Cardiovascular Physiology

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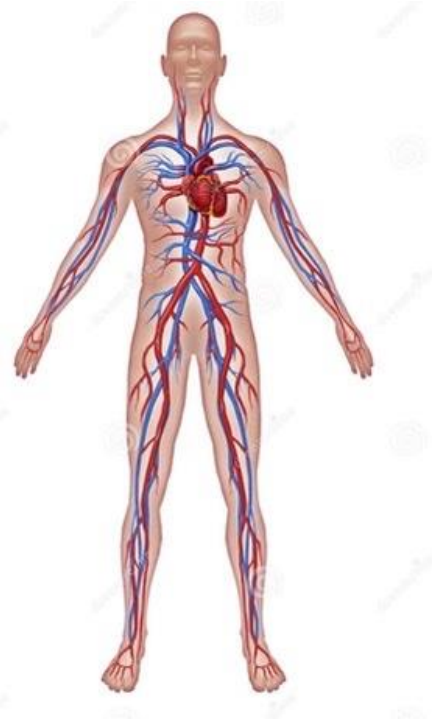

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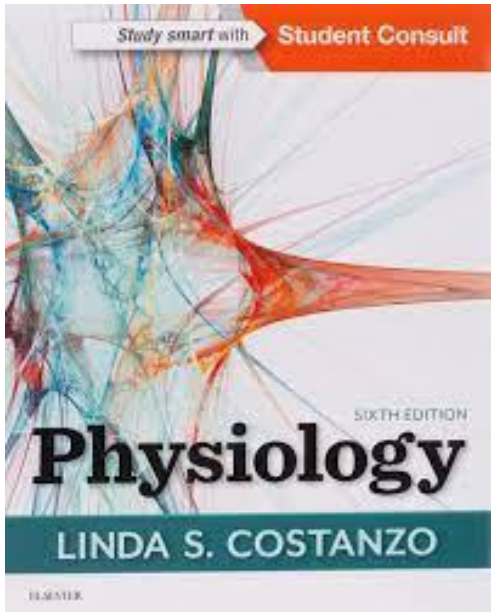
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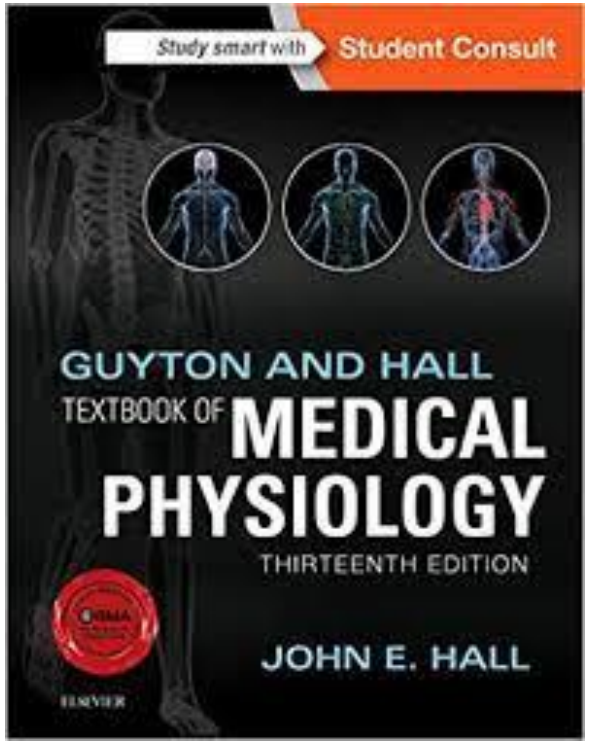
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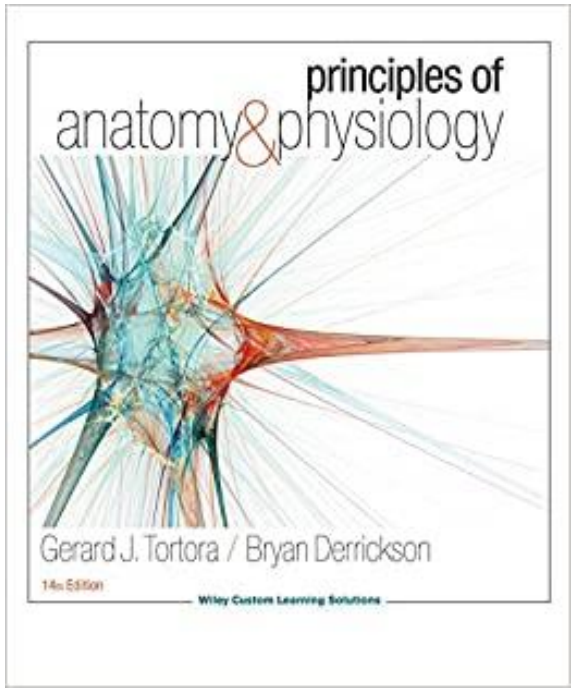
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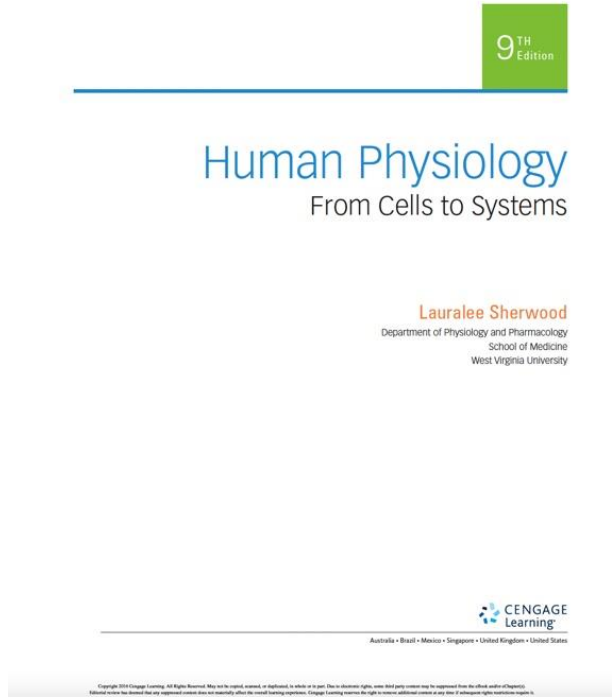
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ABP Regulation

Why ABP need to be regulated

- Mean arterial pressure is the main driving force for propelling blood to the tissues. This pressure must be closely regulated for two reasons.
- First, it must be high enough to ensure sufficient driving pressure; without this pressure, the brain and other organs do not receive adequate flow, no matter what local adjustments are made in the resistance of the arterioles supplying them.
- Second, the pressure must not be so high that it creates extra work for the heart and increases the risk of vascular damage and possible rupture of small blood vessels.

