

Chapter 20.2

Blood Pressure



Blood Pressure

Blood pressure (BP) is the force blood exerts against a vessel wall

The blood's high pressure and low pressure are measured in mmHg (i.e. milliliter of mercury): e.g. 120/80

Measured at brachial artery // measurement should be at level of the heart

Systolic pressure: peak arterial BP taken during ventricular contraction (ventricular systole)

Diastolic pressure: minimum arterial BP taken during ventricular relaxation (diastole - between heart beats)

Pulse Pressure

Normal blood pressure value for young adult: 120/80 mm Hg
(benchmark used for exam!)

Pulse pressure – difference between systolic and diastolic pressure (40 mmHg)

- Important measurement of stress exerted on small
- arteries by pressure surges generated by the heart

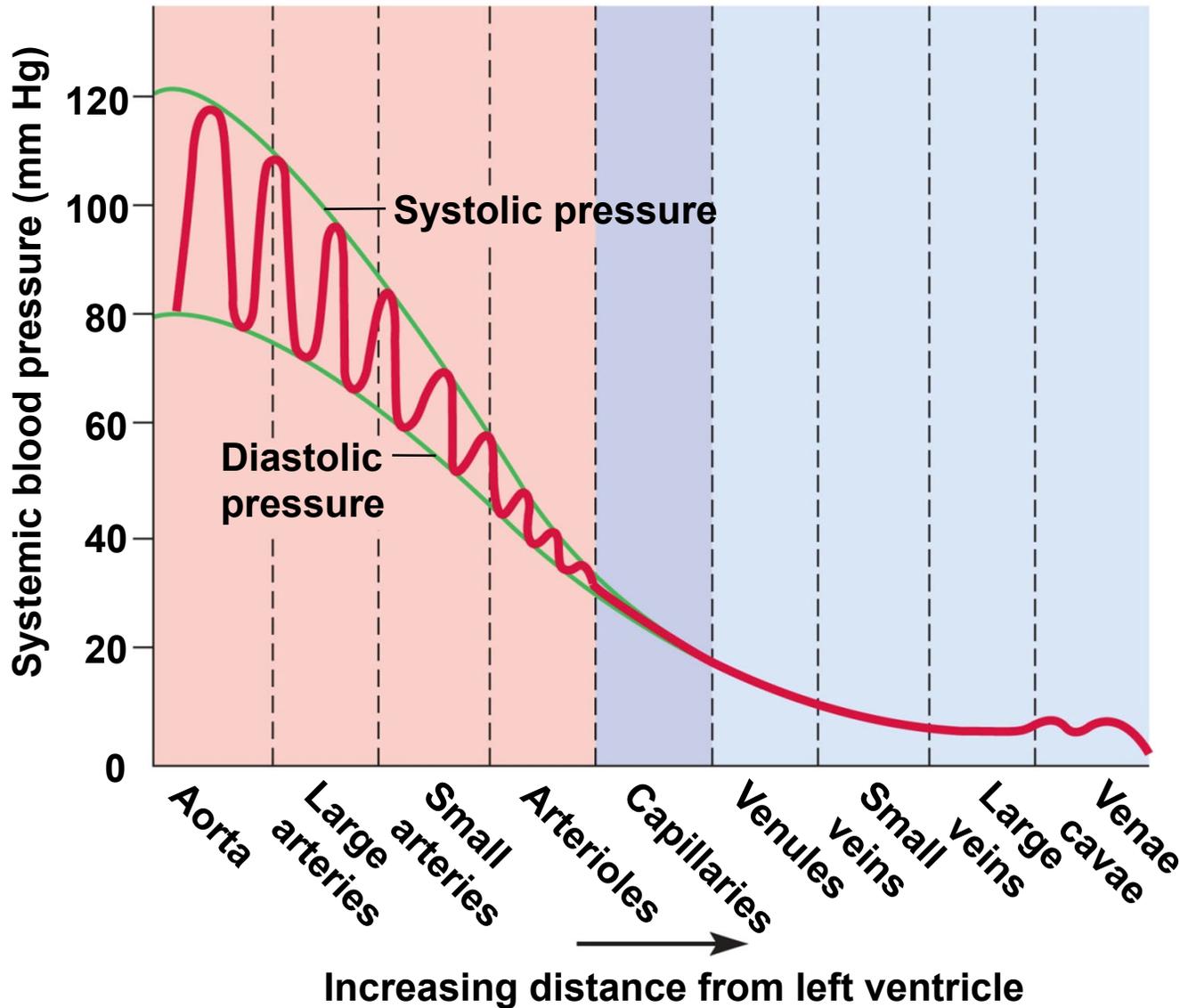
High blood pressure damages blood vessels // called the **silent killer!**

Do you know if you have high blood pressure?

Why is high blood pressure called the silent killer?

Blood Vessel Pressure

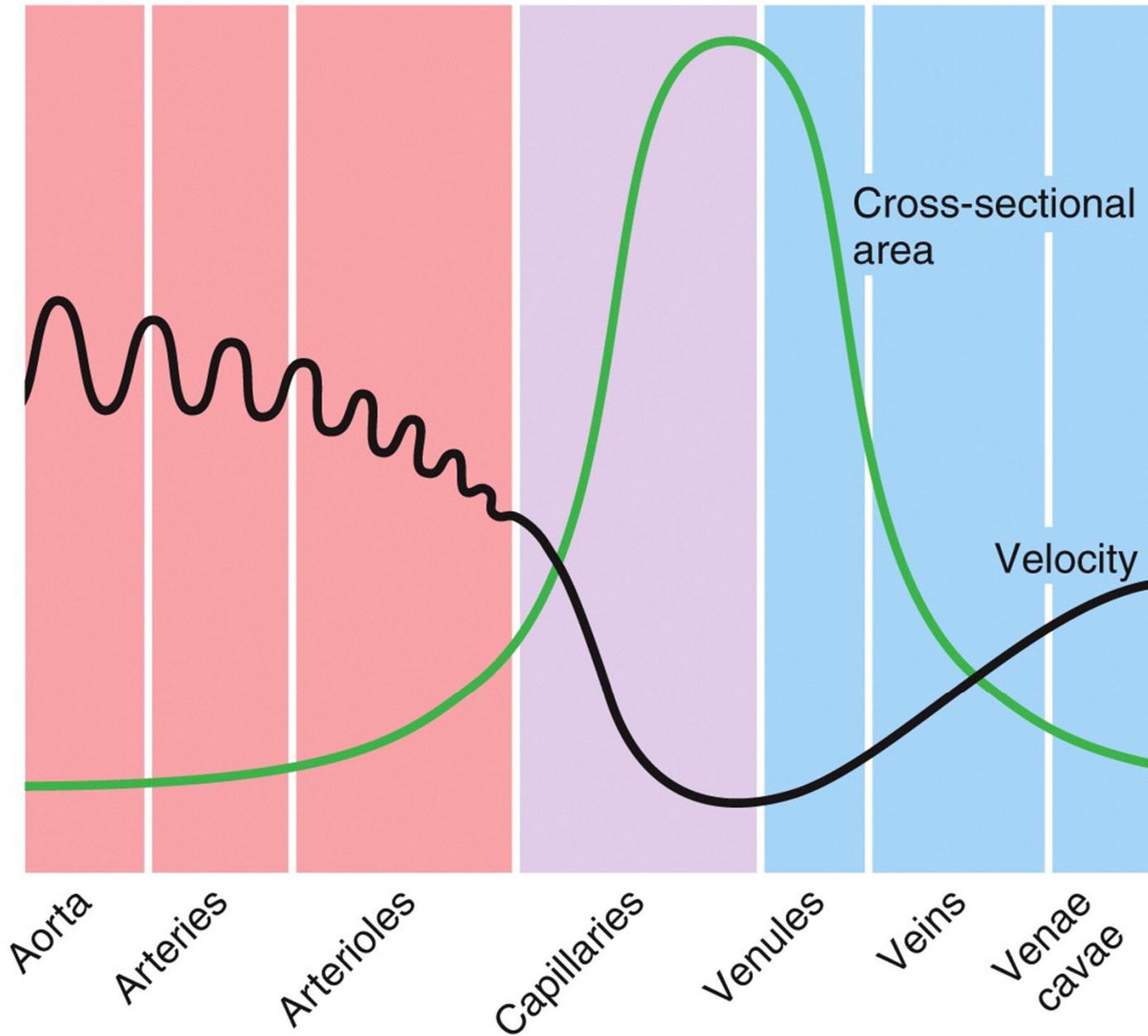
(Changes With Distance From Heart)



What is pulse pressure?

