Cardiovascular Physiology

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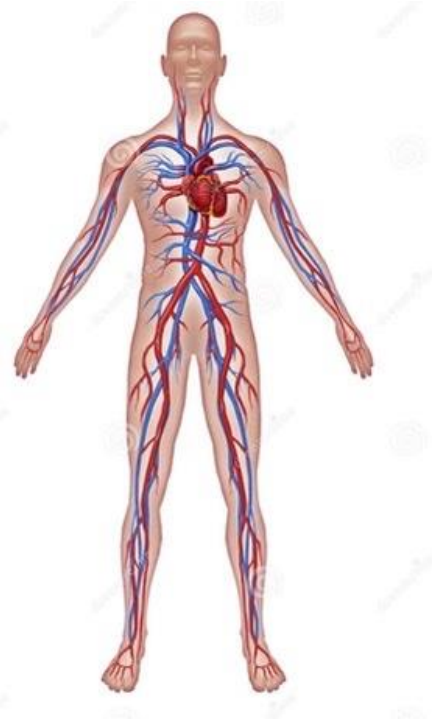

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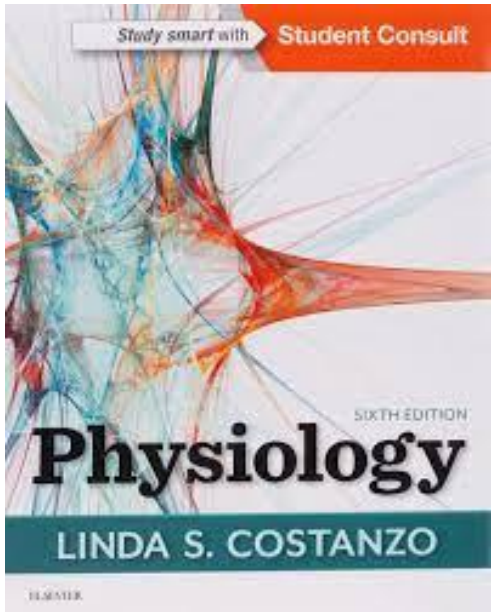
