turn off cell phones

Start Recording

check audio connection

dim lights

Regulation of Heart & Blood Pressure

Keep **Blood Pressure** constant
- if too low, not enough blood (oxygen, glucose) reaches tissues
- if too high, blood vessels damaged & fluid lost from capillaries

Increase **Blood Flow** if needed
- exercising tissue needs higher throughput of blood
- pick up more oxygen from lungs
- faster delivery of oxygen to tissue

Cardiac Output and Blood Pressure

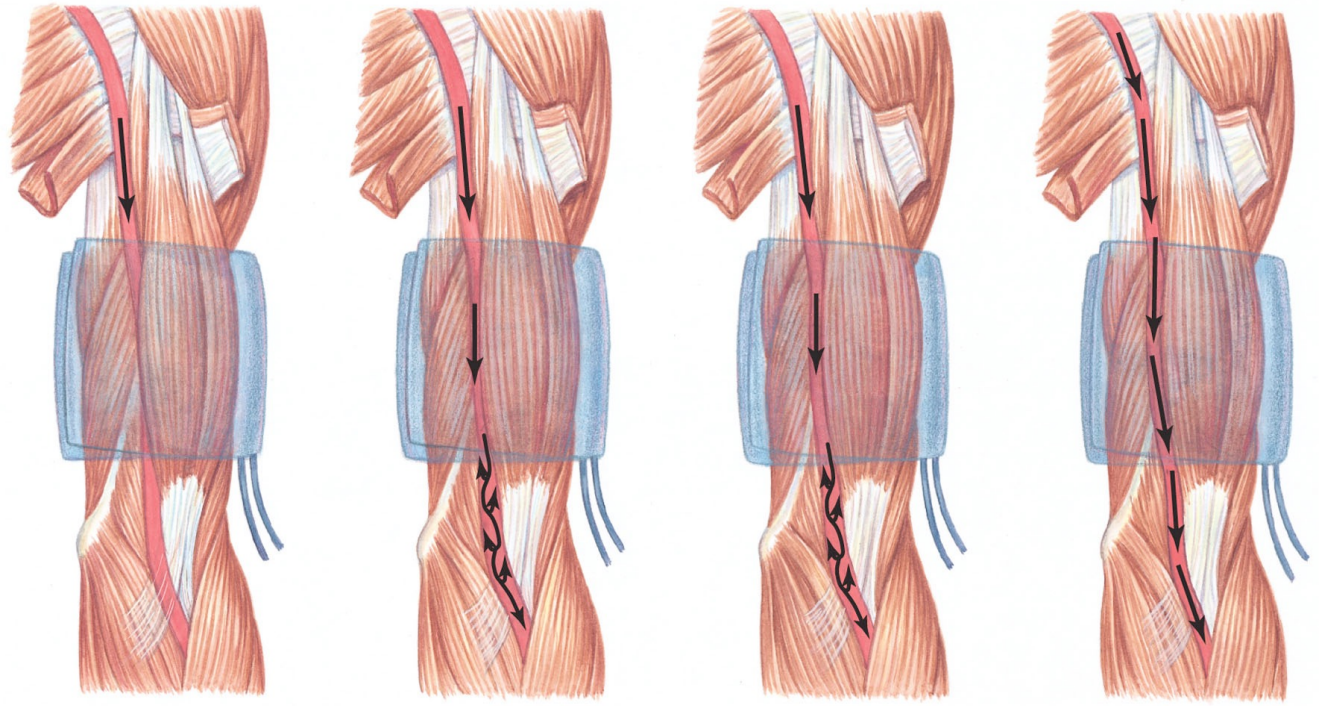
Arterial Blood Pressure is measured with a pressure cuff and **sphygmomanometer**.
1st Korotkoff sound occurs at systolic pressure, 2nd sound at diasystolic pressure.

Blood pressure is highest in aorta and large arteries; drops off in smaller arteries and capillaries because of increased resistance to blood flow.
(high on one side of resistance, low on the other side)

Figure 14.29

Pressure at which blood can **first** get past cuff (i.e. when blood is at highest pressure) gives systolic pressure

Pressure at which **all** blood can get past cuff (i.e. even when blood is at lowest pressure) gives diastolic pressure



No sounds

Cuff pressure = 140

First Korotkoff sounds

Cuff pressure = 120

Systolic pressure = 120 mmHg

Sounds at every systole

Cuff pressure = 100

Last Korotkoff sounds

Cuff pressure = 80

Diastolic pressure = 80 mmHg

Blood pressure = 120/80

Figure 14.30

Korotkoff Sounds start at ~130, end at ~90 mmHg

Pressure at which blood can **first** get past cuff (i.e. when blood is at highest pressure) gives systolic pressure

Pressure at which **all** blood can get past cuff (i.e. even when blood is at lowest pressure) gives diastolic pressure