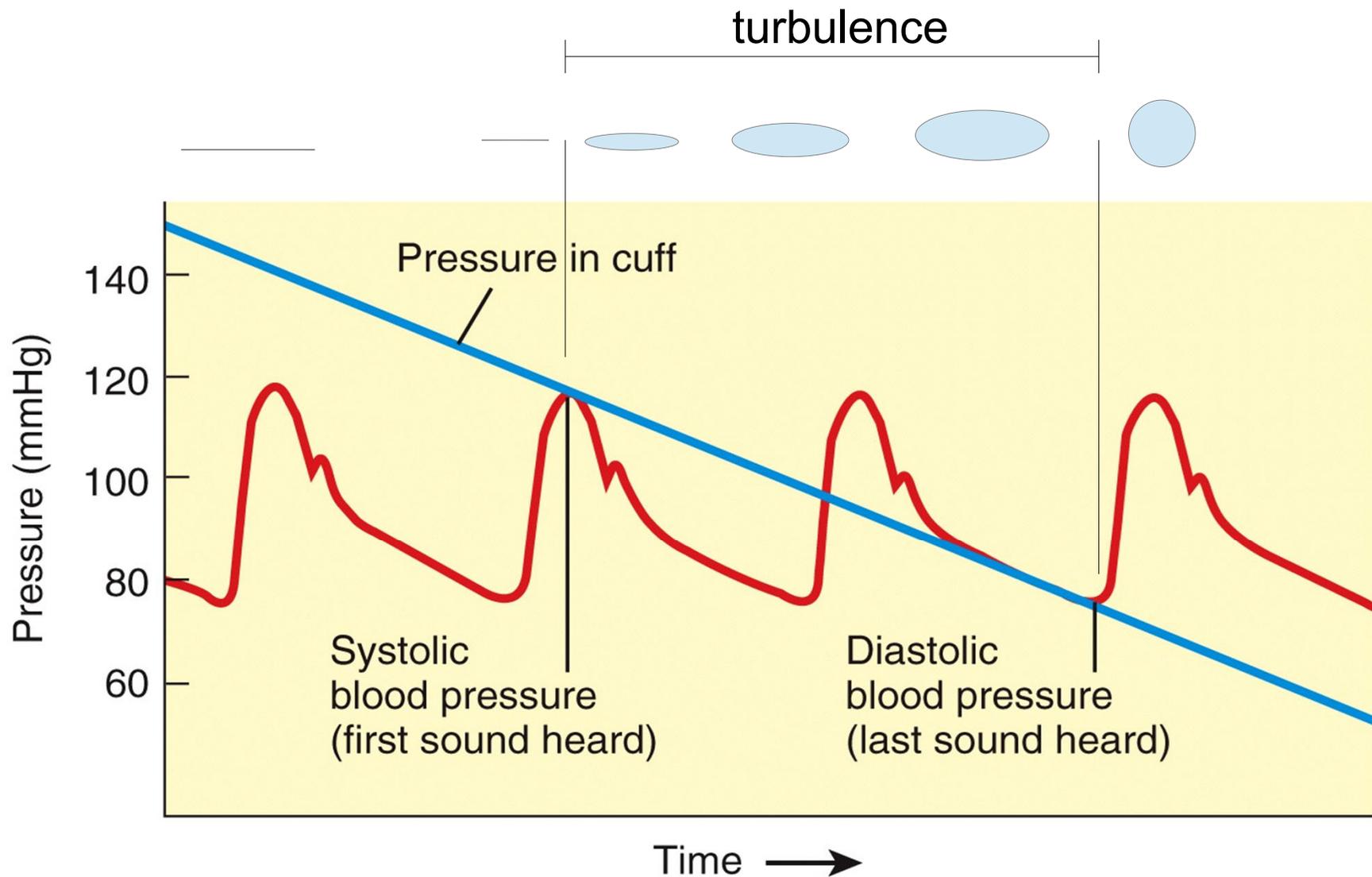
Why do you want a large pulse pressure at the aorta?

Why no pulse pressure in a capillary?



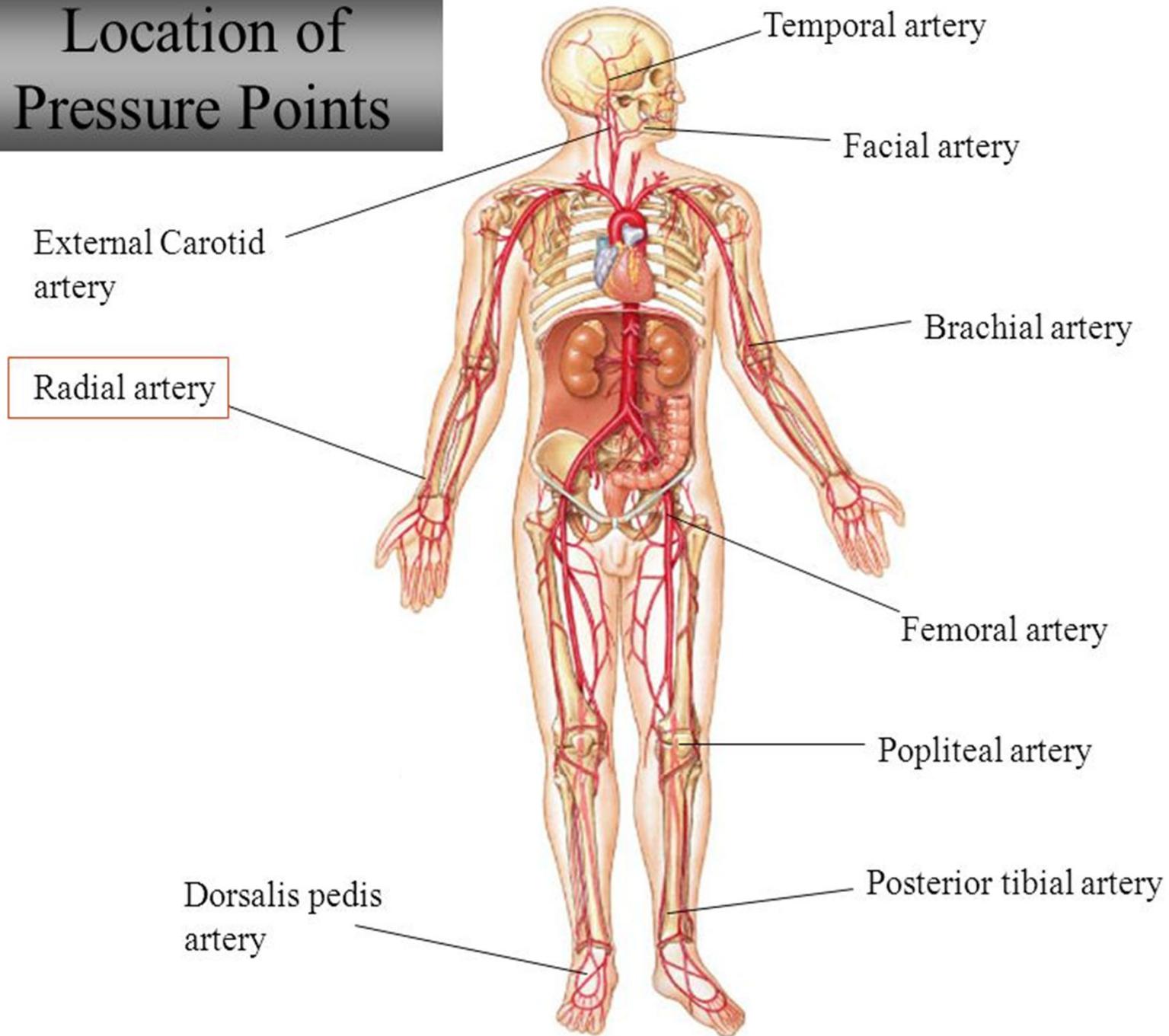
> Explain why this make sense!

