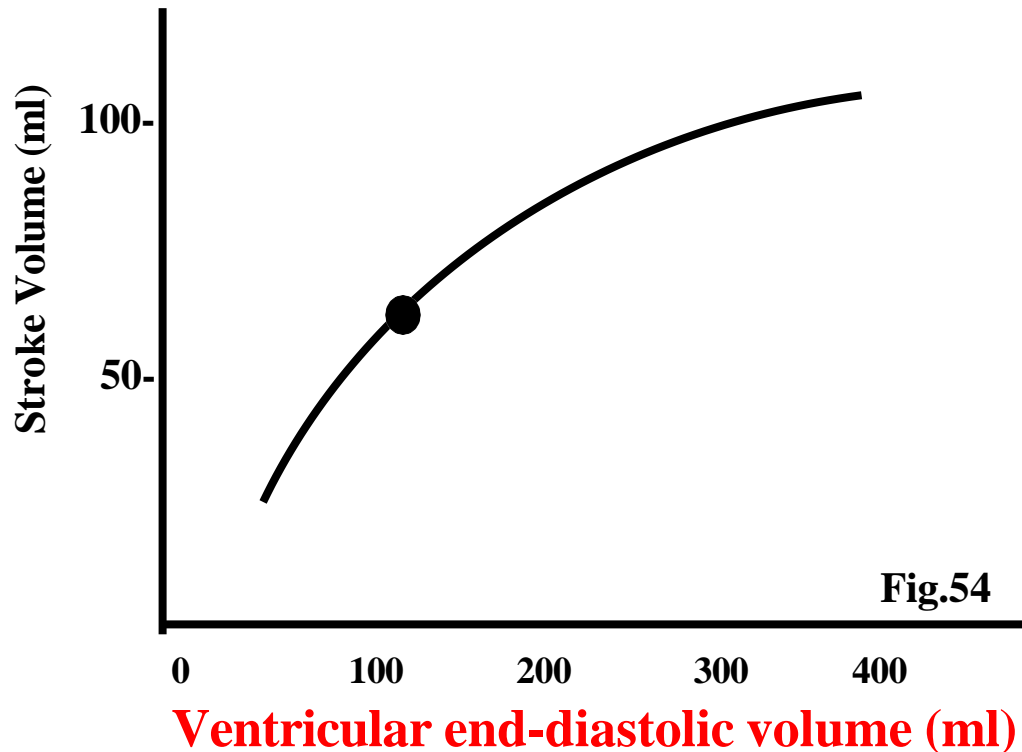


Fig. 54

1. Changing the Force of Contraction

a. Ventricular Function Curve – Frank Starling Mechanism

Stroke volume increases as EDV increases.



?What factors influence EDV?

Total Blood Volume -

Atrial filling -

Ventricular compliance -

Venous tone -

1. Body position
2. Intrathoracic pressure
/ respiration
3. Skeletal muscle pump

If ventricular muscle is stretched more, it will eject blood with more force.

Factors that Influence Ventricular End-Diastolic Volume

- Total Blood Volume - **Blood loss, transfusion, kidney function**
- Atrial filling - **Atrial fibrillation, loss of compliance**
- Ventricular compliance - **Aging, tachycardia (rapid contractions)**
- Venous tone -
 1. Body position **Gravity**
 2. Intrathoracic pressure / respiration **Inspiration, expiration**
 3. Skeletal muscle pump **Increase venous wall pressure**