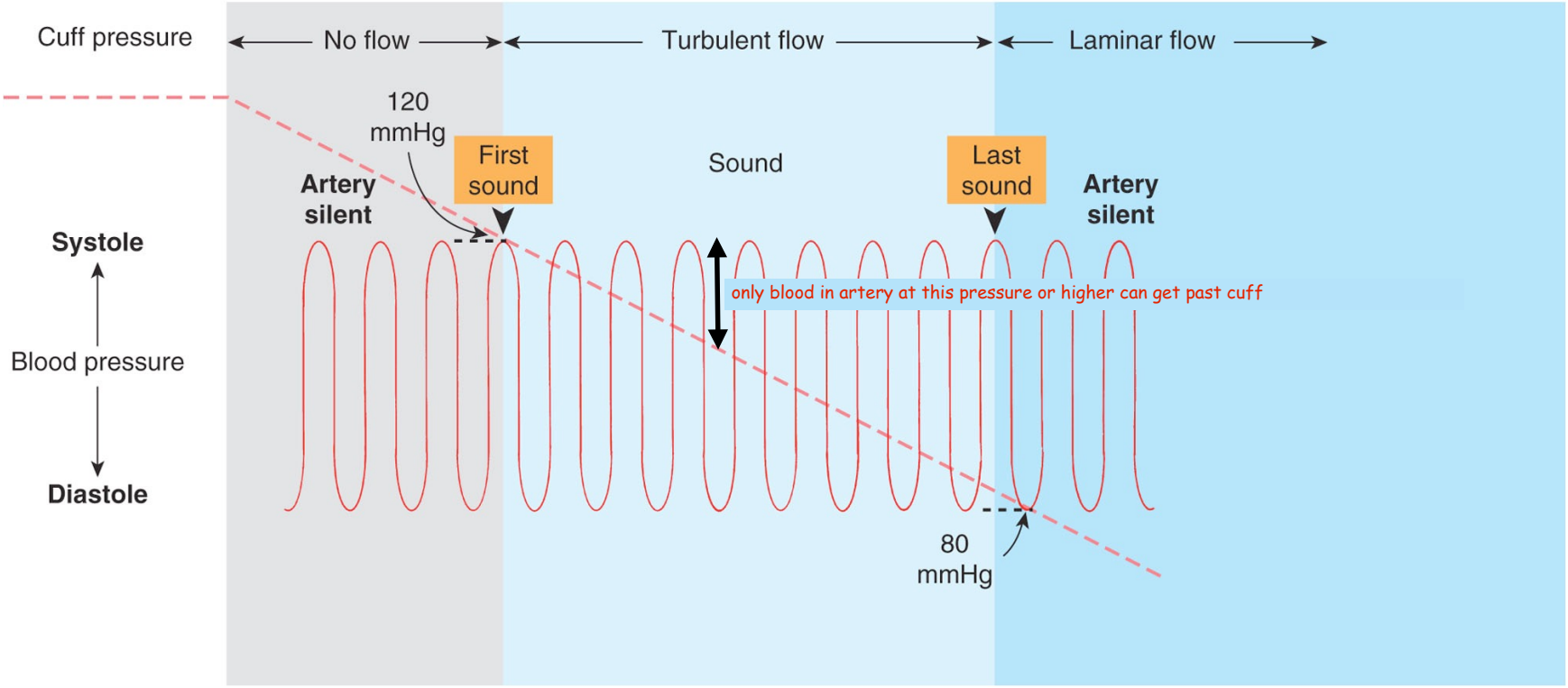
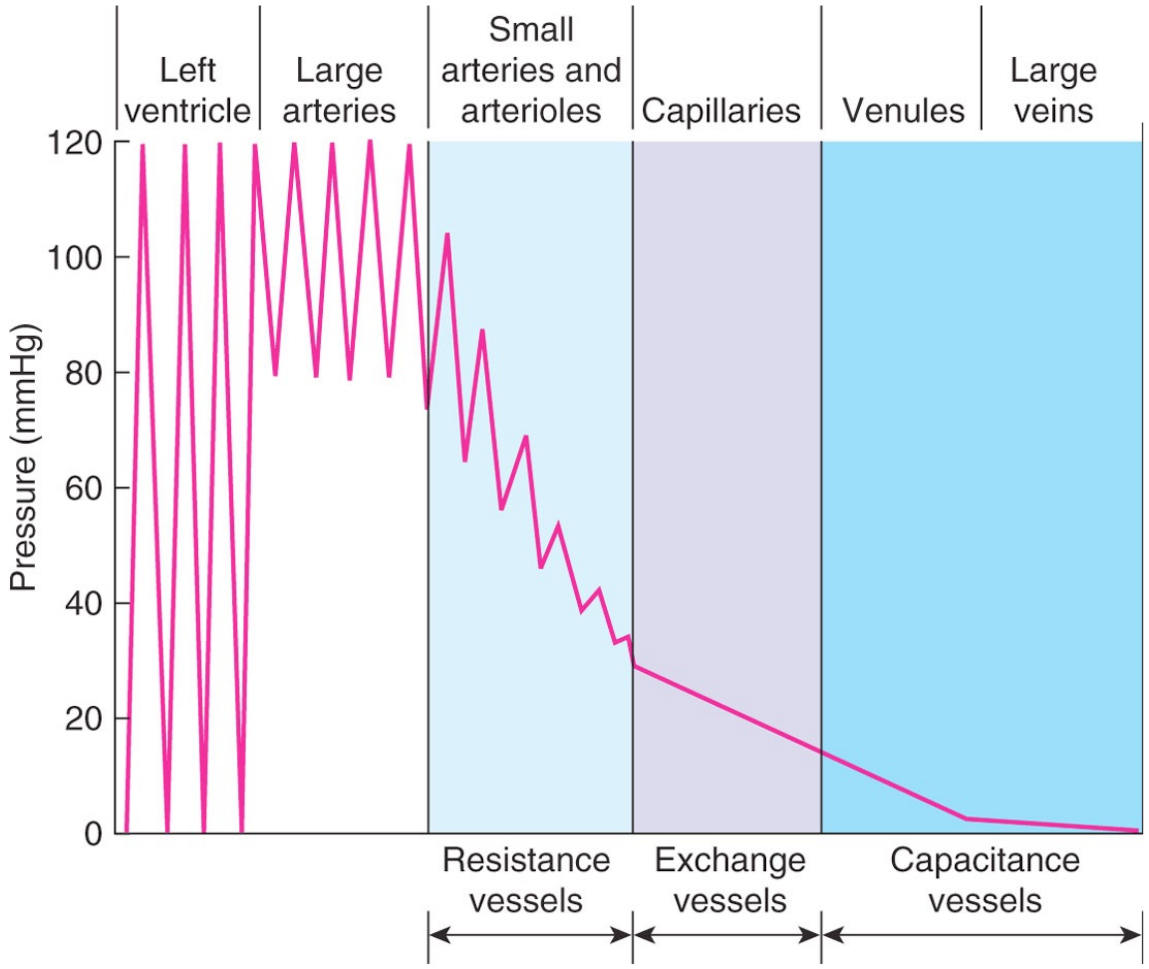


Figure 14.31

Figure 14.16


Left ventricle
Large arteries
Small arteries and arterioles
Capillaries
Venules
Large veins
120
100
80
60
40
20
0
Pressure (mmHg)
Resistance vessels
Exchange vessels
Capacitance vessels

Cardiac Output

- **cardiac output** = volume of blood pumped each minute by each ventricle.
- Product of **cardiac rate** and **stroke volume**.
- Regulation of heart and vasculature lead to changes in cardiac output
- e.g. exercise -> **increased** cardiac output.
- e.g. myocardial infarction -> cardiac output that is **too low** to maintain blood supply to body = **cardiac failure**
- To change cardiac output, need to change cardiac rate and/or stroke volume.

