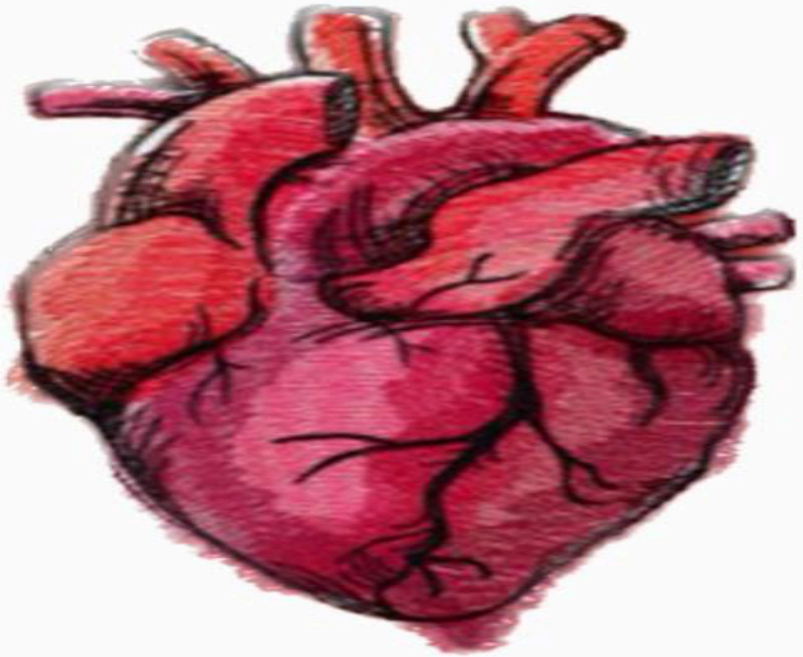
CARDIOVASCULAR SYSTEM



SUBJECT : فيسو

LEC NO. : *Lec 13*

DONE BY : *Basil & mass*

وقل رب زدني علماً

There is two types of responses mechanisms for blood pressure regulation

1) fast short
2) slow long

Fast short

1) Baroreceptor (immediately)
2) Chemoreceptors (seconds)
3) CNS

Slow long

1) Capillary fluid shift
2) Renin angiotensin aldosterone system
3) Renal blood volume pressure control
4) stress relaxation reflux

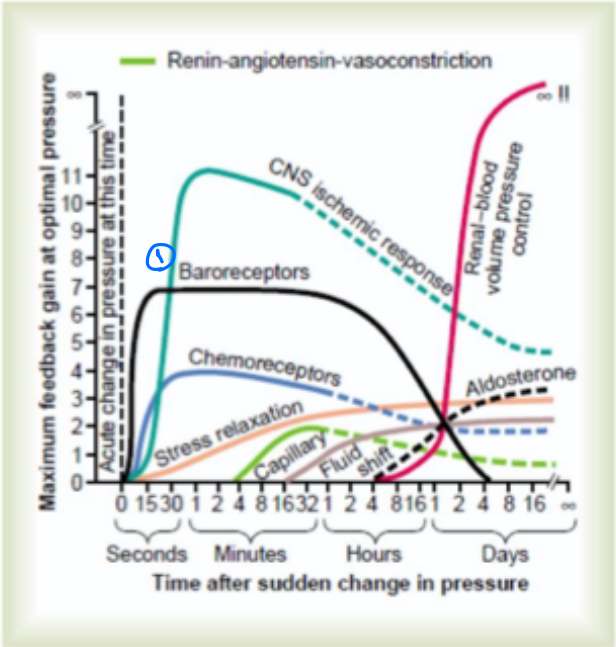


Figure 19–15

Approximate potency of various arterial pressure control mechanisms at different time intervals after onset of a disturbance to the arterial pressure. Note especially the infinite gain (∞) of the renal body fluid pressure control mechanism that occurs after a few weeks' time. (Redrawn from Guyton AC: Arterial Pressure and Hypertension. Philadelphia: WB Saunders Co, 1980.)

why there is Slow Long mechanism?
because: resting could occurs in the baroreceptor.

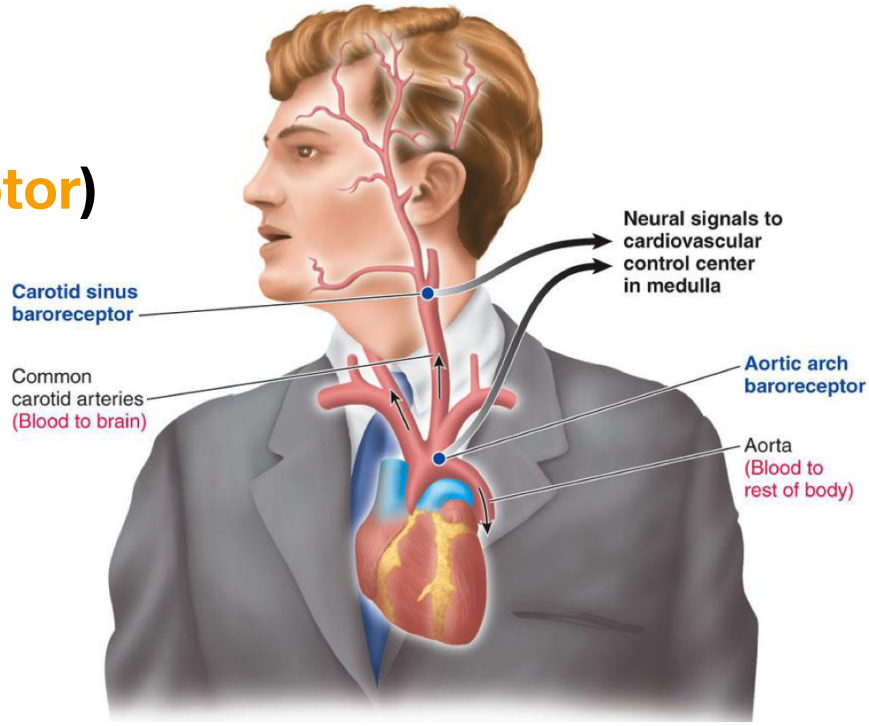
The resting mean, if the mean blood pressure increases and the fast mechanism couldn't regulate it back to normal, like (essential hypertension fast mechanism couldn't regulate it successfully), this lead Baroreceptor rest and adapt to this new high pressure and regulate pressure on the new set point.

The pressure is monitored by:-

Baroreceptor(stretch receptor)

1) Aortic receptor

2) Carotid receptor

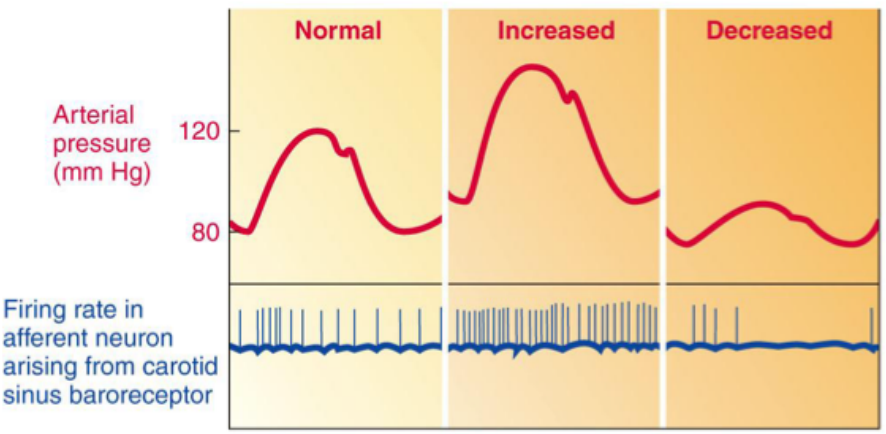


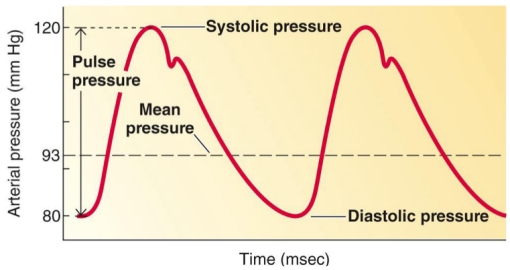
How:- they measure the stretch on the blood vessels

The stretch is proportional to the mean blood pressure

Mean blood pressure = diastolic + ⅓ pules pressure

pules pressure → systole - diastole





increase in blood pressure lead to increase spike frequency, this how receptor detect the elevation in blood pressure and transfer this spike signal to brain.

vasomotor will work efficiently to regulate the pressure. By inhibiting or stimulating the sympathetic system

Baroreceptor
(stretch receptor)

In high blood pressure

↓

Inhibition sympathetic

- ↓ Heart rate
- ↓ Ventricular contractility
- Vasodilation of arterial vessels

This will need decrease pressure to its normal values

vice versa to increase blood pressure

In low blood pressure

↓

Stimulation sympathetic

- ↑ Heart rate
- ↑ Ventricular contractility
- Vasoconstriction of arterial vessels
- Vasoconstriction of venous vessels

↓

this will increase venous return

↓

Increase blood pressure

Baroreceptor didn't see systole or diastole the see the mean blood pressure which is lowest in venous and veins reaching right atrium near **ZERO**

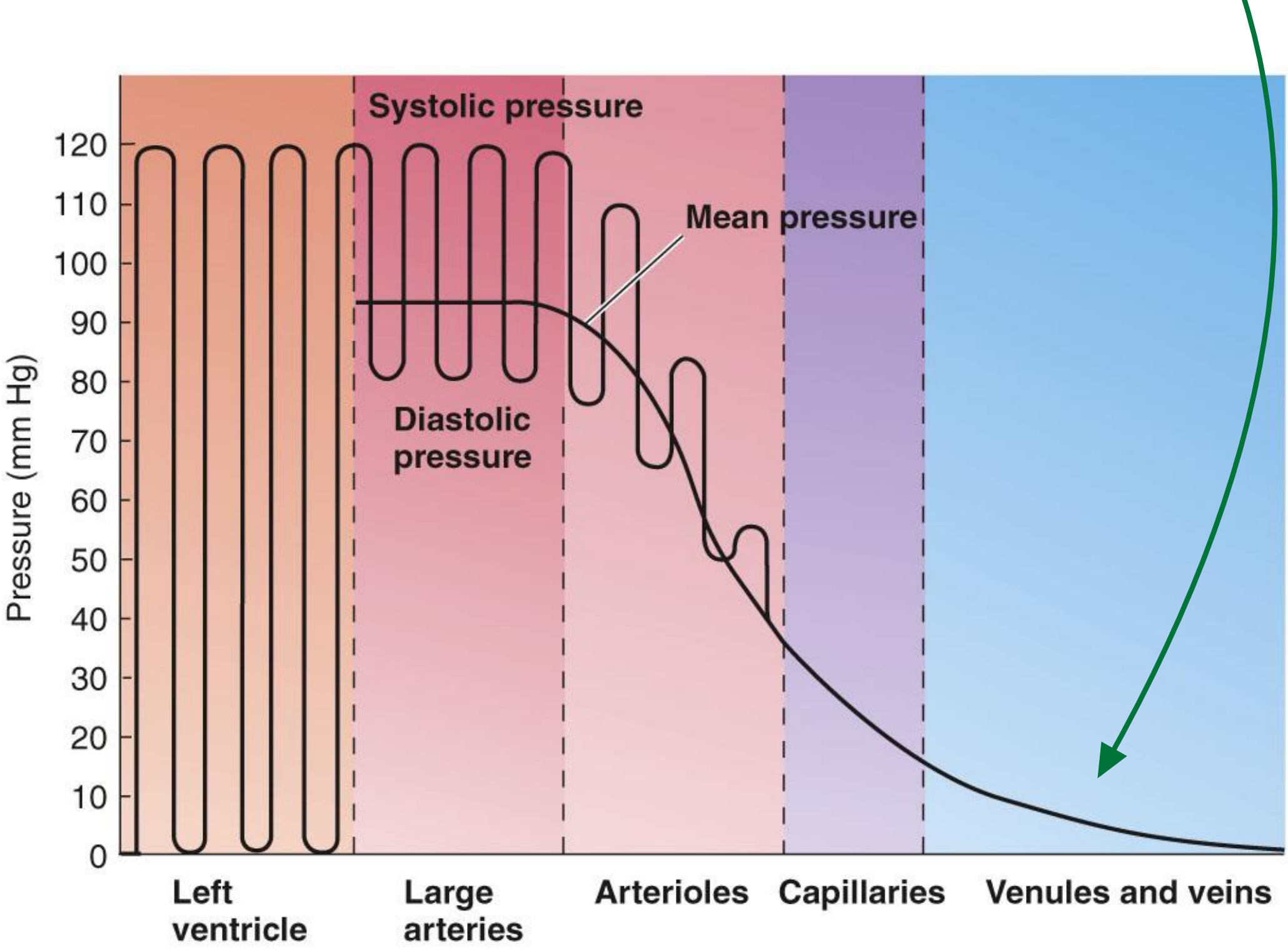

Systolic pressure
Mean pressure
Diastolic pressure
Pressure (mm Hg)
120
110
100
90
80
70
60
50
40
30
20
10
0
Left ventricle
Large arteries
Arterioles
Capillaries
Venules and veins

Fig. 10-9, p. 352

Chemoreceptors

Chemoreceptors: is effected by CO_2 in blood, if CO_2 elevated lead to form HCO_3, then make acidic environment near the chemoreceptor this will increase the activity of the receptor center then increase deep and rate lead to reduce CO_2

This mechanism need to high blood flow so it spontaneously stimulate Cardiovascular system

Cardiovascular and Respiratory collaborate to succeed the process.