Blood pressure can be measured at the brachial artery with a **sphygmomanometer** */// This is a lab requirement // (See posted video)*



NOTE – Sound is turbulence!!!

Location of Pressure Points



Locations where you can check pulse.

Table 10.2 The correlation between palpable pulses and systolic blood pressure

Palpable pulse site	Systolic blood pressure
Radial	>80 mmHg
Femoral	>70 mmHg
Carotid	>60 mmHg

Source: Greaves et al. (2001). Reproduced by permission of Edward Arnold (Publishers) Limited.

Abnormal Blood Pressures

Hypertension – high blood pressure

- chronic is resting BP > 140/90
- weaken small arteries and may cause aneurysms
- may cause enlarged heart with less cardiac output

Hypotension – chronic low resting BP

- caused by blood loss
- dehydration, anemia

Hypertension

Hypertension – most common cardiovascular disease affecting about 30% of Americans over 50

Primary hypertension caused by obesity, sedentary behavior, diet, and nicotine

Secondary to other disease /// e.g. kidney disease, hyperthyroidism

“The silent killer” // major cause of heart failure, stroke, and kidney failure

Damages heart by **increasing afterload** /// **myocardium enlarges** until overstretched and inefficient

Renal arterioles thicken in response to stress /// drop in renal BP leads to salt retention (aldosterone) and worsens the overall hypertension

Do you know your blood pressure? Most young people do not!

Factors That Increase Blood Pressure

BP is a function of:

cardiac output

total blood volume

increase venous return

peripheral resistance

Know This !!!!!!

Peripheral resistance is a function of:

This too is important !!!!!!

> blood viscosity

> vessel length

> vessel radius (most important of three) // vasomotor regulated!

Peripheral Resistance Control by Arterioles

Arterioles are the most significant point of control over peripheral resistance and blood flow

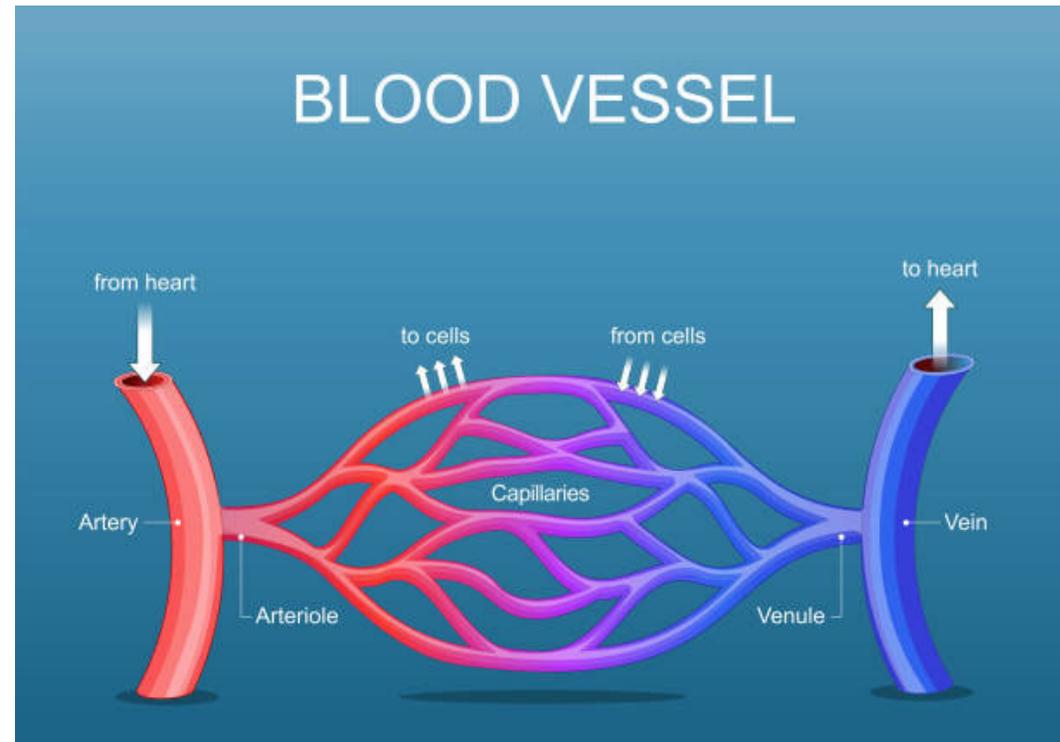
On proximal side of capillary beds and best positioned to regulate flow into the capillaries

Outnumber any other type of artery, providing the most numerous control points

More muscular in proportion to their diameter

Regulated by vasomotion // medulla oblongata

Arterioles responsible for half of the total peripheral resistance



Three Variables of Peripheral Resistance

Arterioles resist the flow of blood away from the heart /// these are the small vessels right before the capillary beds

Peripheral resistance depends on three variables

1. blood viscosity // “thickness of blood”

RBC count and albumin concentration elevate viscosity the most

Decreased viscosity with anemia and hypoproteinemia speed flow

Increased viscosity with polycythemia and dehydration slow flow

Three Variables of Peripheral Resistance

2. vessel length

The farther liquid travels through a tube, the more cumulative friction it encounters

Pressure and flow decline with distance

3. vessel radius */// most powerful influence over flow*

Only significant way of controlling peripheral resistance.

Vasomotion - change in vessel radius

Vasoconstriction - by muscular effort that results in smooth muscle contraction

Vasodilation - by relaxation of the smooth muscle

(see next slide)

