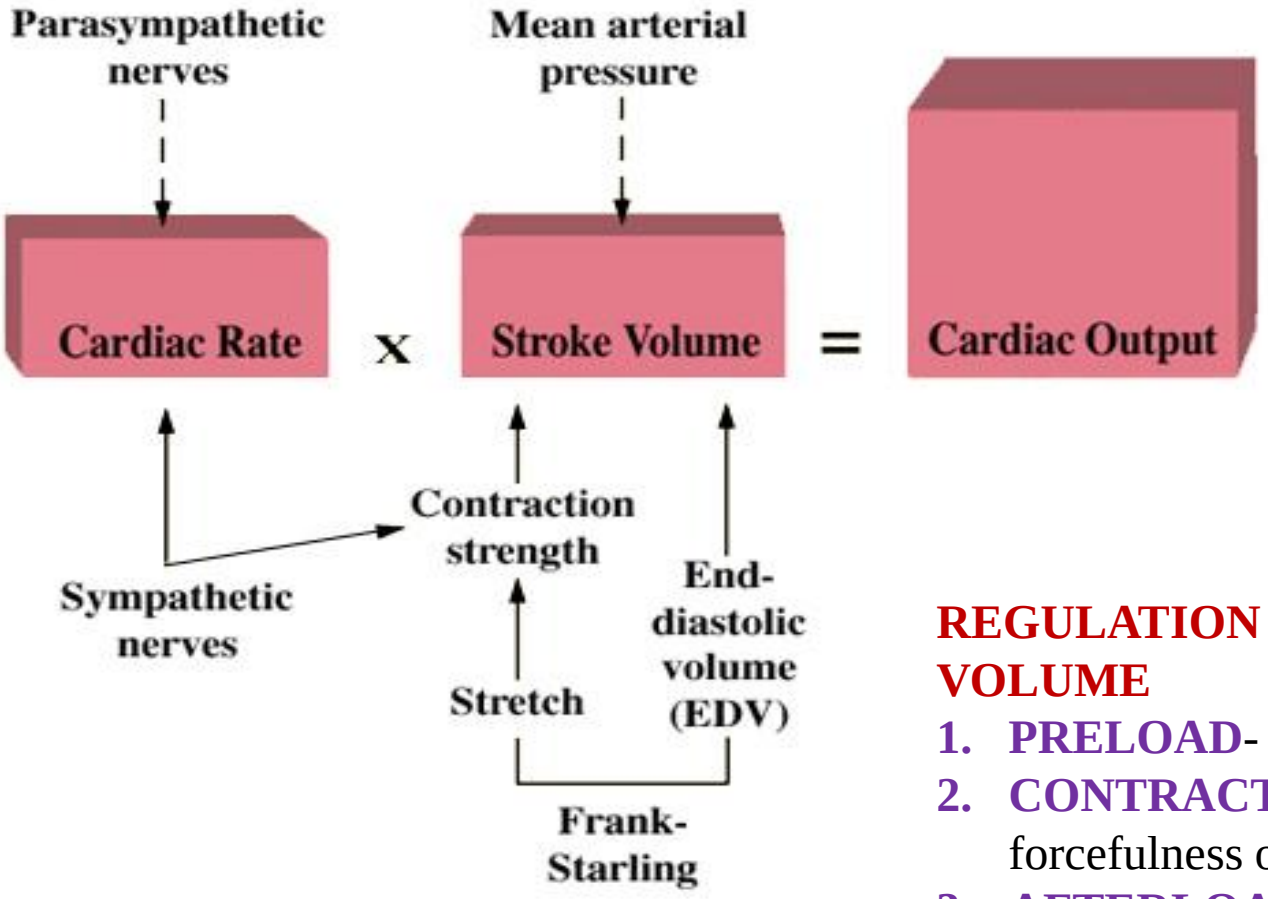
CARDIAC OUTPUT



REGULATION OF STROKE VOLUME

1. **PRELOAD**- Effect of stretching
2. **CONTRACTILITY**- forcefulness of contraction
3. **AFTERLOAD**- pressure

- In **congestive heart failure (CHF),** there is a loss of pumping efficiency by the heart. Causes of CHF include coronary artery disease , congenital defects, long-term high blood pressure (which increases the afterload), myocardial infarctions (regions of dead heart tissue due to a previous heart attack), and valve disorders.

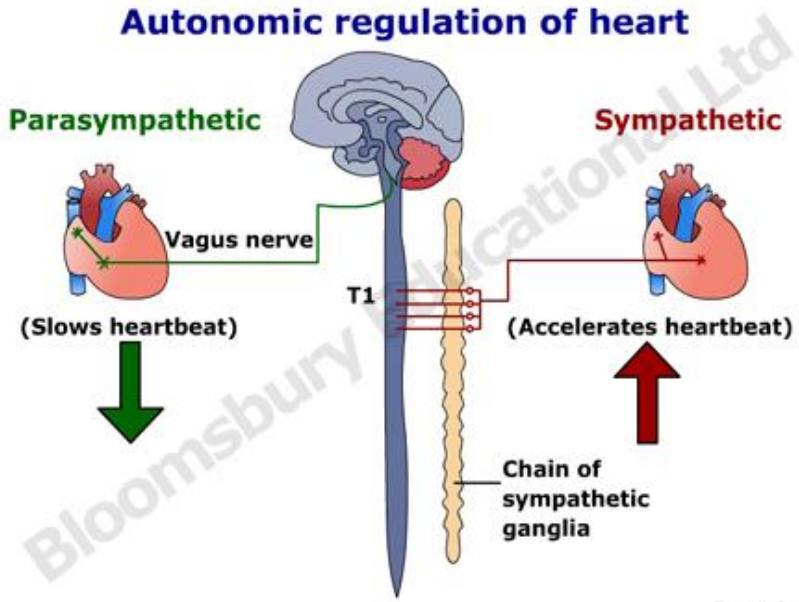
- As the pump becomes less effective, more blood remains in the ventricles at the end of each cycle, and gradually the end-diastolic volume (preload) increases.

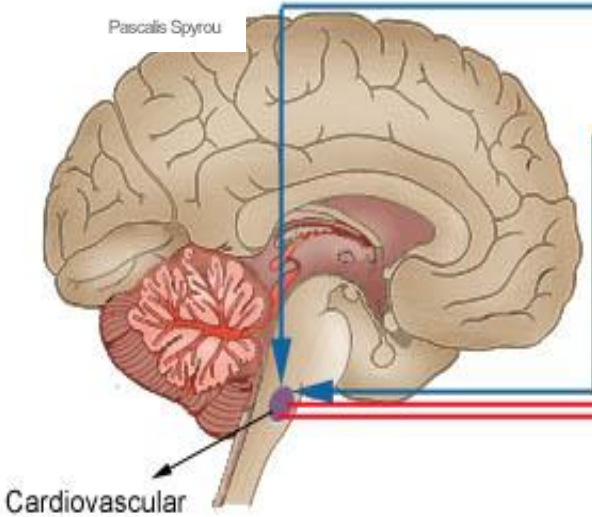
- Often, one side of the heart starts to fail before the other.

- If the **left ventricle** fails first, it can't pump out all the blood it receives. As a result, blood backs up in the lungs and causes ***pulmonary edema***, fluid accumulation in the lungs that can cause suffocation if left untreated.

- If the **right ventricle** fails first, blood backs up in the systemic veins and, over time, the kidneys cause an increase in blood volume. In this case, the resulting ***peripheral edema*** usually is most noticeable in the feet and ankles.

REGULATION OF HEART RATE





INPUT TO CARDIOVASCULAR CENTER
(nerve impulses)

From higher brain centers: cerebral cortex limbic system, and hypothalamus

From sensory receptors:
Proprioceptors - monitor movements
Chemoreceptors - monitor blood chemistry
Baroreceptors - monitor blood pressure

OUTPUT TO HEART
(increased frequency of nerve impulses)

Increased rate of spontaneous depolarization in SA node (and AV node) increases heart rate

Increased contracility of atria and ventricles increases stroke volume

Decreased rate of spontaneous depolarization in SA node (and AV node) decreases heart rate.