The partial chemoreceptor is sensitive to dissolved O_2, and the Chemoreceptor will not be activated until the O_2 saturation is less than 60%, so if person inhaled CO the chemoreceptor will not detect it.

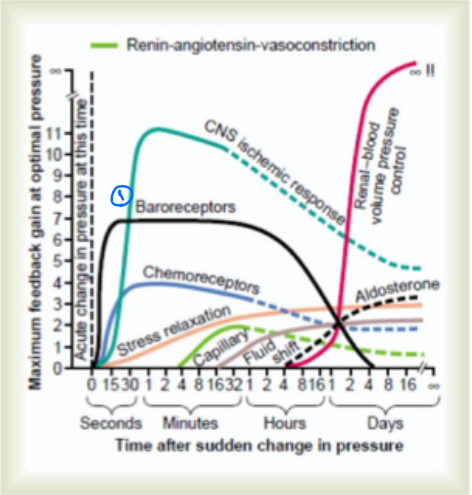
CNS

If the blood pressure reach the brain is decreased and the blood flow decreased the CNS will work immediately to regulate, if the CNS insufficient, the situation will be worse by time til death; the CNS will be unable to respond by time because of hypoxia and ishcemic.

CNS
1) Fast
2) strongest
But if its fails it death occurs

**** Short term mechanism work by sympathetic and parasympathetic thats way they are fast**

Short mechanism



Generally work on blood volume

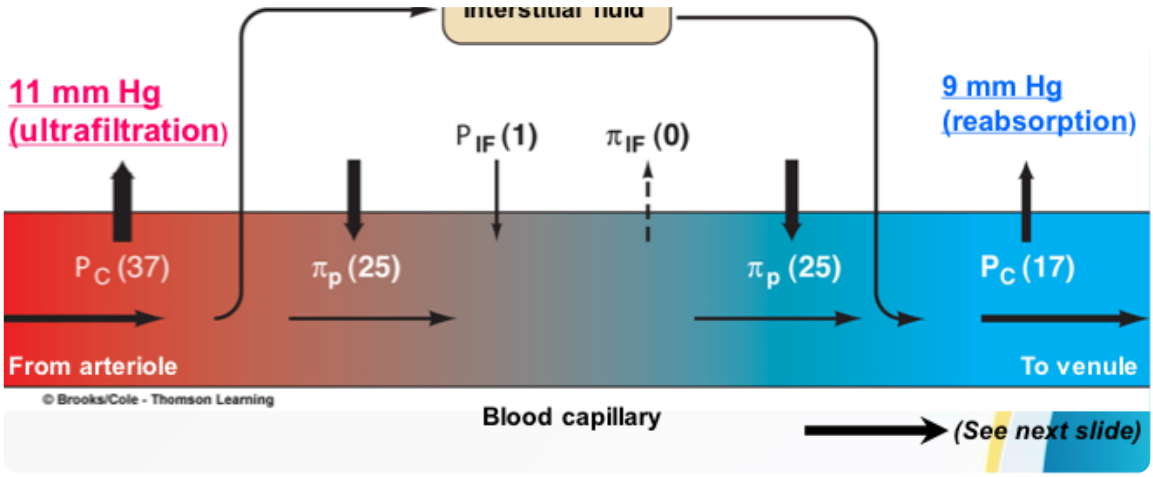
except , works tension on
wall of arterioles (حكينا عنه فوق)

The mechanisms that work on blood volume are:

1) Capillary fluid shift
2) Renin angiotensin aldosterone system
3) Renal blood volume pressure control

How blood volume effects blood pressure?
The blood circulation is closed loop if the fluid increases more then the optimal volume (5 letter), **this will increase the venous return which lead to increase cardiac output** that directly effects the blood pressure.

Capillary fluid shift



The mechanism of Capillary fluid shift

If the blood pressure **increases** the hydrostatic pressure increases, and the filtration mechanism will exceed the reabsorption which lead to accumulation fluid in the interstitial space.

If the blood pressure **decreases** the hydrostatic pressure decreases , and the reabsorption mechanism will exceed the filtration which lead to increase fluid in the capillary increase blood volume.

Renin angiotensin aldosterone system

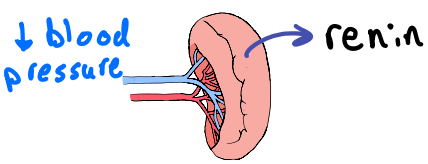
Angiotensin is a systemic vasoconstrictor
Normally angiotensin 2 found in our body in regulated amount to maintain the blood pressure

On the kidney the are sensor for **blood flow and Na amount called** (glomlarapprats)

glomlarapprats monitored the flow and Na in urine

*And the blood flow related to blood pressure as we mentioned.

If the pressure decreases the glomalary system will strat working, synthesis renin, which converts angiotensinogen to angiotensin 1, angiotensin 1 converted to angiotensin 2 in lung, finally angiotensin 2 make vasoconstriction, increasing



Renin
Angiotensinogen → **Angiotensin 1** → **Angiotensin 2**
lung

Angiotensin 2 → **Vasoconstriction**
Angiotensin 2 → ↑ **Aldosterone**
Angiotensin 2 → **Directly effect the nephron to increase sodium reabsorption**
This effect strong but its occur

Aldosterone

Aldosterone is a hormone that is responsible for control of potassium in the blood

Two ions in our body must be accurately regulated
1) Ca 1.8 mmol/liter
2) K 4.5~5 mmol/liter

There are strict regularly hormone for Ca.

Aldosterone can increase potassium loss and increase sodium gain, increasing sodium gain will increase water reabsorption and increase blood volume this way blood pressure increases.

Renal blood volume pressure control

This mechanism need few hours to be activated

In low blood pressure
Nephron adapted to increase Na reabsorption and water reabsorption this will increase blood pressure

In high blood pressure
Nephron adapted to decrease Na reabsorption and less water reabsorption this will decrease blood pressure

Its late mechanism but its very strong and fast

ما تبقى من سلايدات هو ملخصات و مراجعات لكل الموضوع
الفكرة انه ضغط الدم اتقسم الى اكثر من جزء و عرفنا شو العوامل
يلي بتاثر فيه خلال المحاضرات الماضية و المحاضرة هاي عرفنا
كيف الجسم بضبط الضغط و شو الالية المحاضرة هاي فش الها
سلايدات الشرح يلي انا كاتبة هو كل معلومه و فكرة حكاها
الدكتور وان شاءالله تكون شاملة

Control of heart rate:

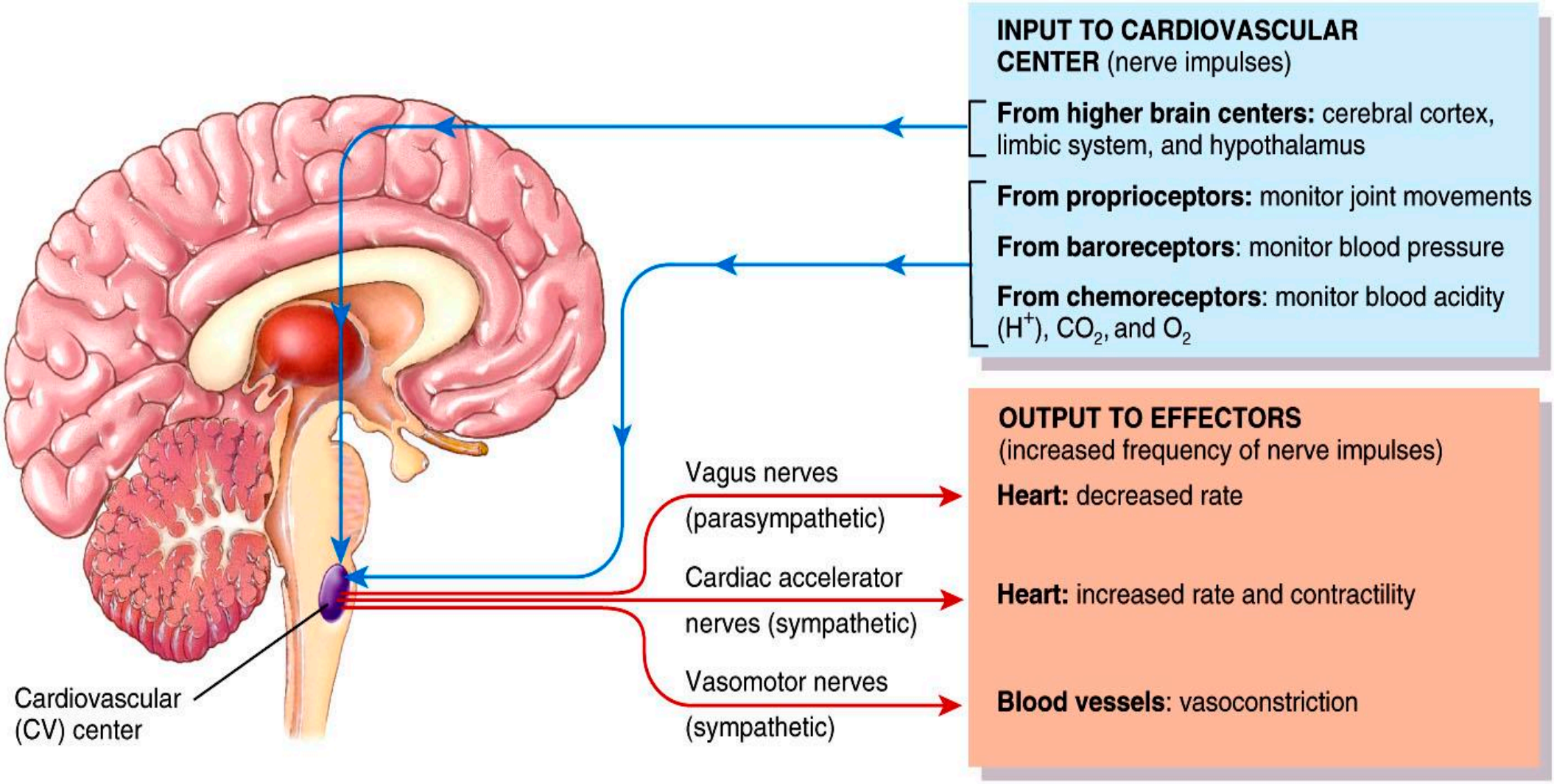


Figure 21.12 Tortora - PAP 12/e

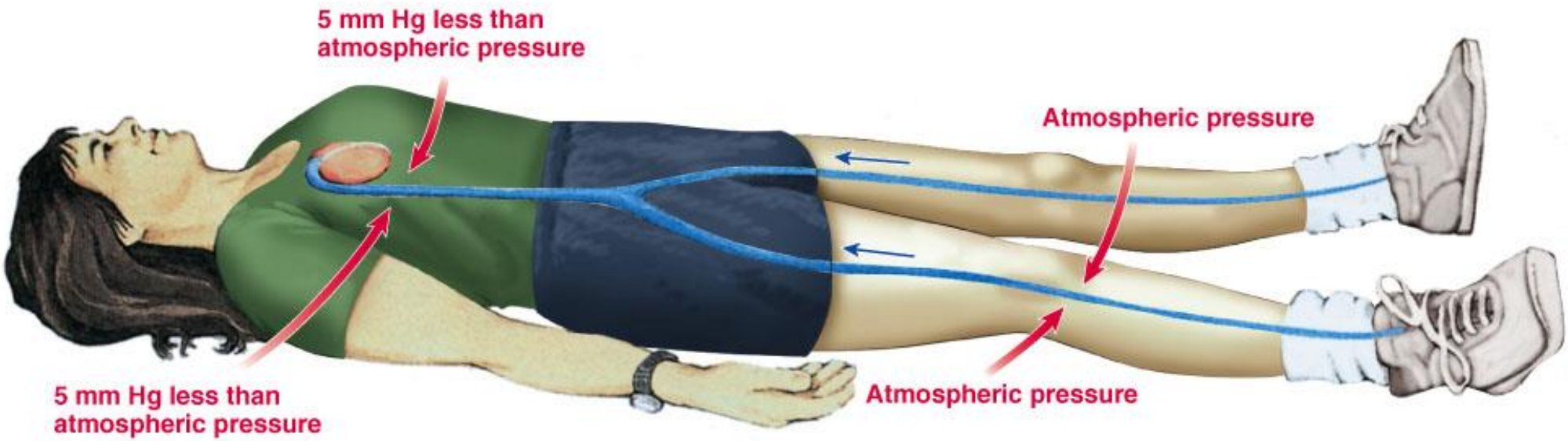


Fig. 10-33, p. 376

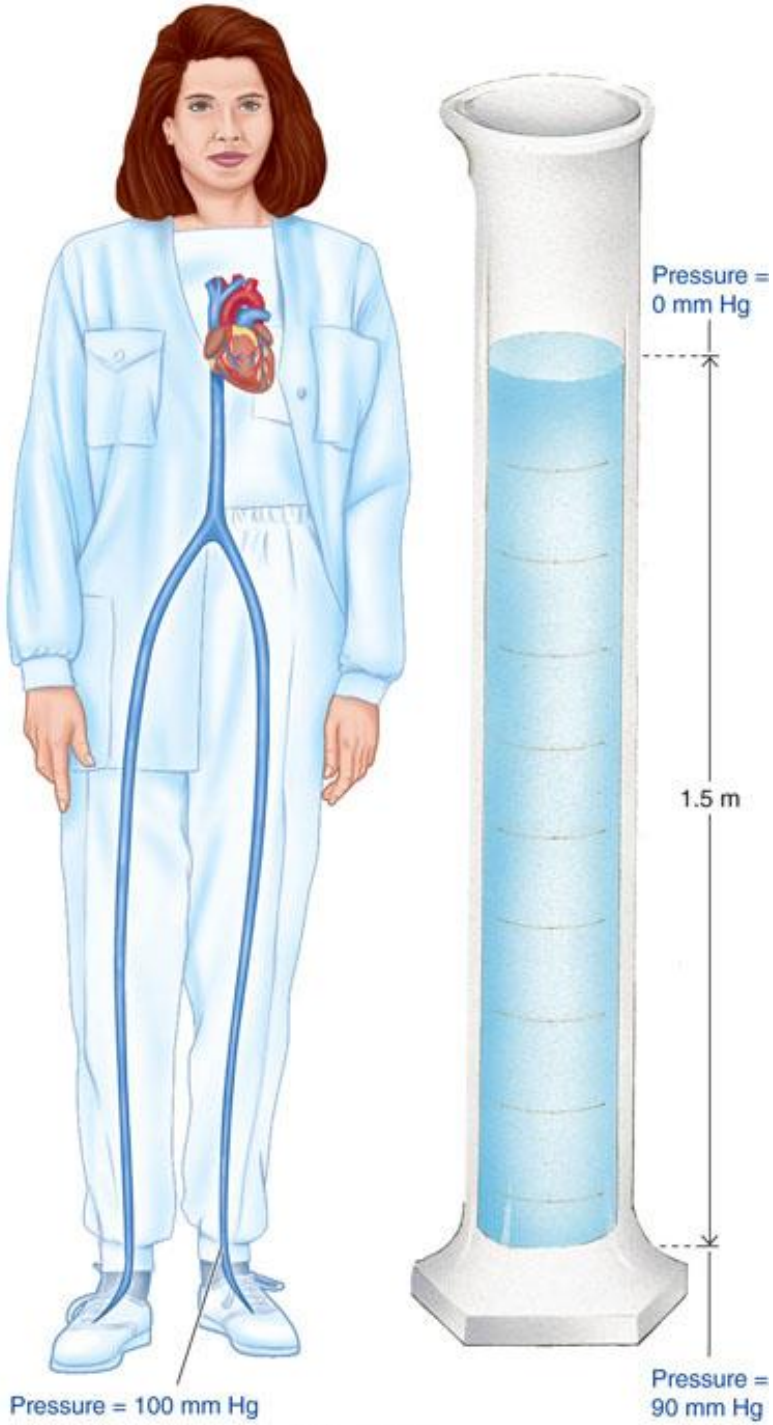
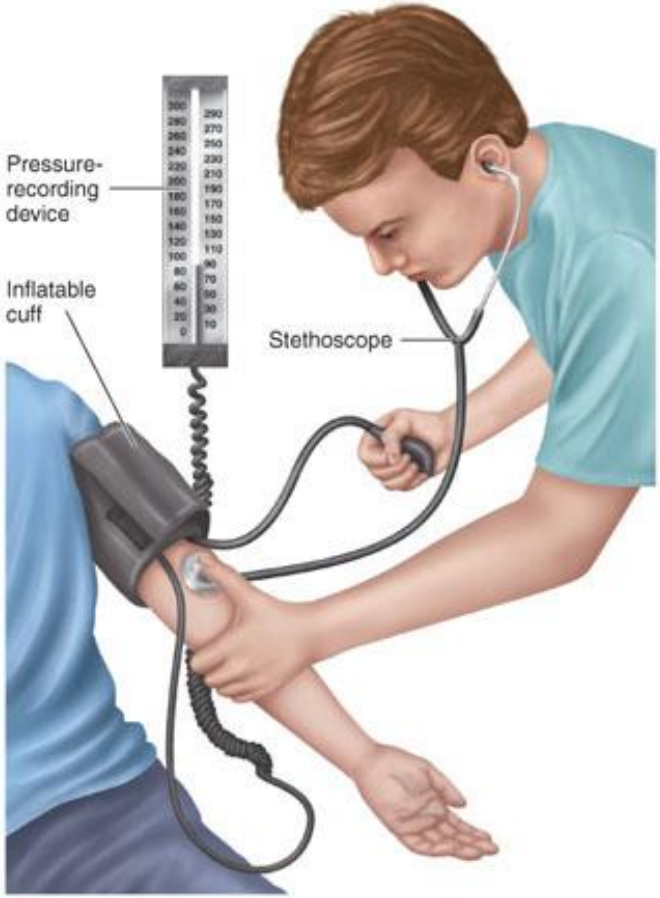
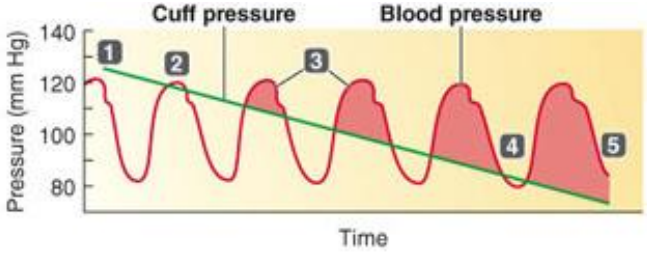


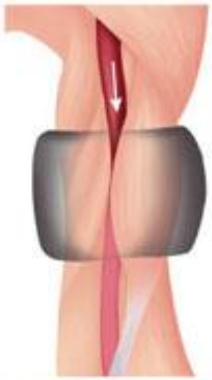
Fig. 10-30, p. 374



(a) Use of a sphygmomanometer in determining blood pressure



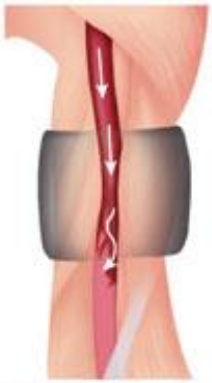
When blood pressure is 120/80:



When cuff pressure is greater than 120 mm Hg and exceeds blood pressure throughout the cardiac cycle:

No blood flows through the vessel.

1 No sound is heard because no blood is flowing.



When cuff pressure is between 120 and 80 mm Hg:

Blood flow through the vessel is turbulent whenever blood pressure exceeds cuff pressure.

2 The first sound is heard at peak systolic pressure.

3 Intermittent sounds are produced by turbulent spurts of flow as blood pressure cyclically exceeds cuff pressure.

When cuff pressure is less than 80 mm Hg and is below blood pressure throughout the cardiac cycle:

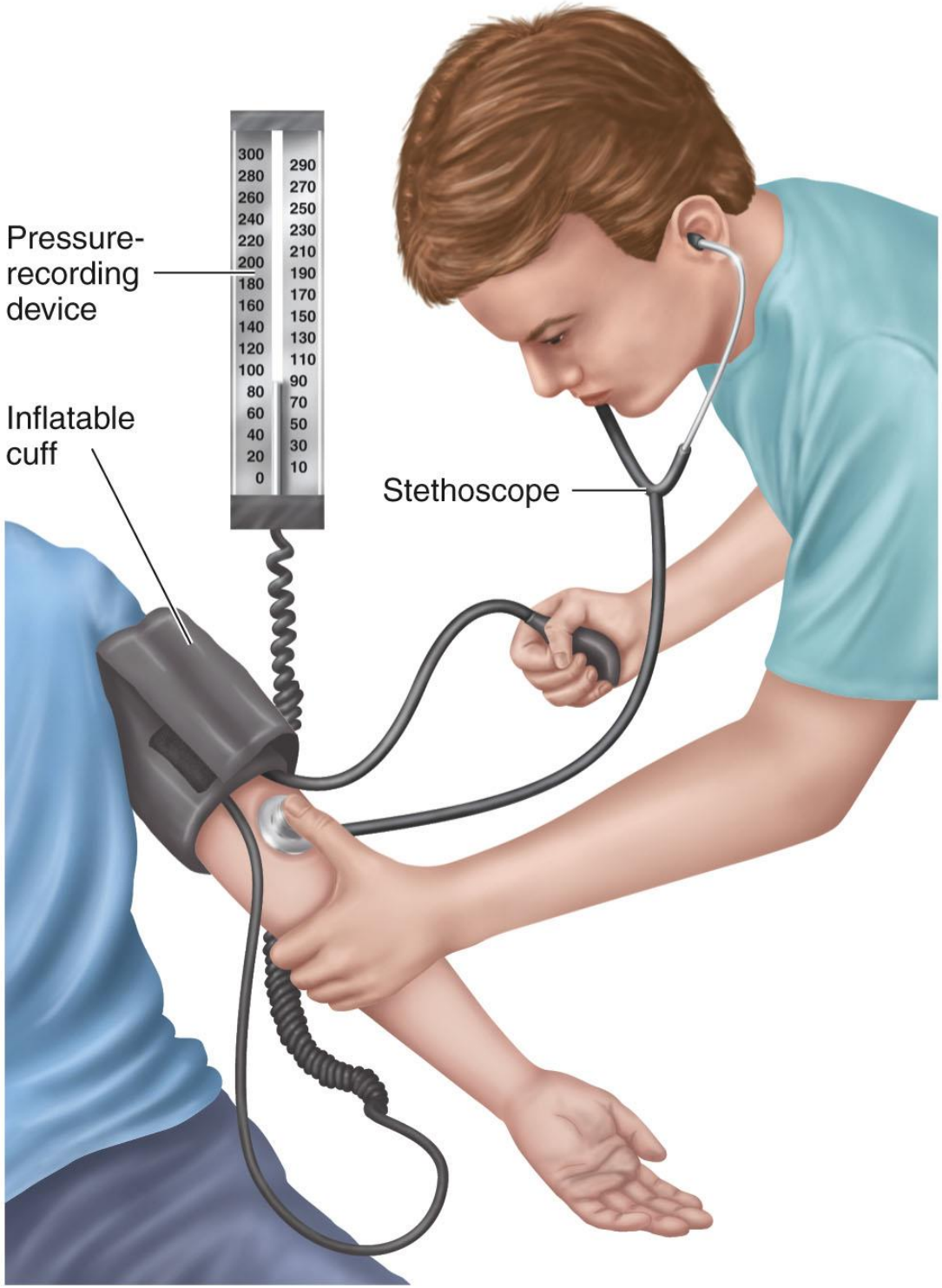
Blood flows through the vessel in smooth, laminar fashion.