Three Variables of Peripheral Resistance

vessel radius markedly affects **blood velocity**

Laminar flow - flows in layers, faster in center

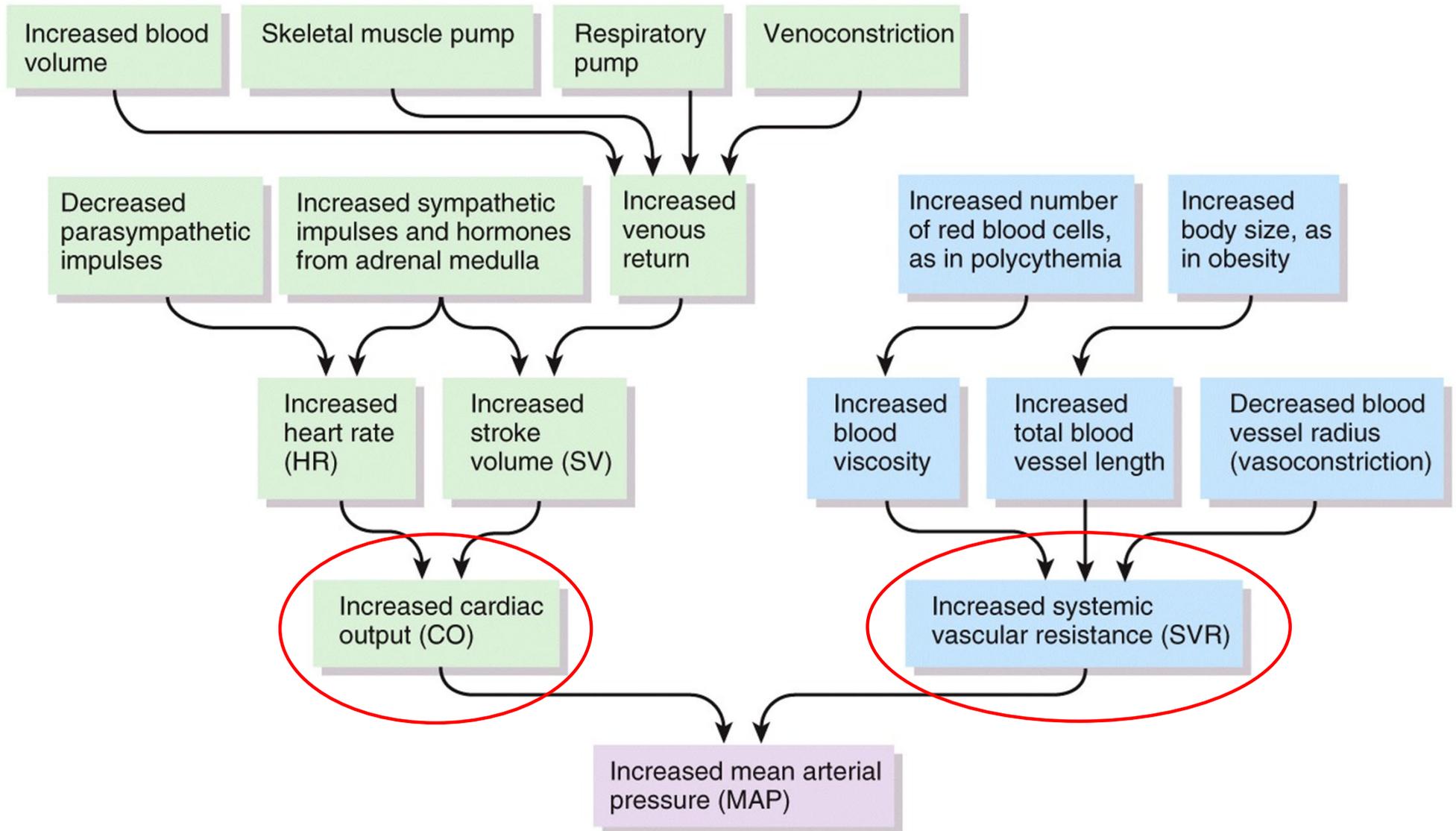
Blood flow (F) proportional to the fourth power of radius (r),
 $F \propto r^4$

Arterioles can constrict to 1/3 of fully relaxed radius

If $r = 3$ mm, $F = (3^4) = 81$ mm/sec; if $r = 1$ mm, $F = 1$ mm/sec

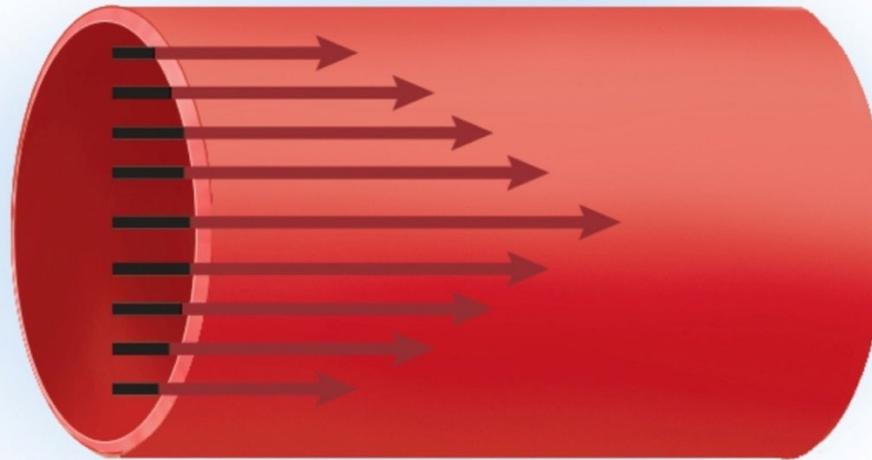
An increase of three times the radius of a vessel results in eighty-one times the flow

Summary of Factors That Increase Blood Pressure

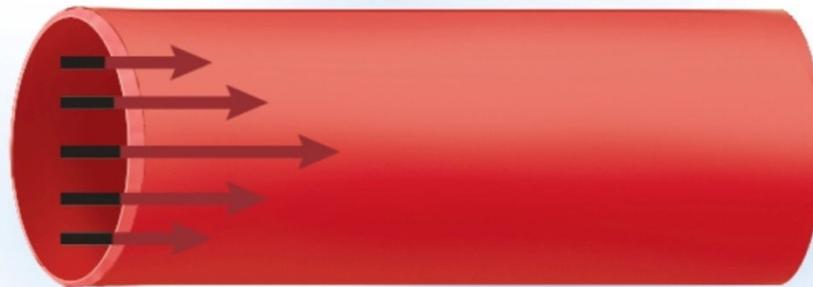


Laminar Flow and Vessel Radius

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(a)



(b)

Other Conditions Affecting Local Blood Flow

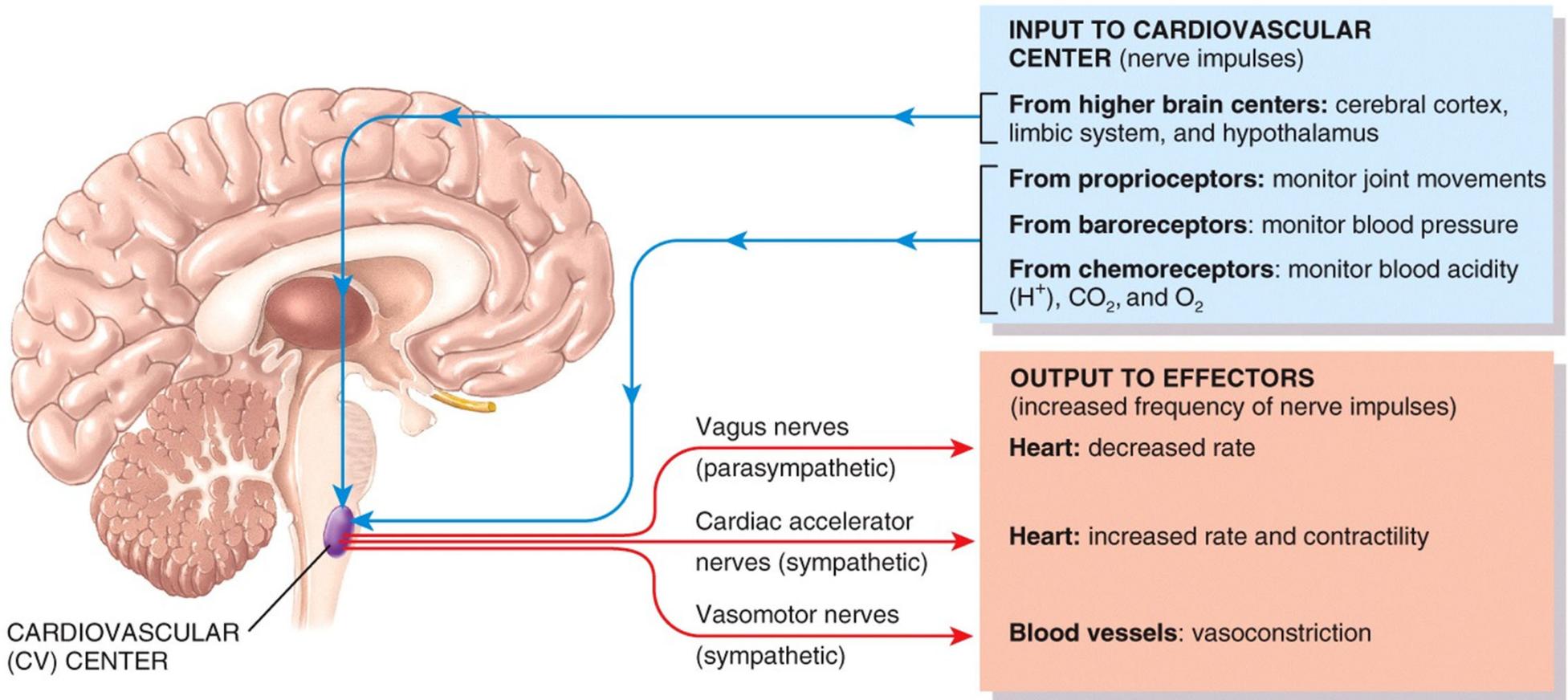
Reactive hyperemia

If blood supply is first cut off so carbon dioxide builds up in tissue so when blood flow is restored then high carbon dioxide concentration results in above normal blood flow through tissue. Think about your rosy cheeks when you come into the house after a winter walk.