Vagus nerves (cranial nerve X, parasympathetic

1) - **Cardiac accelerator nerves** (sympathetic)

Factors that increases cardiac output

Increased end diastolic volume (stretches the heart)

Positive inotropic agents such as increased sympathetic stimulation; catecholamines, glucagon, or thyroid hormones in the blood; increased Ca^{2+} in extracellular fluid

Decreased arterial blood pressure during diastole

Increased PRELOAD

Increased CONTRACTILITY

Decreased AFTERLOAD

Within limits, cardiac muscle fibers contract more forcefully with stretching (Frank–Starling law of the heart)

Positive inotropic agents increase force of contraction at all physiological levels of stretch

Semilunar valves open sooner when blood pressure in aorta and pulmonary artery is lower

Increased STROKE VOLUME

Increased CARDIAC OUTPUT

Increased HEART RATE

Increased sympathetic stimulation and decreased parasympathetic stimulation

Catecholamine or thyroid hormones in the blood; moderate increase in extracellular Ca^{2+}

Infants and senior citizens, females, low physical fitness, increased body temperature

NERVOUS SYSTEM

Cardiovascular center in medulla oblongata receives input from cerebral cortex, limbic system, proprioceptors, baroreceptors, and chemoreceptors

CHEMICALS

OTHER FACTORS

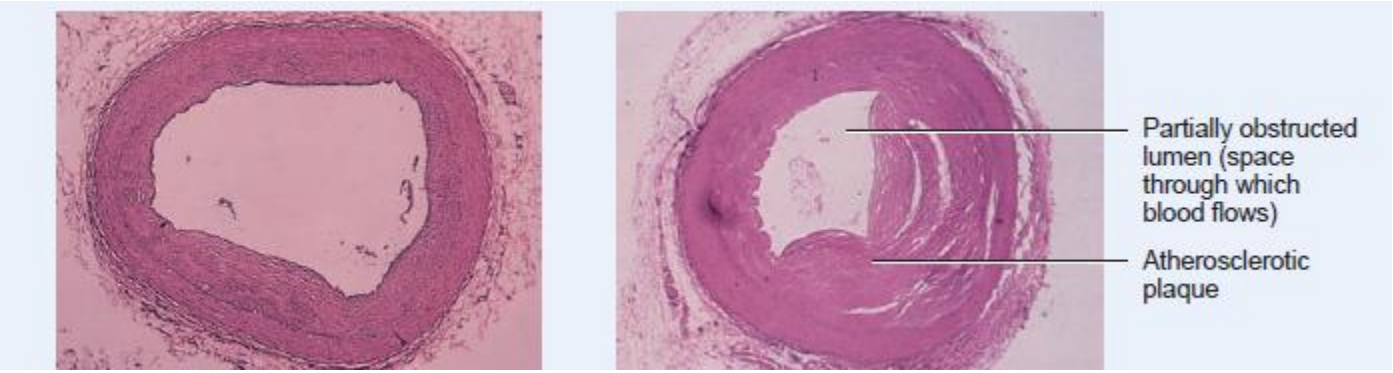
Coronary Artery Disease

- CAD results from the effects of the accumulation of atherosclerotic plaques in coronary arteries, which leads to a reduction in blood flow to the myocardium. Some individuals have no signs or symptoms; others experience angina pectoris (chest pain), and still others suffer from hear attack

Risk Factors for CAD

- smoking, high blood pressure, diabetes, high cholesterol levels, obesity, personality, sedentary lifestyle, and a family history of CAD.

Formation in the walls of large and medium-sized arteries of lesions called **atherosclerotic plaques**



Normal artery Obstructed artery

Diagnosis of CAD

- **Stress testing**
- **echocardiography** , a technique that uses ultrasound waves to image the interior of the heart. Echocardiography allows the heart to be seen in motion and can be used to determine the size, shape, and functions of the chambers of the heart; the volume and velocity of blood pumped from the heart; the status of heart valves; the presence of birth defects; and abnormalities of the pericardium.
- **electron beam computerized tomography (EBCT),** which detects calcium deposits in coronary arteries. These calcium deposits are indicators of atherosclerosis
- **Coronary angiography** (an-je¯-OG-ra-fe¯; *angio-* blood vessel;-*grapho* to write) is an invasive procedure used to obtain information about the coronary arteries

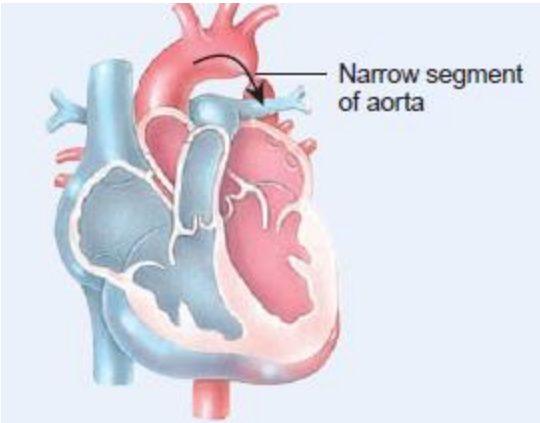
Treatment of CAD

- **Drugs**
- **Coronary artery bypass grafting (CABG)** is a surgical procedure in which a blood vessel from another part of the body is attached ("grafted") to a coronary artery to bypass an area of blockage. A piece of the grafted blood vessel is sutured between the aorta and the unblocked portion of the coronary artery

Congenital Heart Defects

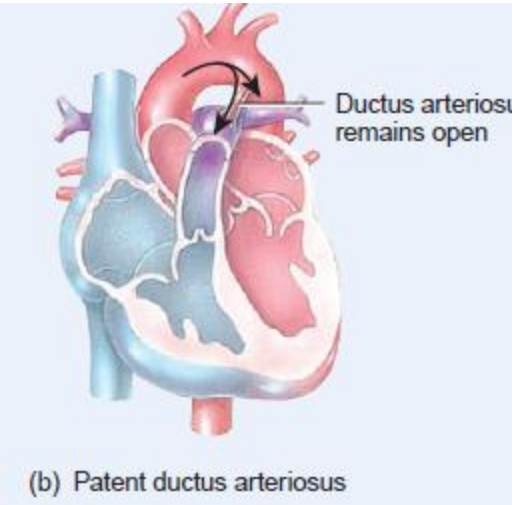
A defect that is present at birth, and usually before, is called a **congenital defect.** Many such defects are not serious and may go unnoticed for a lifetime.

- **Coarctation of the aorta.** In this condition, a segment of the aorta is too narrow, and thus the flow of oxygenated blood to the body is reduced, the left ventricle is forced to pump harder, and high blood pressure develops. Coarctation is usually repaired surgically by removing the area of obstruction



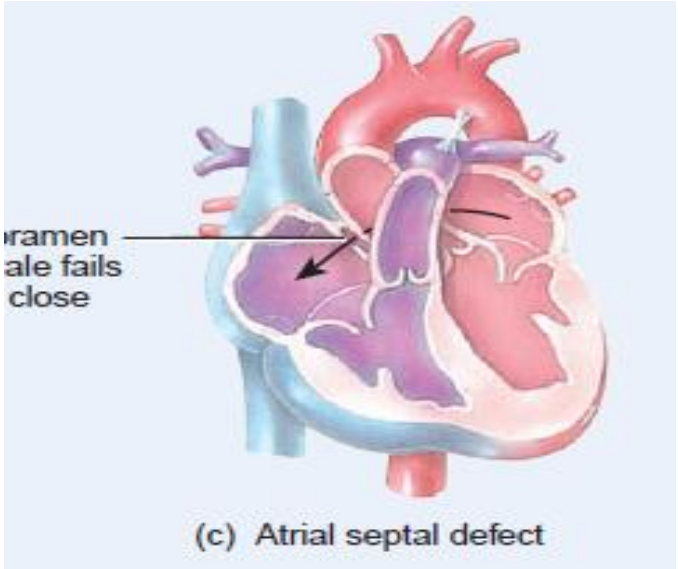
(a) Coarctation of the aorta

- **Patent ductus arteriosus (PDA).** In some babies, the ductus arteriosus, a temporary blood vessel between the aorta and the pulmonary trunk, remains open rather than closing shortly after birth. As a result, aortic blood flows into the lower-pressure pulmonary trunk, thus increasing the pulmonary trunk blood pressure and overworking both ventricles.

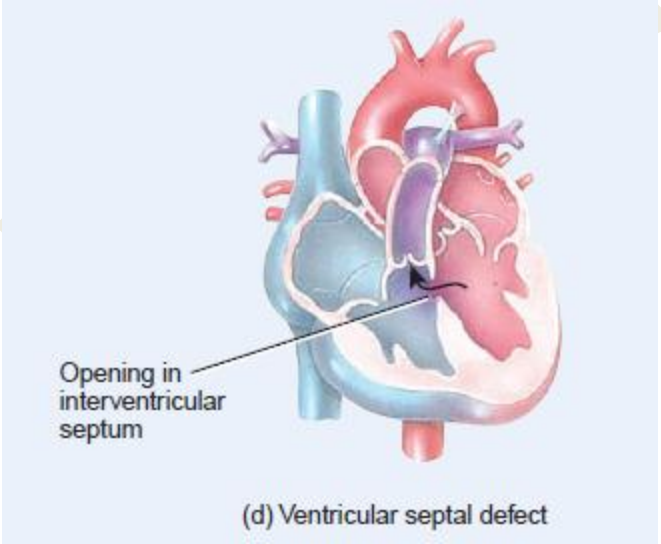


(b) Patent ductus arteriosus

- **Septal defect.** A septal defect is an opening in the septum that separates the interior of the heart into left and right sides. In an **atrial septal defect** the fetal foramen ovale between the two atria fails to close after birth.