4 The last sound is heard at minimum diastolic pressure.

5 No sound is heard thereafter because of uninterrupted, smooth, laminar flow.

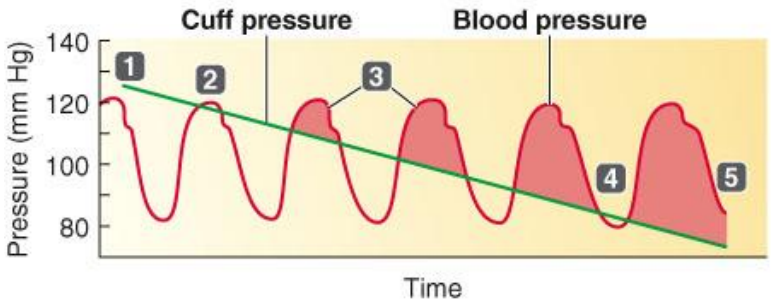
(b) Blood flow through the brachial artery in relation to cuff pressure and sounds

Fig. 10-8, p. 351

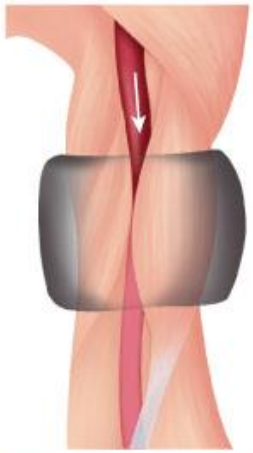


(a) Use of a sphygmomanometer in determining blood pressure

Fig. 10-8a, p. 351



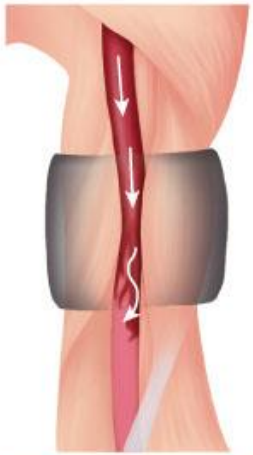
When blood pressure is 120/80:



When cuff pressure is greater than 120 mm Hg and exceeds blood pressure throughout the cardiac cycle:

No blood flows through the vessel.

1 No sound is heard because no blood is flowing.

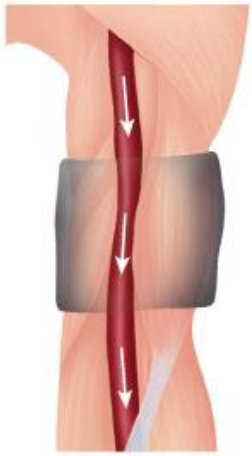


When cuff pressure is between 120 and 80 mm Hg:

Blood flow through the vessel is turbulent whenever blood pressure exceeds cuff pressure.

2 The first sound is heard at peak systolic pressure.

3 Intermittent sounds are produced by turbulent spurts of flow as blood pressure cyclically exceeds cuff pressure.



When cuff pressure is less than 80 mm Hg and is below blood pressure throughout the cardiac cycle:

Blood flows through the vessel in smooth, laminar fashion.

4 The last sound is heard at minimum diastolic pressure.

5 No sound is heard thereafter because of uninterrupted, smooth, laminar flow.

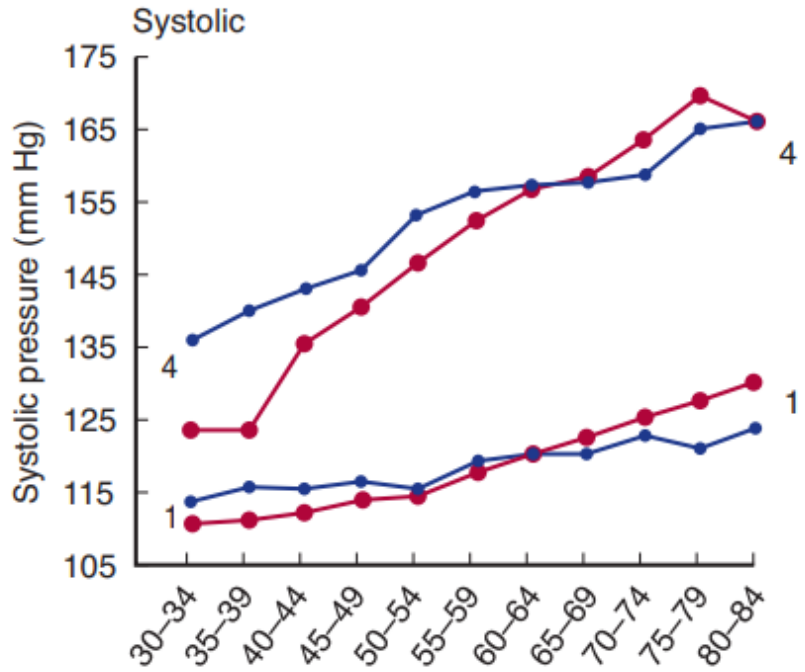
(b) Blood flow through the brachial artery in relation to cuff pressure and sounds

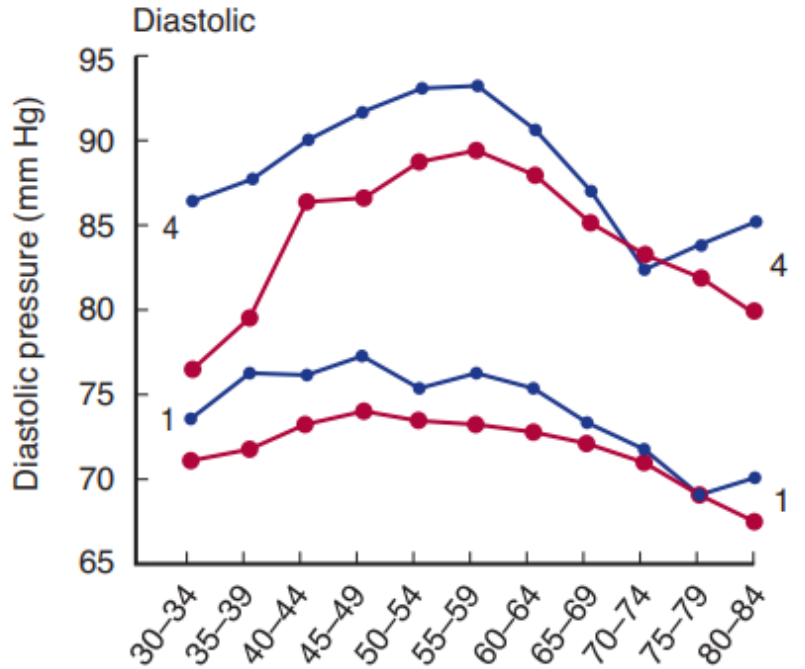
Fig. 10-8b, p. 351

Changes inpressure over age in male & female

blue color in male

Red in female





Regulation of Blood Pressure

Two types of regulatory mechanisms

1- Short term (Baroreceptors, hormonal, capillary fluid shift)

2- Long term (Hormonal, Capillary fluid shift , and Renal (Renin- Angiotensin system

Blood Pressure regulation

BP = CO x TPR

i.e. = ((HR x SV x TPR))