1. Changing the Force of Contraction

b. Sympathetic activation increases Contractility

Contractility = Force of contraction for a given sarcomere length

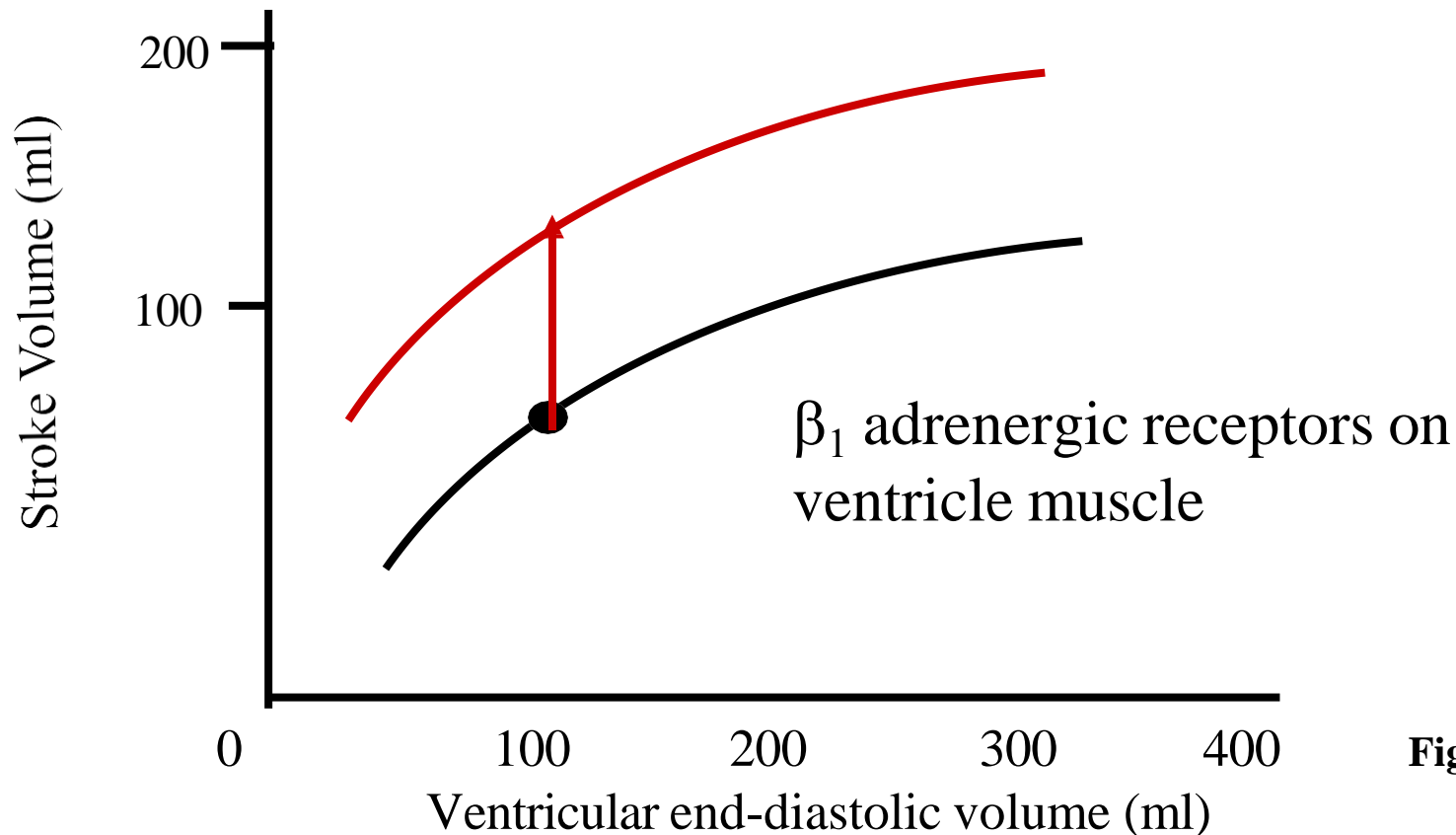
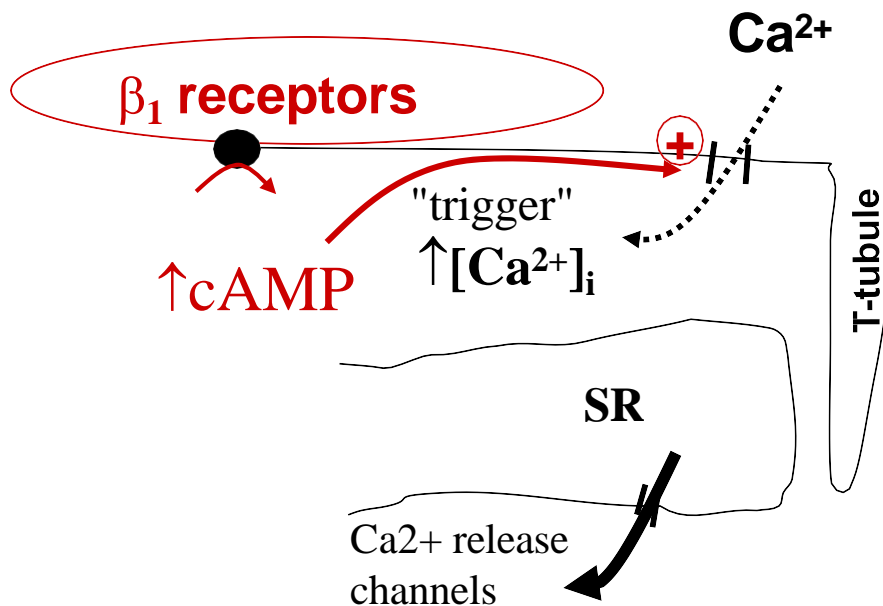