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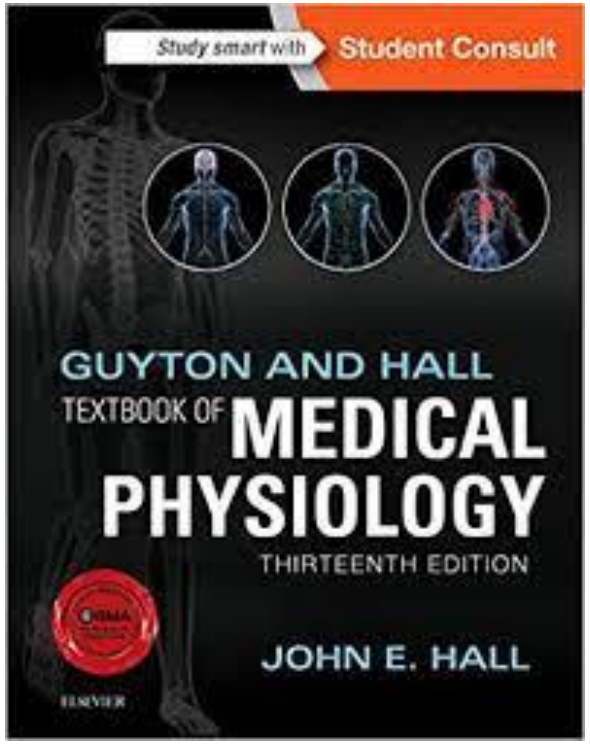
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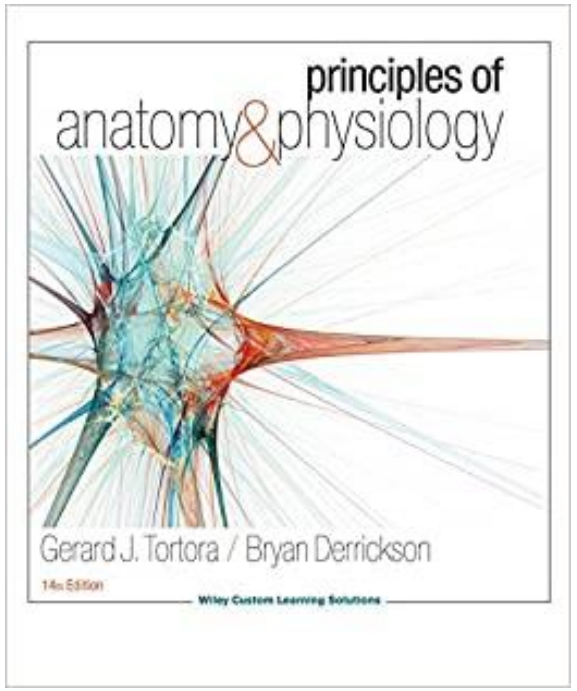
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