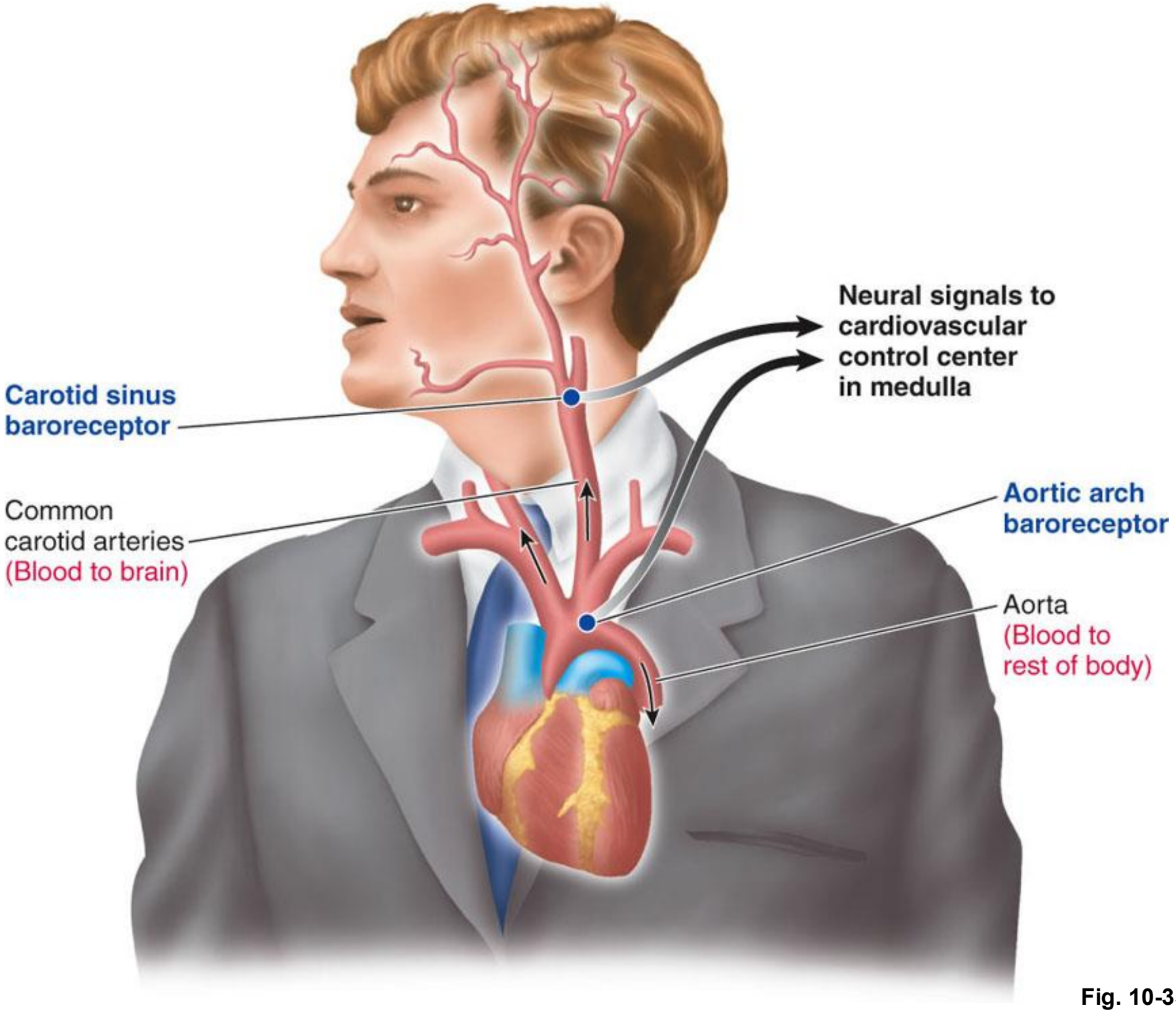


Fig. 10-35, p. 378

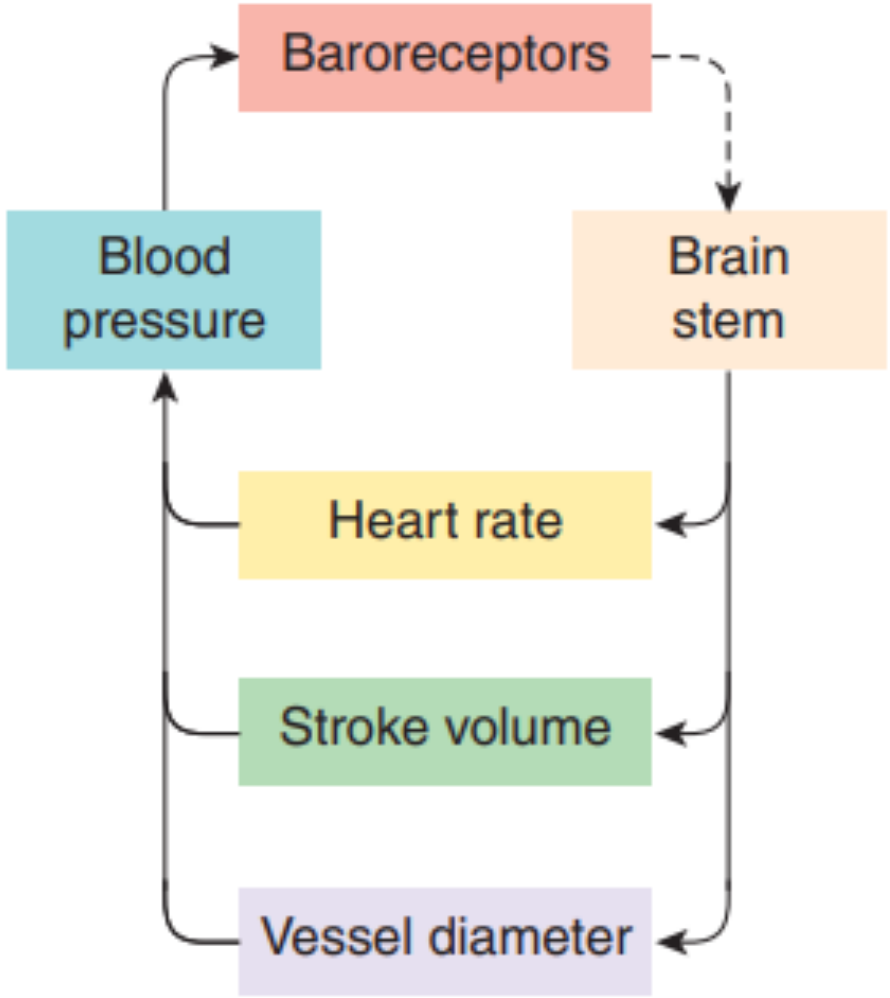
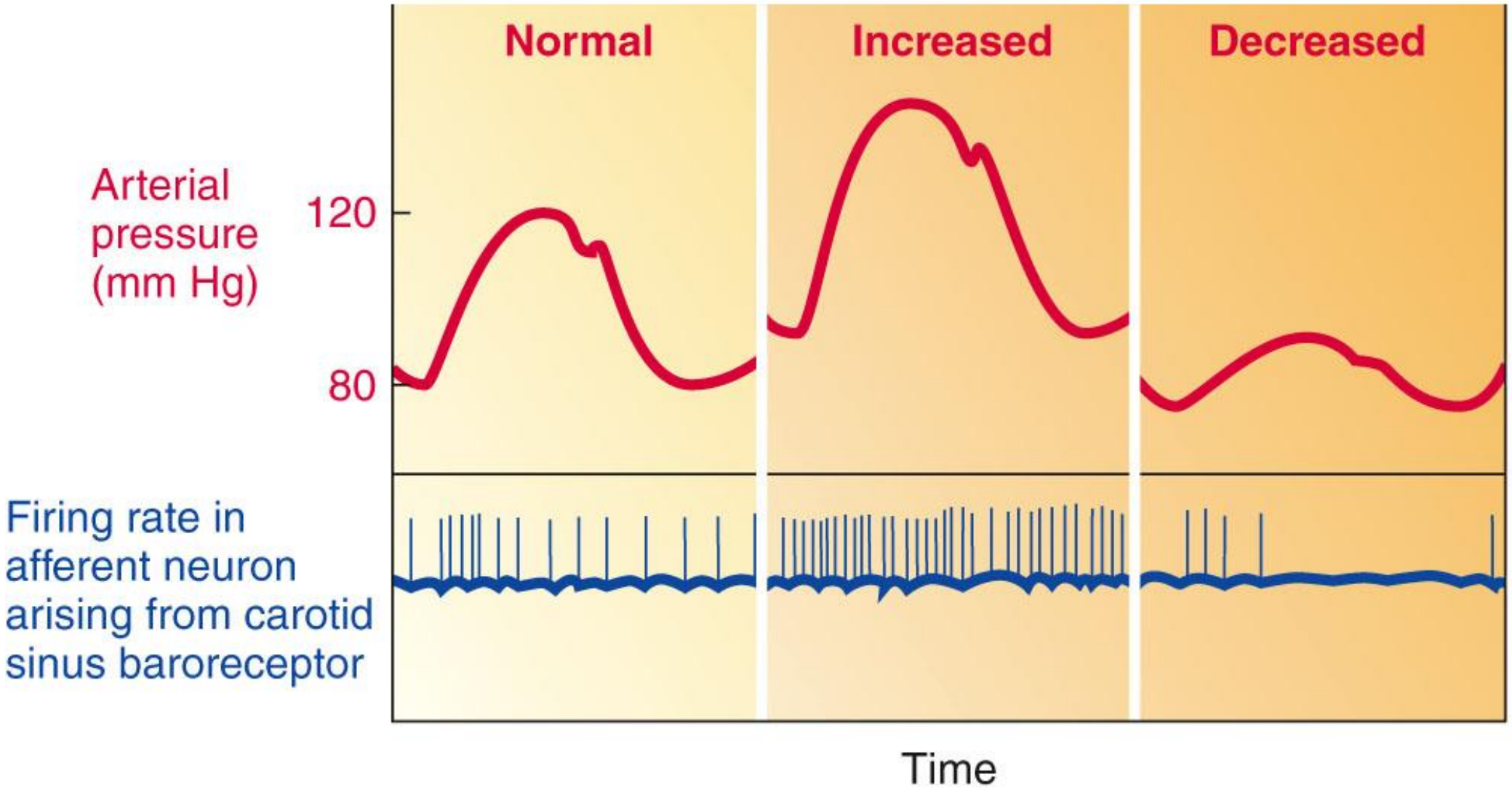


FIGURE 32–1 **Feedback control of blood pressure.** Brain stem excitatory input to sympathetic nerves to the heart and vasculature increases heart rate and stroke volume and reduces vessel diameter. Together these increase blood pressure, which activates the baroreceptor reflex to reduce the activity in the brain stem.

Barorexceptors reflex:



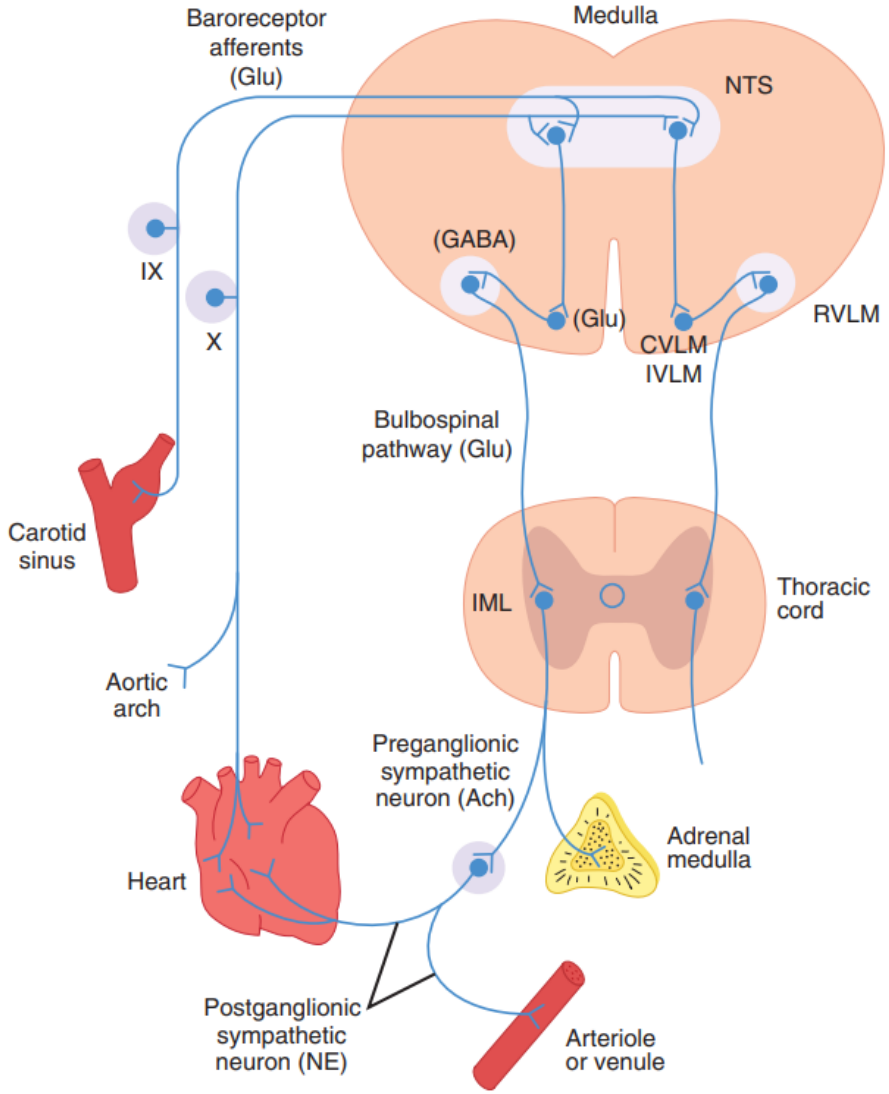


FIGURE 32–2 **Basic pathways involved in the medullary** (NTS) is the site of termination of baroreceptor afferent fibers.

FIGURE 32–2 **Basic pathways involved in the medullary control of blood pressure.** The rostral ventrolateral medulla (RVLM) is one of the major sources of excitatory input to sympathetic nerves controlling the vasculature. These neurons receive inhibitory input from the baroreceptors via an inhibitory neuron in the caudal ventrolateral medulla (CVLM). The nucleus of the tractus solitarius (NTS) is the site of termination of baroreceptor afferent fibers. The putative neurotransmitters in the pathways are indicated in parentheses. Ach, acetylcholine; GABA, γ-aminobutyric acid; Glu, glutamate; IML, intermediolateral gray column; IVLM, intermediate ventrolateral medulla; NE, norepinephrine; NTS, nucleus of the tractus solitarius; IX and X, glossopharyngeal and vagus nerves.

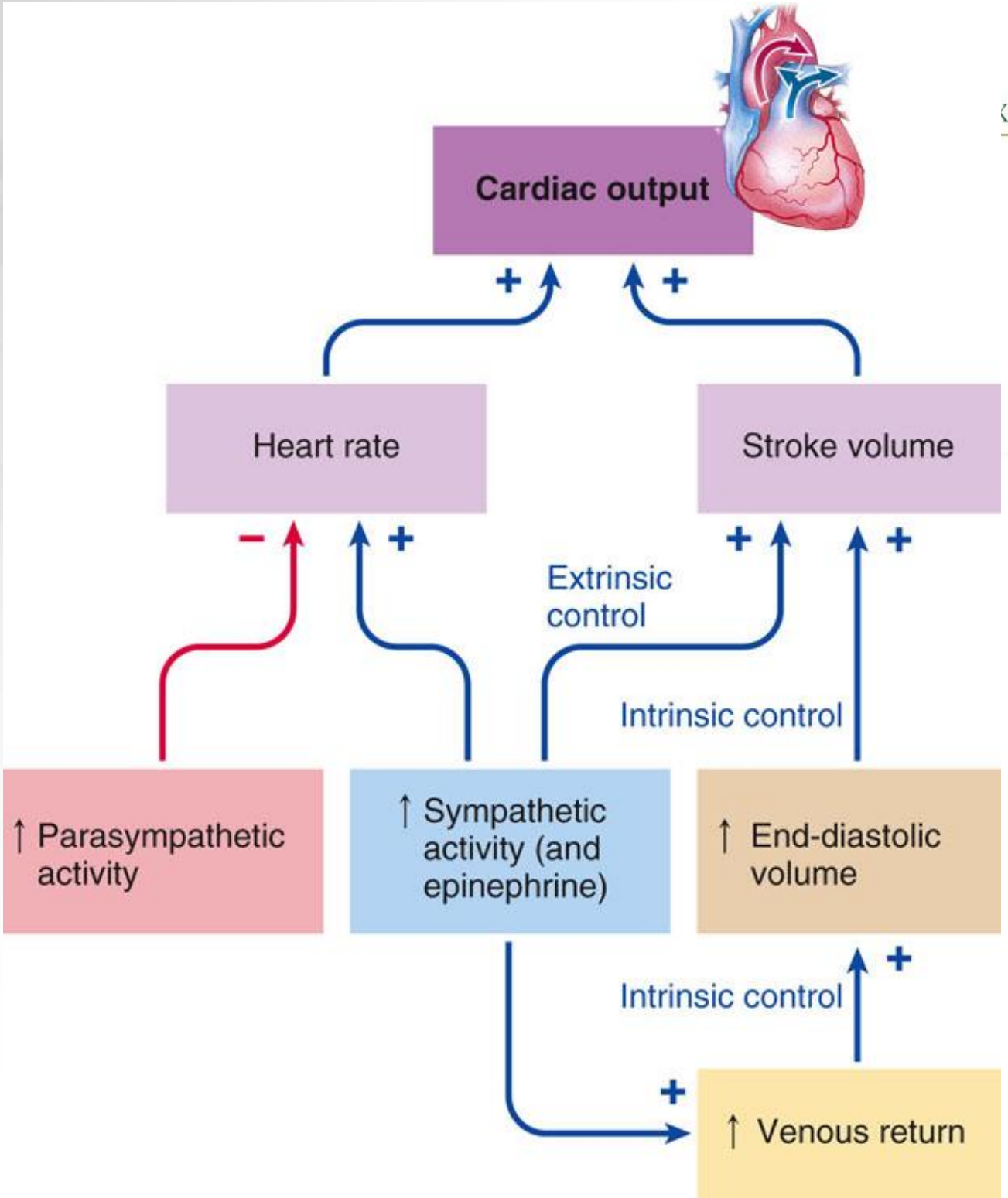

Cardiac output
+
+
Heart rate
Stroke volume
−
+
+
+
Extrinsic control
Intrinsic control
↑ Parasympathetic activity
↑ Sympathetic activity (and epinephrine)
↑ End-diastolic volume
Intrinsic control
+
+
↑ Venous return