(note: cardiac output must be the same for both ventricles, but we'll use left ventricle as example)

Adjust cardiac output & Total Peripheral Resistance & blood volume to maintain a constant blood pressure of 120/80 mmHg

arterial blood pressure = cardiac output (total blood flow) X total peripheral resistance

$V = I \times R$

circulation at rest vs. exercise

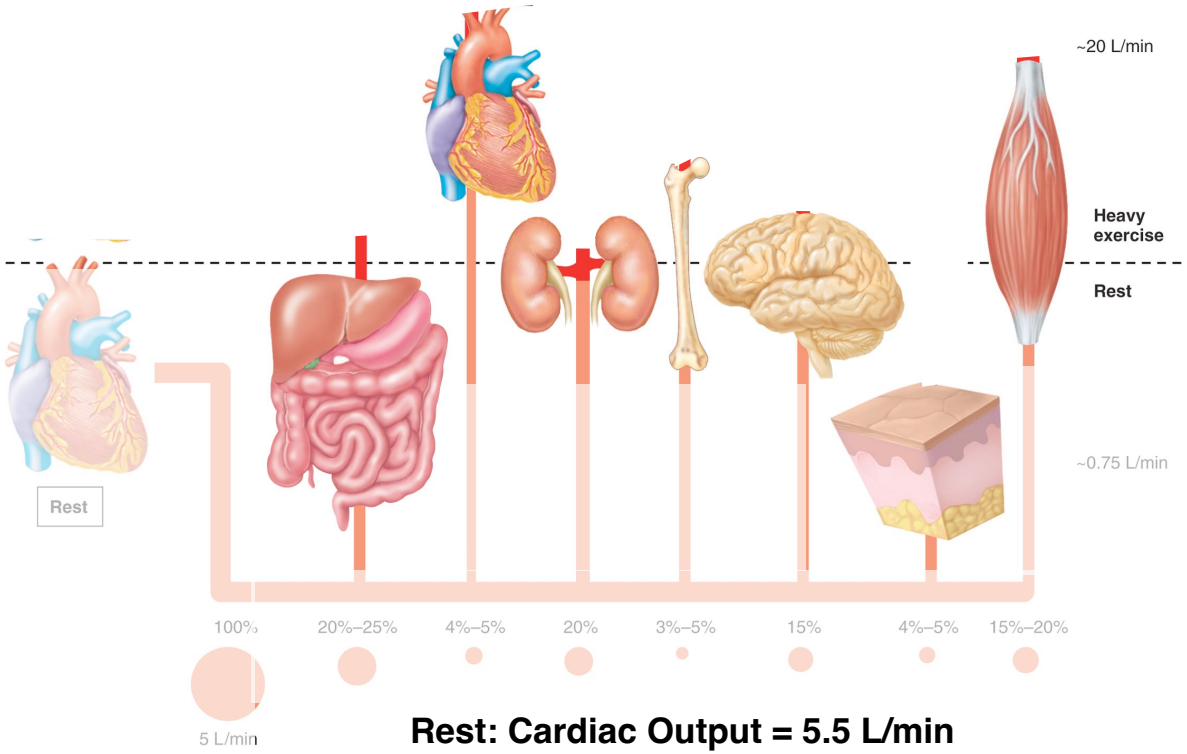


Figure 14.20

Exercise increases **cardiac output** and causes **redistribution** of blood flow

Organs	Blood Flow (ml/min)	
	At Rest	Exercising
Gastrointestinal tract and liver	1,400	750
Kidneys	1,100	500
Brain	750	750
Heart	250	1000
Skeletal muscles	1,200	21,250
Skin	500	0
Other organs	600	750
Total organs	5,800	25,000
Cardiac Output:	5.8 L/min	25 L/min

Blood Pressure = Blood flow x Resistance

blood pressure stays pretty constant
so, to increase blood flow:
increase **cardiac output** and/or **decrease resistance** at muscles

Cardiac Output

Cardiac output	=	Cardiac rate	X	Stroke Volume
(ml/min) amount of blood pumped out by heart every minute		(beats/min) how often the heart beats each minute		(ml/beat) how much blood is ejected from each ventricle with each beat

Average Resting Values:

5.5k ml / min	=	**70 beats/min**	X	**80 ml/beat**

Total blood volume = 5.5 liters, so all blood pumped each minute.

Figure 14.5

Cardiac Output

Cardiac output	=	Cardiac rate	X	Stroke Volume
(ml/min) amount of blood pumped out by heart every minute		(beats/min) how often the heart beats each minute		(ml/beat) how much blood is ejected from each ventricle with each beat

How to increase cardiac output?

Increase heart rate.

Increase stroke volume.

- increase contractility of ventricle.
- increase venous return.

Regulation of Cardiac Rate

Chronotropic effects

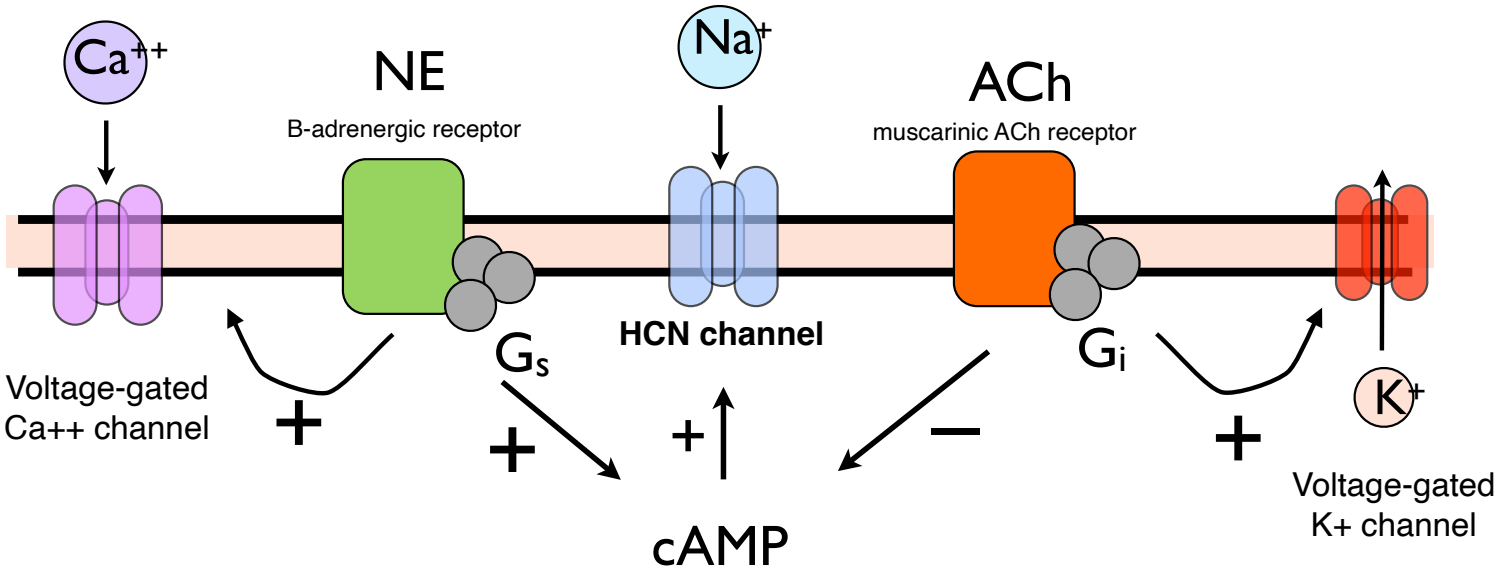
- Sympathetic Nervous System: **norepi** from **sympathetic nerves** and **epi** from **adrenal medulla** -> beta-adrenergic receptors -> increased cAMP -> open HCN channels in SA node -> **faster heart rate**

