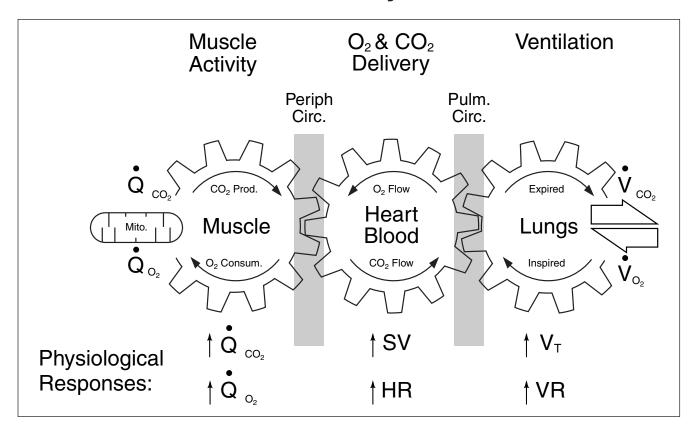
Cardiac Hemodynamics



Cardiac hemodynamics refers to the forces involved in circulating blood through the body.

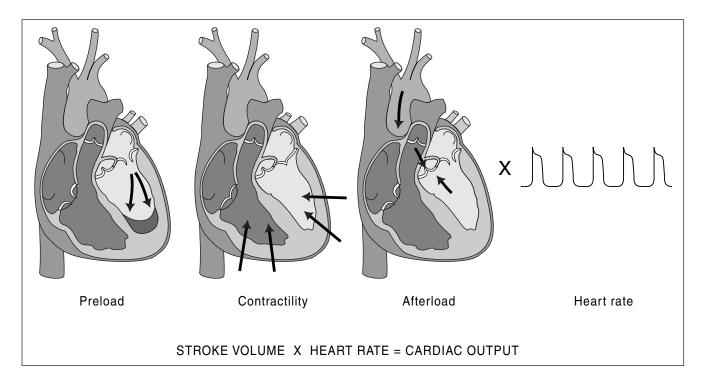
The heart adapts its *hemodynamic* performance to the needs of the body for oxygen and nutrients, increasing its output of blood when muscles are working and decreasing it when the body is at rest. The heart also adapts to changes that occur as the cardiovascular system ages and in the presence of disease.

Notes

The forces that keep blood flowing under various conditions are known as hemodynamics.

Question:		
The <i>forces</i> that circulate blood are called		

Cardiac Output



Cardiac output is the volume of blood ejected by the heart in one minute.

Cardiac output = stroke volume x heart rate

Heart rate is the number of beats per minute. Stroke volume is the amount of blood ejected from the ventricles per heartbeat. Thus, if the heart rate is 60 beats per minute and the stroke volume is 75 milliliters, the cardiac output is:

60 bpm x 75 ml = 4,500 milliliters/minute - or 4.5 liters/minute.

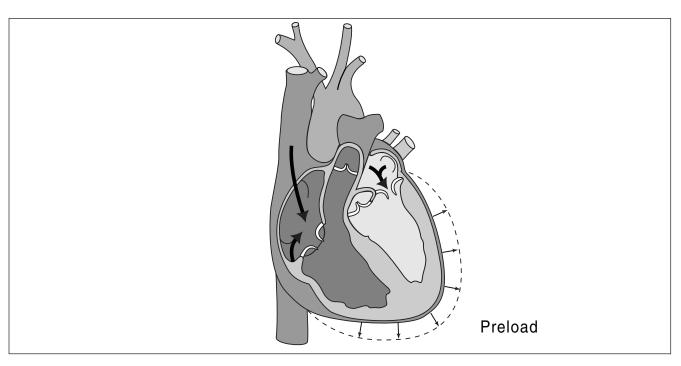
Normal cardiac output in an adult at rest is 4 to 6 liters per minute, depending on the size of the individual. Each ventricle ejects about 65% of its volume per stroke, but this amount varies with changes in metabolic need.

Notes

Cardiac output is the product of heart rate and stroke volume.

Questions:	
The volume of blood ejected per heartbeat is	
The volume of blood ejected in one minute is	_•

Preload



Preload is the expansion / stretching of the myocardium to produce a more forceful contraction.

The more the cardiac muscle is stretched (within limits), the more force it can deliver per contraction, and the more blood it can pump into circulation. This is stated in the *Frank-Starling law*: Within limits, an increase in *preload* leads to a more powerful contraction and an increase in the volume of blood ejected.