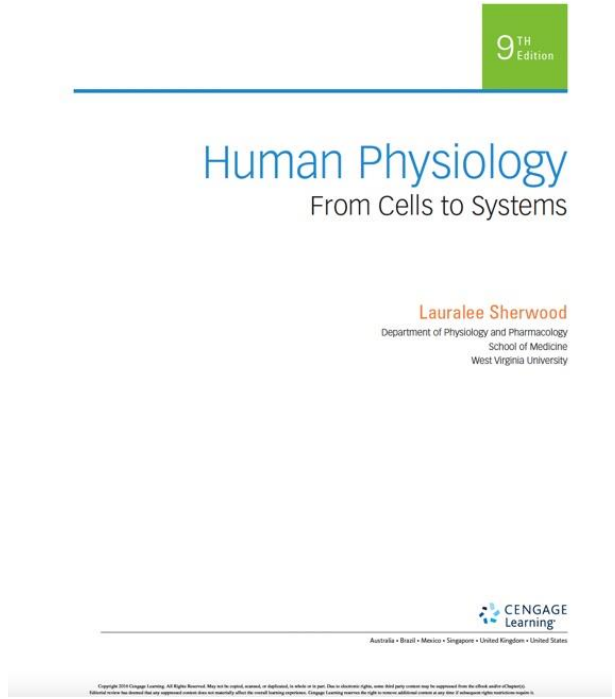
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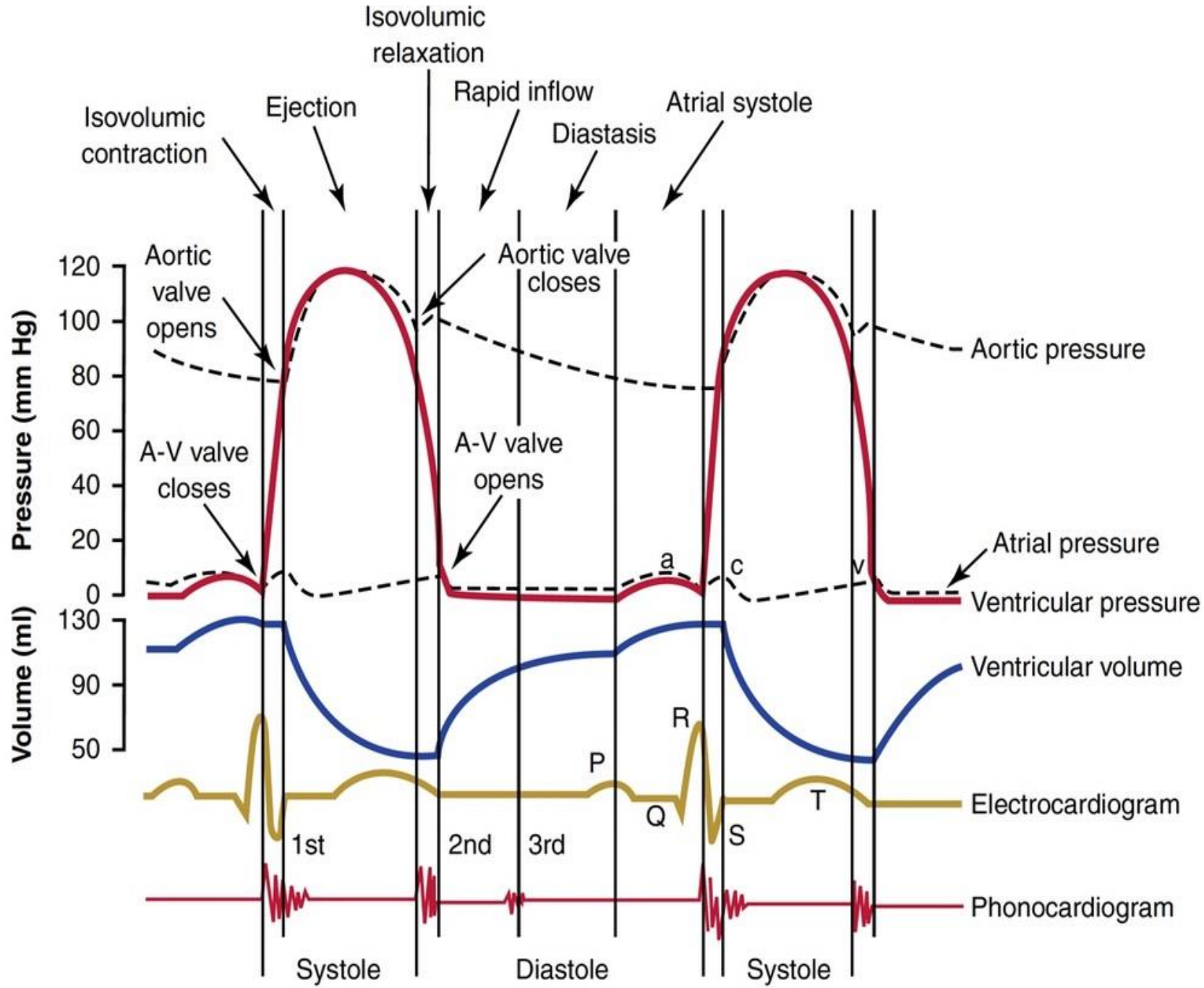
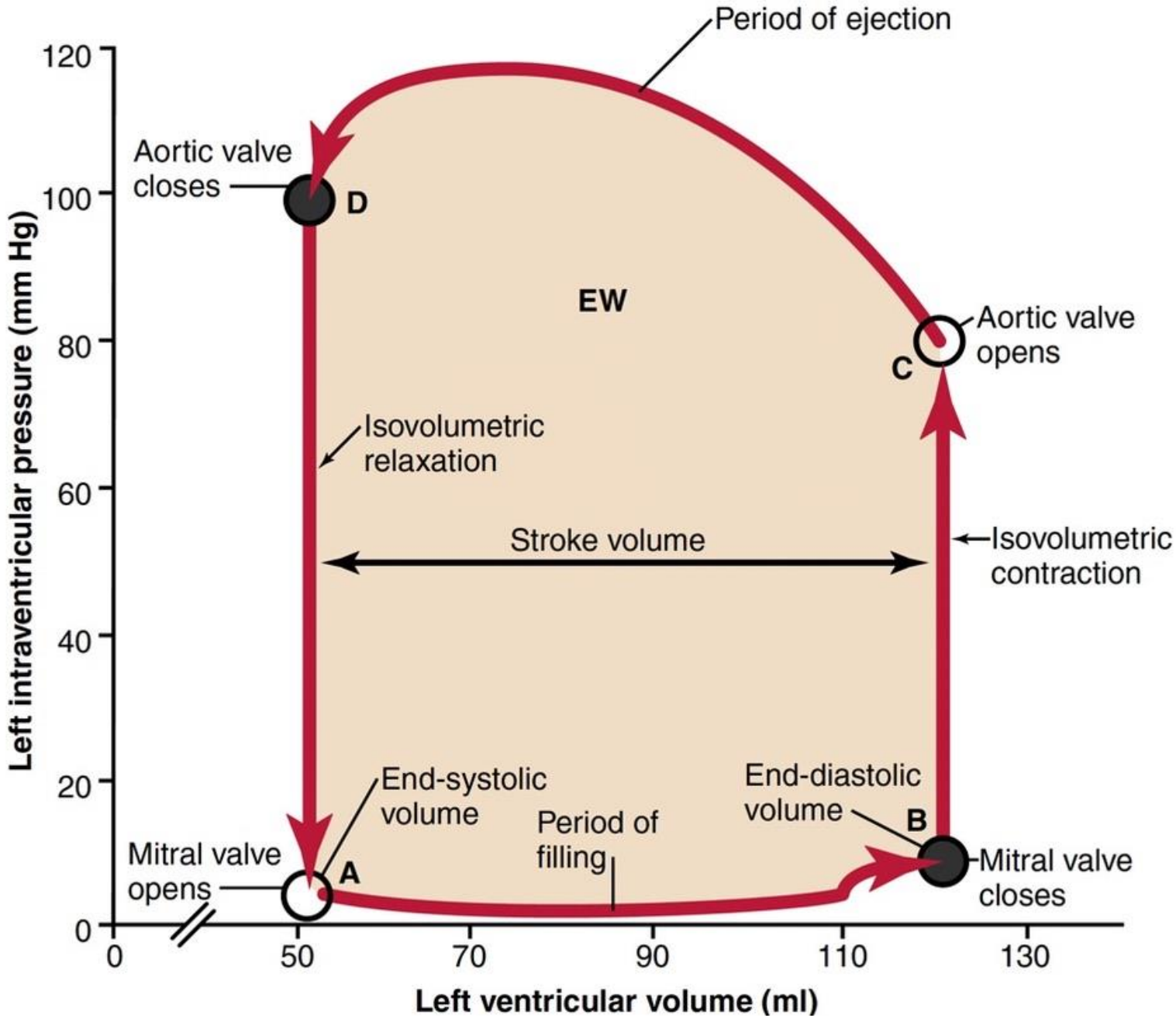
Mechanical events of the cardiac cycle


