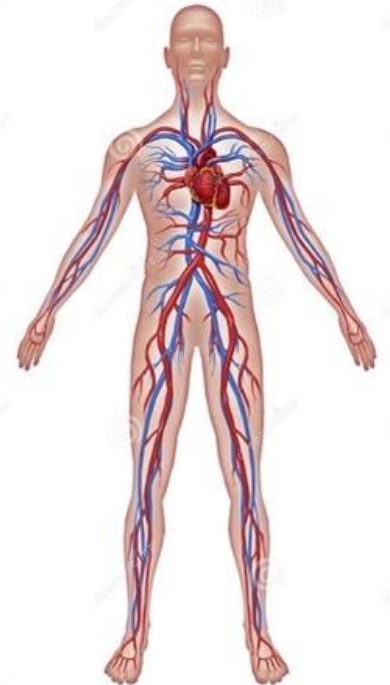


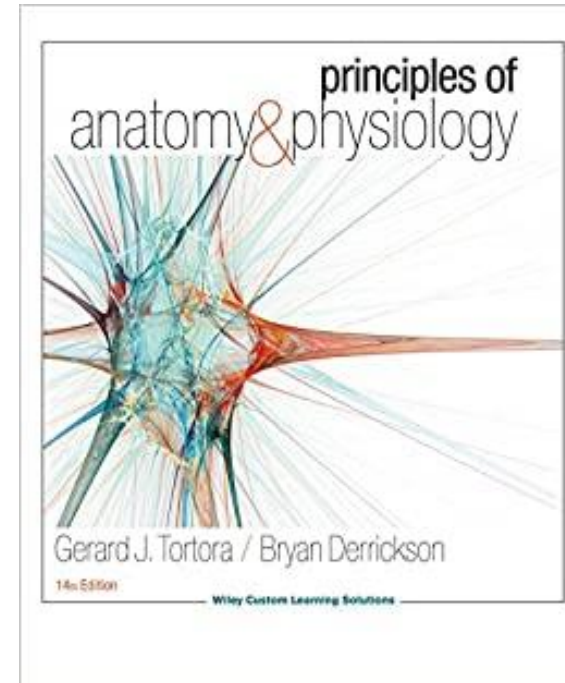
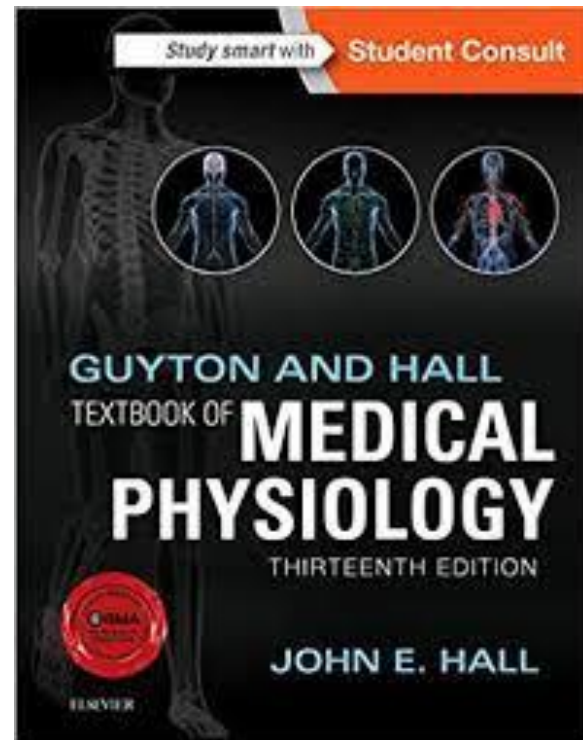
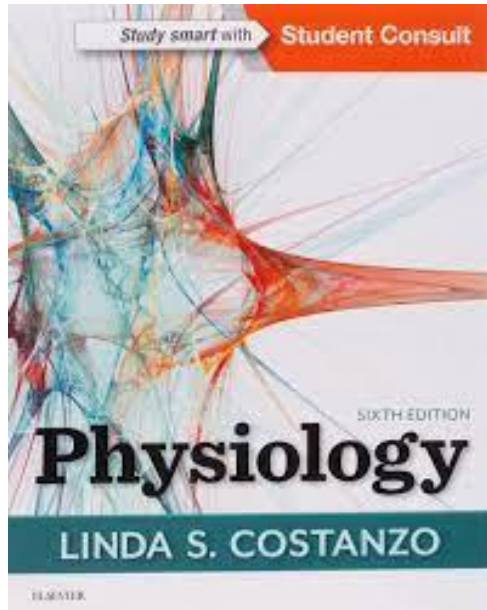
Cardiovascular Physiology