Preload is influenced by the volume of blood that enters the ventricle during diastole. Several factors improve filling: an adequate rate of venous return, sufficient filling time, atrial kick, and properly functioning atrioventricular valves.

As the ventricles fill, ventricular pressure rises; but if the preload pressure is too high, the muscle begins to lose elasticity. The preload volume of blood is assessed by measuring the *end-diastolic pressure* of the left ventricle. Normal values are 4-12 millimeters mercury (Hg).

Notes

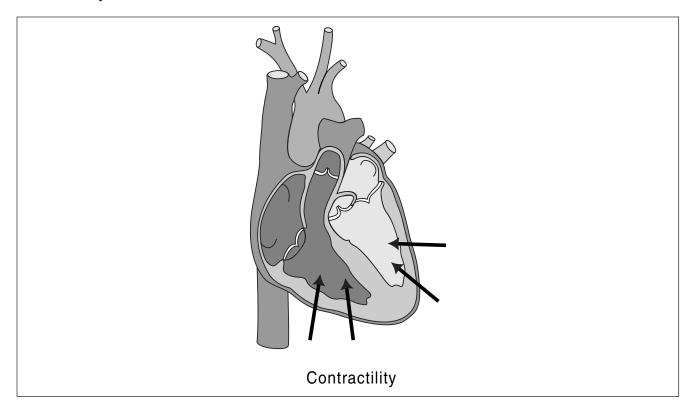
Preloading the ventricle with blood to the point at which muscle fibers stretch produces a more forceful contraction and increases the volume of blood ejected.

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The myocardial stretching that produces a strong contraction is _

Answer: preload

Contractility



Contractility is the ability of the myocardium to contract and relax. An increase in contractility leads to an increase in stroke volume independently of preload and afterload.

The *contractility* of the myocardium is controlled by the forces generated by myofibrils - the strings of protein that shorten when stimulated. Efficient contraction (shortening) and complete relaxation of these muscle fibers results in a longer diastolic filling time and less resistance to filling.

The contractile state of the myocardium is expressed clinically as the *ejection fraction* - the ratio between the stroke volume and end-diastolic volume for the left ventricle:

Ejection fraction = stroke volume
end-diastolic volume

The normal ejection fraction is about 65%.

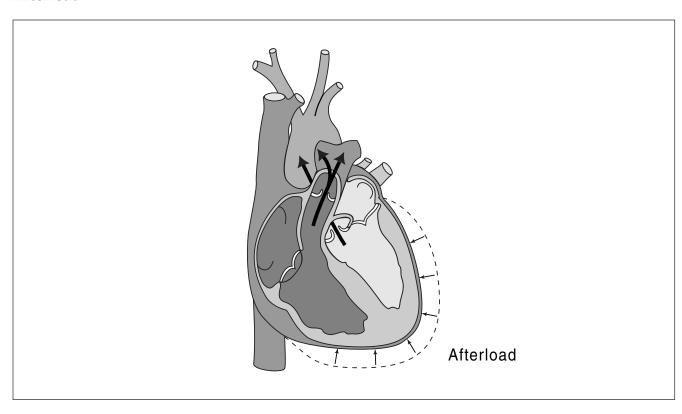
Notes

An increase in contractility leads to an increase in stroke volume.

Q	u	es	st	io	n	ľ

The ability of the heart to contract and relax is_

Afterload



Afterload is the resistance the left ventricle must overcome to eject blood into circulation.

Pressure in the systemic arteries is fairly high so that blood can be carried to distant parts of the body. The left ventricle works against this arterial (aortic) pressure when pumping its volume of blood. *Afterload* is the force that resists the ejection of blood, so an increase in afterload leads to a decrease in stroke volume.*

Afterload is kept at a healthy level by several factors: a properly functioning aortic valve, an aorta that expands easily to accommodate blood, low peripheral vascular resistance, normal blood volume, and low viscosity blood (flows easily).

Clinicians determine if afterload is within normal limits by measuring the *aortic pressure* - that is, the systemic blood pressure at aortic valve opening. Normal values are 90 -140 millimeters Hg.

Notes

Afterload is the pressure against which the left ventricle ejects blood. An increase in afterload leads to a decrease in stroke volume.