- Parasympathetic Nervous System: **ACh** from **vagus nerve** -> decreased cAMP -> closed HCN channels & open K+ channels in SA node -> **slower heart rate**

- Autonomic innervation regulated by **cardiac control center** in brainstem medulla. Changes in blood pressure detected by **baroreceptors** (pressure sensors, like barometer) cause **reflexive change** in heart rate to restore normal blood pressure.

(Inotropic Effect:
Norepi and Epi increase Ca++ influx into ventricular muscle)

Autonomic Nervous System & Heart Rate



What would the effects of NE and ACh agonists & antagonists be on heart rate?

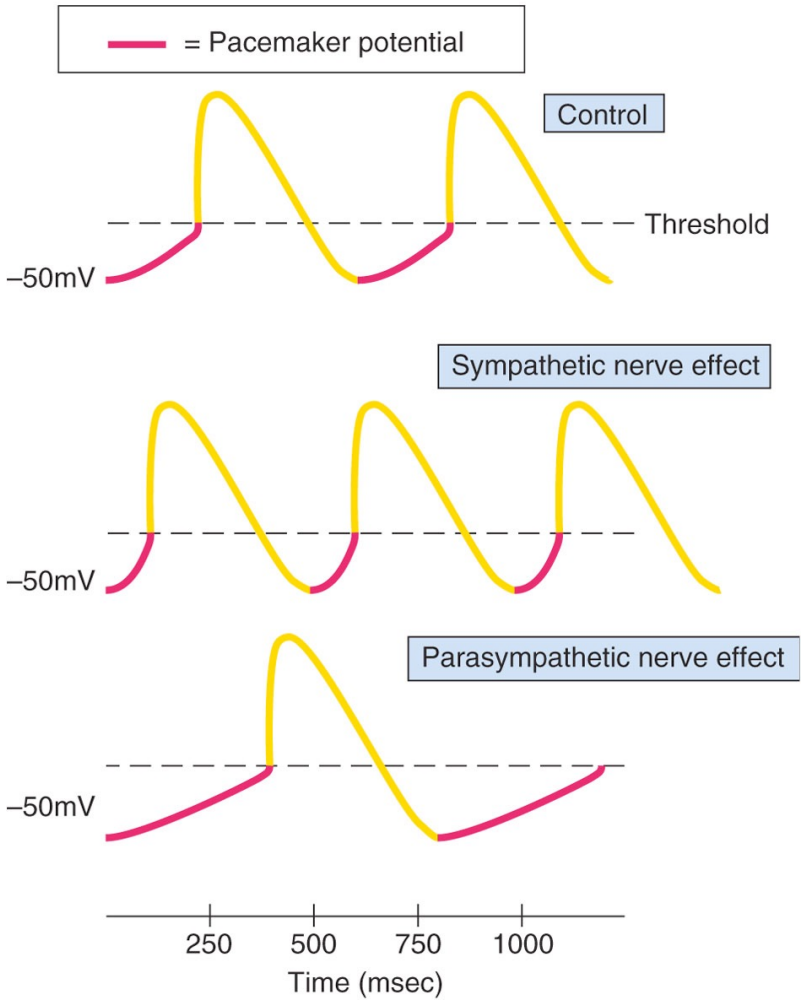

= Pacemaker potential
Control
Threshold
–50mV
Sympathetic nerve effect
–50mV
Parasympathetic nerve effect
–50mV
250
500
750
1000
Time (msec)

Figure 14.1

Regulation of Heart & Blood Pressure

Keep **Blood Pressure** constant
- if too low, not enough blood (oxygen, glucose) reaches tissues
- if too high, blood vessels damaged & fluid lost from capillaries

Increase **Blood Flow** if needed
- exercising tissue needs higher throughput of blood
- pick up more oxygen from lungs
- faster delivery of oxygen to tissue

Blood flow = ΔP / resistance

cardiac output = MAP / TPR

(I = V/R)