(c) Atrial septal defect

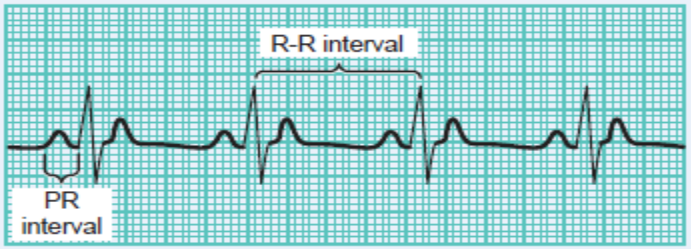


(d) Ventricular septal defect

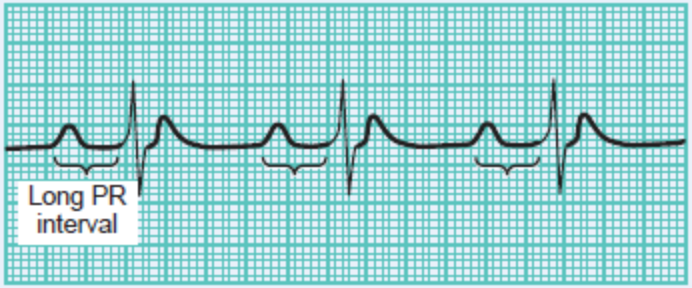
A **ventricular septal defect** is caused by incomplete development of the interventricular septum. In such cases, oxygenated blood flows directly from the left ventricle in to the right ventricle, where it mixes with deoxygenated blood. The condition is treated surgically

Arrhythmias

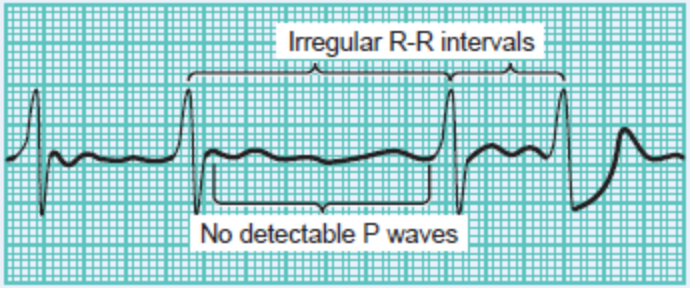
- The usual rhythm of heartbeats, established by the SA node, is called **normal sinus rhythm.** The term **arrhythmia** refers to an abnormal rhythm as a result of a defect in the conduction system of the heart.



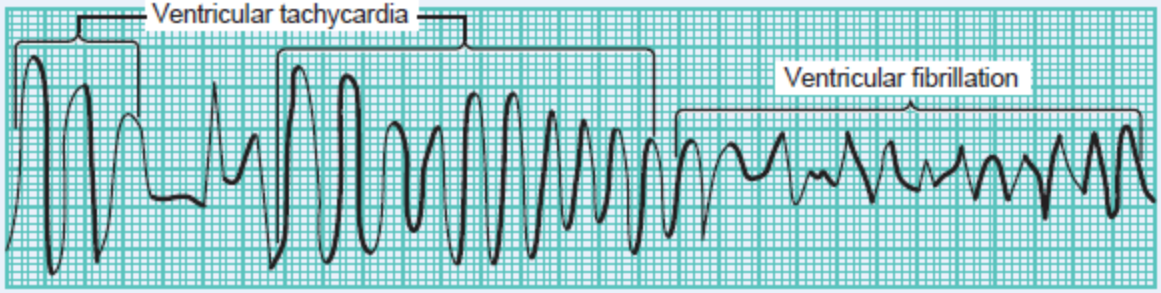
(a) Normal electrocardiogram (ECG)



(b) First-degree AV block



(c) Atrial fibrillation



(d) Ventricular tachycardia

(e) Ventricular fibrillation

- **Bradycardia** (*brady-* slow)
  refers to a slow heart rate (below 50 beats per minute)

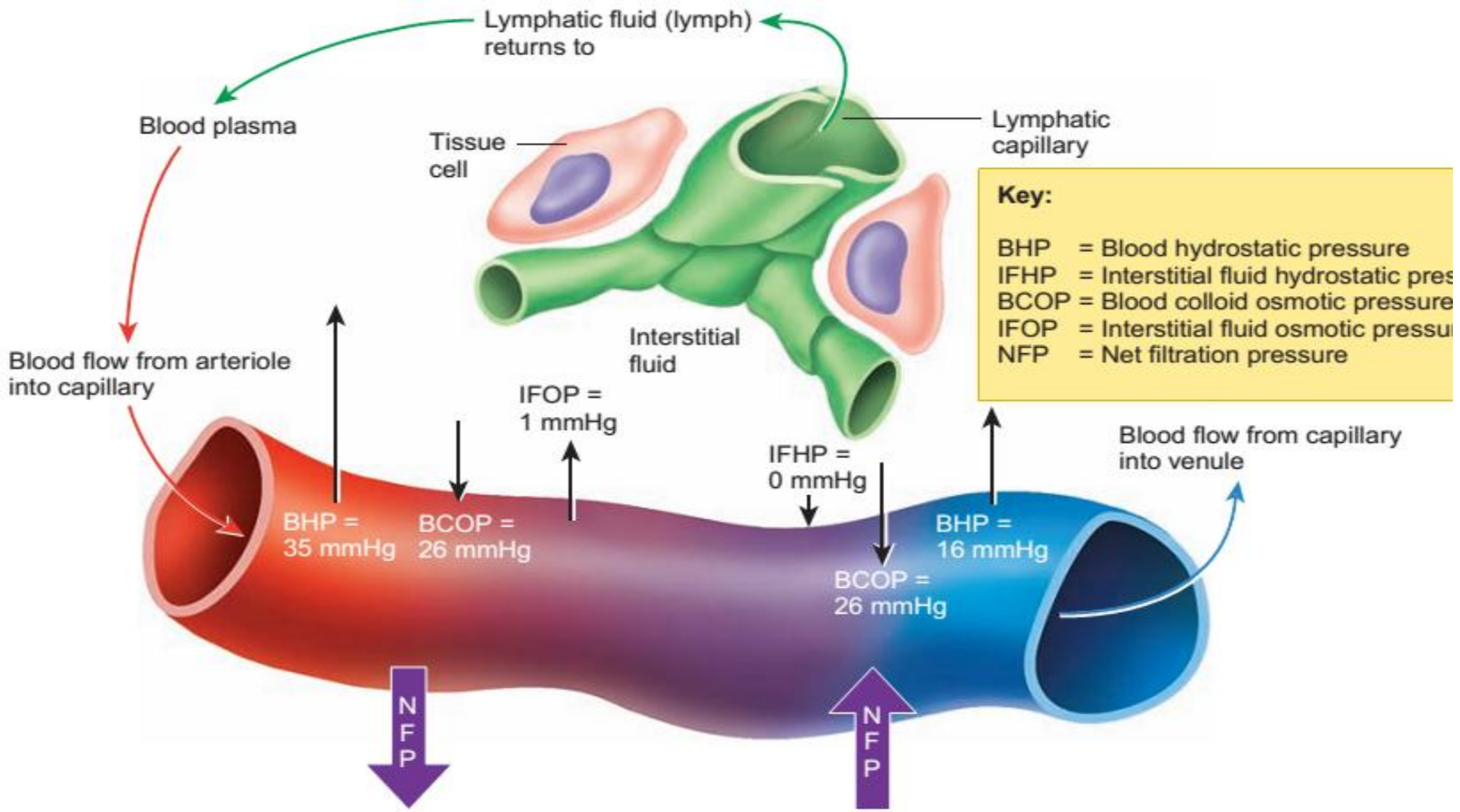
- **tachycardia**(*tachy-* quick) refers to a rapid heart rate (over 100 beats per minute); and

- **fibrillation** refers to rapid, uncoordinated heartbeats

- ***Asystole*** (*a-* without) Failure of the myocardium to contract.