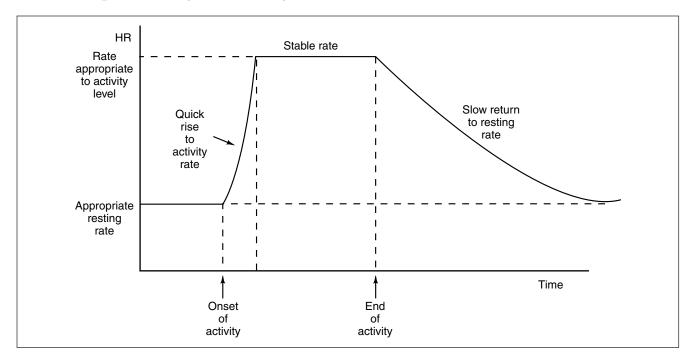
Question:

The force against which the left ventricle ejects blood is_

Answer: afterload

^{*} The healthy heart balances the forces of preload and afterload to maintain adequate cardiac output.

Cardiac Response to Physical Activity



The heart responds quickly to an increase in activity by increasing its rate of contraction. This increases cardiac output and provides working muscles with the oxygen they need.

Cardiac output increases in proportion to increases in heart rate and is limited by the maximum rate an individual can attain. As heart rate increases, the diastolic phase of the cardiac cycle becomes progressively shorter. This decreases the time for ventricular filling. When the heart rate reaches its upper limit, diastolic filling becomes insufficient to further increase cardiac output. In an average healthy person, an increase in heart rate can double or triple cardiac output.*

The maximum heart rate attainable varies with age and conditioning. A fit, young adult may have a maximum rate of 180 to 200 bpm. A healthy 70-year-old typically has a maximum rate of about 145 bpm.

The ability of the heart to increase its pacing rate in response to increased metabolic need is known as *chronotropic competence*.

An increase in heart rate is the fastest way to increase cardiac output.

Question:

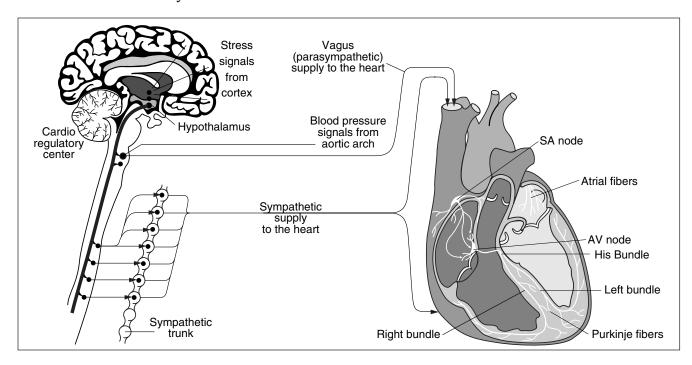
Cardiac output increases in proportion to increases in _

Answer: heart rate

Notes

^{*} Well-conditioned athletes may produce four to five-fold increases in cardiac output because their resting rates are lower and ventricular end-diastolic volume is higher.

Role of the Nervous System



The heart rate and work capacity of the heart are regulated by the autonomic nervous system.

Sympathetic nerves increase cardiac activity. When sympathetic nerves in the heart are stimulated, the sinus node sets a faster pace, conduction speed increases, and contractions become more forceful. Parasympathetic nerves inhibit cardiac activity. When the vagus nerve - the parasympathetic pathway in the heart is stimulated, the sinus node sets a slower pace, conduction speed slows, and contractions become less forceful.

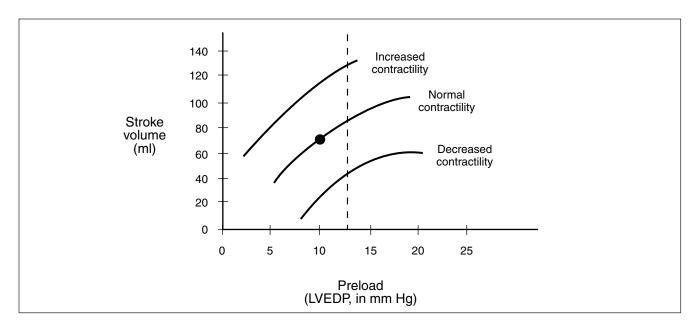
Stimulation of sympathetic nerve endings causes the release of *catecholamines* (epinephrine and norepinephrine) at the sinus node (which increase pacing rate). There is also an increase in catecholamines circulated to the heart. These dilate the coronary arteries and increase oxygen delivery to the myocardium, enabling it to contract more forcefully.