Isovolumic relaxation
Isovolumic contraction
Ejection
Rapid inflow
Diastasis
Atrial systole
Aortic valve opens
Aortic valve closes
A-V valve closes
A-V valve opens
Pressure (mm Hg)
120
100
80
60
40
20
0
Volume (ml)
130
90
50
a
c
v
Aortic pressure
Atrial pressure
Ventricular pressure
Ventricular volume
R
P
Q
S
T
Electrocardiogram
1st
2nd
3rd
Phonocardiogram
Systole
Diastole
Systole


Period of ejection
Aortic valve closes
D
EW
Aortic valve opens
C
Isovolumetric relaxation
Stroke volume
Isovolumetric contraction
End-systolic volume
Period of filling
End-diastolic volume
B
Mitral valve opens
A
Mitral valve closes
Left intraventricular pressure (mm Hg)
120
100
80
60
40
20
0
0
50
70
90
110
130
Left ventricular volume (ml)

Volume pressure diagram (loop)

- Phase I: Period of Filling.
- begins at a ventricular volume of about 50 ml and a diastolic pressure of 2 to 3 mm Hg.
- The amount of blood that remains in the ventricle after the previous heartbeat, 50 ml, is called the end-systolic volume.
- As venous blood flows into the ventricle from the left atrium, the ventricular volume normally increases to about 120 ml, called the end-diastolic volume, an increase of 70 ml.

Volume pressure diagram (loop)

- Phase II: Period of Isovolumic Contraction.
- During isovolumic contraction, the volume of the ventricle does not change because all valves are closed.
- However, the pressure inside the ventricle increases to equal the pressure in the aorta, at a pressure value of about 80 mm Hg.