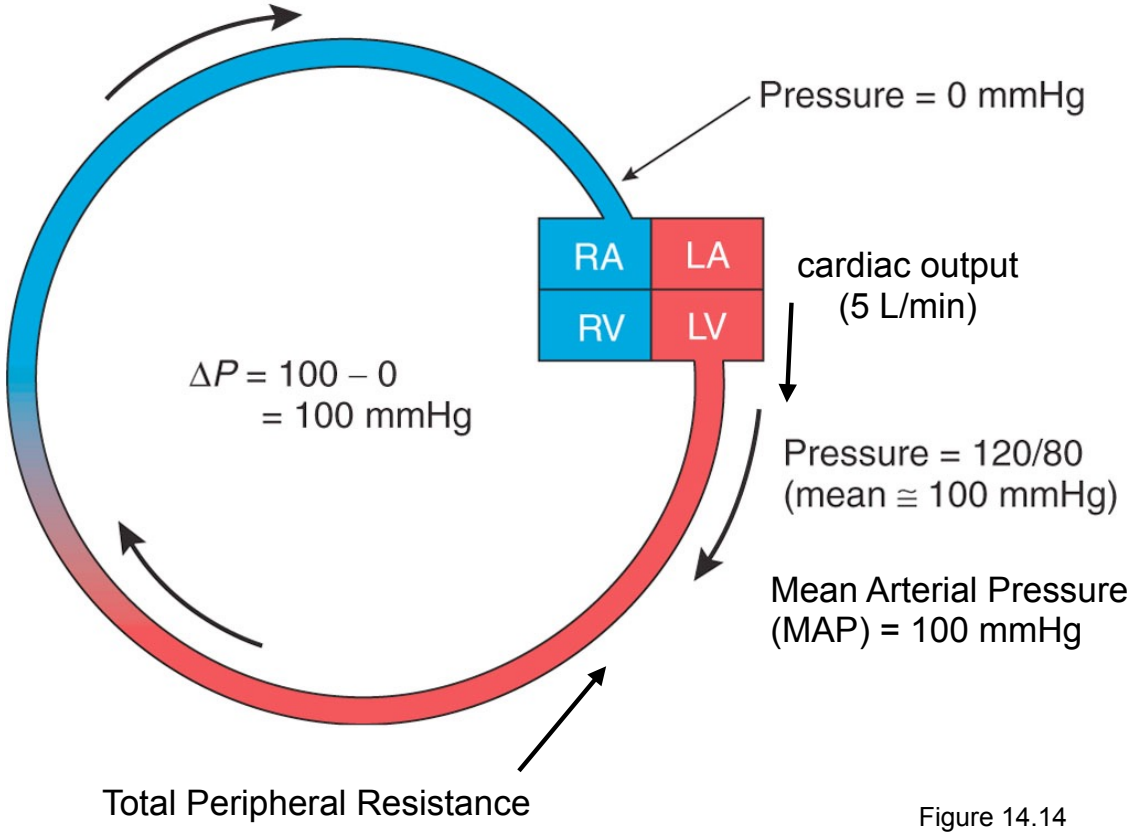


Figure 14.14

baroreceptors in aorta & carotid sinus -> medullary