Nervous regulation of the circulation

- in most tissues, all the vessels except the capillaries are innervated.
- The innervation of the small arteries and arterioles allows sympathetic stimulation to increase resistance to blood flow and thereby decrease the rate of blood flow through the tissues.
- The innervation of the large vessels, particularly of the veins, makes it possible for sympathetic stimulation to decrease the volume of these vessels. This decrease in volume can push blood into the heart and thereby plays a major role in regulation of heart pumping

Nervous regulation of the circulation

- sympathetic stimulation markedly increases the activity of the heart, both increasing the heart rate and enhancing its strength and volume of pumping.
- parasympathetic stimulation causes a marked decrease in heart rate and a slight decrease in heart muscle contractility.
- This sympathetic vasoconstrictor effect is especially powerful in the kidneys, intestines, spleen, and skin but is much less potent in skeletal muscle, heart, and the brain.

Rapid control of arterial pressure by nervous system

- three major changes occur due to sympatsimultaneously, each of which helps increase arterial pressure:
- 1. Most arterioles of the systemic circulation are constricted, which greatly increases the total peripheral resistance, thereby increasing the arterial pressure.
- 2. The veins especially (but the other large vessels of the circulation as well) are strongly constricted. This constriction displaces blood out of the large peripheral blood vessels toward the heart, thus increasing the volume of blood in the heart chambers.
- The stretch of the heart then causes the heart to beat with greater force and therefore to pump increased quantities of blood. This also increases the arterial pressure.

Rapid control of arterial pressure by nervous system

- Finally, the heart is directly stimulated by the autonomic nervous system, further enhancing cardiac pumping.
- Much of this enhanced cardiac pumping is caused by an increase in the heart rate, which sometimes increases to as much as three times normal.
- In addition, sympathetic nervous signals directly increase the contractile force of the heart muscle, increasing the capability of the heart to pump larger volumes of blood.
- During strong sympathetic stimulation, the heart can pump about two times as much blood as under normal conditions, which contributes still more to the acute rise in arterial pressure.

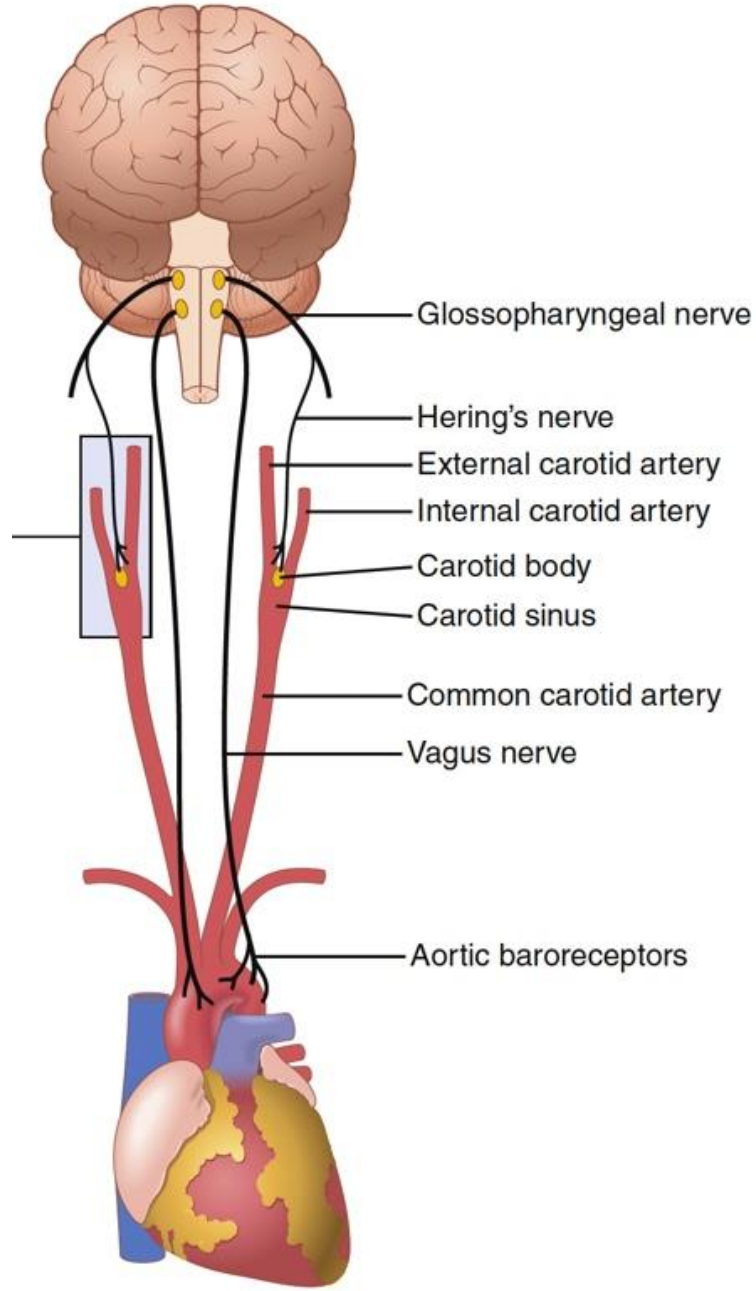
Baroreceptor reflex

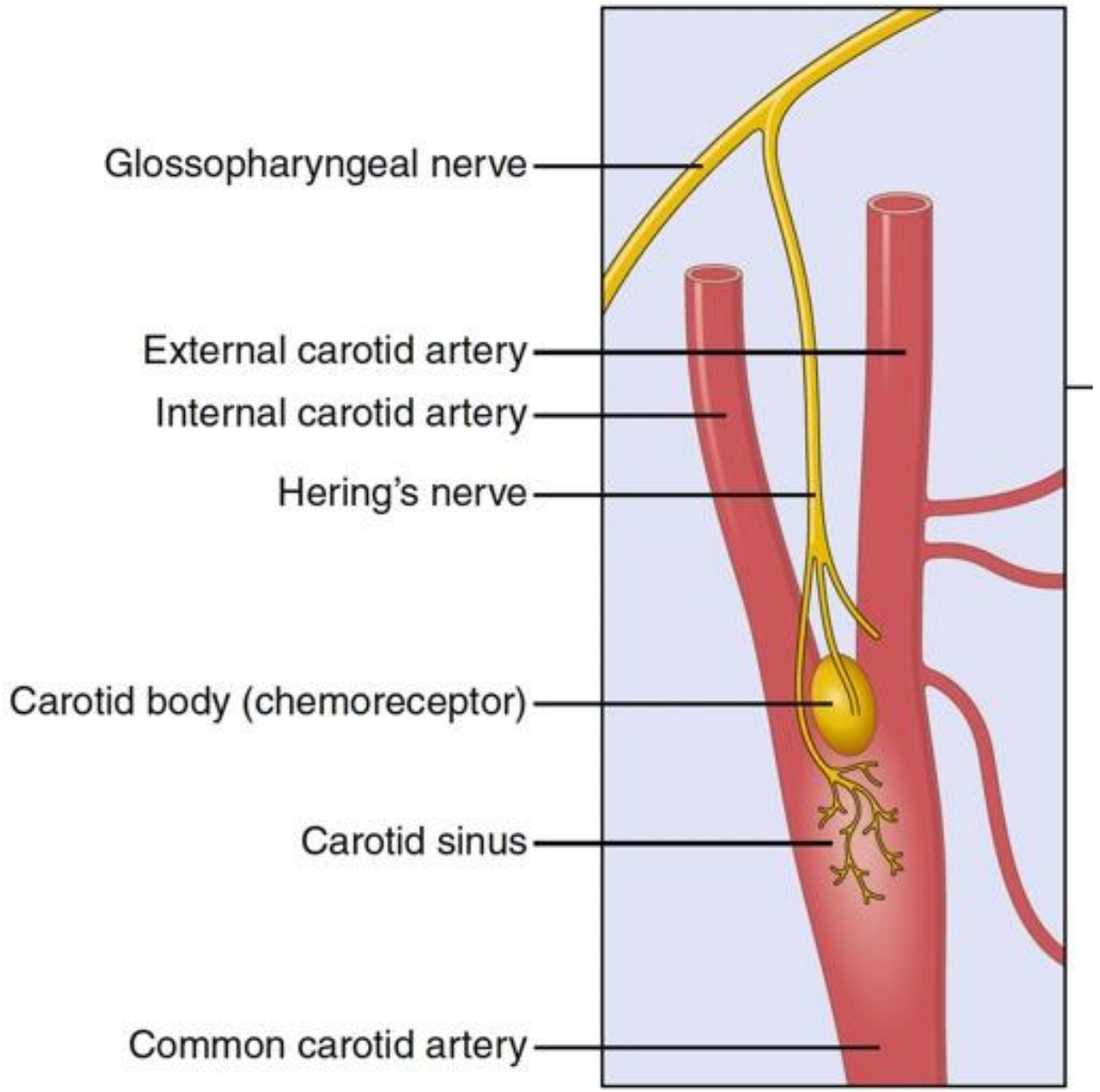
- blood pressure is sensed by baroreceptors in the carotid sinus and aortic arch.
- Afferent information about blood pressure is then sent to the medulla via the glossopharyngeal (CN IX) and vagus (CN X) nerves.
- This information is integrated in the nucleus tractus solitarius, which then directs changes in the activity of several cardiovascular centers.
- These cardiovascular centers are tonically active, and the nucleus tractus solitarius simply directs, via the centers, increases or decreases in outflow from the sympathetic and parasympathetic nervous systems.