- ***Cardiac arrest*** A clinical term meaning cessation of an effective heartbeat. The heart may be completely stopped or in ventricular fibrillation.

- ***Cardiomegaly*** Heart enlargement.



Net filtration at arterial end of capillaries (20 liters per day)

Net reabsorption at venous end of capillaries (17 liters per day)

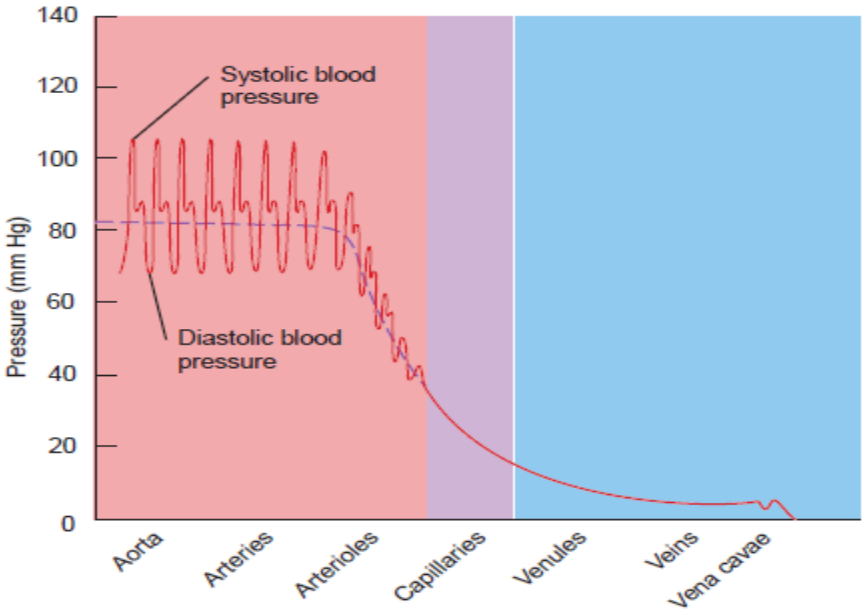
Net filtration pressure (NFP) = (BHP + IFOP) – (BCOP + IFHP)

Pressures promoting filtration

Pressure promoting reabsorption

	Arterial end	Venous end
	NFP = (35 + 1) – (26 + 0) = 10 mmHg	NFP = (16 + 1) – (26 + 0) = – 9 mmHg
Result	Net filtration	Net reabsorption

- ***Blood pressure*** **(BP),** the hydrostatic pressure exerted by blood on the walls of a blood vessel.
- BP is determined by cardiac output, blood volume, and vascular resistance
- BP is highest in the aorta and large systemic arteries; in a resting, young adult, BP rises to about *110 mmHg during systole* (ventricular contraction) and drops to about *70 mmHg during diastole* (ventricular relaxation).
- **Systolic blood pressure** is the highest pressure attained in arteries during systole, and **diastolic blood pressure** is the lowest arterial pressure during diastole



Mean arterial pressure (MAP), the average blood pressure in arteries, is roughly one-third of the way between the diastolic and systolic pressures. It can be estimated as follows:

MAP = diastolic BP + 1/3 (systolic BP diastolic BP)

Thus, in a person whose BP is 110/70 mmHg, MAP is about 83 mmHg (70 1/3(110 70)).

- Blood pressure also depends on the total volume of blood in the cardiovascular system.
- **vascular resistance** is the opposition to blood flow due to friction between blood and the walls of blood vessels.
- Vascular resistance depends on

1. size of the blood vessel lumen-The smaller the diameter of the blood vessel, the greater the resistance it offers to blood flow
2. Blood viscosity-The viscosity (thickness) of blood depends mostly on the ratio of red blood cells to plasma (fluid) volume, and to a smaller extent on the concentration of proteins in plasma. The higher the blood's viscosity, the higher the resistance.

(3) total blood vessel length-Resistance to blood flow through a vessel is directly proportional to the length of the blood vessel. The longer a blood vessel, the greater the resistance

Systemic vascular resistance (SVR), also known as *total peripheral resistance (TPR),* refers to all the vascular resistances offered by systemic blood vessels.

- The diameters of arteries and veins are large, so their resistance is very small because most of the blood does not come into physical contact with the walls of the blood vessel. The smallest vessels—arterioles, capillaries, and venules—contribute the most resistance

- A major function of arterioles is to control SVR—and therefore blood pressure and blood flow to particular tissues—by changing their diameters.

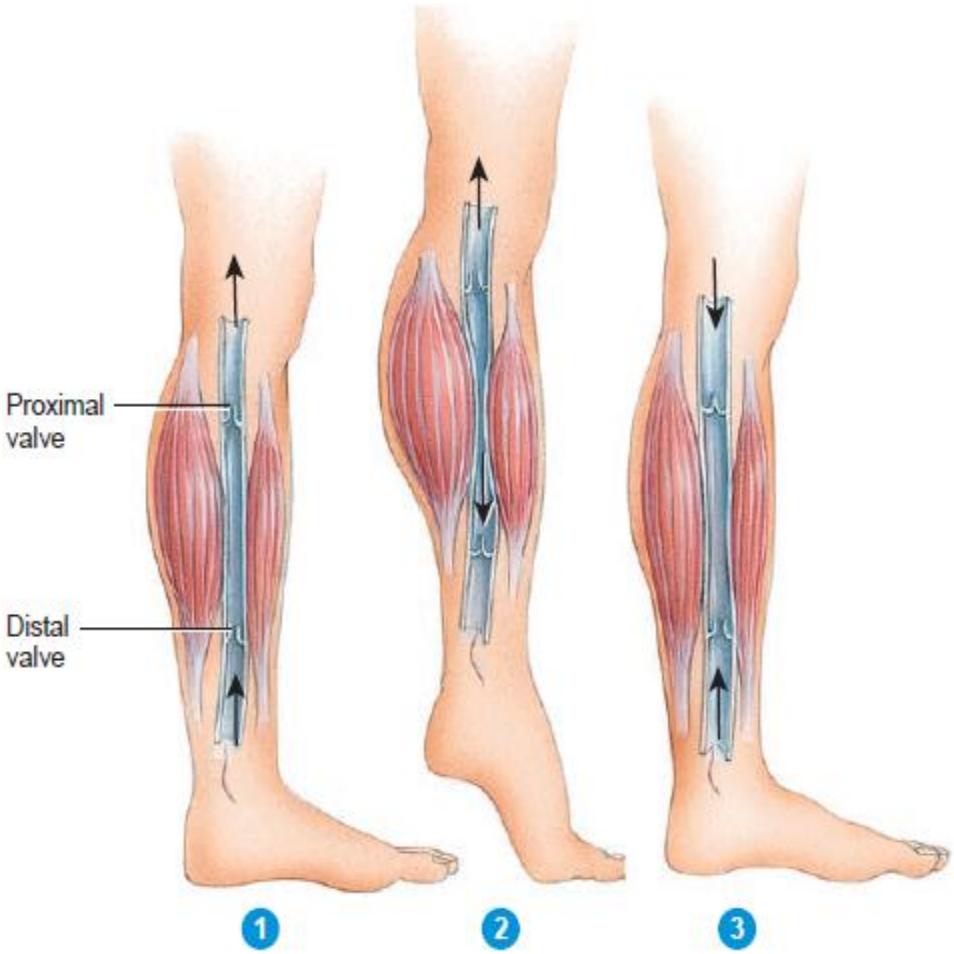
- Arterioles need to vasodilate or vasoconstrict only slightly to have a large effect on SVR. The main center for regulation of SVR is the vasomotor center in the brain stem