cardiac control center

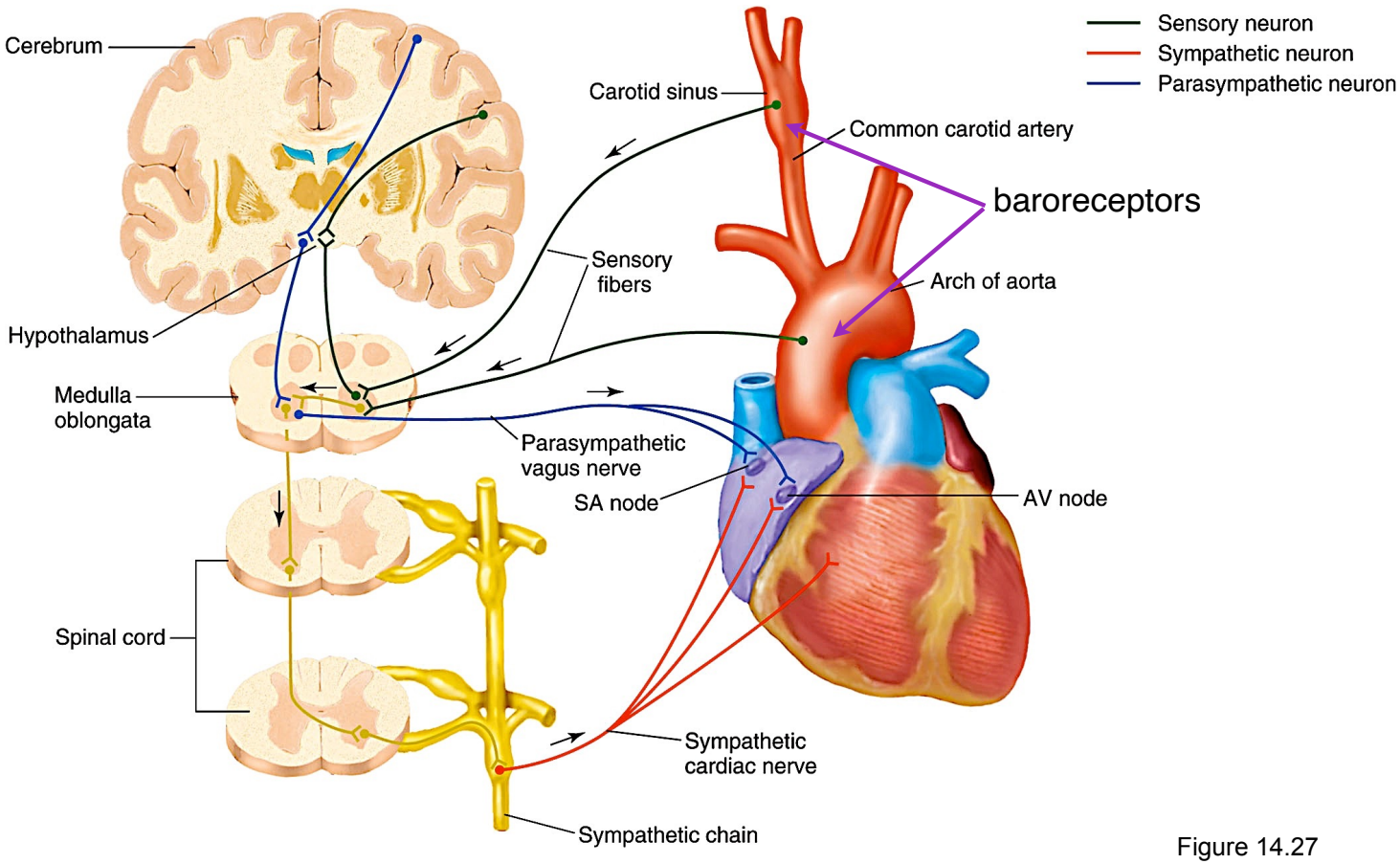
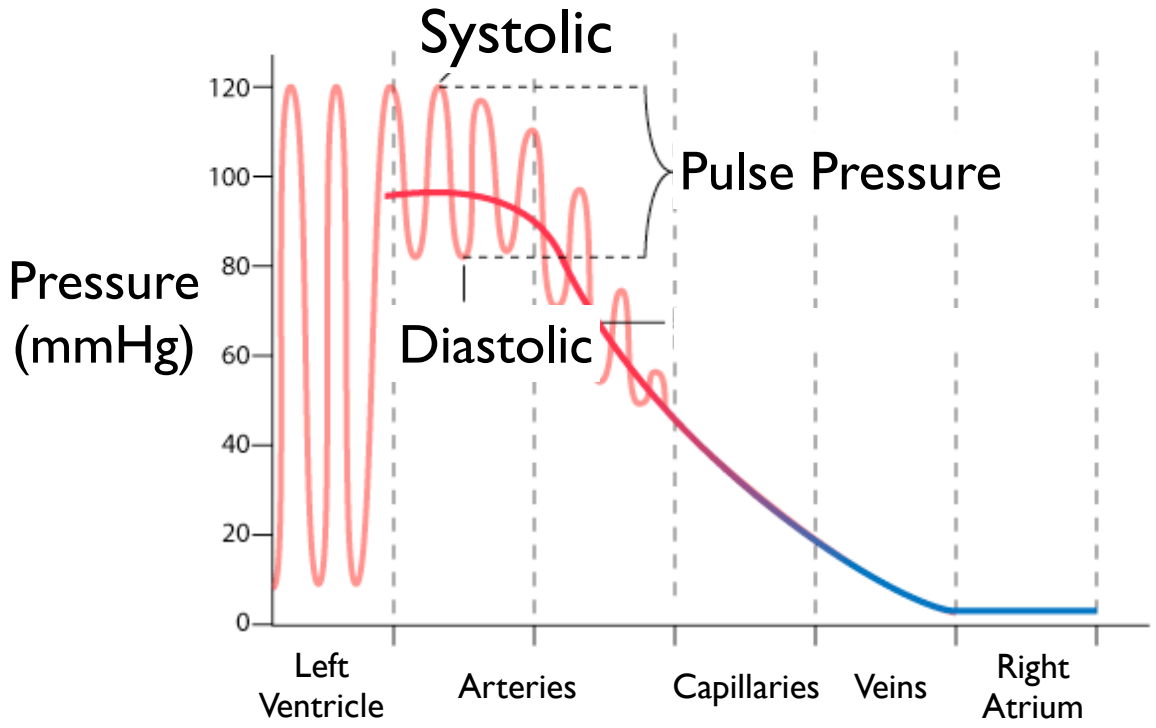