Volume pressure diagram (loop)

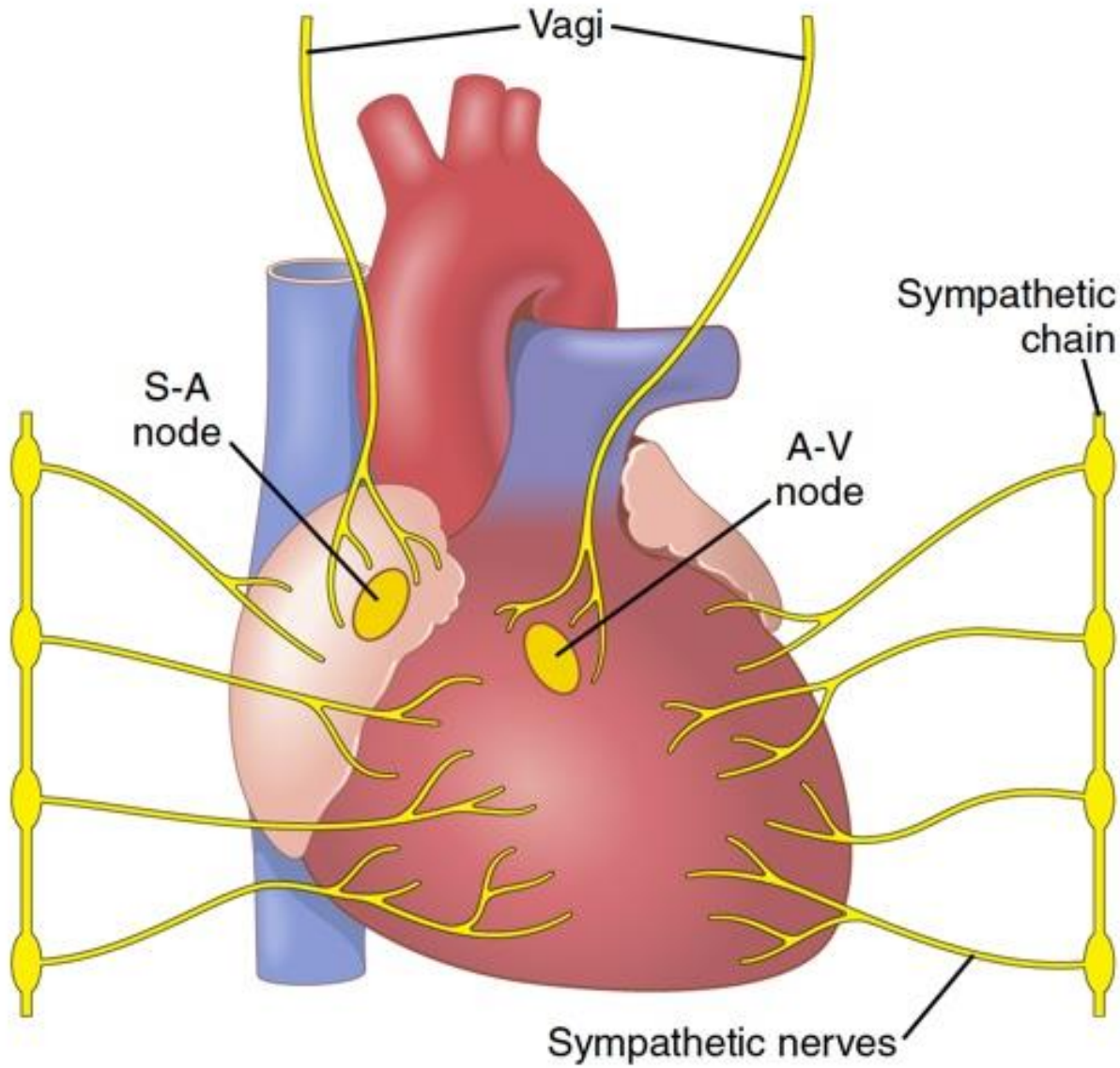
- Phase III: Period of Ejection.
- During ejection, the systolic pressure rises even higher because of still more contraction of the ventricle.
- At the same time, the volume of the ventricle decreases because the aortic valve has now opened, and blood flows out of the ventricle into the aorta.