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References



9TH
Edition

Human Physiology From Cells to Systems

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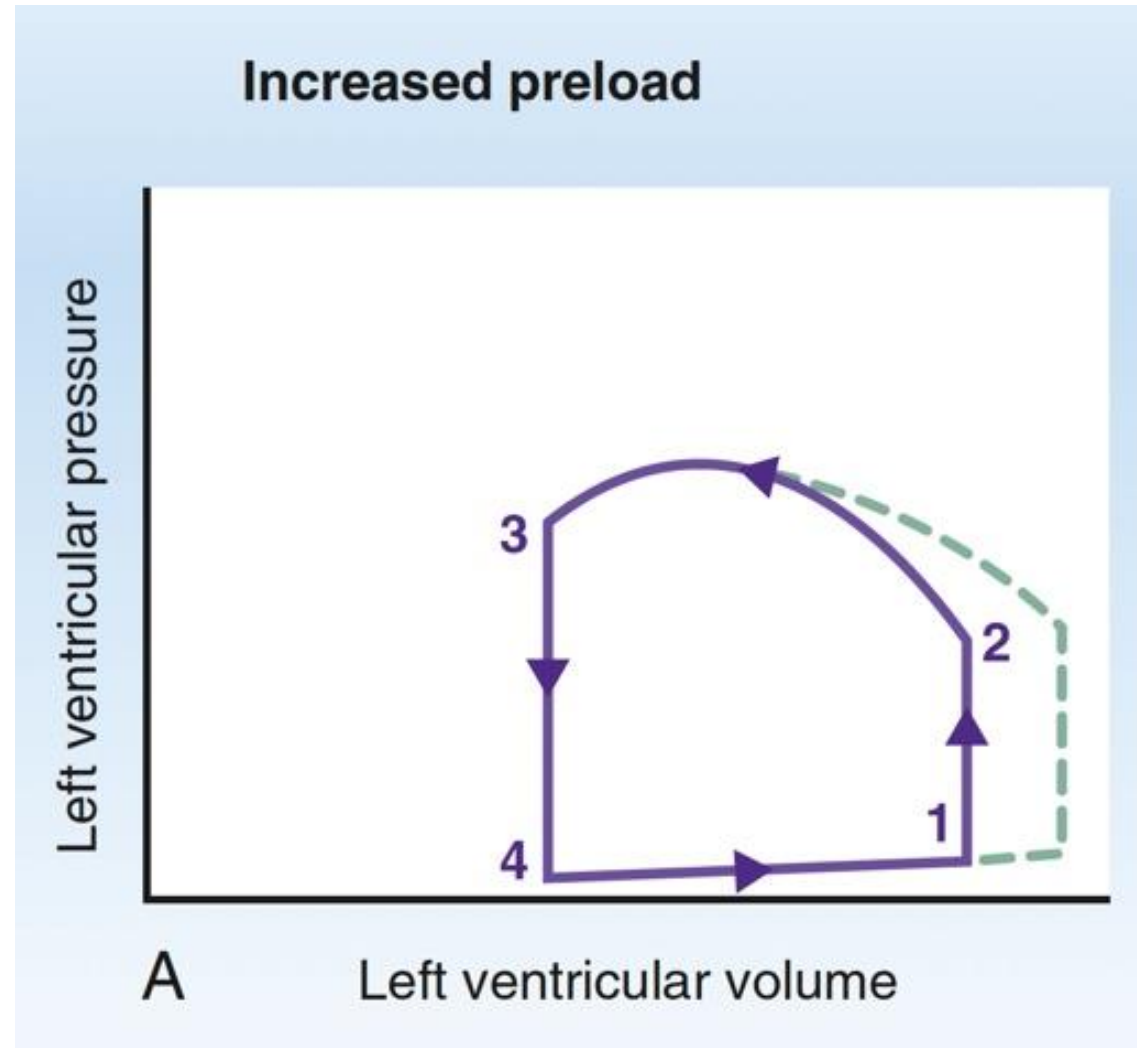
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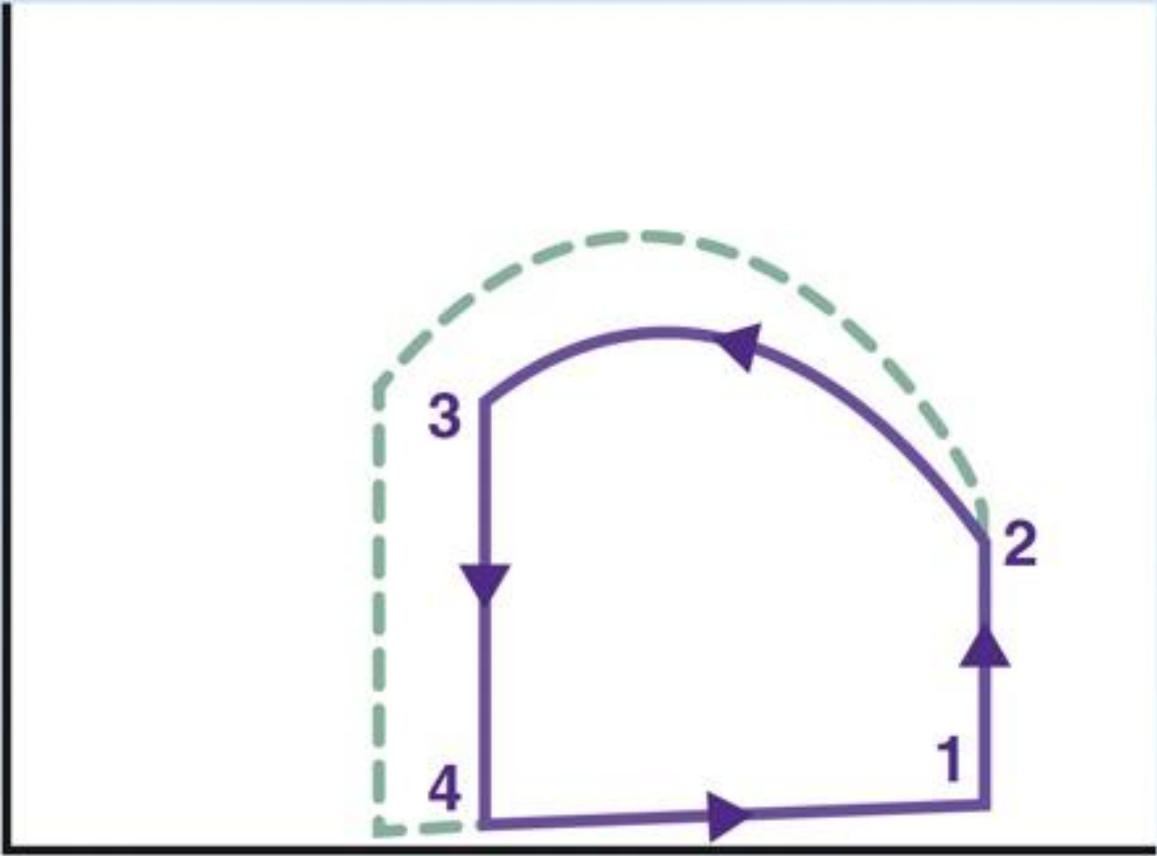
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Mechanical events of the cardiac cycle-2