Figure 14.27

Blood Pressure:

Arterial Pressure > Venous Pressure



http://www.adinstruments.com

Baroreceptor Sensory Neurons report Blood Pressure

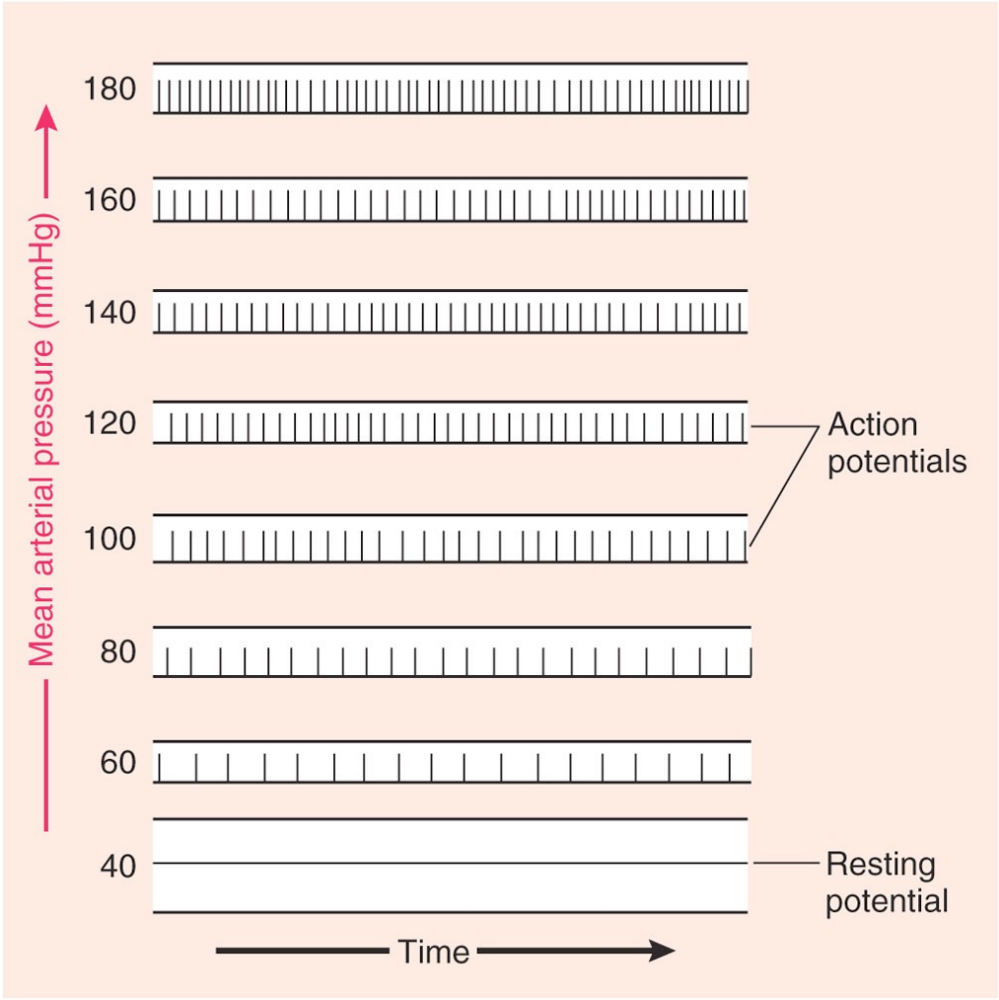


Figure 14.26

Orthostatic Regulation of Blood Pressure

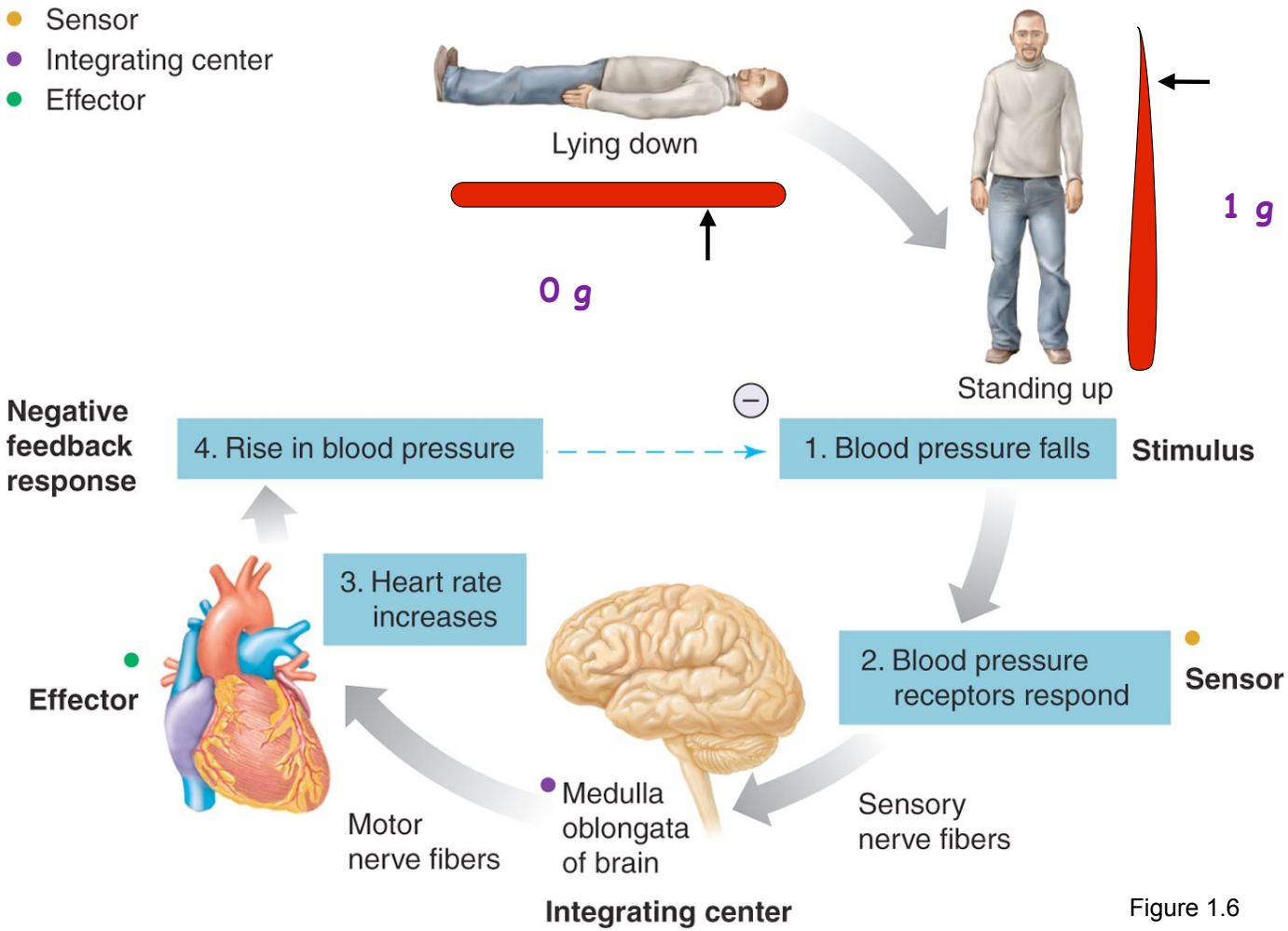


Figure 1.6

G-Forces

In aircraft particularly, vertical g-forces are often positive (forcing blood towards the feet and away from the head); this causes problems with the eyes and brain in particular. As positive vertical g-force is progressively increased (such as in a centrifuge) the following symptoms may be experienced:

- **Grey-out,** where the vision loses hue, easily reversible on levelling out. **(2-3g)**
- **Tunnel vision**, where peripheral vision is progressively lost.
- **Blackout**, a loss of vision while consciousness is maintained, caused by a lack of blood to the head. **(4 g)**
- **G-LOC** a loss of consciousness ("LOC" stands for "Loss Of Consciousness"). **(5 g; 9 g with a g-suit)**
- **Death**, if g-forces are not quickly reduced, death can occur. **(50 g)**

Table 14.1

Table 14.1 | Effects of Autonomic Nerve Activity on the Heart

	Region Affected	Sympathetic Nerve Effects	Parasympathetic Nerve Effects
chronotropic	SA node	Increased rate of diastolic depolarization; increased cardiac rate	Decreased rate of diastolic depolarization; decreased cardiac rate
chronotropic	AV node	Increased conduction rate	Decreased conduction rate
Inotropic	Atrial muscle	Increased strength of contraction	No significant effect
Inotropic	Ventricular muscle	Increased strength of contraction	No significant effect