Volume pressure diagram (loop)

- Phase IV: Period of Isovolumic Relaxation.
- At the end of the period of ejection, the aortic valve closes, and the ventricular pressure falls back to the diastolic pressure level, without any change in volume.
- Thus, the ventricle returns to its starting point.

Regulation of heart pumping

- The basic mechanisms for regulating heart pumping are as follows:
- (1) intrinsic cardiac pumping regulation in response to changes in volume of blood flowing into the heart.
- (2) control of heart rate and heart strength by the autonomic nervous system.


Vagi
Sympathetic chain
S-A node
A-V node
Sympathetic nerves

Regulation: autonomic nervous system

- The pumping effectiveness of the heart is controlled by the sympathetic and parasympathetic (vagus) nerves, which abundantly supply the heart.

Sympathetic stimulation effects

- The overall effect of sympathetic stimulation on the heart is to improve its effectiveness as a pump by increasing HR, decreasing the delay between atrial and ventricular contraction, decreasing conduction time throughout the heart, increasing the force of contraction, and speeding up the relaxation process.

Parasympathetic stimulation

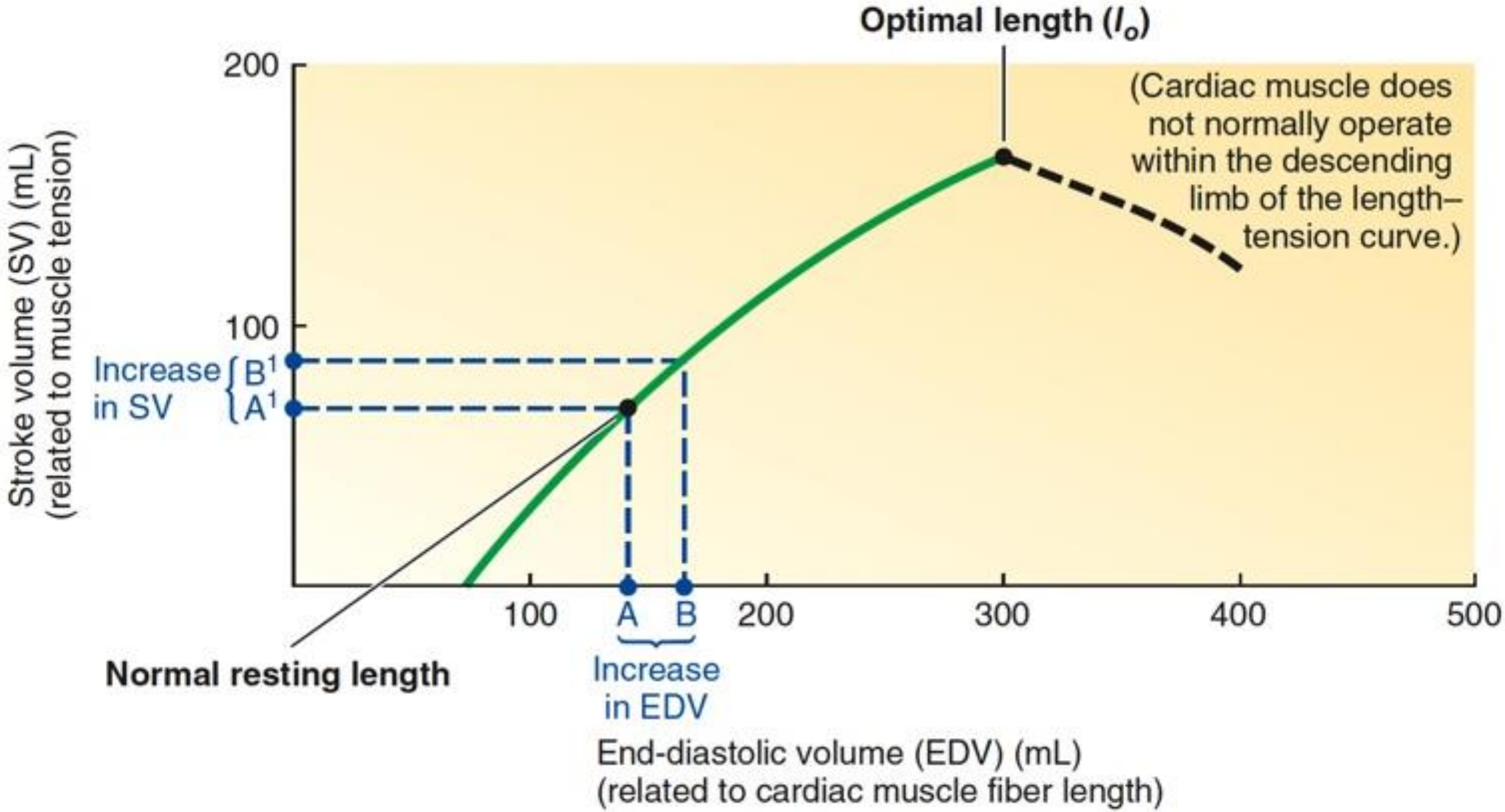
- The vagal fibers are distributed mainly to the atria and not much to the ventricles, where the power contraction of the heart occurs.
- This distribution explains why the effect of vagal stimulation is mainly to decrease the heart rate rather than to decrease greatly the strength of heart contraction.
- Nevertheless, the great decrease in heart rate, combined with a slight decrease in heart contraction strength, can decrease ventricular pumping by 50% or more.