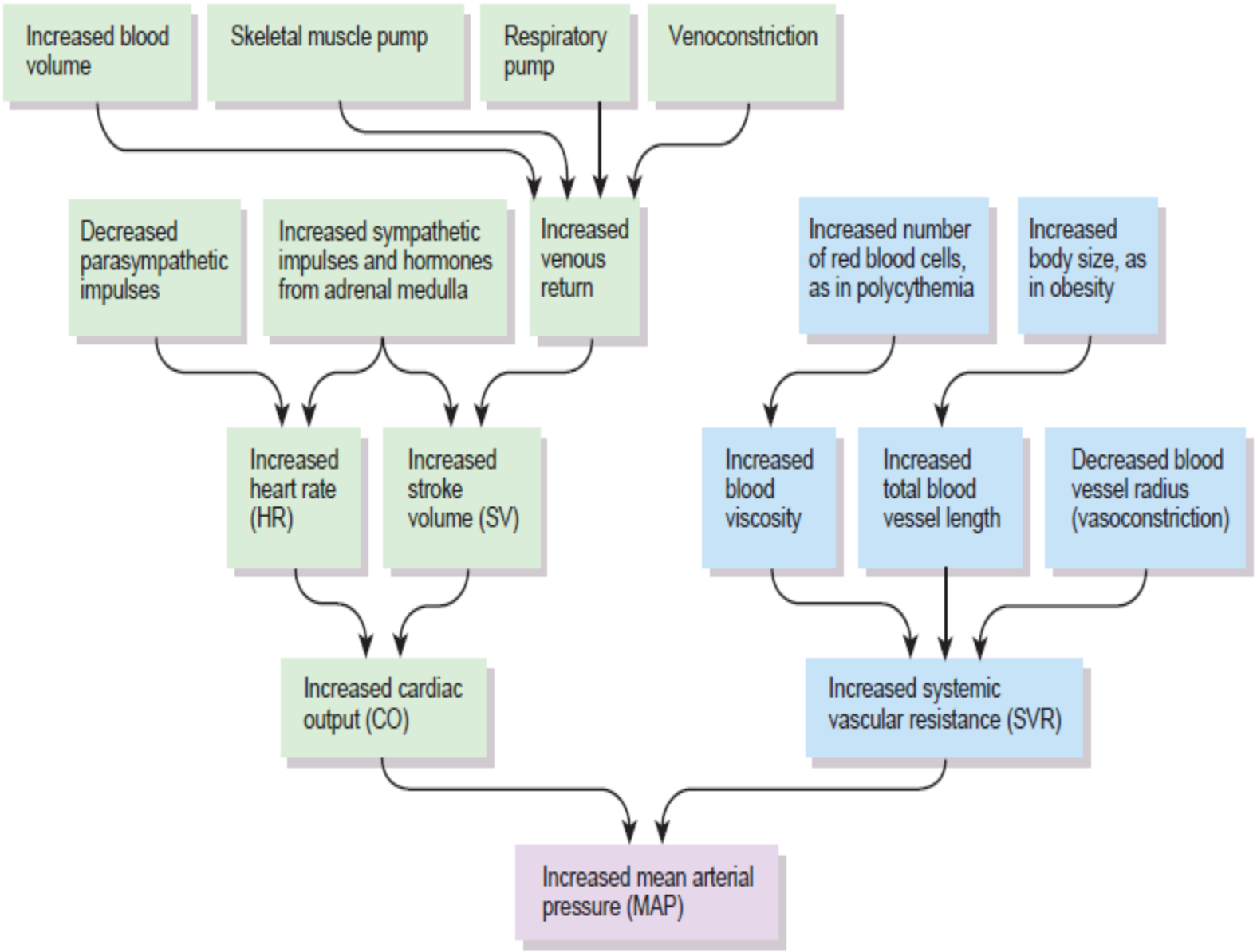
Venous return, the volume of blood flowing back to the heart through the systemic veins, occurs due to the pressure generated by contractions of the heart's left ventricle.

- The pressure difference from venules (averaging about 16 mmHg) to the right ventricle (0 mmHg), although small, normally is sufficient to cause venous return to the heart.
- If pressure increases in the right atrium or ventricle, venous return will decrease.

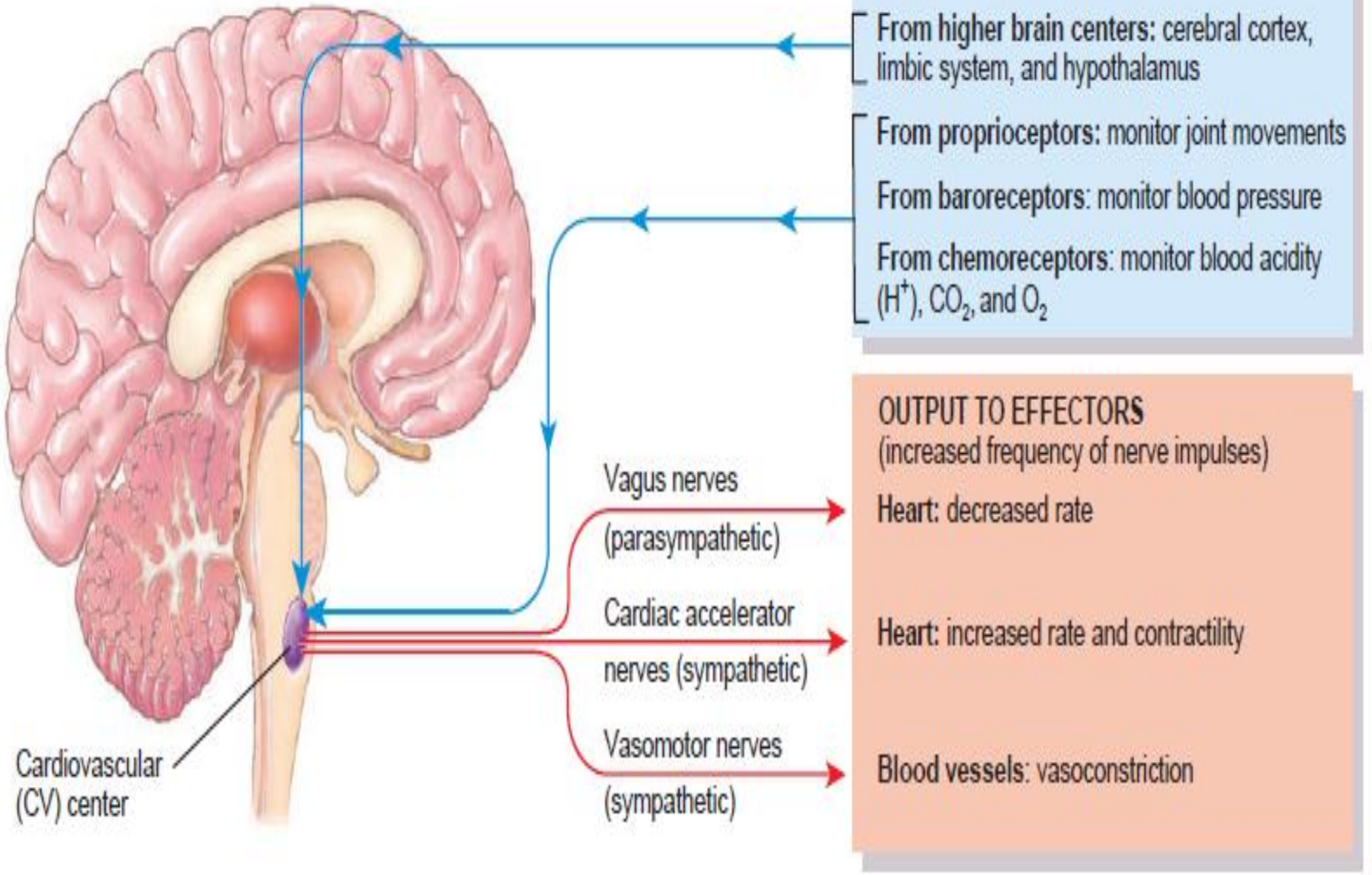
Besides the heart, two other mechanisms "pump" blood from the lower body back to the heart:

(1) the skeletal muscle pump, and
(2) the respiratory pump. Both pumps depend on the presence of valves in veins.