Baroreceptor reflex

- A rise in arterial pressure stretches the baroreceptors and causes them to transmit signals into the CNS.
- Feedback signals are then sent back through the autonomic nervous system to the circulation to reduce arterial pressure down toward the normal level.

- in the normal operating range of arterial pressure, around 100 mm Hg, even a slight change in pressure causes a strong change in the baroreflex signal to readjust arterial pressure back toward normal. Thus, the baroreceptor feedback mechanism functions most effectively in the pressure range where it is most needed


Glossopharyngeal nerve
Hering's nerve
External carotid artery
Internal carotid artery
Carotid body
Carotid sinus
Common carotid artery
Vagus nerve
Aortic baroreceptors


Glossopharyngeal nerve
External carotid artery
Internal carotid artery
Hering's nerve
Carotid body (chemoreceptor)
Carotid sinus
Common carotid artery

Baroreceptor reflex

- Although the baroreceptors are sensitive to the absolute level of pressure, they are even more sensitive to changes in pressure and the rate of change of pressure.
- The strongest stimulus for the baroreceptors is a rapid change in arterial pressure.
- That is, if the mean arterial pressure is 150 mm Hg but at that moment is rising rapidly, the rate of impulse transmission may be as much as twice that when the pressure is stationary at 150 mm Hg.

Baroreceptor reflex

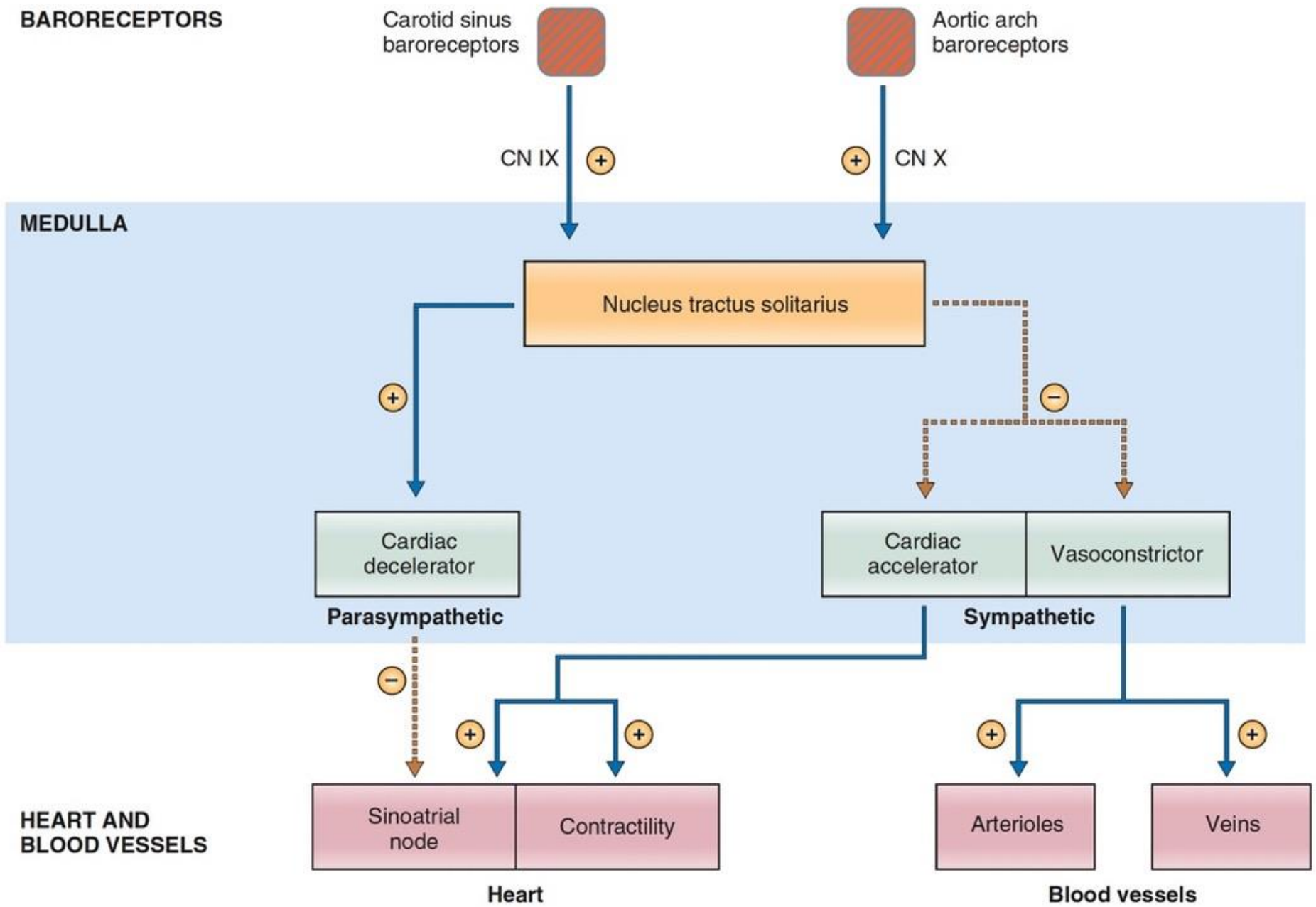
- Pressure buffer system:
- A primary purpose of the arterial baroreceptor system is therefore to reduce the minute by minute variation in arterial pressure to about one-third that which would occur if the baroreceptor system were not present.
- the extreme variability of pressure caused by simple events of the day, such as lying down, standing, excitement, eating, defecation, and noises is minimized.
- when the baroreceptors were functioning normally, the mean arterial pressure remained within a narrow range of between 85 and 115 mm Hg throughout the day and, for most of the day, it remained at about 100 mm Hg.