Notes

The heart rate and work capacity of the heart are regulated by sympathetic and parasympathetic nerves.

Questions:	
	increase cardiac activity.
	inhibit cardiac activity.

Stroke Volume Factors



The heart can increase cardiac output by increasing the amount of blood ejected per stroke.

Several factors enable the left ventricle to pump more blood with each stroke (normally about 65% of the volume is pumped per contraction). Higher levels of circulating catecholamines *increase contractility*, so a higher percentage of blood is ejected per contraction (see figure). Stimulation by sympathetic nerves causes the veins to constrict and return more blood to the heart. The higher rate of venous return *increases preload*. Various stimuli cause the arteries to dilate, which reduces vascular resistance and therefore *decreases afterload*.

An increase in stroke volume can increase cardiac output by about 50%, compared to increases in heart rate which double or triple output. The heart's use of stroke volume reserves is important at low activity levels or when heart rate increases are too low to meet metabolic need.

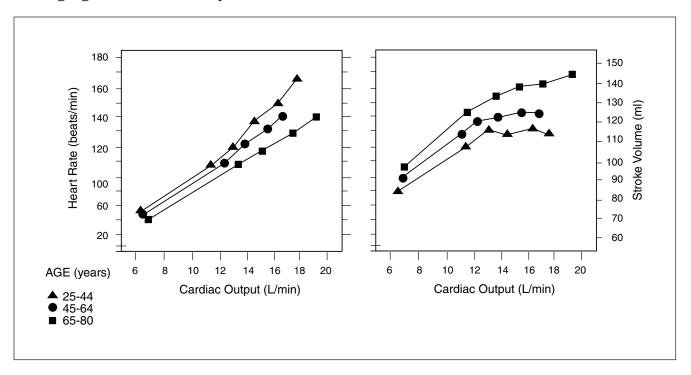
Notes

The heart can increase cardiac output by using stroke volume reserves.

Question:

Increased preload, increased contractility, and decreased afterload all contribute to increases in

The Aging Cardiovascular System



Age-related cardiovascular changes alter the hemodynamic performance of the heart.

With age, the *blood vessels* become thick and less compliant. This increases arterial pressure and therefore increases afterload. The *myocardium* also stiffens and can no longer completely contract and relax. This hinders diastolic filling and reduces cardiac output. The aging *heart valves* become stiff or flabby and may fail to fully open (which reduces forward flow) or fully close (which causes backflow). The aging myocardium becomes less sensitive to catecholamines, which reduces the minimum and maximum heart rate and the heart's work capacity.

The gradual degeneration of the cardiovascular structures affects the *conduction system*. The increase in fibrotic (thickened) and calcified (hardened) tissue often causes partial or complete block of electrical signals. Abnormal rhythms are therefore more common in the elderly.

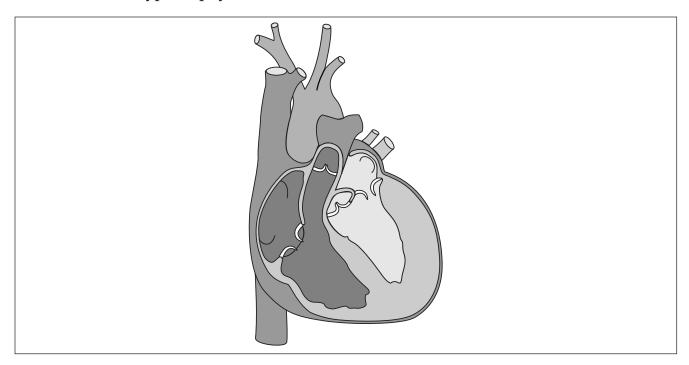
Notes

Normal age-related changes in cardiovascular structures alter the heart rate and work capacity of the heart.

Question:

As a person ages, the heart rate and work capacity of the heart

Left Ventricular Hypertrophy



Left ventricular hypertrophy is an enlarged (thickened) myocardium that results from overwork.

Hypertrophy develops when the left ventricle is required to work harder to compensate for problems such as high arterial pressure (increased afterload).* This maintains cardiac output until the muscle becomes too enlarged to pump efficiently. Significant enlargement prevents full relaxation and reduces diastolic filling. It also reduces the size of the ventricular chamber and may decrease the volume of blood available for ejection.