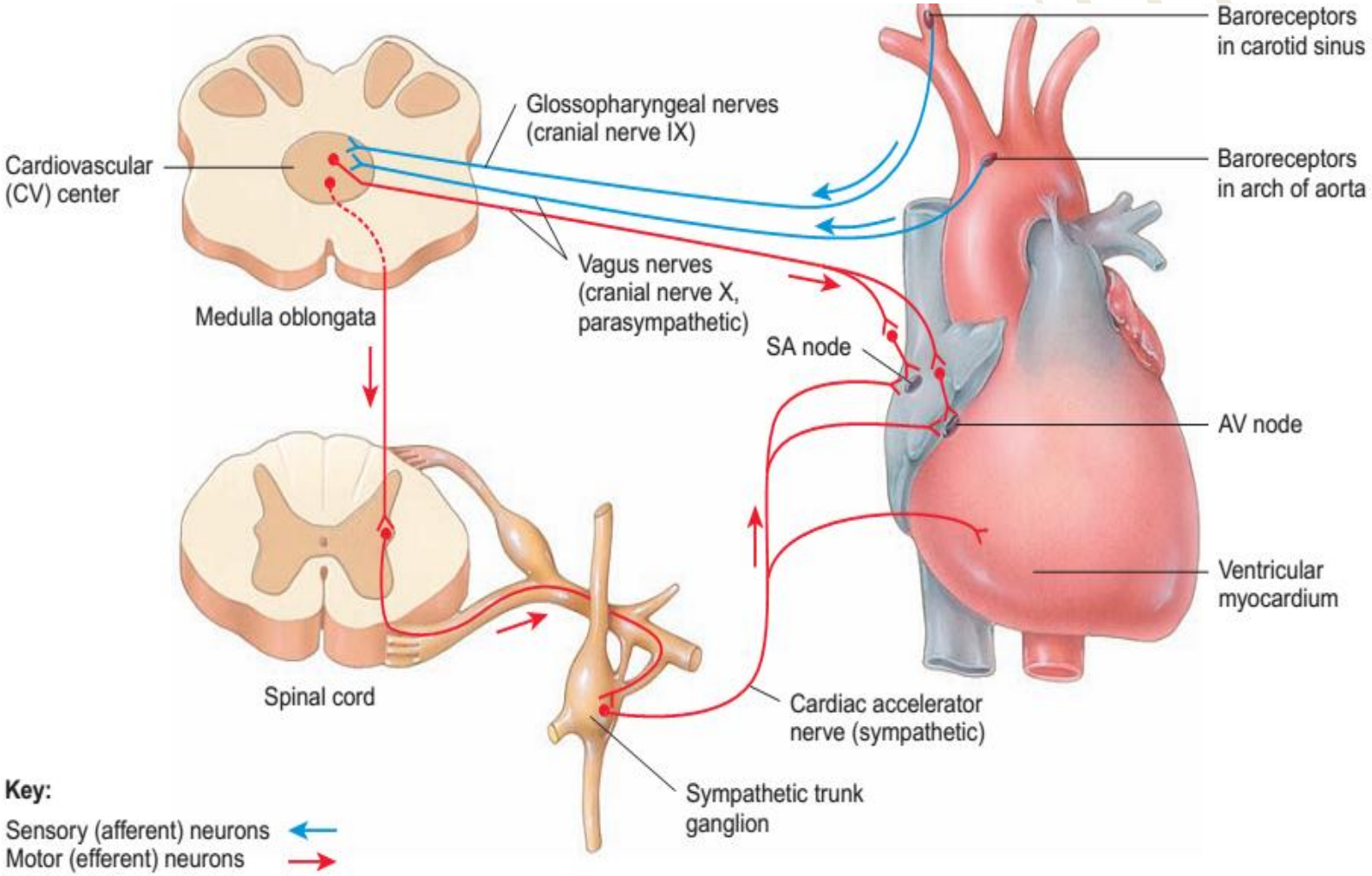



Increased blood volume
Skeletal muscle pump
Respiratory pump
Venoconstriction
Decreased parasympathetic impulses
Increased sympathetic impulses and hormones from adrenal medulla
Increased venous return
Increased number of red blood cells, as in polycythemia
Increased body size, as in obesity
Increased heart rate (HR)
Increased stroke volume (SV)
Increased blood viscosity
Increased total blood vessel length
Decreased blood vessel radius (vasoconstriction)
Increased cardiac output (CO)
Increased systemic vascular resistance (SVR)
Increased mean arterial pressure (MAP)

Location and function of the cardiovascular (CV) center in the medulla oblongata

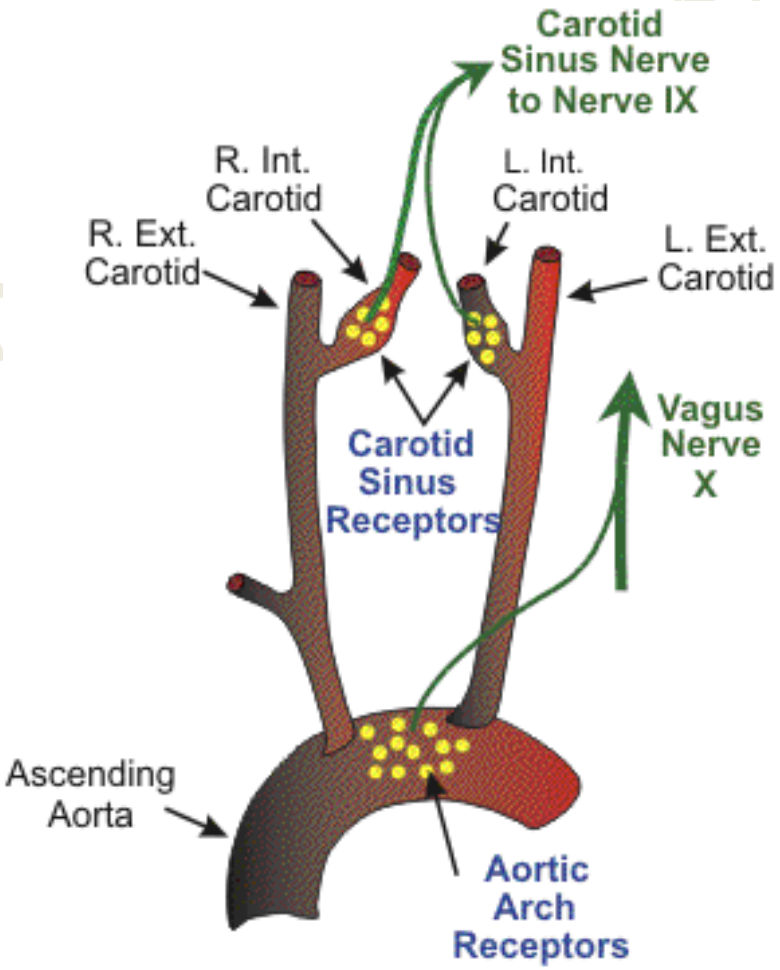


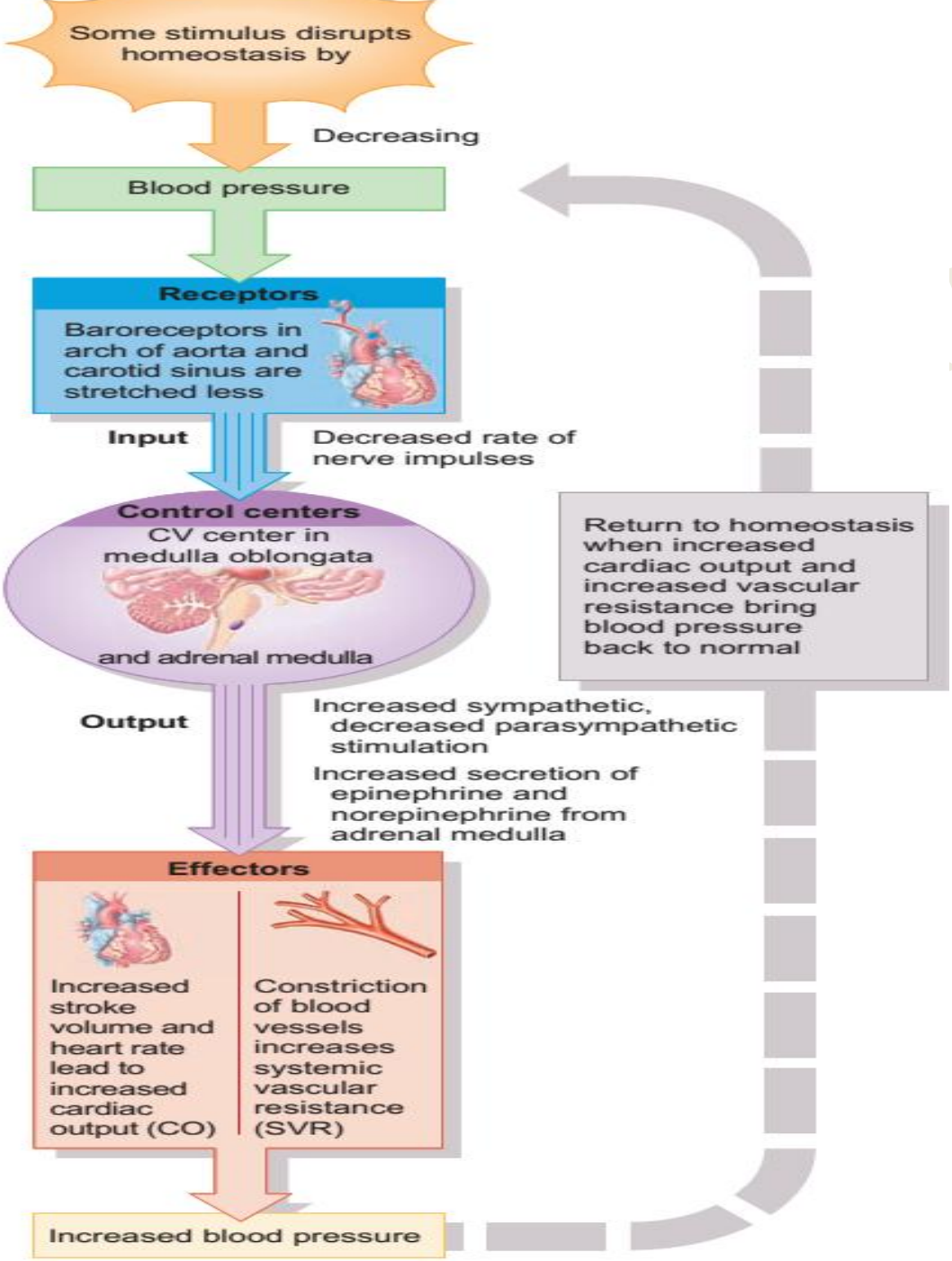
ANS innervation of the heart and the baroreceptor reflexes that help regulate blood pressure.