Changes in ventricular volume-pressure loop



Increased contractility



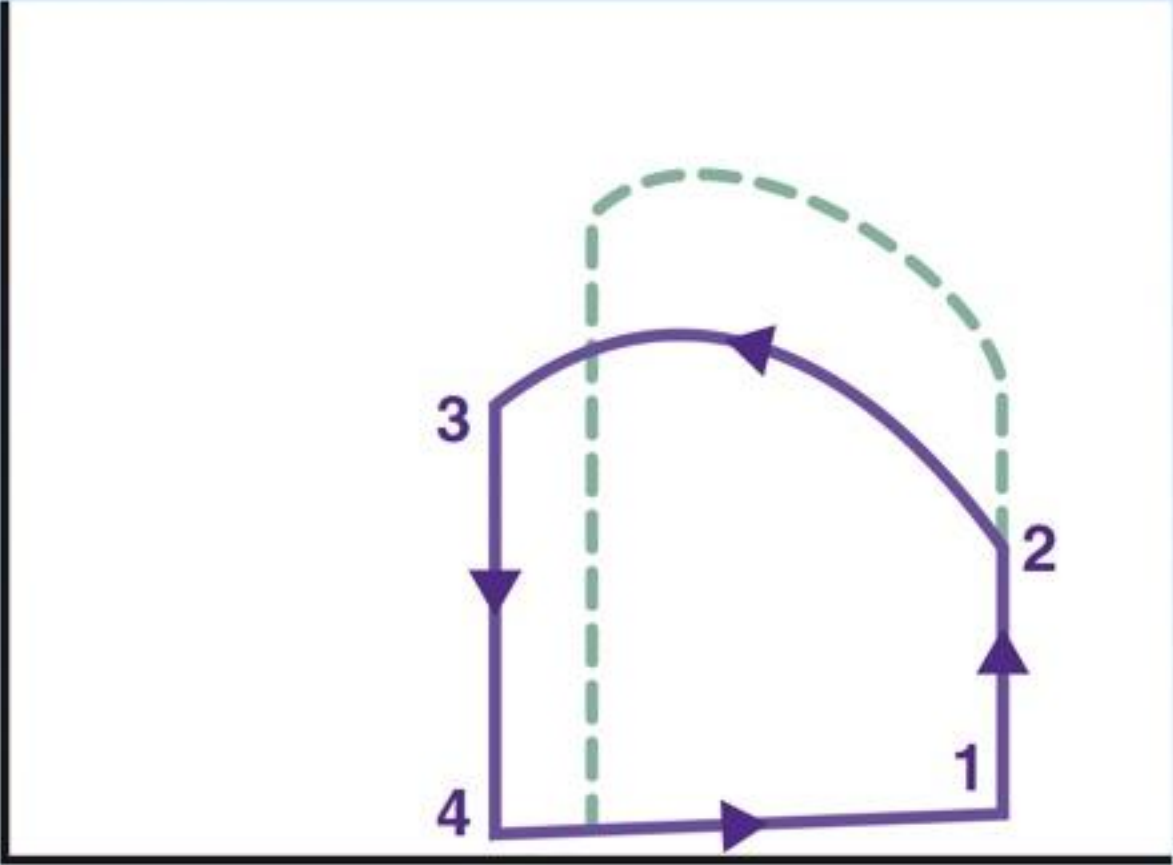
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Left ventricular volume

Stroke volume

- Sympathetic stimulation increases SV not only by strengthening cardiac contractility but also by enhancing venous return.
- Sympathetic stimulation constricts the veins, which squeezes more blood forward from the veins to the heart, increasing the EDV and subsequently increasing SV even further.