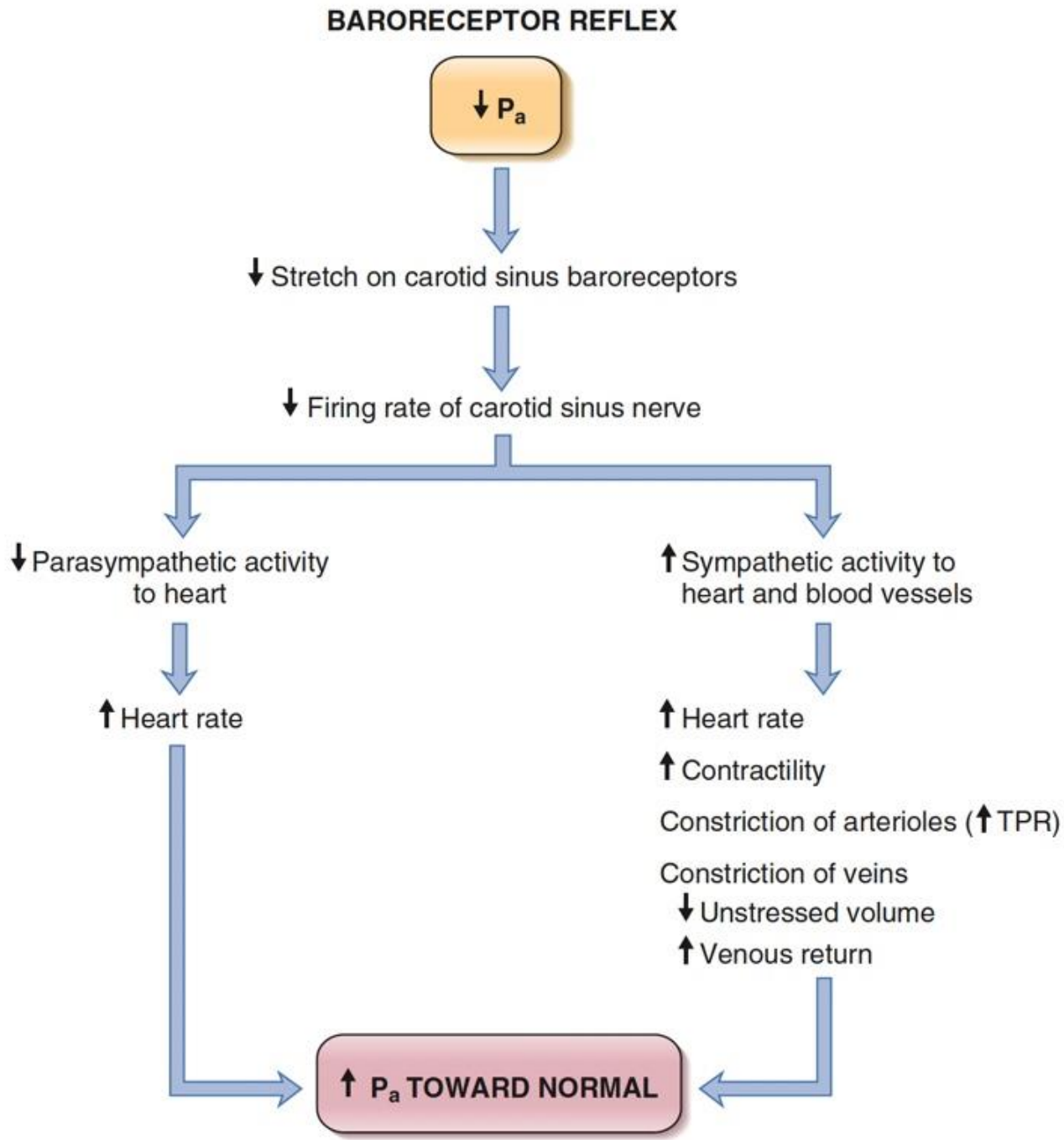
Orthostatic hypotension

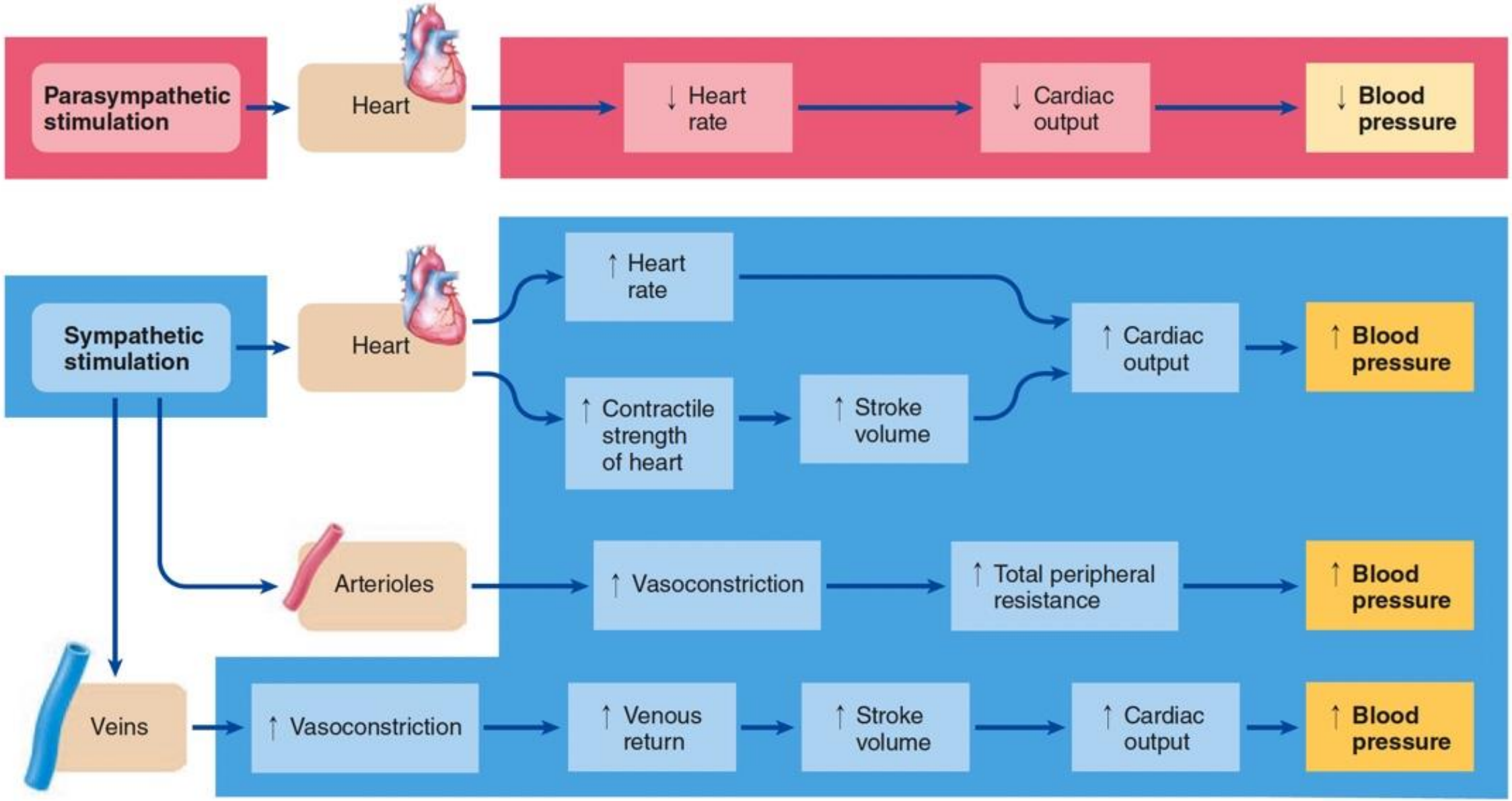
- The ability of the baroreceptors to maintain relatively constant arterial pressure in the upper body is important when a person stands up after lying down.
- Immediately on standing, the arterial pressure in the head and upper part of the body tends to fall, and marked reduction of this pressure could cause loss of consciousness.
- However, the falling pressure at the baroreceptors elicits an immediate reflex, resulting in strong sympathetic discharge throughout the body that minimizes the decrease in pressure in the head and upper body.