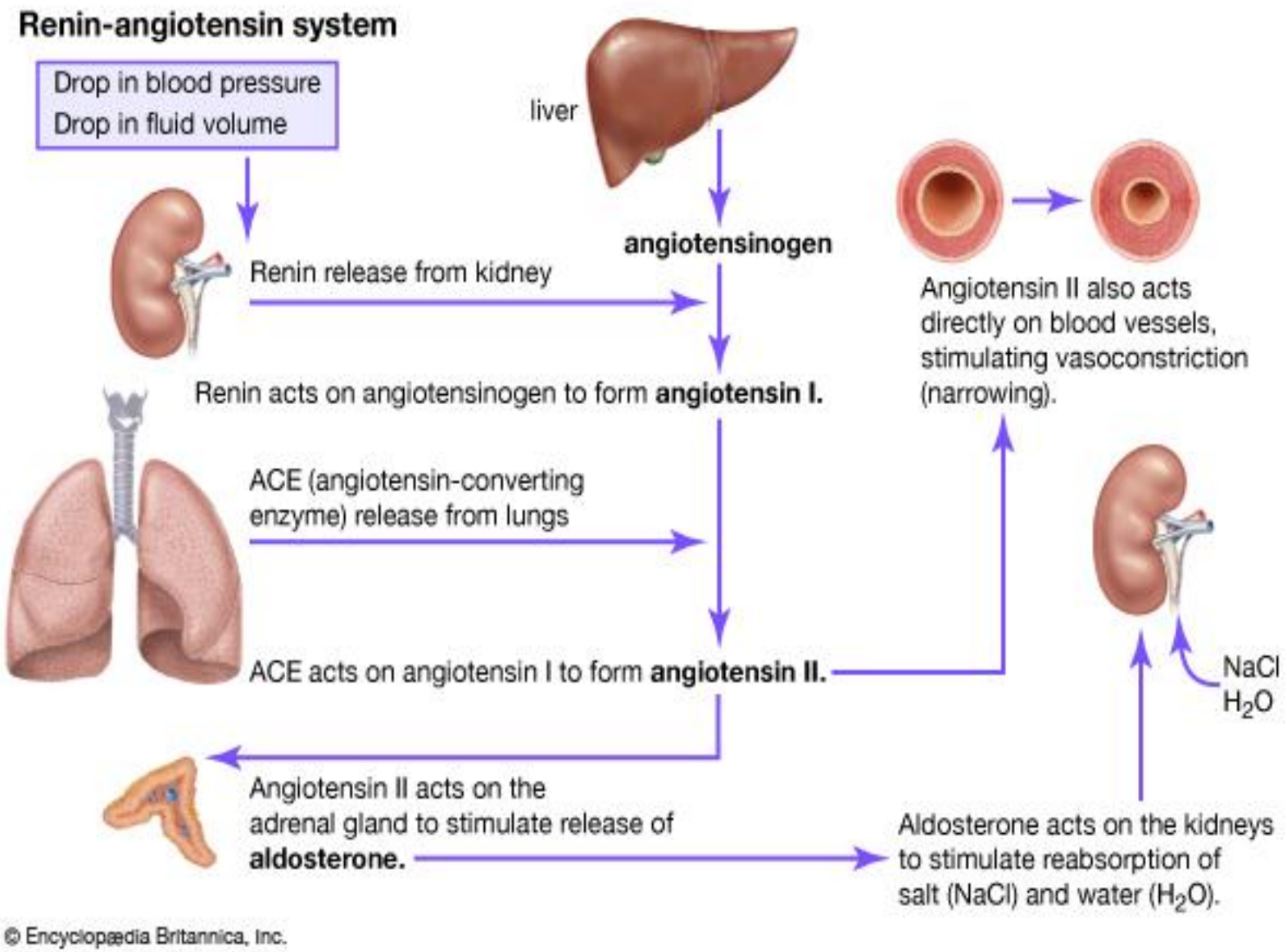
Location of baroreceptors

- Baroreceptors sense stretch and rate of stretch by generating action potentials (voltage spikes)
- Located in highly distensible regions of the circulation to maximise sensitivity

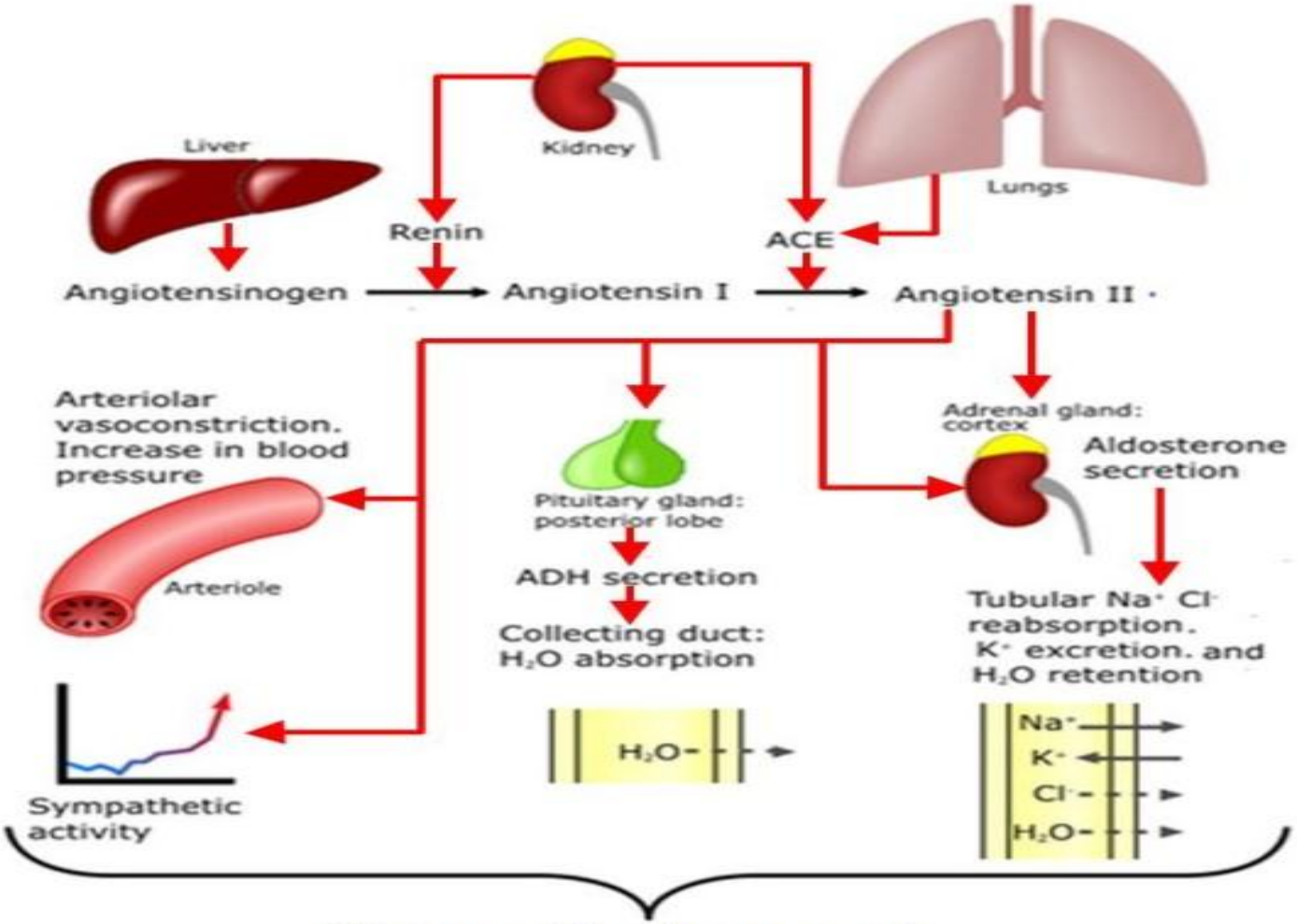




Negative feedback regulation of blood pressure via baroreceptor reflexes.