Angiogenesis = growth of new blood vessels /// controlled by growth factors

- occurs as you repair a bruised area
- occurs in re-growth of uterine lining
- around a coronary artery obstructions
- in exercised muscle
- in malignant tumors

Cardiovascular Center in Medulla Oblongata Related to Blood Pressure



What are the two functions of the vasomotor center?

- = Method to raise or lower BP throughout the whole body

increasing BP requires medullary vasomotor center

or widespread circulation of a hormone like epinephrine

important in supporting cerebral perfusion during a hemorrhage or dehydration

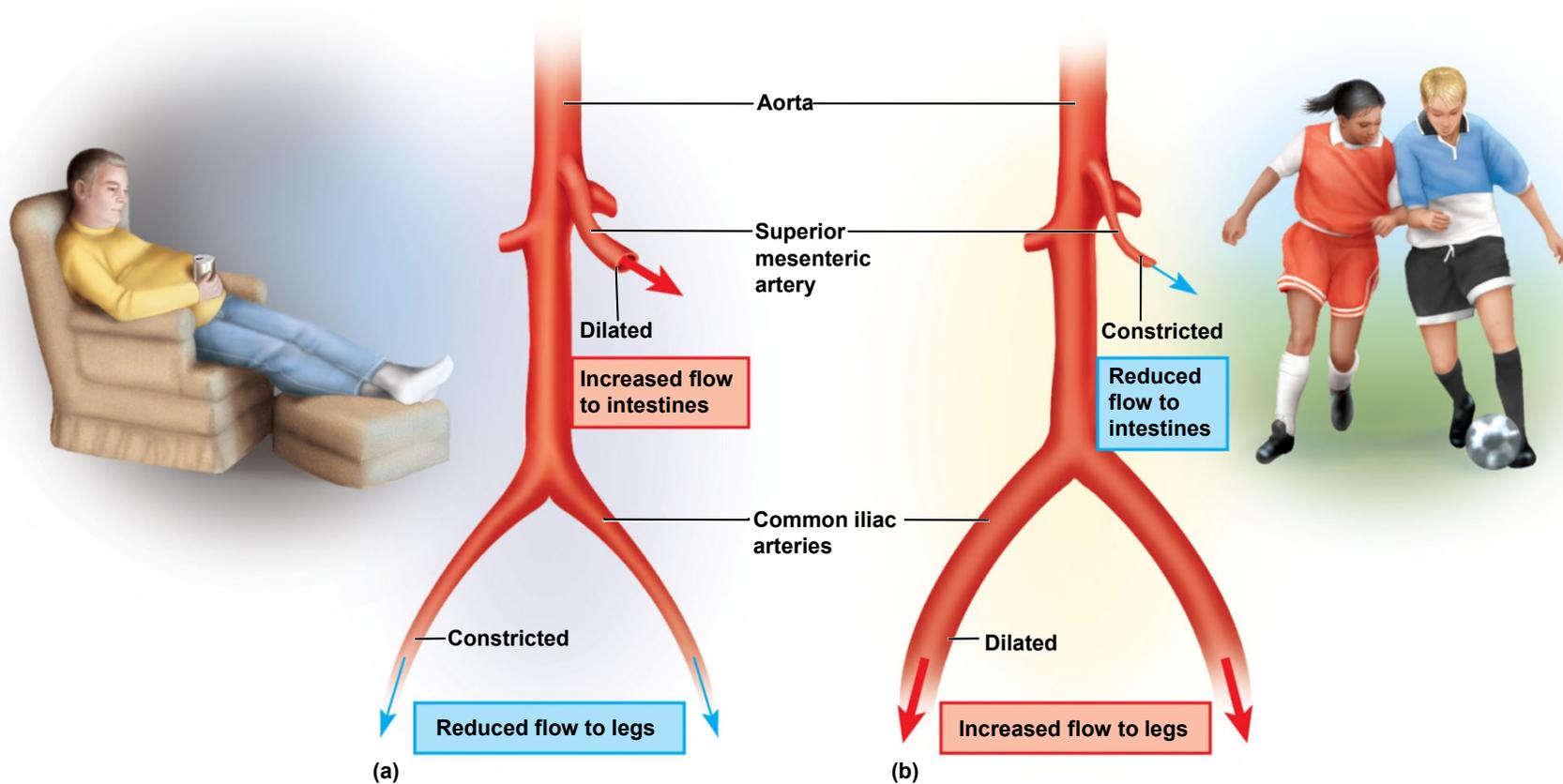
Second Function of the Vasomotor Center

2 = Method to re-route blood from one region to another region in order to meet metabolic need of individual organs

- either may be centrally or locally controlled
- during exercise, sympathetic system reduces blood flow to kidneys and digestive tract and increases blood flow to skeletal muscles
- metabolite accumulation in a tissue affects local circulation without affecting circulation elsewhere in the body