Increased afterload



B

Left ventricular volume

Afterload

- If arterial blood pressure is chronically elevated (high blood pressure) or if the exit valve is stenotic, the ventricle must generate more pressure to eject blood.
- The heart may be able to compensate for a sustained increase in afterload by hypertrophying, that is, by increasing the thickness of the cardiac muscle fibers.
- This enables it to contract more forcefully and maintain a normal SV despite an abnormal impediment to ejection.
- However, a diseased heart or a heart weakened with age may not be able to compensate completely; in that case, heart failure ensues.

Cardiac output

- Cardiac output (CO) is the volume of blood pumped by each ventricle per minute (not the total amount of blood pumped by the heart).
- During any period, the volume of blood flowing through the pulmonary circulation is the same as the volume flowing through the systemic circulation.
- This is the quantity of blood that flows through the circulation.

Cardiac output

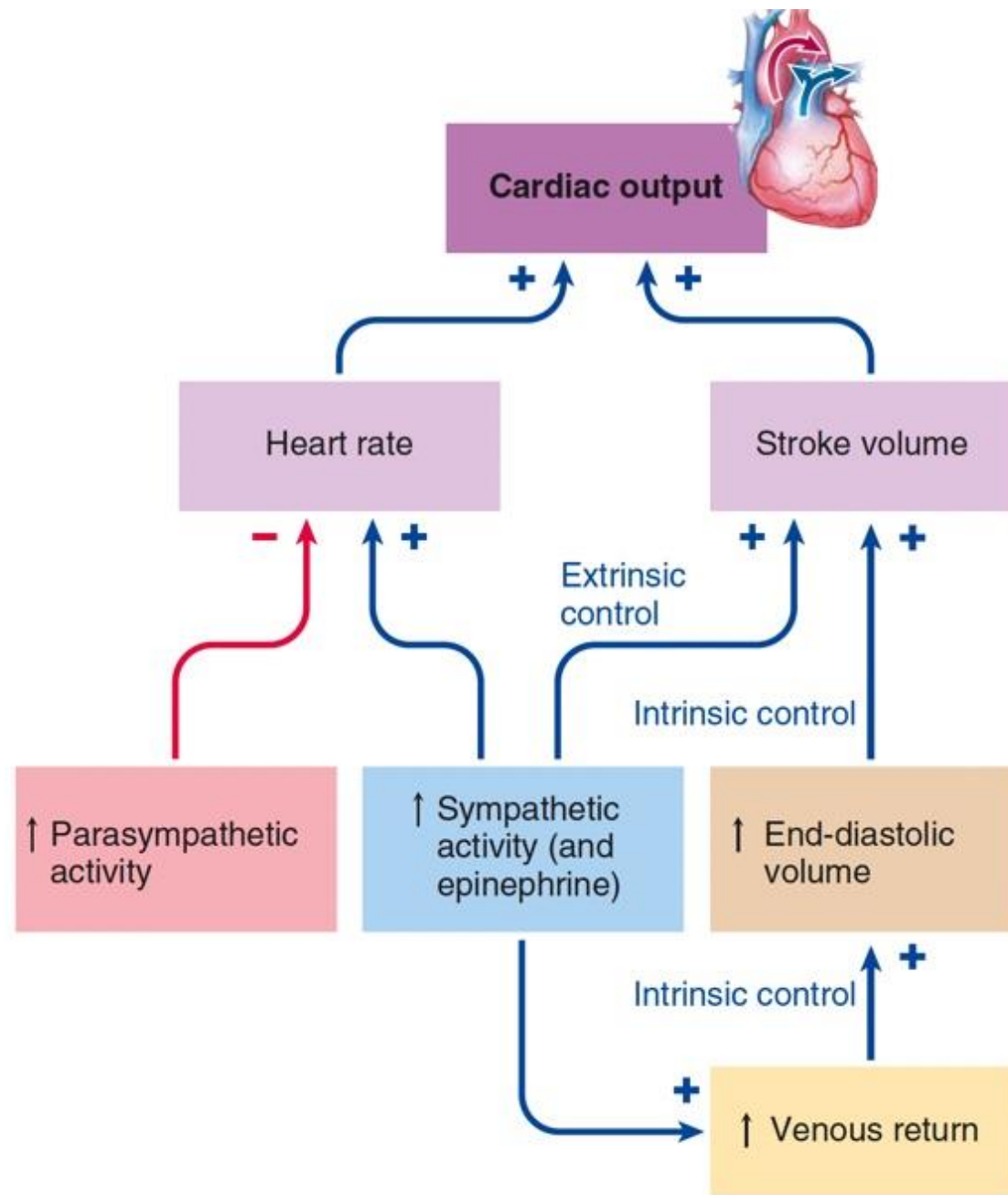
- The two determinants of cardiac output are heart rate (HR) (beats per minute) and stroke volume (SV) (volume of blood pumped per beat or stroke).
- $CO = HR \times SV$
- The difference between the cardiac output at rest and the maximum volume of blood the heart can pump per minute is called the cardiac reserve.