Baroreceptor reflex

- relatively unimportant in chronic regulation of arterial pressure is that they tend to reset in 1 to 2 days to the pressure level to which they are exposed. That is, if the arterial pressure rises from the normal value of 100 to 160 mm Hg, a very high rate of baroreceptor impulses is at first transmitted. During the next few minutes, the rate of firing diminishes considerably. Then, it diminishes much more slowly during the next 1 to 2 days, at the end of which time the rate of firing will have returned to nearly normal, despite the fact that the mean arterial pressure still remains at 160 mm Hg. Conversely, when the arterial pressure falls to a very low level, the baroreceptors at first transmit no impulses but gradually, over 1 to 2 days, the rate of baroreceptor firing returns toward the control level.


BARORECEPTORS
Carotid sinus baroreceptors
Aortic arch baroreceptors
CN IX
CN X
MEDULLA
Nucleus tractus solitarius
Cardiac decelerator
Cardiac accelerator
Vasoconstrictor
Parasympathetic
Sympathetic
HEART AND BLOOD VESSELS
Sinoatrial node
Contractility
Arterioles
Veins
Heart
Blood vessels


BARORECEPTOR REFLEX
↓ P_a
↓ Stretch on carotid sinus baroreceptors
↓ Firing rate of carotid sinus nerve
↓ Parasympathetic activity to heart
↑ Sympathetic activity to heart and blood vessels
↑ Heart rate
↑ Heart rate
↑ Contractility
Constriction of arterioles (↑ TPR)
Constriction of veins
↓ Unstressed volume
↑ Venous return
↑ P_a TOWARD NORMAL


Parasympathetic stimulation
Heart
↓ Heart rate
↓ Cardiac output
↓ Blood pressure
Sympathetic stimulation
Heart
↑ Heart rate
↑ Contractile strength of heart
↑ Stroke volume
↑ Cardiac output
↑ Blood pressure
Arterioles
↑ Vasoconstriction
↑ Total peripheral resistance
↑ Blood pressure
Veins
↑ Vasoconstriction
↑ Venous return
↑ Stroke volume
↑ Cardiac output
↑ Blood pressure

Chemoreceptor reflex

- Chemoreceptor organs: carotid bodies and aortic bodies.
- Sensitive to lowO_2 or elevated CO_2 and H^+.
- The signals transmitted from the chemoreceptors excite the cardiovascular center, and this response elevates the arterial pressure back toward normal.
- However, this chemoreceptor reflex is not a powerful arterial pressure controller until the arterial pressure falls below 80 mm Hg. Therefore, it is at the lower pressures that this reflex becomes important to help prevent further decreases in arterial pressure.

Central chemoreceptors

- Central chemoreceptors are located in the medulla. They are most sensitive to CO2 and pH and less sensitive to O2.
- If the brain becomes ischemic (i.e., there is decreased cerebral blood flow), cerebral PCO2 immediately increases and pH decreases. The medullary chemoreceptors detect these changes and direct an increase in sympathetic outflow that causes intense arteriolar vasoconstriction in many vascular beds and an increase in TPR. Blood flow is thereby redirected to the brain to maintain its perfusion. As a result of this vasoconstriction, BP increases dramatically, even to life-threatening levels.

Atrial and pulmonary artery reflexes

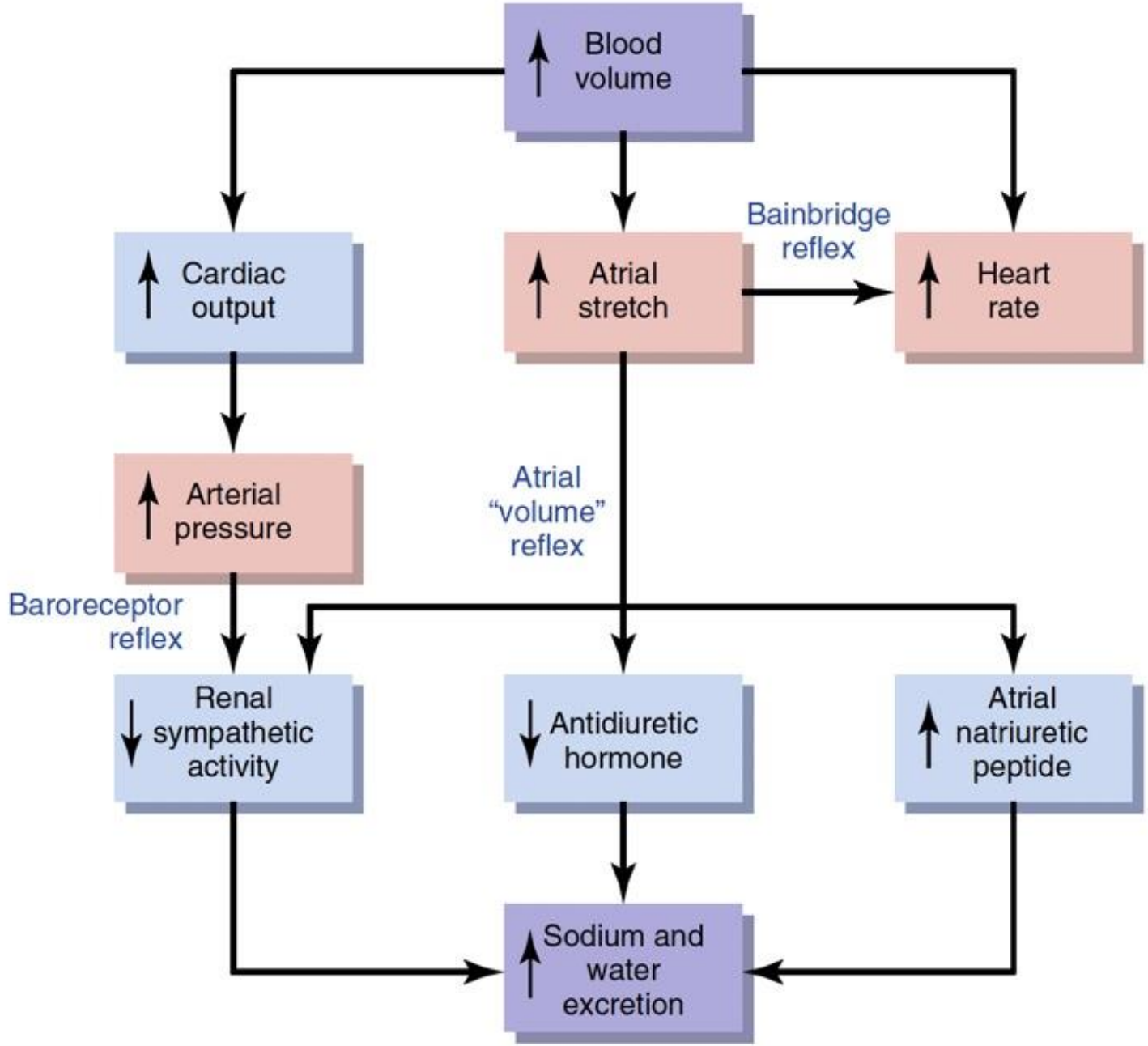
- The atria and pulmonary arteries have stretch receptors in their walls called low-pressure receptors.
- Low-pressure receptors are similar to the baroreceptor stretch receptors of the large systemic arteries.
- These low-pressure receptors play an important role, especially in minimizing arterial pressure changes in response to changes in blood volume.