Blood Flow in Response to Needs Vasomotion

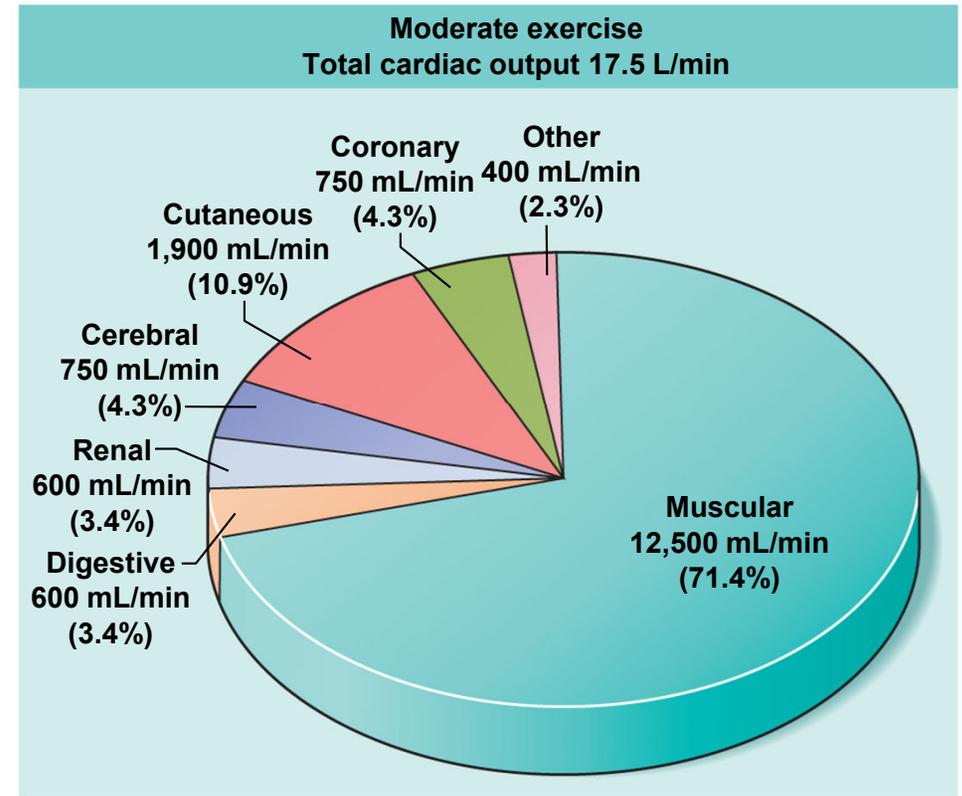
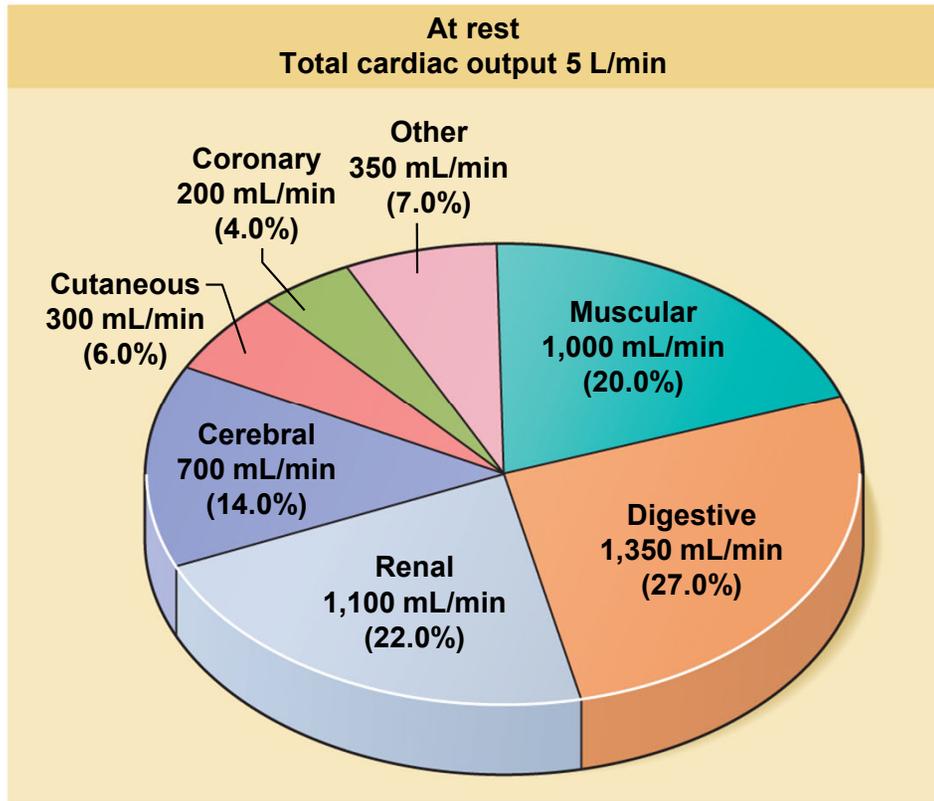
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arterioles shift blood flow with changing priorities

Blood Flow Comparison

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during exercise // increased perfusion of lungs, myocardium, and skeletal muscles
/// decreased perfusion of kidneys and digestive tract

Mean Arterial Blood Pressure

Mean arterial pressure (MAP) – the mean arterial pressure is an estimate of what would be the “average pressure” throughout the cardiac cycle

MAP = diastolic pressure + (1/3 of pulse pressure)

$$\text{MAP} = 80 + (1/3) 40 = 93.2$$

Best risk level indicator for edema, fainting (syncope), atherosclerosis, kidney failure, and aneurysm

Preventing Excess Blood Pressure in Blood Vessels

The body's chief mechanisms in preventing excessive blood pressure is the **stretch and recoil of arteries** during the cardiac cycle

The importance of arterial elasticity

expansion and recoil maintains steady flow of blood throughout cardiac cycle

smooth out pressure fluctuations

decreases stress on small arteries

Note /// BP rises with age // caused by change in amount of elastic connective tissue fibers /// arteries less distend able and absorb less systolic force

Blood Flow Between Different Points

From aorta to capillaries, blood velocity (speed) decreases for three reasons:

greater distance, more friction to reduce speed

smaller radius of arterioles and capillaries offers more resistance

farther from heart, the number of vessels and their total cross-sectional area becomes greater and greater

From capillaries to vena cava, flow increases again

decreased resistance going from capillaries to veins

large amount of blood forced into smaller channels

however /// never regains velocity of large arteries

How is blood flow in the capillaries regulated?

Neural Control – Hormonal Control - Autoregulation

Vasomotion is a quick and powerful way to change blood flow into the capillary bed by adjusting smooth muscle. However, there are two other ways to influence blood flow in the capillaries.

- 1) **neural** control (vasomotor center in medulla oblongata)
- 2) **hormonal control** (secretions from adrenal gland - catecholamines)
- 3) **autoregulation** - from within the capillary bed by using carbon dioxide to adjusting tension on the smooth muscles of the per-capillary sphincters. Increasing CO₂ relaxes smooth muscle and more blood flows into the capillary bed.

Other Local Control Mechanisms

Vasoactive chemicals /// substances secreted by platelets, endothelial cells, and perivascular tissue may also stimulate vasomotion /// Eg. Mast cells secrete histamine to cause

Endothelial cells secrete

Prostacyclin (makes inside surface of blood vessels “slippery” // less likely for platelets to stick to lining of blood vessels and release chemicals which would cause vasoconstriction)

Nitric oxide (vasodilators)

Endothelins (vasoconstrictor)

Vasomotor Center As An Integrating Center

Vasomotor center is the integrating center for three autonomic reflexes:

- baroreflexes
- chemoreflexes
- medullary ischemic reflex

Smooth muscle of arterioles regulated by the sympathetic division of the autonomic nervous systems // SNS is the motor pathway

Vasomotor center located in medulla oblongata

- causes some arterioles in the body to **constrict**
- **dilates** vessels in skeletal and cardiac muscle to during exercise

Note: precapillary sphincters respond only to local and hormonal control due to lack of innervation

Vasomotor Center As An Integrating Center

ANS negative feedback response to changes in blood pressure

Mechanism:

- 1) An increase in BP detected by **carotid sinuses**
- 2) Signals sent to brainstem by way of **glossopharyngeal nerve**
- 3) Inhibit the sympathetic cardiac and vasomotor neurons reducing sympathetic tone → blood pressure drops