Cardiac output

- The following factors, among others, directly affect cardiac output:
 - (1) the basic level of body metabolism;
 - (2) whether the person is exercising;
 - (3) the person's age;
 - (4) the size of the body.

Cardiac index

- cardiac output increases approximately in proportion to the surface area of the body.
- Therefore, cardiac output can be stated as cardiac index, which is the cardiac output per square meter of body surface area.
- declining cardiac index is indicative of declining activity and/or declining muscle mass with age.



Heart failure

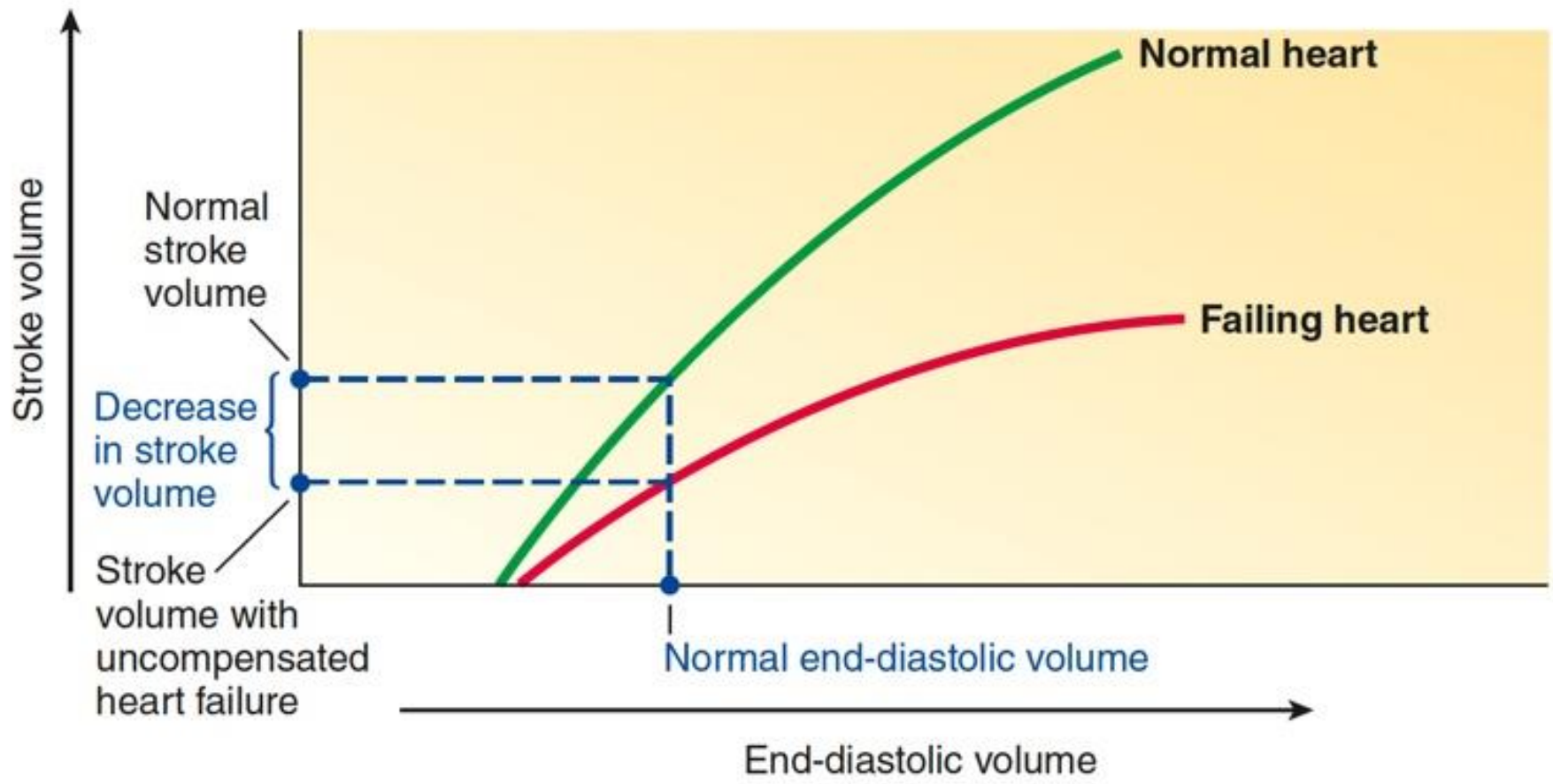
- Heart failure (HF) is the inability of CO to keep pace with the body's demands for supplies and removal of wastes.
- Heart failure can be of two types: systolic HF, in which the heart has difficulty pumping blood out, or diastolic HF, in which the heart has trouble filling.