As hypertrophy becomes more severe, coronary circulation may be insufficient to supply the enlarged myocardium with oxygenated blood. This further decreases pumping efficiency. Symptoms of hypertrophy are shortness of breath (dyspnea), chest pain, or arrhythmias.

Notes

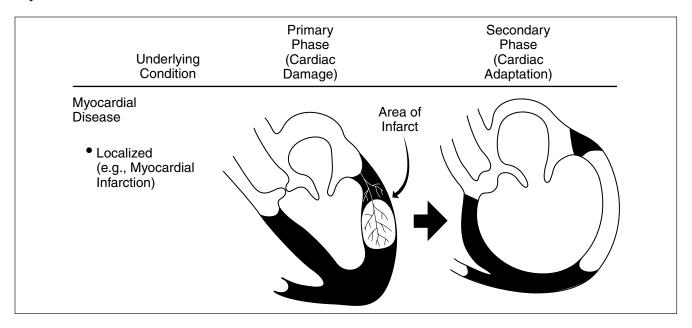
Left ventricular hypertrophy occurs when the left ventricle is required to work harder to compensate for high arterial pressure. It leads to decreased cardiac output.

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()	11	est	ion	١.

An *enlarged myocardium* caused by overwork is _

^{*} Hypertrophy also affects the right ventricle, but the effect is greater in the left ventricle because the left ventricle has a greater workload.

Myocardial Ischemia



Myocardial ischemia is insufficient coronary circulation. The myocardial cells are deprived of oxygen, so they die (infarct). Conduction may be disturbed (see figure).

The usual cause of *myocardial ischemia* is obstruction of the coronary arteries caused by the build-up of calcium deposits, common with aging. These harden and narrow the arteries, so blood flow slows or becomes blocked. If the muscle cells die, they cannot depolarize. Electrical impulses may be delayed or blocked at the infarcted site, or they may travel in a circular path (called re-entrant conduction). The ECG shows variations in the ST segment because the injured cells are unresponsive (refractory) to electrical impulses.

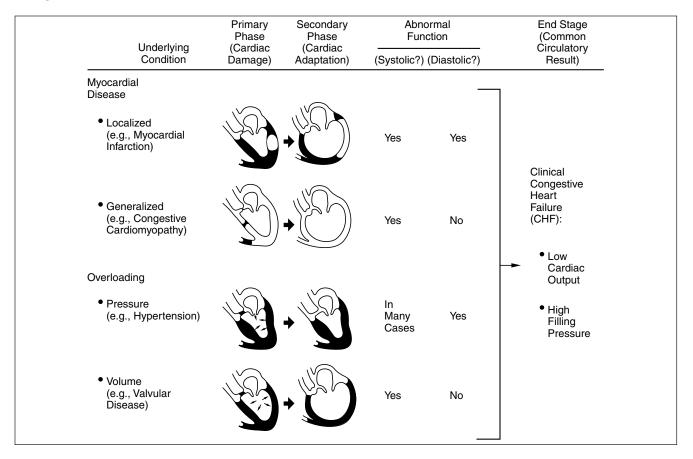
An ischemic myocardium cannot contract or relax fully, so diastolic filling is impaired and stroke volume is reduced. The patient may have no symptoms (silent ischemia) or suffer chest pain, heart attack, ventricular arrhythmias, or sudden death.

Notes

Myocardial ischemia disturbs conduction and without treatment may cause pain, heart attack, ventricular arrhythmias, or death.

Question:	
Insufficient coronary circulation is	

Congestive Heart Failure (CHF)



Untreated problems of the left ventricle may lead to congestive heart failure, a condition in which blood circulates poorly and backs up (congests) in vessels or body tissue.

Congestive heart failure may be backward or forward. In backward failure, the heart cannot pump its volume of blood, so blood backs up in the pulmonary vessels and lung tissue. This increases the volume and pressure in the pulmonary veins and left chambers. In forward failure, blood does not flow effectively from the left ventricle, so blood is poorly circulated and tends to pool in the extremities (feet and legs).

Typically, heart failure is both backward and forward, and the left ventricle fails before the right. With mild failure, the patient may become short of breath during exercise. With severe failure, the patient may be short of breath even at rest and may have cyanosis (blue skin coloring due to inadequate oxygen). The clinical indicator is increased end-diastolic pressure of the left ventricle.

Notes

Congestive heart failure is the result of untreated left ventricular dysfunction.

The failure of the heart to maintain adequate cardiac output is ______.