Fig. 55

Sympathetic activation increases contractility of the myocardium

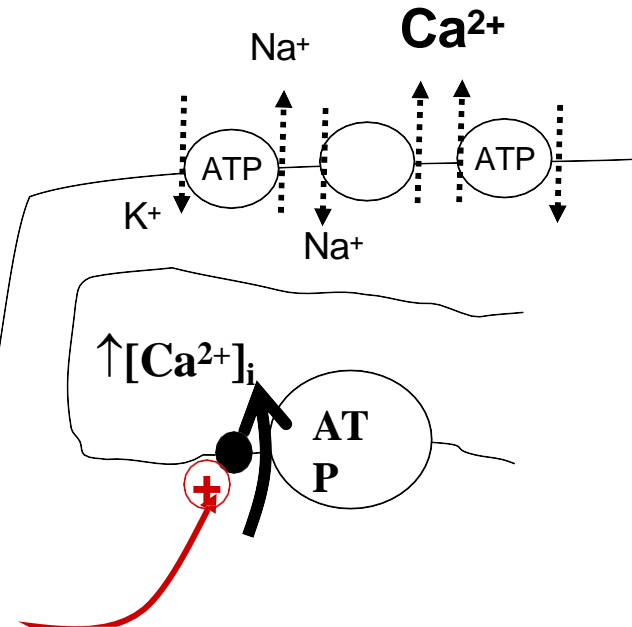
MECHANISMS: Modulation of Excitation-Contraction Coupling
cAMP regulation of intracellular calcium release and sequestration

1. Excitation



More cross bridges
Faster and stronger contraction

2. Relaxation



Increased sequestration
Faster relaxation

Fig. 56

1. Changing the Force of Contraction

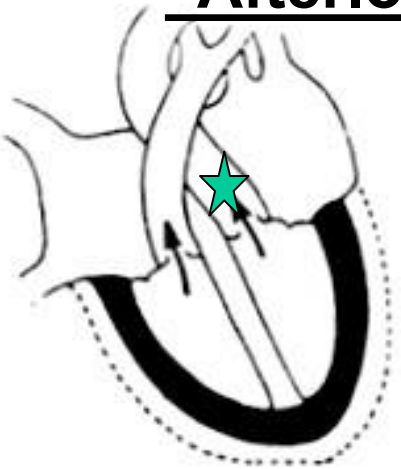
c. Exercise can alter Muscle Fiber Structure

ex.) Changes can be induced by Long-Term Aerobic Training
↑ actin / myosin and ↑ cardiac muscle fiber size



Clipart © Microsoft, Inc.

2. Changes in flow by pressure or resistance “Afterload” of ventricular pressure



Ventricular
ejection



Aortic and pulmonary pressures

Increased aortic and pulmonary pressure can cause decreased ejection volume ($F \propto \Delta P$)

Fig. 57

Cardiac Output: Key Points

Cardiac Output = Volume of Blood pumped by each ventricle per minute.