Ejection fraction

- The ejection fraction is the ratio of stroke volume to end-diastolic volume (ejection fraction = SV/EDV); that is, it is the proportion of the blood in the ventricle that is pumped out.
- The ejection fraction is often used clinically as an indication of contractility.
- A healthy heart normally has an ejection fraction of 50% to 75% under resting conditions and may go as high as 90% during strenuous exercise, but a failing heart may pump out 30% or less.

Systolic Heart failure

- The prime defect in systolic HF is decreased cardiac contractility—that is, weakened cardiac muscle cells contract less effectively, resulting in a greatly reduced ejection fraction.
- With systolic HF, the intrinsic ability of the heart to develop pressure and eject a SV is reduced so that the heart operates on a lower length–tension curve.
- The Frank–Starling curve shifts downward and to the right such that, for a given EDV, a failing heart pumps out a smaller SV than a normal healthy heart does.



Compensated Systolic Heart failure

- In the early stages of systolic HF, two major compensatory measures help restore SV to normal.
- First, sympathetic activity to the heart is increased, which increases heart contractility toward normal.
- Sympathetic stimulation can help compensate only for a limited time, however, because the heart becomes less responsive to norepinephrine after prolonged exposure, and furthermore, norepinephrine stores in the heart's sympathetic nerve terminals become depleted.

Compensated Systolic Heart failure

- Second, when CO is reduced, the kidneys, in a compensatory attempt to improve their reduced blood flow, retain extra salt and water in the body during urine formation to expand the blood volume.
- The increase in circulating blood volume increases the EDV. The resultant stretching of the cardiac muscle fibers enables the weakened heart to pump out a normal SV. The heart is now pumping out the blood returned to it but is operating at a greater cardiac muscle fiber length.

Decompensated Systolic Heart failure

- As the disease progresses and heart contractility deteriorates further, the heart reaches a point at which it can no longer pump out a normal SV despite compensatory measures. At this point, the heart slips from compensated HF into a state of decompensated HF.
- Now the cardiac muscle fibers are stretched to the point that they are operating in the descending limb of the length–tension curve.
- Forward failure occurs as the heart fails to pump an adequate amount of blood forward to the tissues because the SV becomes progressively smaller.

Decompensated Systolic Heart failure

- Backward failure occurs simultaneously as the failing heart cannot pump out all of the blood returned to it (SV cannot keep pace with venous return) so that the returning blood continues to dam up in the venous system.
- Congestion of blood in the venous system behind a failing ventricle is the reason this condition is sometimes termed congestive heart failure.
- Left-sided failure has more serious consequences than right-sided failure.

Decompensated Systolic Heart failure

- Backward failure of the left side leads to pulmonary edema (excess tissue fluid in the lungs) because blood dams up in the lungs. This fluid accumulation in the lungs reduces exchange of O₂ and CO₂, reducing arterial oxygenation and elevating levels of acid-forming CO₂ in the blood.
- In addition, one of the more serious consequences of left-sided forward failure is inadequate blood flow to the kidneys, which causes a twofold problem.
- First, vital kidney function is depressed;

Decompensated Systolic Heart failure

- Second, the kidneys retain even more salt and water in the body during urine formation as they try to expand the blood volume even further to improve their reduced blood flow.
- Excessive fluid retention worsens the already existing problems of venous congestion.
- Treatment of congestive heart failure therefore includes measures that reduce salt and water retention and increase urinary output and drugs that enhance the contractile ability of the weakened heart—digitalis, for example.
- Digitalis increases cardiac contractility by causing accumulation of cytosolic Ca.