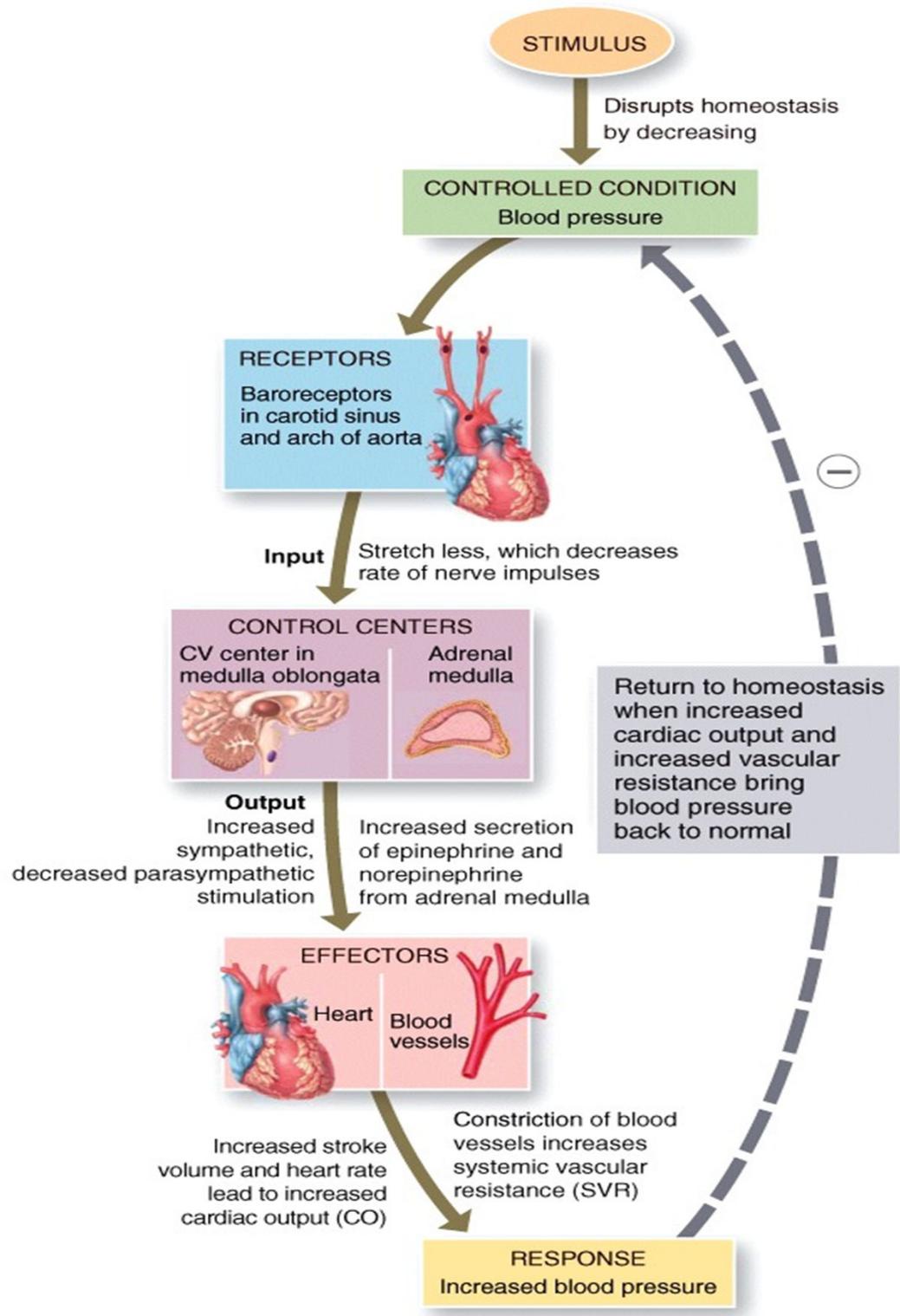
Excite vagal fibers (parasympathetic)

»slow heart rate

»reduce cardiac output

»reducingg BP

- 4) Note: decrease in BP will have opposite effects



Baroreflex

- Baroreflexes important in **short-term regulation of BP**

–e.g. adjustments for rapid changes in posture / like when you “hop” out of bed!

- **This reflex is unable to correct for chronic hypertension**

Chemoreflex

An automatic response to changes in blood chemistry

Especially pH, and concentrations of O₂ and CO₂

Chemoreceptors called aortic bodies and carotid bodies ///
located in aortic arch, subclavian arteries, external carotid arteries

Primary role /// adjust respiration to changes in blood chemistry

Secondary role /// vasomotion

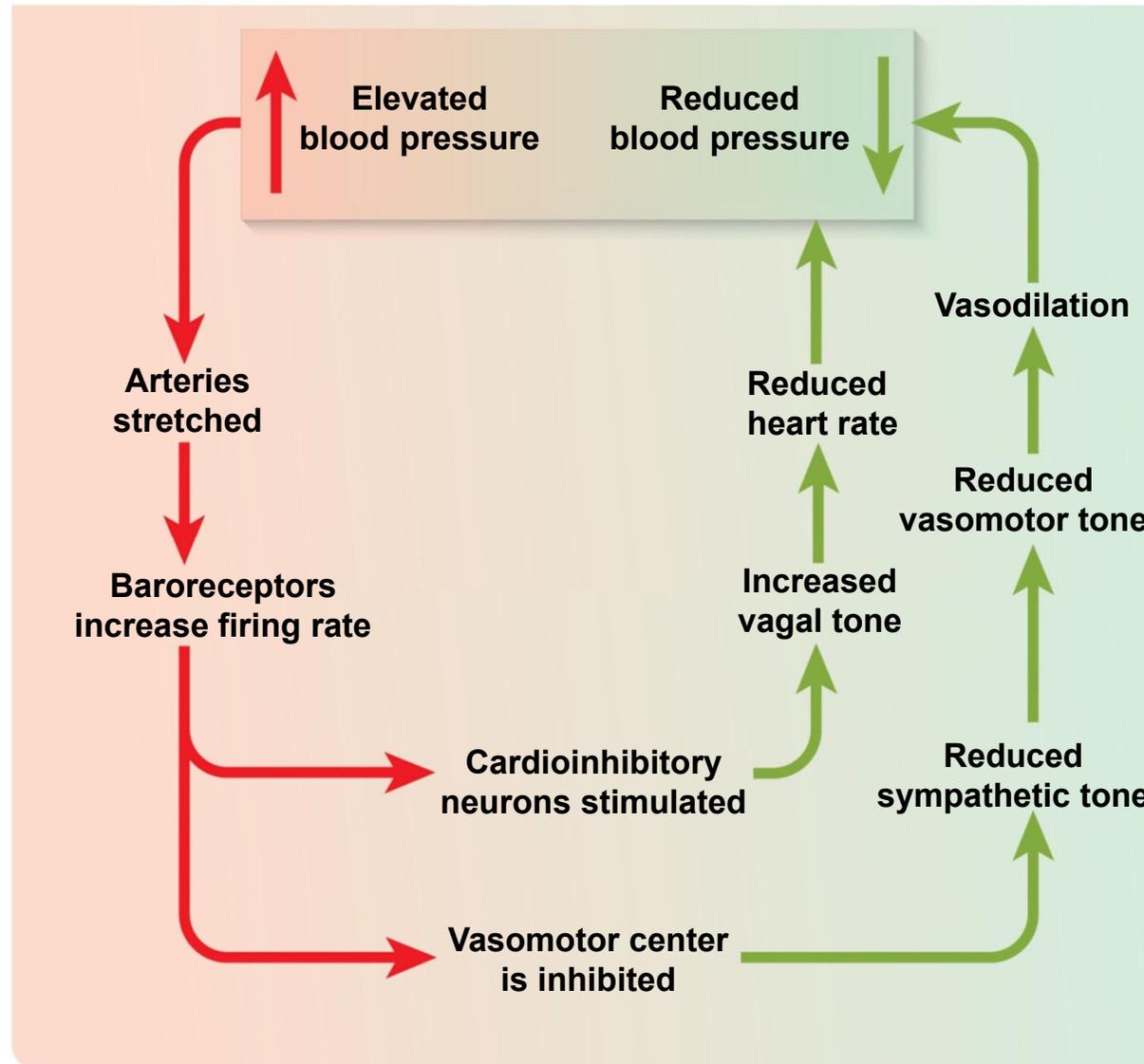
hypoxemia, hypercapnia, and acidosis stimulate chemoreceptors

acting through vasomotor center to cause widespread vasoconstriction

increasing BP, increasing lung perfusion and gas exchange

Response to Elevated Blood Pressure

Negative Feedback Control



Medulla Oblongata Ischemic Reflex

Automatic response to a reduced blood perfusion in the brain

Medulla oblongata monitors its own blood supply

Activates corrective reflexes when it senses ischemia (insufficient perfusion of the brain)

Cardiac and vasomotor centers send **sympathetic signals** to heart and blood vessels

- increases heart rate and contraction force
- causes widespread vasoconstriction below the neck
- goal - raise overall BP and restores normal perfusion to the brain**

Other areas of the brain can also have an effect on vasomotor center /// stress, anger, and arousal can influence medulla oblongata to increase BP (Think about the Triune Brain Theory!)

Hormonal Control

Hormones also influence blood pressure.....