1. $CO = HR \times SV$.

2. HR is controlled by Sympathetic and Parasympathetic activation of SA node.

3. SV is controlled by three factors related to force:

- a. End-diastolic volume (stretch)
- b. Contractility (strength)
- c. Actin/myosin content (size)

and one factor related to pressure and resistance:

- d. Afterload (ex.: increased aortic pressure)

See Flow Diagram Figure 12-28 Vander p. 411

Summary:

Cardiac Output = Volume of Blood pumped by each ventricle per minute.

1. $CO = HR \times SV$.

2. HR is controlled by Sympathetic and Parasympathetic activation of SA node.

3. SV is controlled by three factors related to force:

a. End-diastolic volume (stretch)

b. Contractility (strength) β_1 receptors

c. Actin/myosin content (size)

- Blood volume
- Ventricular compliance
- Atrial contraction
- Venous tone

and one factor related to pressure:

d. Afterload (ex.: increased aortic pressure)

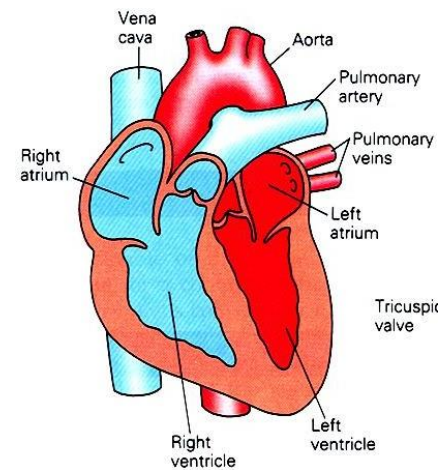
How is Cardiac Output Measured?

1. Indirectly via Fick's principle

$O_2 \text{ consumed} = O_2 \text{ removed} \times \text{Flow rate}$

$$\text{Flow} = \frac{\text{total } O_2 \text{ consumed}}{[O_2]_{\text{art}} - [O_2]_{\text{ven}}}$$

2. Dye or thermo (heat) dilution



Actual Past Dental Board Questions:

1. Both systemic and pulmonary circulations have the same:
 - A. pulse pressure.
 - B. total capacitance.
 - C. diastolic pressure.
 - D. resistance.
 - E. flow rate.

1. E
2. Increased parasympathetic activity results in
 - A. decreased salivary secretion.
 - B. increased cardiac contractility
 - C. decreased gastric motility and tone.
 - D. increased bronchiolar smooth muscle contraction.

2. D
3. D
3. Which of the following is MOST likely to result from increased vagal activity?
 - A. Increased heart rate
 - B. Increased stroke volume
 - C. Increased cardiac output
 - D. Decreased cardiac oxygen consumption
 - E. Decreased transit time through the AV node

Exam will cover Lectures, Handouts and Text Assignments

Text: Vander, Sherman, & Luciano's HUMAN PHYSIOLOGY, 10th Edition

| <u>Date</u> | <u>Lecture topic</u> | <u>Text Pages</u> |
|--|--|-------------------|
| 9/13 | - Autonomic nervous system | 199-204 |
| | - Cardiovascular system: organization | 387-395 |
| 9/15 1st Midterm Exam (Cell Physiology) | | |
| 9/20 | - Functional requirements of the heart | 395-399 |
| | - Electrocardiogram | 389-403 |
| 9/22 | - The cardiac cycle | 403-408 |
| | - Cardiac output | 408-414 |
| 10/13 2nd Midterm Exam (ANS and Cardiovascular) | | |

WebCT and Dent Website information:

Slides and Supplementary info: <http://www.dent.ohio-state.edu/Courses/physiology>

Practice self-tests: <https://enigma.optometry.ohio-state.edu/>

Select: view my courses

User ID: pcbcardio-dent

password: pcbcardio

PCB Cardiovascular; Course Materials; Course Content

Self test. Note- Questions #1-16 of each test for Dr. Jakeman, #17-30 for Dr. Ward