Diastolic Heart failure

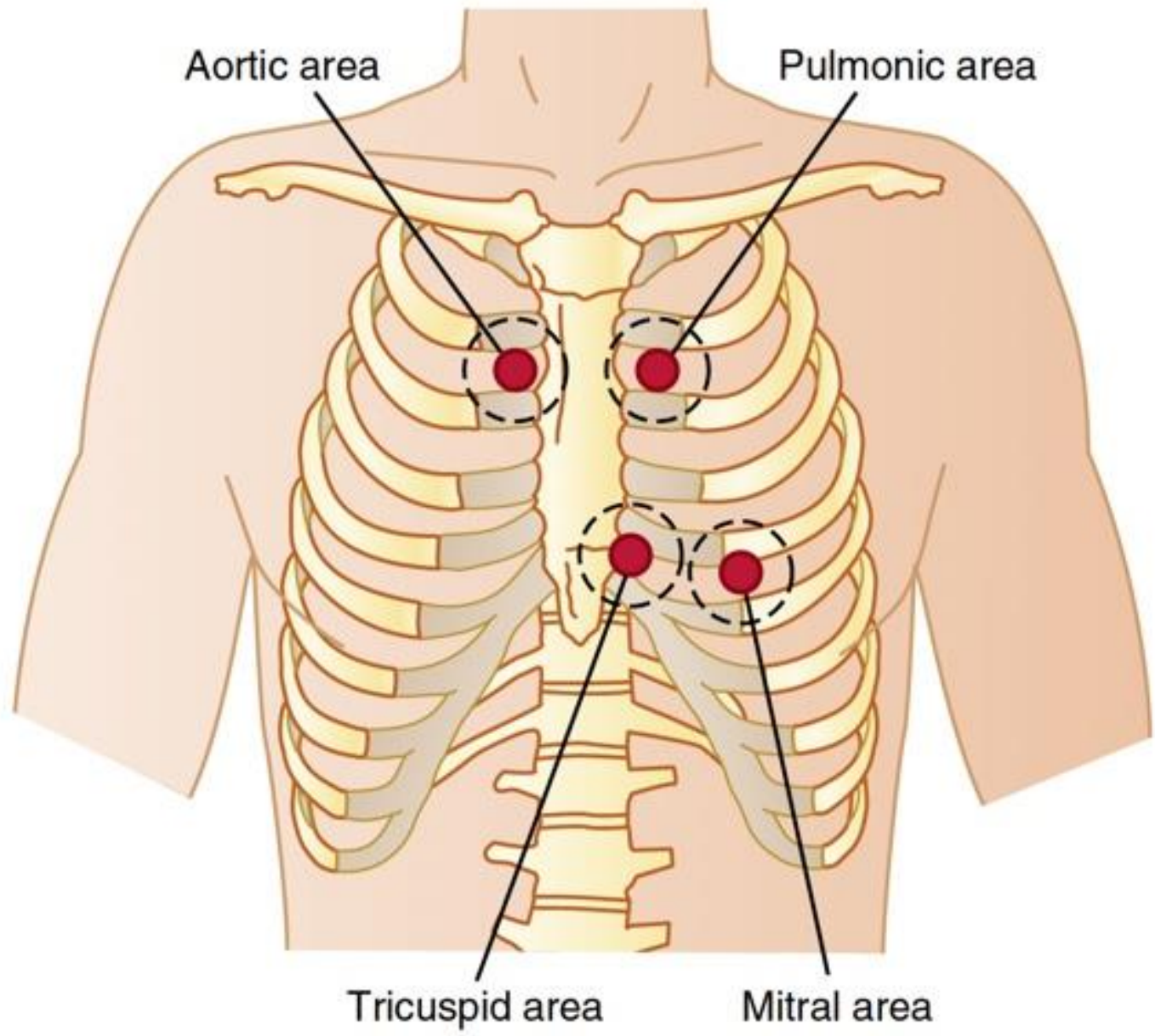
- With the more recently recognized diastolic HF, the ventricles do not fill normally either because the heart muscle does not adequately relax between beats or because it stiffens and cannot expand as much as usual.
- The heart can pump properly and the ejection fraction is normal. For this reason, diastolic HF is alternatively called heart failure with preserved ejection fraction.
- However, even though the ejection fraction is normal (that is, the ventricles pump out a normal percentage of the blood present in their chambers), less blood than normal is pumped out by a diastolic failing heart because the ventricles are inadequately filled with blood.

Diastolic Heart failure

- Both forward and backward failure, including congestive heart failure, can result, so symptoms are similar to systolic HF.
- No drugs are available yet that reliably help the heart relax, so treatment of diastolic HF is aimed at relieving symptoms, halting underlying causes, or lessening aggravating factors, such as by controlling high blood pressure.

Heart sounds

- when the valves close, the vanes of the valves and the surrounding fluids vibrate under the influence of sudden pressure changes, giving off sound that travels in all directions through the chest.
- When the ventricles contract, one first hears a sound caused by closure of the A-V valves.
- The vibration pitch is low and relatively long-lasting and is known as the first heart sound (S1).
- When the aortic and pulmonary valves close at the end of systole, one hears a rapid snap because these valves close rapidly, and the surroundings vibrate for a short period.
- This sound is called the second heart sound (S2).