Fig. 9-24, p. 330

Contractility Curves (Frank – Starling law

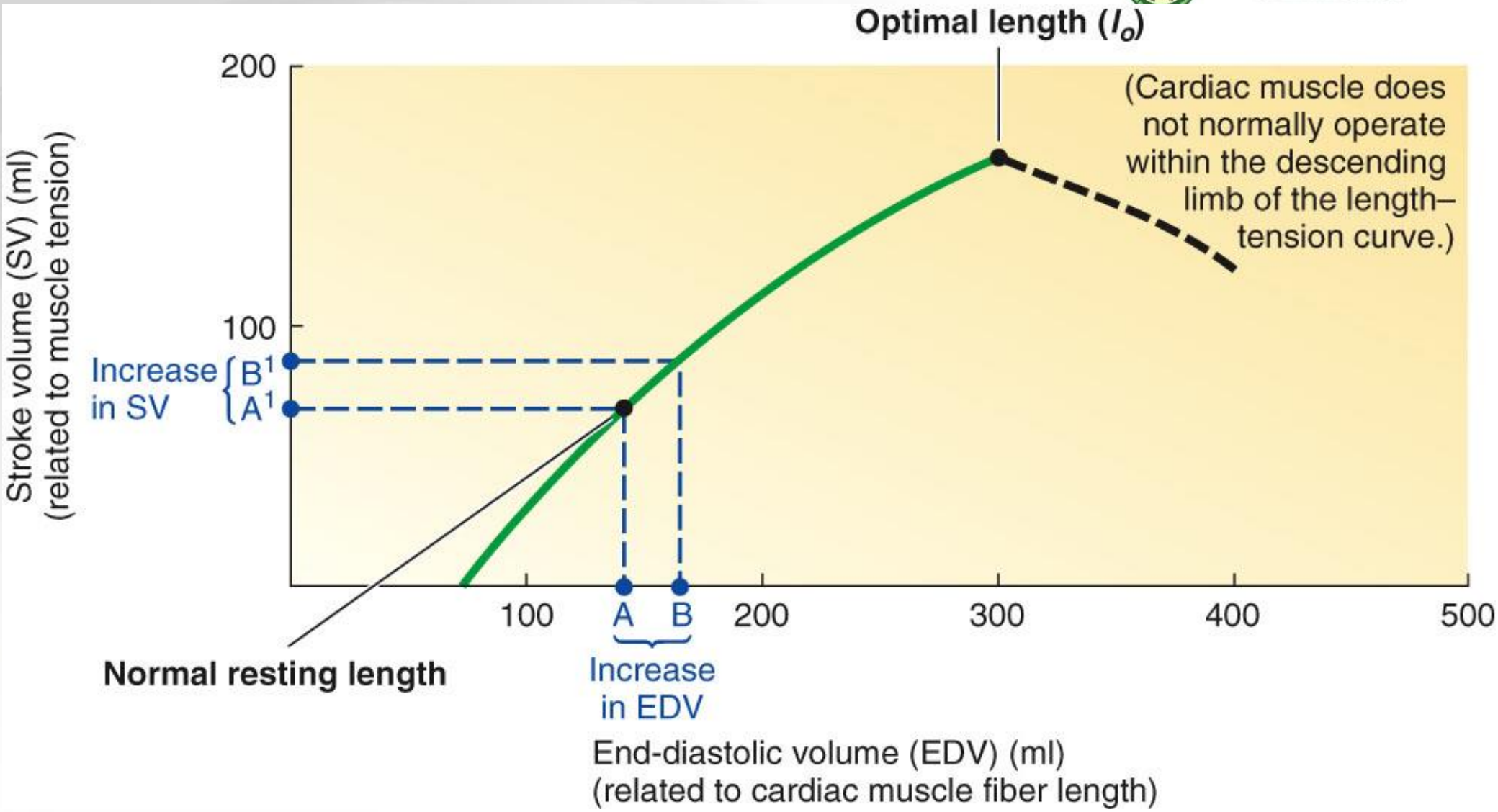
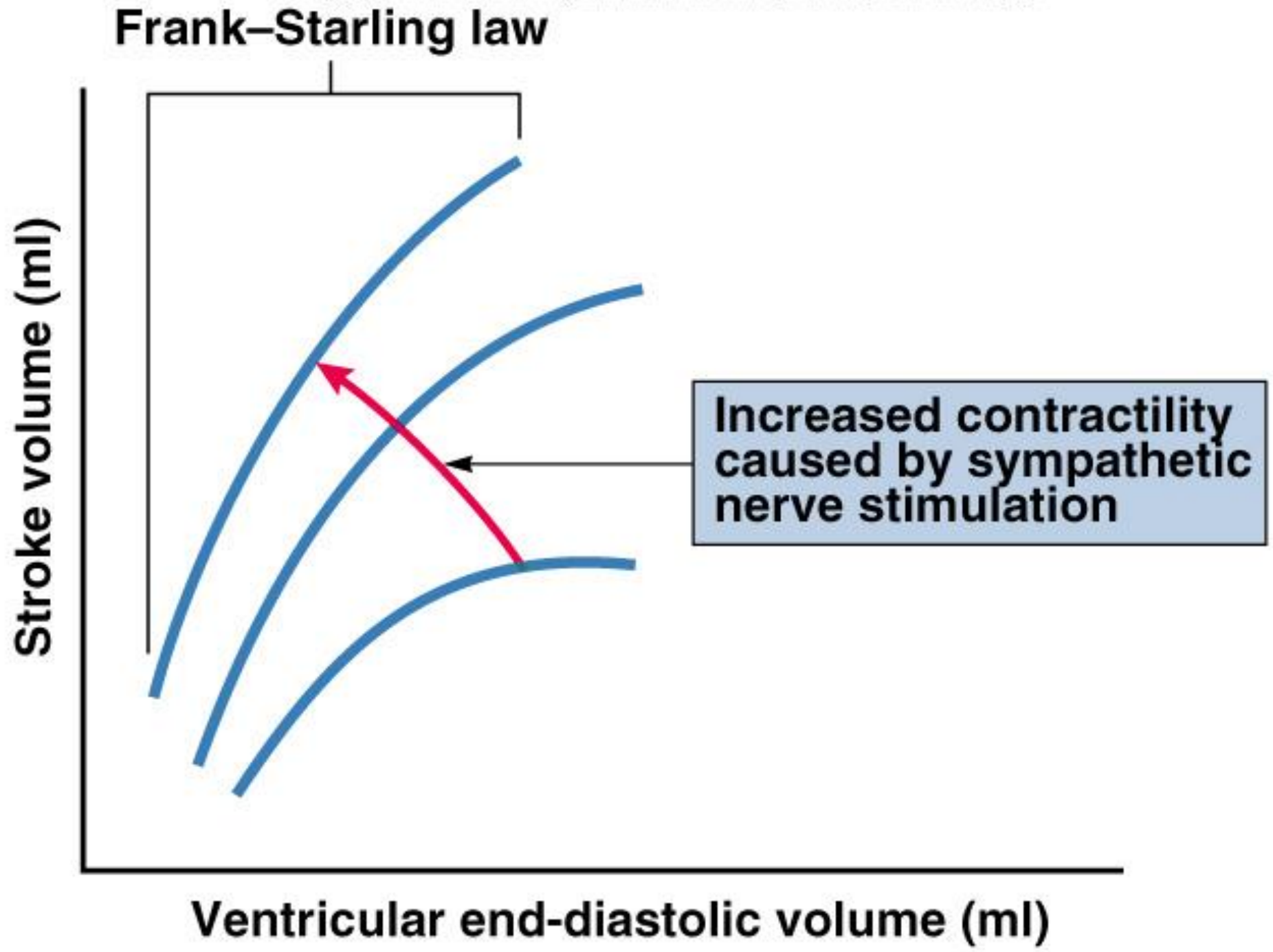