Intrinsic regulation: Frank-Starling law

- The intrinsic ability of the heart to adapt to increasing volumes of inflowing blood is called the Frank-Starling mechanism of the heart.

- The more the heart muscle is stretched during filling, the greater is the force of contraction, and the greater is the quantity of blood pumped into the aorta.

- Within physiological limits, the heart pumps all the blood that returns to it by way of the veins.


Optimal length (l_o)
(Cardiac muscle does not normally operate within the descending limb of the length–tension curve.)
Stroke volume (SV) (mL)
(related to muscle tension)
200
100
Increase in SV
B1
A1
100
A
B
200
300
400
500
Normal resting length
Increase in EDV
End-diastolic volume (EDV) (mL)
(related to cardiac muscle fiber length)

Intrinsic regulation

- Intrinsic control of stroke volume, which refers to the heart's inherent ability to vary SV, depends on the direct correlation between end-diastolic volume (EDV) and SV.
- As more blood returns to the heart, the heart pumps out more blood, but the relationship is not as simple as it might seem because the heart does not eject all the blood it contains.
- This intrinsic control depends on the length–tension relationship of cardiac muscle, which is similar to that of skeletal muscle.
- For skeletal muscle, the resting muscle length is approximately the optimal length at which maximal tension can be developed during a subsequent contraction. When the skeletal muscle is longer or shorter, the subsequent contraction is weaker.

Intrinsic regulation

- For cardiac muscle, the resting cardiac muscle fiber length is less than optimal length. Therefore, the length of cardiac muscle fibers normally varies along the ascending limb of the length–tension curve.
- An increase in cardiac muscle fiber length, by moving closer to optimal length, increases the contractile tension of the heart on the following systole.
- Unlike in skeletal muscle, the length–tension curve of cardiac muscle normally does not operate at lengths that fall within the region of the descending limb. That is, within physiologic limits, cardiac muscle does not get stretched beyond its optimal length to the point that contractile strength diminishes with further stretching.

Intrinsic regulation

- What causes cardiac muscle fibers to vary in length before contraction?

- The main determinant of cardiac muscle fiber length is the degree of diastolic filling.
- The greater the diastolic filling, the larger the EDV, and the more the heart is stretched. The more the heart is stretched, the longer the cardiac fibers before contraction. The increased length results in a greater force on the subsequent cardiac contraction and thus in a greater SV.