Aortic area

Pulmonic area

Tricuspid area

Mitral area

Valve stenosis

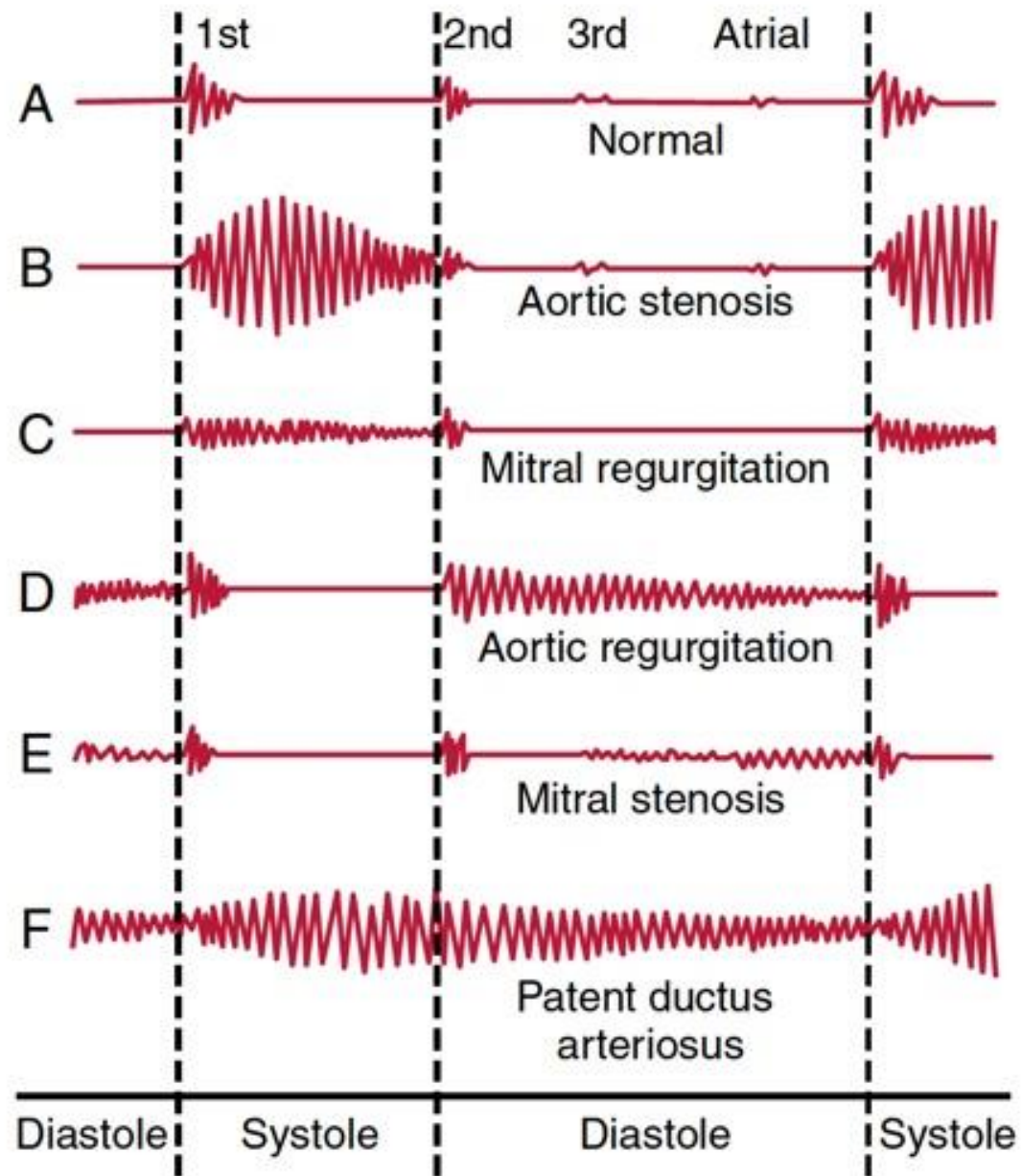
- Abnormal heart sounds, or murmurs, are usually (but not always) associated with cardiac disease. Result from turbulent blood flow rather than laminar flow.
- A stenotic valve is a stiff, narrowed valve that does not open completely.
- Blood must be forced through the constricted opening at tremendous velocity, resulting in turbulence that produces an abnormal whistling sound.

Valve incompetency

- An insufficient, or incompetent, valve is one that cannot close completely because the valve edges do not fit together properly.
- Turbulence is produced when blood flows backward through the insufficient valve and collides with blood moving in the opposite direction, creating a swishing or gurgling murmur.
- Such backflow of blood is known as regurgitation. An insufficient heart valve is often called a leaky valve because it lets blood leak back through when the valve should be closed.
- rheumatic fever

Murmurs

- The valve involved and the type of defect can usually be detected by the timing and location of the murmur.
- The timing of the murmur refers to the part of the cardiac cycle during which the murmur is heard.
- Recall that the first heart sound signals the onset of ventricular systole and the second heart sound signals the onset of ventricular diastole.
- Thus, a murmur between the first and the second heart sounds is a systolic murmur.
- A diastolic murmur occurs between the second and the first heart sounds.
- The sound of the murmur characterizes it as either a stenotic murmur or an insufficient murmur.



S3

- The rapid flow of blood from the atria to the ventricles produces the third heart sound (S3).
- normal in children.
- but in adults indicates volume overload, as in congestive heart failure or advanced mitral or tricuspid regurgitation.

S4

- The fourth heart sound (S4) is not audible in normal adults, although it may be heard in ventricular hypertrophy, where ventricular compliance is decreased.
- When present, S4 coincides with atrial contraction. The sound is caused by the atrium contracting against, and trying to fill, a stiffened ventricle.

Spilt

- Closure of the AV valves produces the first heart sound (S1), which may be split because the mitral valve closes slightly before the tricuspid valve.
- The aortic valve closes slightly before the pulmonic valve, producing the second heart sound (S2).
- Inspiration delays closure of the pulmonic valve and causes splitting of the second heart sound; because the associated decrease in intrathoracic pressure produces an increase in venous return to the right side of the heart.
- The resulting increase in right ventricular end-diastolic volume causes an increase in right ventricular stroke volume, and prolongs right ventricular ejection time; so delays closure of the pulmonic valve relative to the aortic valve.

Thank you