Atrial reflexes

- Stretch of the atria and activation of low-pressure atrial receptors also causes reflex reductions in renal sympathetic nerve activity, decreased tubular reabsorption, and dilation of afferent arterioles in the kidneys
- Signals are also transmitted simultaneously from the atria to the hypothalamus to decrease secretion of antidiuretic hormone (ADH).
- The combination of these effects—an increase in glomerular filtration and a decrease in reabsorption of the fluid—increases fluid loss by the kidneys and attenuates the increased blood volume.
- Atrial stretch caused by increased blood volume also elicits release of atrial natriuretic peptide (ANP), a hormone that adds further to the excretion of sodium and water in the urine and return of blood volume toward normal.

Bainbridge reflex

- Increases in atrial pressure sometimes increase the heart rate as much as 75%, particularly when the prevailing heart rate is slow.
- When the heart rate is rapid, atrial stretch cause by infusion of fluids may reduce the heart rate due to activation of arterial baroreceptors. Thus, the net effect of increased blood volume and atrial stretch on heart rate depends on the relative contributions of the baroreceptor reflexes (which tends to slow the heart rate) and the Bainbridge reflex which tends to accelerate the heart rate,


Blood volume
Cardiac output
Atrial stretch
Bainbridge reflex
Heart rate
Arterial pressure
Atrial "volume" reflex
Baroreceptor reflex
Renal sympathetic activity
Antidiuretic hormone
Atrial natriuretic peptide
Sodium and water excretion

CNS ischemic response

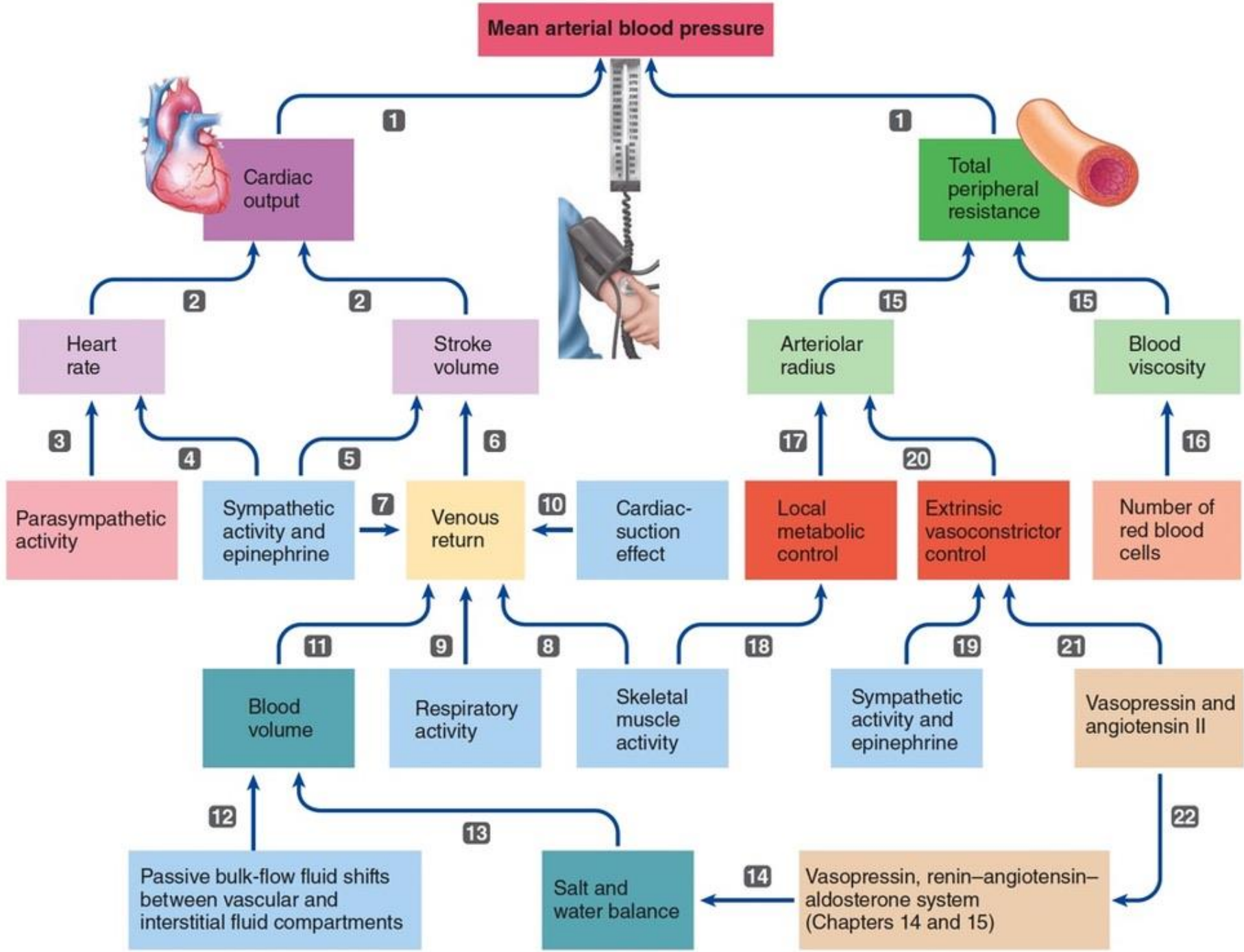
- arterial pressure elevation in response to cerebral ischemia is known as the CNS ischemic response.
- The ischemic effect on vasomotor activity can elevate the mean arterial pressure dramatically, sometimes to as high as 250 mm Hg for as long as 10 minutes.
- The degree of sympathetic vasoconstriction caused by intense cerebral ischemia is often so great that some of the peripheral vessels become totally or almost totally occluded.
- The kidneys, for example, often cease their production of urine entirely because of renal arteriolar constriction in response to the sympathetic discharge. Therefore, the CNS ischemic response is one of the most powerful of all the activators of the sympathetic vasoconstrictor system.