1 - by their direct effect on smooth muscle at arterioles

2 - by regulating water balance // change blood volume through the renin-angiotensinogen-aldosterone pathway

The Renin–Angiotensinogen–Aldosterone Pathway

–Renin is released into blood by kidney in response to low blood pressure

–Renin converts the blood protein angiotensinogen into angiotensin-1

–Angiotensin-1 circulates through lungs where angiotensin converting enzyme changes angiotensin-1 into angiotensin-2

–Angiotensin-2 will cause the adrenal gland to release aldosterone /// aldosterone is a salt retention hormone – it promotes Na^+ and water retention by kidneys // angiotensin-2 is also a vasopressor and will increase peripheral resistance in the arterioles which increases blood pressure

–This mechanism increases blood pressure by increasing both blood volume and peripheral resistance.

More Hormonal Control

Atrial natriuretic peptide

- increases urinary sodium excretion
- reduces blood volume and promotes vasodilation
- lowers blood pressure

Antidiuretic hormone

- promotes water retention and raises BP
- pathologically high concentrations - vasoconstrictor

Epinephrine and norepinephrine

- effect most blood vessels
- binds to α -adrenergic receptors – **vasoconstriction**

- skeletal and cardiac muscle blood vessels
- binds to β - adrenergic receptors - **vasodilation**

Variations in Capillary Activity

Capillaries usually reabsorb 85% of the fluid they filter – key exception:

kidney capillaries in glomeruli do not reabsorb

alveolar capillaries in lung absorb completely to keep fluid out of air spaces

Capillary activity varies from moment to moment

collapsed in resting tissue, reabsorption predominates since BP is low

metabolically active tissue has increase in capillary flow and BP /// increase in muscular bulk by 25% due to accumulation of fluid

Edema

The accumulation of excess fluid in a tissue

Occurs when fluid filters into a tissue faster than it is absorbed

Three primary causes

increased capillary filtration /// kidney failure, histamine release, old age, poor venous return

reduced capillary absorption /// hypoproteinemia, liver disease, dietary protein deficiency

obstructed lymphatic drainage /// surgical removal of lymph nodes

Consequences of Edema

Tissue necrosis /// oxygen delivery and waste removal impaired

Pulmonary edema /// suffocation threat

Cerebral edema /// headaches, nausea, seizures, and coma

Severe edema or circulatory shock /// excess fluid in tissue spaces causes low blood volume and low blood pressure

Circulatory Shock

Any state in which cardiac output is insufficient to meet the body's metabolic needs

Cardiogenic shock - inadequate pumping of heart (MI)

Low venous return (LVR) – cardiac output is low because too little blood is returning to the heart

Three common forms of low venous shock

1.hypovolemic shock - most common /// loss of blood volume /// trauma, burns, dehydration

2.obstructed venous return shock /// tumor or aneurysm compresses a vein

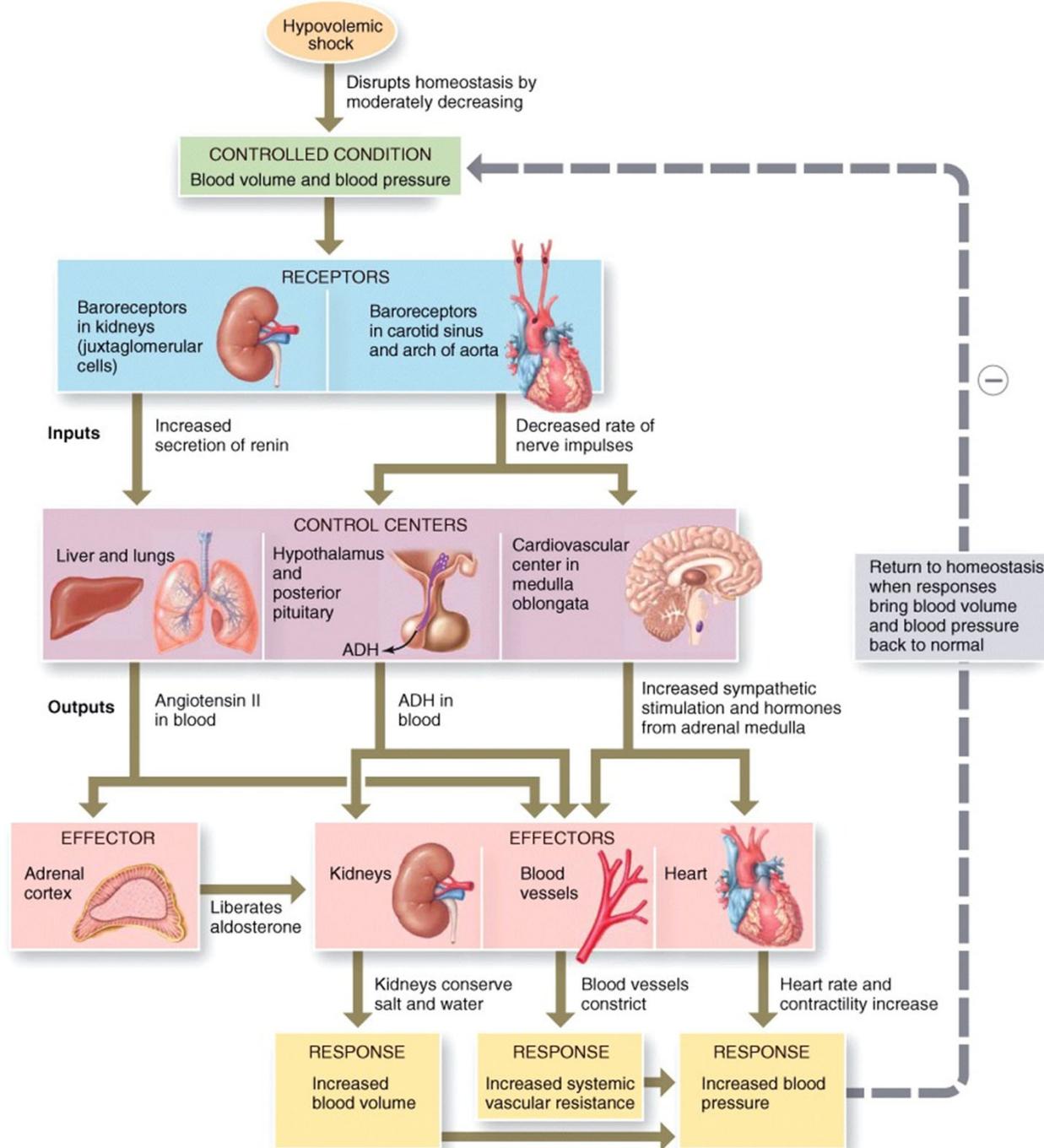
3.venous pooling (vascular) shock

long periods of standing, sitting or widespread vasodilation

neurogenic shock - loss of vasomotor tone, vasodilation

causes from emotional shock to brainstem injury

How do we restore normal blood pressure during hypovolemic shock?



Other Causes of Circulatory Shock

Septic shock

bacterial toxins trigger vasodilation and increased capillary permeability

Anaphylactic shock

severe immune reaction to antigen, histamine release, generalized vasodilation, increased capillary permeability

Compensated Shock

Several homeostatic mechanisms bring about spontaneous recovery

Decreased BP triggers the following

- baroreflex
- production of angiotensin II
- both counteract shock by stimulating vasoconstriction

If person faints (syncope) and then falls into horizontal position, now gravity has helped to restore blood flow to brain // quicker if feet are raised

Decompensated Shock

If shock compensation mechanisms are inadequate, several life-threatening positive feedback loops occur

Poor cardiac output results in myocardial ischemia (ischemia is reduced blood flow) which can result in an infarction (necrosis due to lack of blood flow) /// this further weakens the heart and reduces output

Slow circulation can lead to disseminated intra-vascular coagulation

Vessels become congested with clotted blood // venous return grows worse

Ischemia and acidosis in brainstem depresses vasomotor and cardiac centers

Loss of vasomotor tone, further dilation in systemic circuit, which results in further drop in BP and less cardiac output

Resulting damage to cardiac and brain tissue may be too great to survive – death occurs

Summary of Factors That Increase Blood Pressure