Intrinsic regulation

- This intrinsic relationship between EDV and SV is known as the Frank–Starling law of the heart. Stated simply, the law says that the heart normally pumps out during systole the volume of blood returned to it during diastole; increased venous return results in increased SV.
- The extent of filling is referred to as the preload because it is the workload imposed on the heart before contraction begins.

Intrinsic regulation

- Advantages of the Cardiac Length–Tension Relationship

- The built-in relationship matching SV with venous return has two important advantages.
- First, this intrinsic mechanism equalizes output between the right and the left sides of the heart so that blood pumped out by the heart is equally distributed between the pulmonary and systemic circulations.

Intrinsic regulation

- If, for example, the right side of the heart ejects a larger SV, more blood enters the pulmonary circulation, so venous return to the left side of the heart increases accordingly.
- The increased EDV of the left side of the heart causes it to contract more forcefully, so it too pumps out a larger SV. In this way, output of the two ventricular chambers is kept equal.
- If such equalization did not happen, too much blood would be dammed up in the venous system before the ventricle with the lower output.

Intrinsic regulation

- Second, when a larger CO is needed, as during exercise, venous return is increased by the sympathetic nervous system constricting the veins to drive blood forward and by the contracting muscles compressing the veins, which squeezes more blood toward the heart.
- The resulting increase in EDV automatically increases SV correspondingly.
- Because exercise also increases HR, these two factors act together to increase CO so that more blood can be delivered to the exercising muscles.

Length-tension relationship

- Although the length–tension relationship in cardiac muscle fibers depends to a degree on the extent of overlap of thick and thin filaments, similar to the length–tension relationship in skeletal muscle, the key factor relating cardiac muscle fiber length to tension development is the dependence of myofilament Ca sensitivity on the fiber's length.
- Specifically, as a cardiac muscle fiber is stretched as a result of greater ventricular filling, its myofilaments are pulled closer together side by side.
- As a result of this reduction in distance between the thick and thin filaments, more cross-bridge interactions between myosin and actin can take place when Ca pulls the troponin– tropomyosin complex away from actin's cross-bridge binding sites—that is, myofilament Ca sensitivity increases.
- Thus, the length–tension relationship in cardiac muscle depends not on muscle fiber length per se but on the resultant variations in the lateral spacing between the myosin and actin filaments.

Sympathetic stimulation

- Sympathetic stimulation and epinephrine enhance the heart's contractility, which is the strength of contraction at any given EDV.
- In other words, on sympathetic stimulation the heart contracts more forcefully and squeezes out a greater percentage of the blood it contains, leading to more complete ejection.
- This increased contractility results from the increased Ca influx triggered by norepinephrine and epinephrine.
- The extra cytosolic Ca lets the myocardial fibers generate more force through greater cross-bridges.

Sympathetic stimulation

- In effect, sympathetic stimulation shifts the Frank–Starling curve to the left.
- Depending on the extent of sympathetic stimulation, the curve can be shifted to varying degrees, up to a maximal increase in contractile strength of about 100% greater than normal.

Other intrinsic mechanisms

- In addition to the important effect of lengthening the heart muscle, another factor increases heart pumping when its volume is increased.
- Stretch of the right atrial wall directly increases the heart rate by 10% to 20%, which also helps increase the amount of blood pumped each minute, although its contribution is much less than that of the Frank-Starling mechanism.
- A stretch of the atrium also activates stretch receptors and a nervous reflex, the Bainbridge reflex, that is transmitted by the vagus nerve and may increase heart rate an additional 40% to 60%.

Preload

- Preload:
- the degree of tension on the muscle when it begins to contract.

- For cardiac contraction:
- Preload is usually considered to be the end-diastolic pressure when the ventricle has become filled.

Afterload

- Afterload:
- the load against which the muscle exerts its contractile force.

- For cardiac contraction:
- Afterload of the ventricle is the pressure in the aorta leading from the ventricle (or the resistance).

Venous return

- Venous return is important because it is the quantity of blood flowing from the veins into the right atrium each minute.

- The venous return and the cardiac output must equal each other except for a few heartbeats when blood is temporarily stored in or removed from the heart and lungs.

Thank you