CNS ischemic response

- Despite the powerful nature of the CNS ischemic response, it does not become significant until the arterial pressure falls far below normal, down to 60 mm Hg and below, reaching its greatest degree of stimulation at a pressure of 15 to 20 mm Hg.
- Therefore, the CNS ischemic response is not one of the normal mechanisms for regulating arterial pressure.
- Instead, it operates principally as an emergency pressure control system that acts rapidly and powerfully to prevent further decrease in arterial pressure whenever blood flow to the brain decreases dangerously close to the lethal level.
- It is sometimes called the last-ditch stand pressure control mechanism.

Cushing reaction

- The Cushing reaction illustrates the role of the cerebral chemoreceptors in maintaining cerebral blood flow. When intracranial pressure increases (e.g., tumors, head injury), there is compression of cerebral arteries, which results in decreased perfusion of the brain. There is an immediate increase in PCO2 and a decrease in pH because CO2 generated from brain tissue is not adequately removed by blood flow. The medullary chemoreceptors respond to these changes in PCO2 and pH by directing an increase in sympathetic outflow to the blood vessels. Again, the overall effect of these changes is to increase TPR and dramatically increase P.


Mean arterial blood pressure
1
1
Cardiac output
Total peripheral resistance
2
2
15
15
Heart rate
Stroke volume
Arteriolar radius
Blood viscosity
3
4
5
6
17
20
16
Parasympathetic activity
Sympathetic activity and epinephrine
7
Venous return
10
Cardiac-suction effect
Local metabolic control
Extrinsic vasoconstrictor control
Number of red blood cells
11
9
8
18
19
21
Blood volume
Respiratory activity
Skeletal muscle activity
Sympathetic activity and epinephrine
Vasopressin and angiotensin II
12
13
22
Passive bulk-flow fluid shifts between vascular and interstitial fluid compartments
Salt and water balance
14
Vasopressin, renin–angiotensin–aldosterone system (Chapters 14 and 15)

TPR effect on MAP

- Thus, the extent of TPR offered collectively by all the systemic arterioles influences MAP immensely.
- generalized, sympathetically induced vasoconstriction reflexly reduces blood flow downstream to the organs while elevating the upstream mean arterial pressure, thereby increasing the main driving force for blood flow to all the organs.

TPR effect on MAP

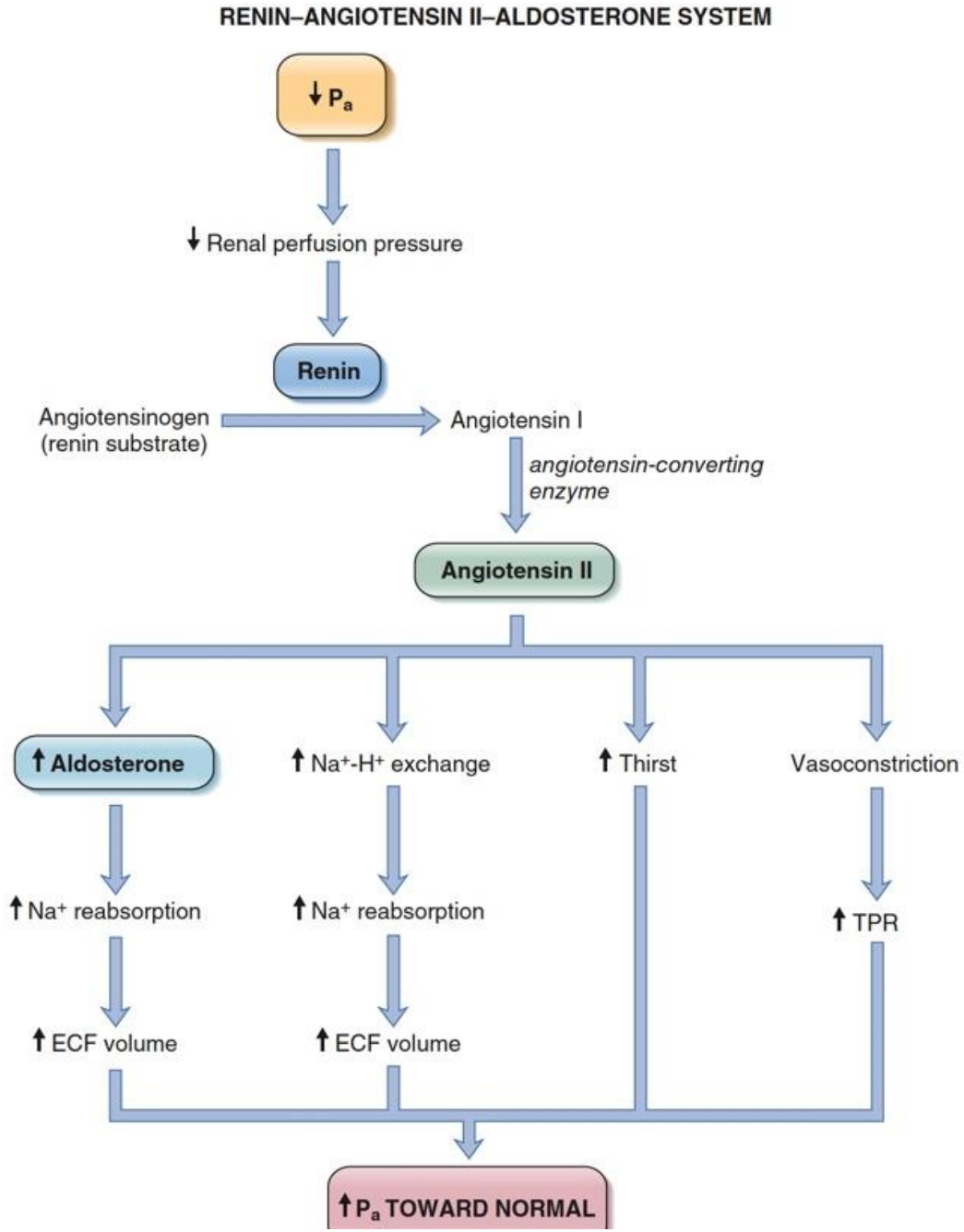
- Why increase the driving force for flow to the organs by increasing MAP while reducing flow to the organs by narrowing the vessels supplying them?
- In effect, the sympathetically induced arteriolar responses help maintain the appropriate driving pressure head (that is, the MAP) to all organs. The extent to which each organ actually receives blood flow is determined by local arteriolar adjustments that override the sympathetic constrictor effect. If all arterioles were dilated, blood pressure would fall substantially, so there would not be an adequate driving force for blood flow.
- Skeletal and cardiac muscles have the most powerful local control mechanisms with which to override generalized sympathetic vasoconstriction.