Fig. 9-21, p. 329



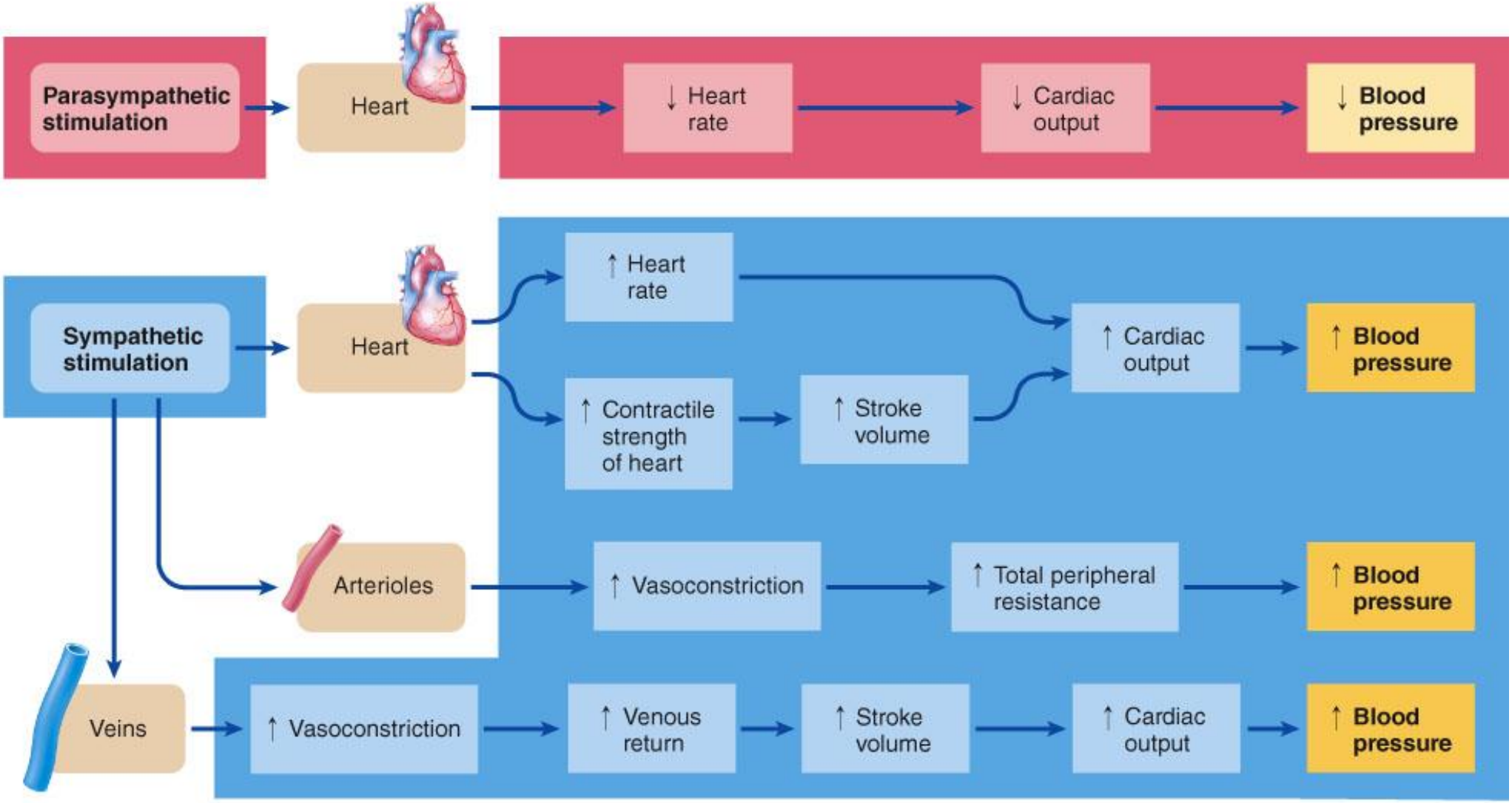


Fig. 10-37, p. 379

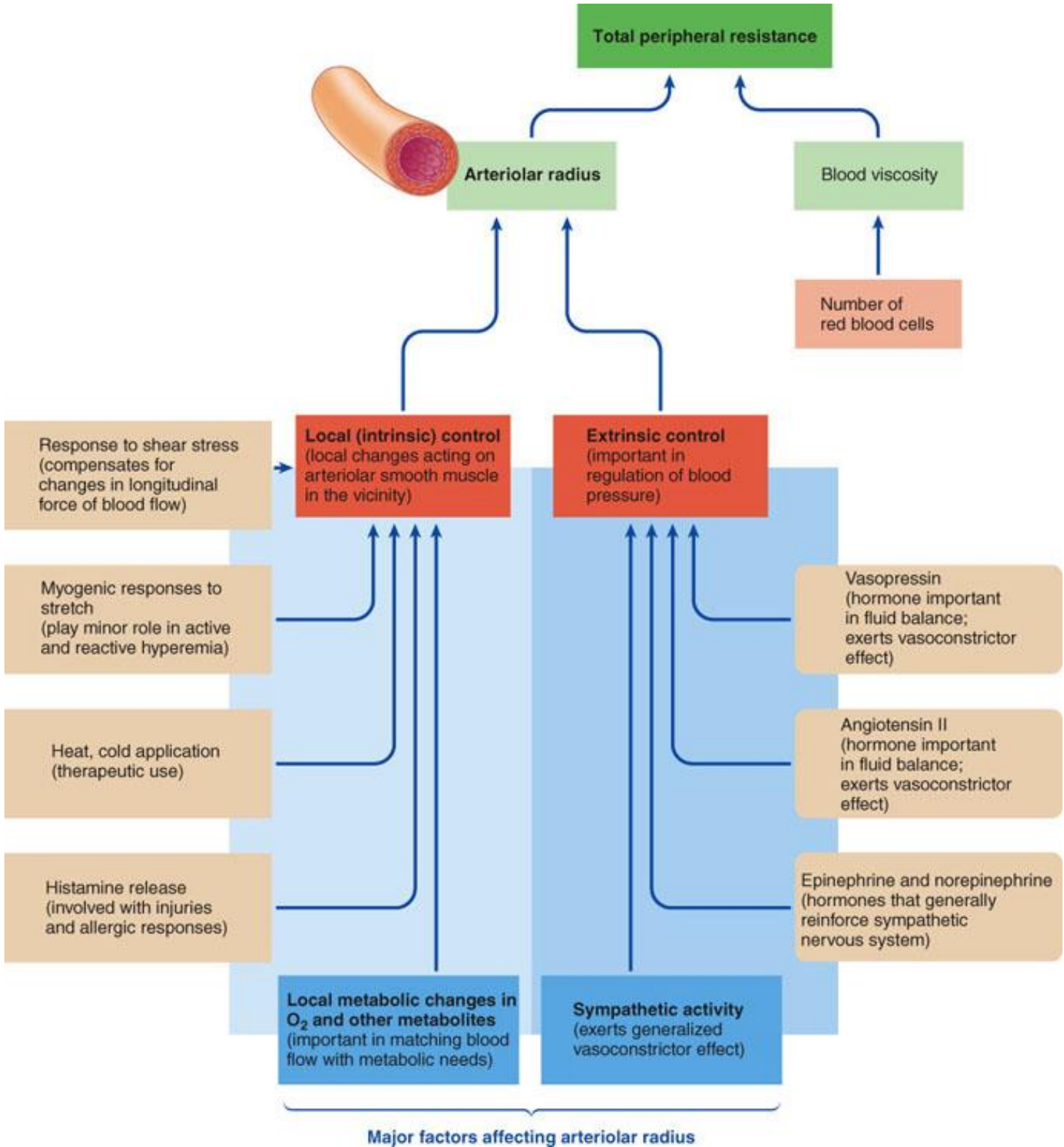
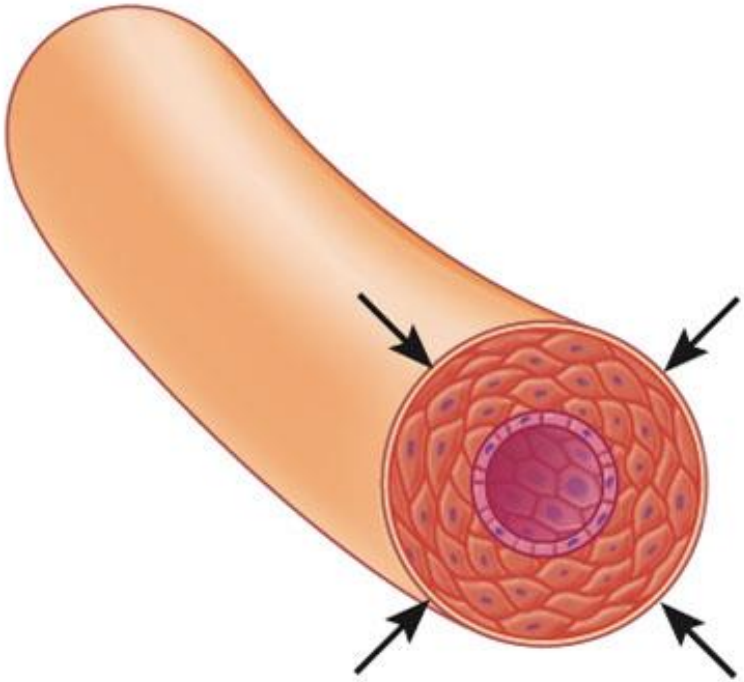


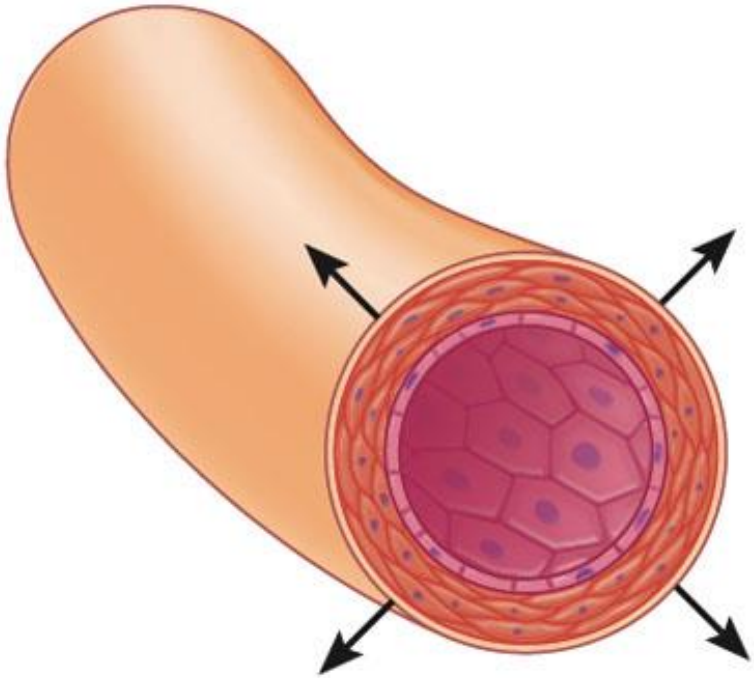
Fig. 10-14, p. 360



Caused by:

- ↑ Myogenic activity
- ↑ Oxygen (O_2)
- ↓ Carbon dioxide (CO_2) and other metabolites
- ↑ Endothelin
- ↑ Sympathetic stimulation
- Vasopressin; angiotensin II
- Cold

(c) Vasoconstriction (increased contraction of circular smooth muscle in the arteriolar wall, which leads to increased resistance and decreased flow through the vessel)



Caused by:

- ↓ Myogenic activity
- ↓ O_2
- ↑ CO_2 and other metabolites
- ↑ Nitric oxide
- ↓ Sympathetic stimulation
- Histamine release
- Heat

(d) Vasodilation (decreased contraction of circular smooth muscle in the arteriolar wall, which leads to decreased resistance and increased flow through the vessel)