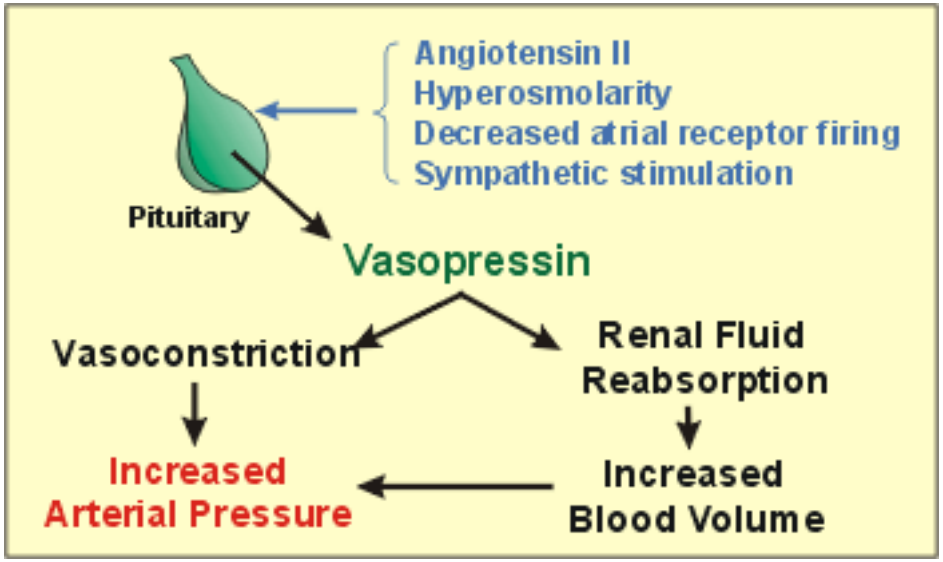
Renin-Angiotensin-Aldosterone System (RAAS)



Water and Sodium retention.
Increased circulating volume. Increased renal perfusion.

Vasopressin

- Enhances water retention
- Causes vasoconstriction
- Secretion increased by unloading of aortic Baroreceptors and atrial sensors



Blood Pressure Regulation by Hormones

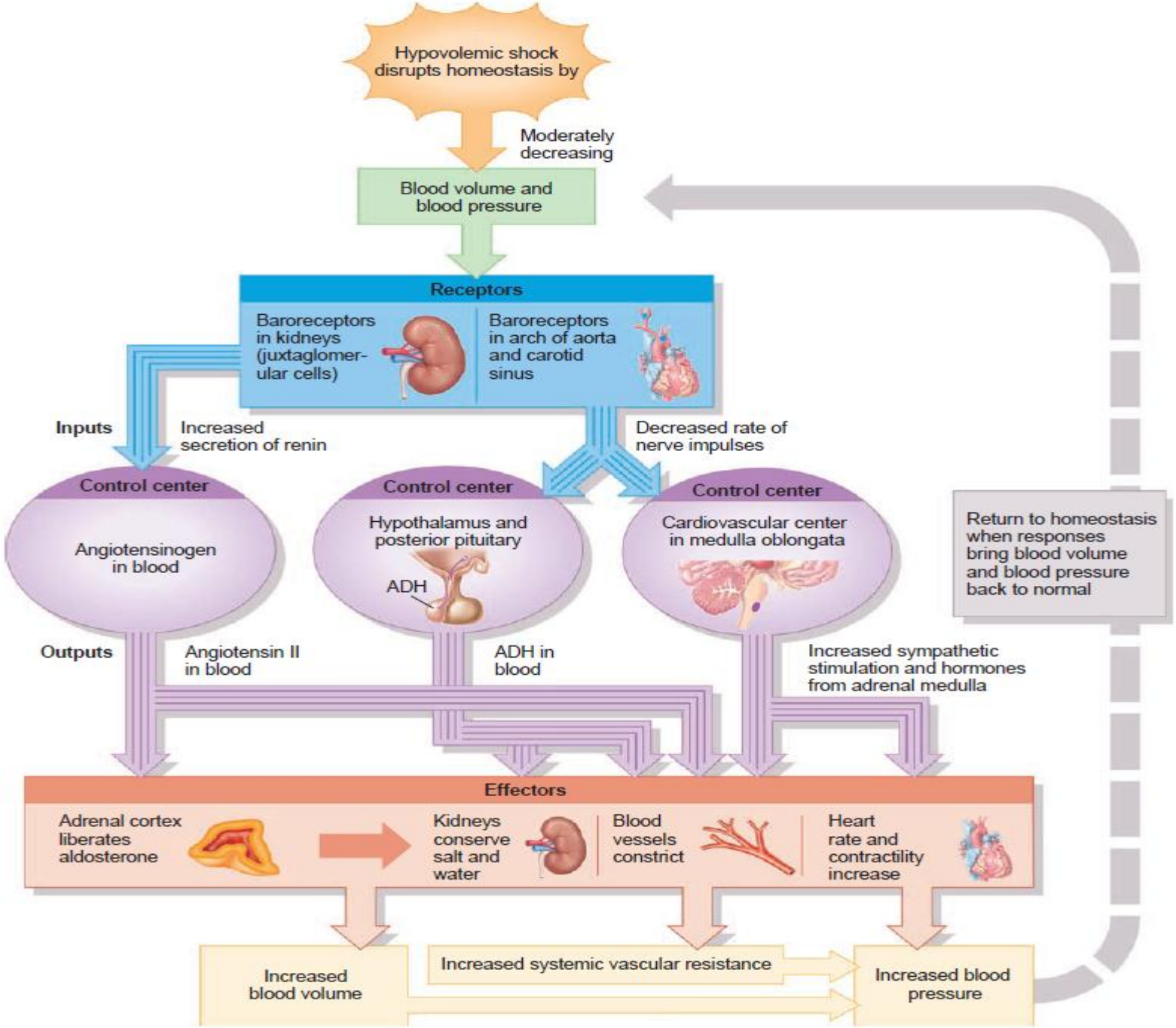
FACTOR INFLUENCING BLOOD PRESSURE	HORMONE	EFFECT ON BLOOD PRESSURE
CARDIAC OUTPUT		
Increased heart rate and contractility	Norepinephrine Epinephrine	Increase
SYSTEMIC VASCULAR RESISTANCE		
Vasoconstriction	Angiotensin II	Increase
	Antidiuretic hormone (vasopressin)	
	Norepinephrine*	
	Epinephrine†	
Vasodilation	Atrial natriuretic peptide	Decrease
	Epinephrine†	
	Nitric oxide	
BLOOD VOLUME		
Blood volume increase	Aldosterone Antidiuretic hormone	Increase
Blood volume decrease	Atrial natriuretic peptide	Decrease

Shock is a failure of the cardiovascular system to deliver enough O_2 and nutrients to meet cellular metabolic needs.

Symptoms

- Cold, clammy skin
- Muscular weakness
- Rapid and shallow breathing
- Rapid and weak pulse
- Low pulse pressure (and sometimes mean pressure)
- Reduced urine output
- Confusion

- **Hypovolaemia** Caused by drop in blood (plasma) volume
  - e.g. haemorrhage, diarrhoea, vomiting, injury
- **Vascular shock** due to inappropriate vasodialation
- **Cardiogenic shock** An acute interruption of cardiac function
  - e.g. myocarditis (inflammation of the heart muscle) or myocardial infarction
- **Obstructive shock** due to obstruction in blood flow


Hypovolemic shock disrupts homeostasis by
Moderately decreasing
Blood volume and blood pressure
Receptors
Baroreceptors in kidneys (juxtaglomer-ular cells)
Baroreceptors in arch of aorta and carotid sinus
Inputs
Increased secretion of renin
Decreased rate of nerve impulses
Control center
Angiotensinogen in blood
Control center
Hypothalamus and posterior pituitary
ADH
Control center
Cardiovascular center in medulla oblongata
Return to homeostasis when responses bring blood volume and blood pressure back to normal
Outputs
Angiotensin II in blood
ADH in blood
Increased sympathetic stimulation and hormones from adrenal medulla
Effectors
Adrenal cortex liberates aldosterone
Kidneys conserve salt and water
Blood vessels constrict
Heart rate and contractility increase
Increased blood volume
Increased systemic vascular resistance
Increased blood pressure