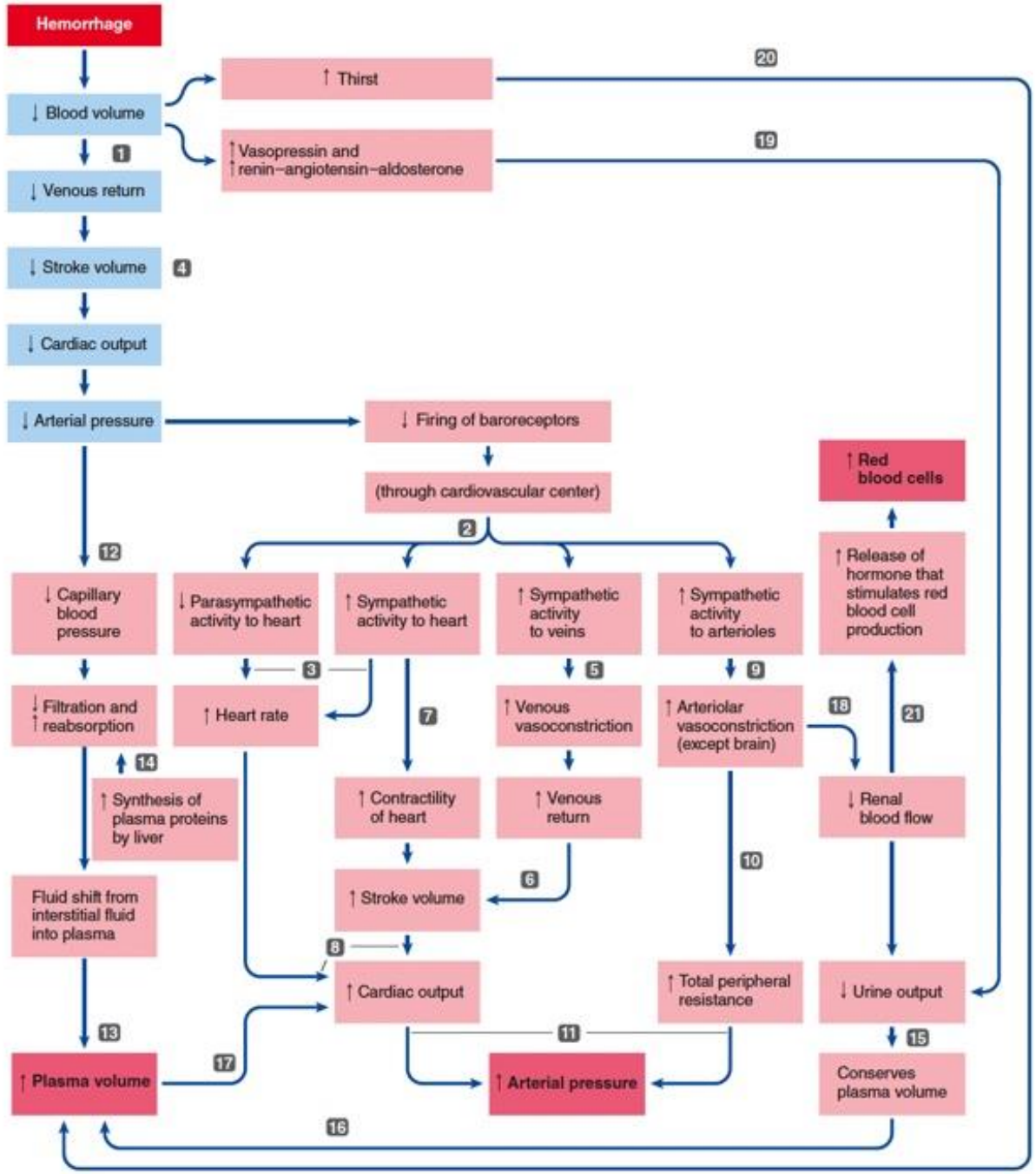
TPR effect on MAP

- The norepinephrine released from sympathetic nerve endings combines with a1-adrenergic receptors on arteriolar smooth muscle to bring about vasoconstriction.
- Cerebral arterioles are the only ones that do not have a1 receptors, so no vasoconstriction occurs in the brain. It is important that cerebral arterioles are not reflexly constricted by neural influences because brain blood flow must remain constant to meet the brain's continuous need for O2, no matter what is going on elsewhere in the body. Cerebral vessels are almost entirely controlled by local mechanisms that maintain a constant blood flow to support a constant level of brain metabolic activity. In fact, reflex vasoconstrictor activity in the remainder of the cardiovascular system is aimed at maintaining an adequate pressure head for blood flow to the vital brain.

Arteriolar Myogenic response to stretch

- Arteriolar smooth muscle responds to being passively stretched by myogenically increasing its tone via vasoconstriction, thereby acting to resist the initial passive stretch.
- Increased vessel stretching brings about opening of mechanically gated cation channels, which leads to a small depolarization that triggers opening of more surface-membrane voltage-gated Ca channels. The resultant Ca entry promotes increased smooth muscle contraction, boosting myogenic vessel tone and causing vasoconstriction. Conversely, a reduction in arteriolar stretching decreases myogenic vessel tone and promotes vasodilation.


RENIN–ANGIOTENSIN II–ALDOSTERONE SYSTEM
↓ P_a
↓ Renal perfusion pressure
Renin
Angiotensinogen (renin substrate)
Angiotensin I
angiotensin-converting enzyme
Angiotensin II
↑ Aldosterone
↑ Na^+-H^+ exchange
↑ Thirst
Vasoconstriction
↑ Na^+ reabsorption
↑ Na^+ reabsorption
↑ TPR
↑ ECF volume
↑ ECF volume
↑ P_a TOWARD NORMAL


Hemorrhage
↓ Blood volume
1
↓ Venous return
↓ Stroke volume
4
↓ Cardiac output
↓ Arterial pressure
↑ Thirst
20
↑ Vasopressin and
↑ renin–angiotensin–aldosterone
19
↓ Firing of baroreceptors
(through cardiovascular center)
2
12
↓ Capillary blood pressure
↓ Filtration and ↑ reabsorption
14
↑ Synthesis of plasma proteins by liver
Fluid shift from interstitial fluid into plasma
13
↑ Plasma volume
17
↓ Parasympathetic activity to heart
3
↑ Heart rate
↑ Sympathetic activity to heart
7
↑ Contractility of heart
↑ Stroke volume
8
↑ Cardiac output
↑ Sympathetic activity to veins
5
↑ Venous vasoconstriction
↑ Venous return
6
↑ Sympathetic activity to arterioles
9
↑ Arteriolar vasoconstriction (except brain)
10
↑ Total peripheral resistance
11
↑ Arterial pressure
18
↑ Red blood cells
↑ Release of hormone that stimulates red blood cell production
21
↓ Renal blood flow
↓ Urine output
15
Conserves plasma volume
16

Thank you