Fig. 10-10d, p. 353

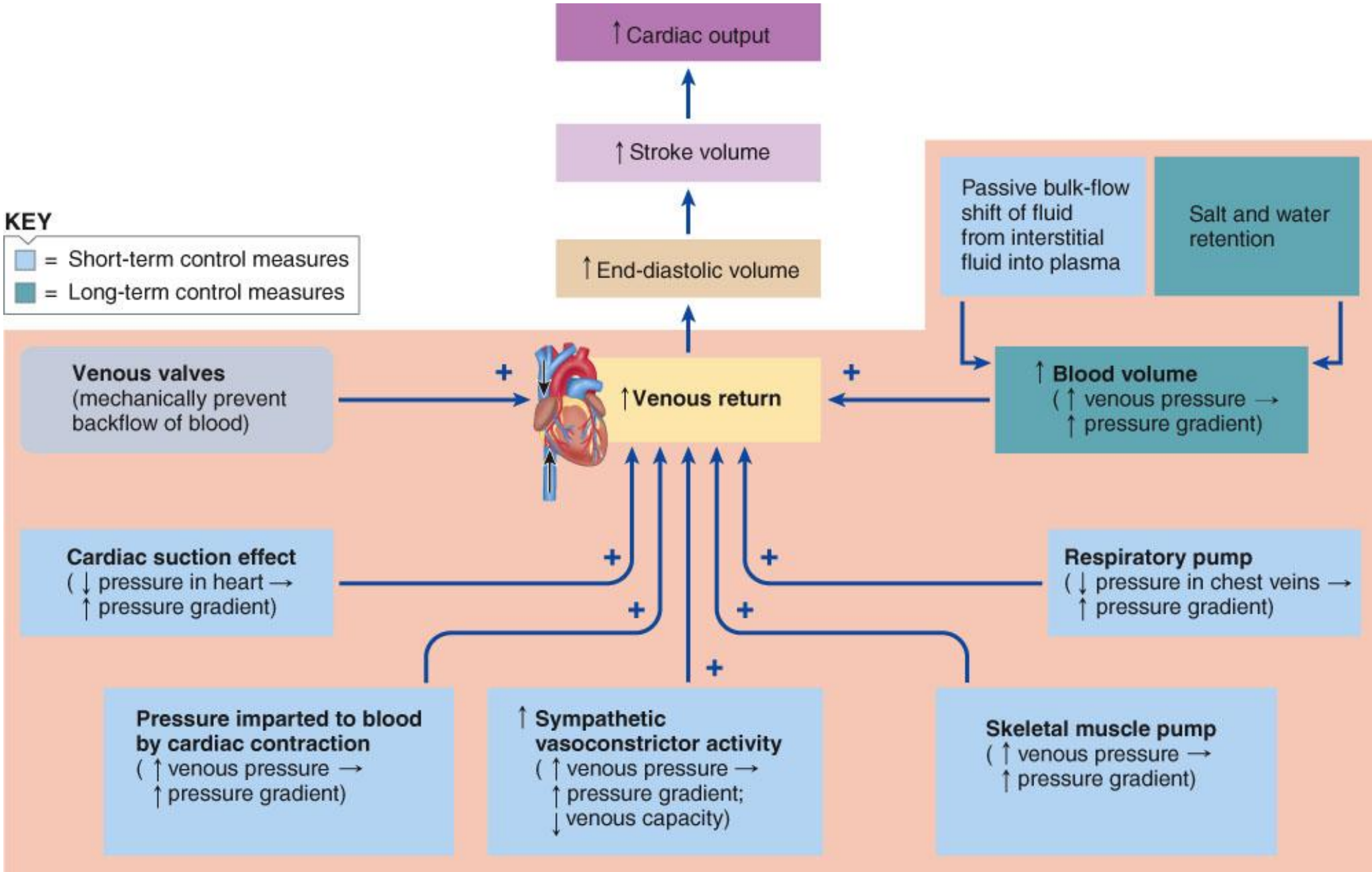


Fig. 10-28, p. 372

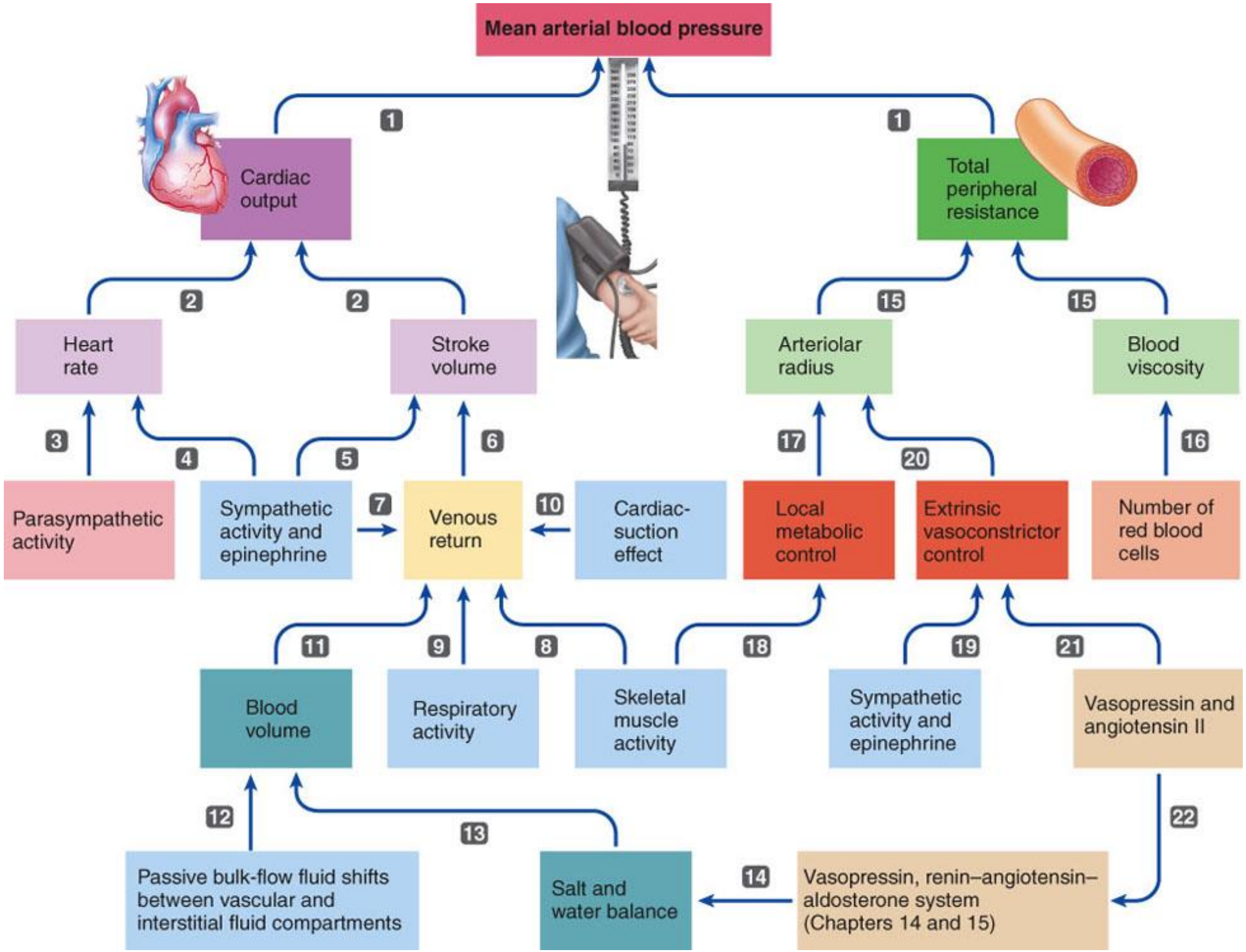
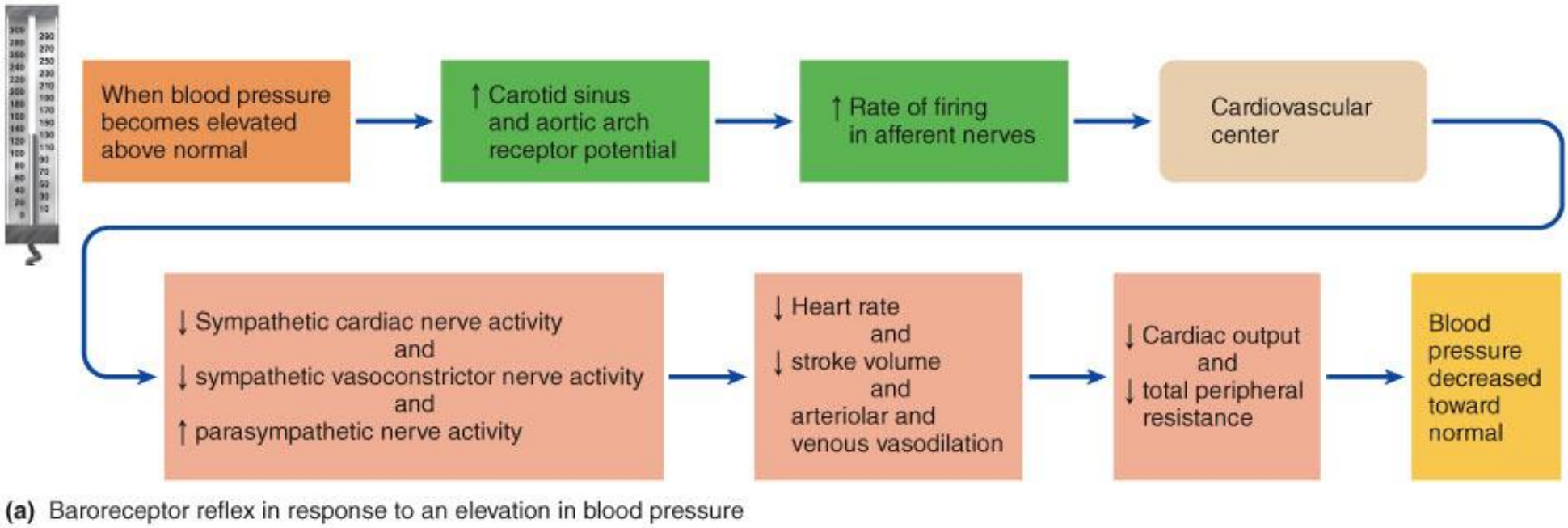
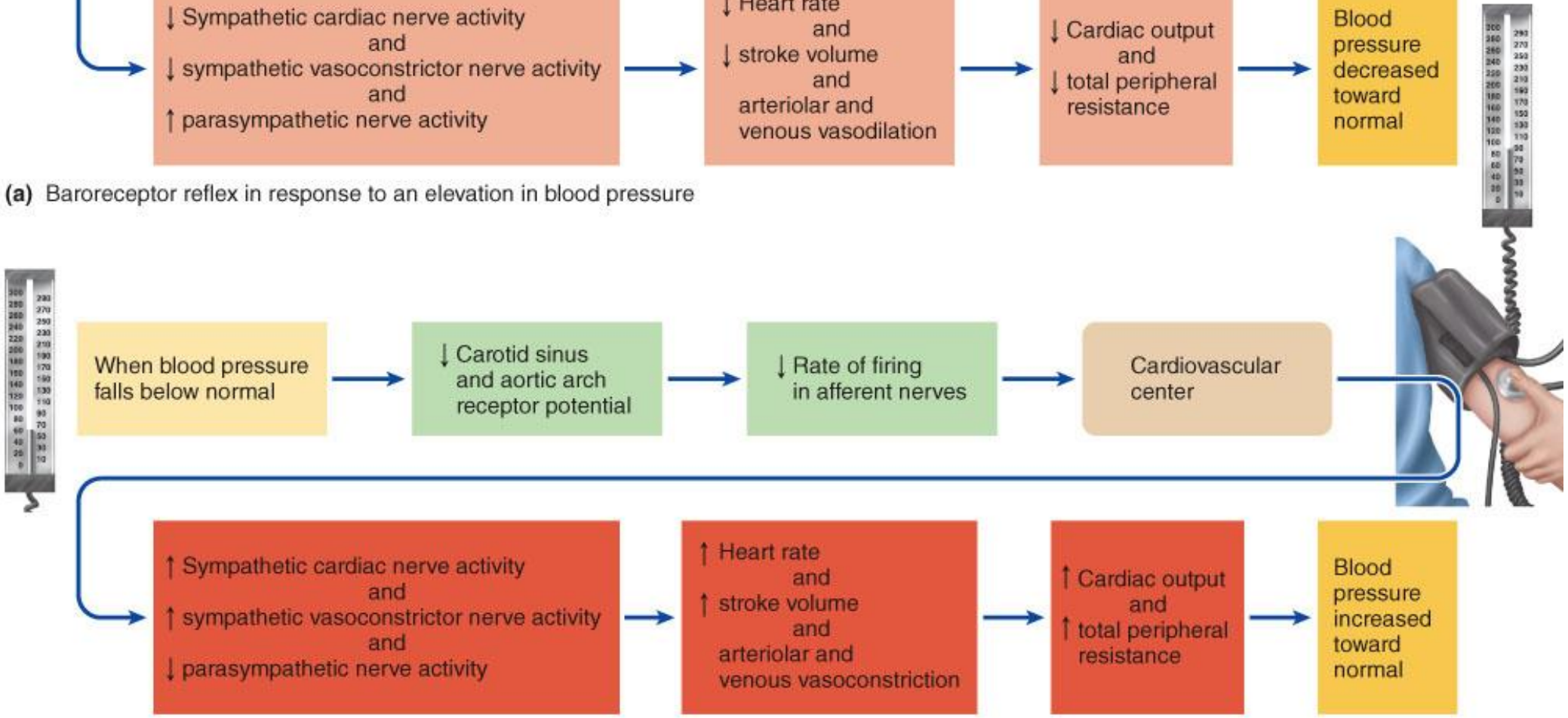


Fig. 10-34, p. 377



(a) Baroreceptor reflex in response to an elevation in blood pressure



(b) Baroreceptor reflex in response to a fall in blood pressure

Fig. 10-38, p. 380

TABLE 10-5 Cardiovascular Changes during Exercise

Cardiovascular Variable	Change	Comment
Heart Rate	Increases	Occurs as a result of increased sympathetic and decreased parasympathetic activity to the SA node
Venous Return	Increases	Occurs as a result of sympathetically induced venous vasoconstriction and increased activity of the skeletal muscle pump and respiratory pump
Stroke Volume	Increases	Occurs both as a result of increased venous return by means of the Frank–Starling mechanism (unless diastolic filling time is significantly reduced by a high heart rate) and as a result of a sympathetically induced increase in myocardial contractility
Cardiac Output	Increases	Occurs as a result of increases in both heart rate and stroke volume
Blood Flow to Active Skeletal Muscles and Heart Muscle	Increases	Occurs as a result of locally controlled arteriolar vasodilation, which is reinforced by the vasodilatory effects of epinephrine and overpowers the weaker sympathetic vasoconstrictor effect
Blood Flow to the Brain	Unchanged	Occurs because sympathetic stimulation has no effect on brain arterioles; local control mechanisms maintain constant cerebral blood flow whatever the circumstances
Blood Flow to the Skin	Increases	Occurs because the hypothalamic temperature control center induces vasodilation of skin arterioles; increased skin blood flow brings heat produced by exercising muscles to the body surface where the heat can be lost to the external environment
Blood Flow to the Digestive System, Kidneys, and Other Organs	Decreases	Occurs as a result of generalized sympathetically induced arteriolar vasoconstriction
Total Peripheral Resistance	Decreases	Occurs because resistance in the skeletal muscles, heart, and skin decreases to a greater extent than resistance in the other organs increases
Mean Arterial Blood Pressure	Increases (modest)	Occurs because cardiac output increases more than total peripheral resistance decreases