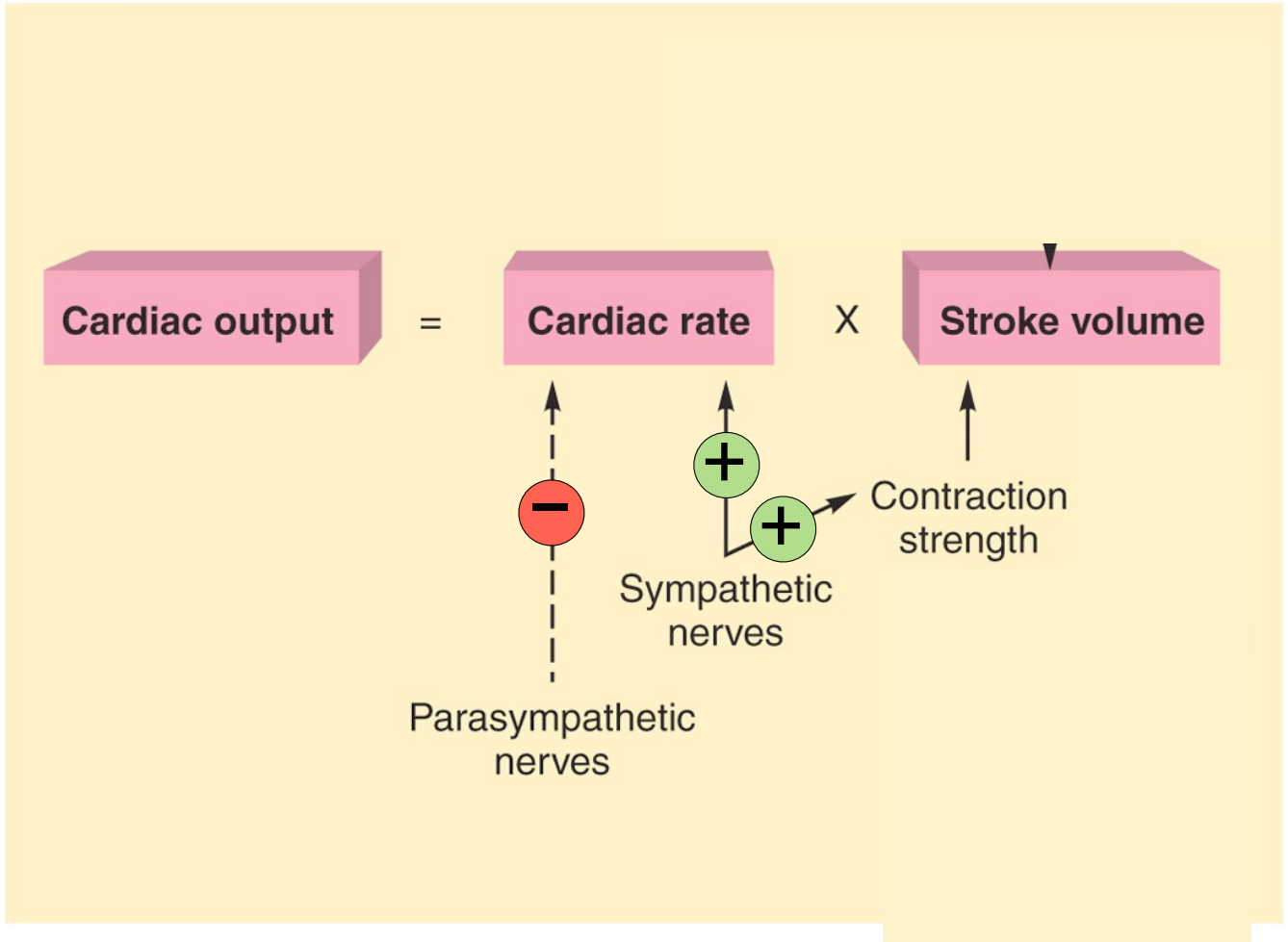

Cardiac output
=
Cardiac rate
X
Stroke volume
Contraction strength
Sympathetic nerves
Parasympathetic nerves

Figure 14.5

Regulation of Stroke Volume

Stroke volume is determined by:

- **end-diastolic volume** (EDV); amount of blood in ventricles right before they contract (systole)
- **contractility** (strength) of ventricle contraction
- **total peripheral resistance**; frictional resistance of arteries to increased blood flow

The more blood in the ventricle (EDV) and the stronger the ventricle contracts (contractility), the more blood is pumped out with each beat (stroke volume).

Resistance from the arteries -> increased arterial pressure -> resists the pumping of blood out of the ventricle.

Intrinsic Control: Frank-Starling Law:
Greater EDV -> increased stretch of ventricle -> increased contractility

Extrinsic Control: Inotropic Effect of Sympathetic Nerves
Norepi & epi -> increased Ca++ influx in cardiac muscle -> increased contractility.

Cardiac Output

Cardiac output	=	Cardiac rate	X	Stroke Volume
(ml/min) amount of blood pumped out by heart every minute		(beats/min) how often the heart beats each minute		(ml/beat) how much blood is ejected from each ventricle with each beat

Average Resting Values:

5.5k ml / min	=	**70 beats/min**	X	**80 ml/beat**

Total blood volume = 5.5 liters, so all blood pumped each minute.

Figure 14.5

Stroke Volume

amount of blood in ventricle before contraction

strength of ventricular contraction

Stroke Volume = **End-Diastolic Volume (EDV)** X **Contractility**

(ml/beat)
how much blood is ejected from each ventricle with each beat

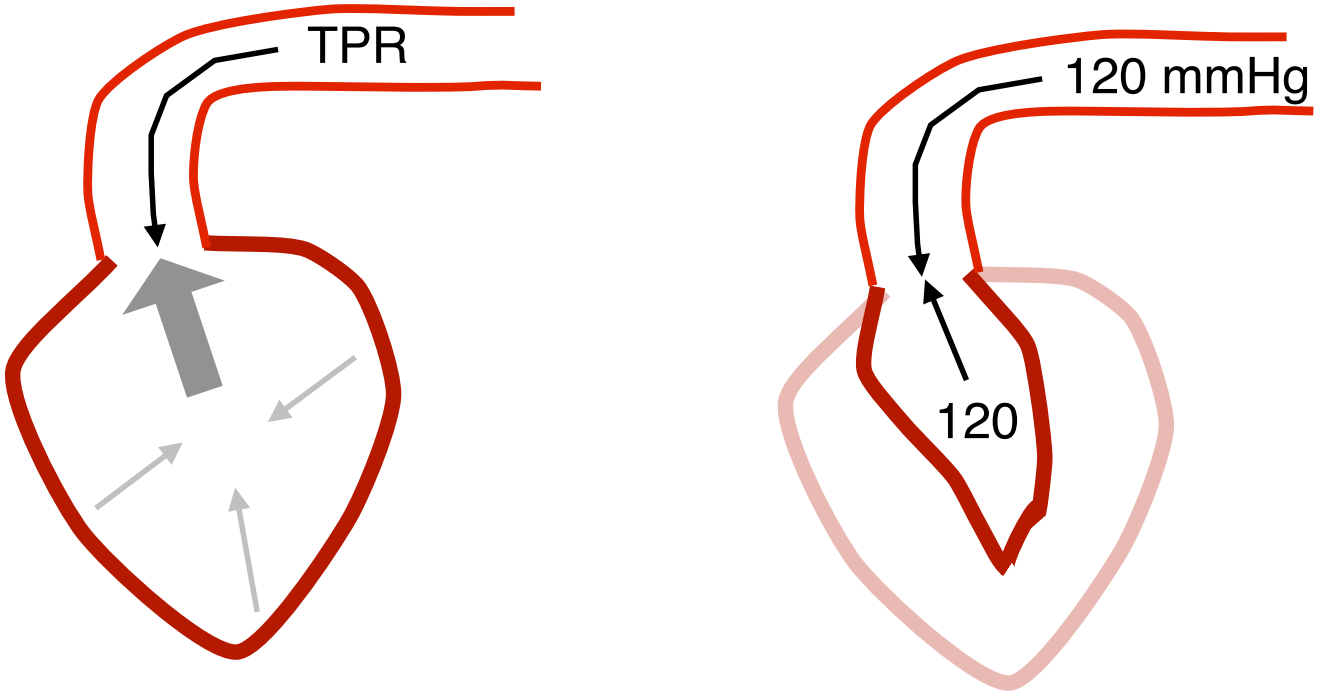
---

total peripheral resistance

resistance of arteries to increased blood flow

$$I = \frac{V}{R}$$

Total peripheral resistance resists cardiac output



ventricles contract until ventricular pressure matches TPR

Vasoconstriction elevates blood pressure

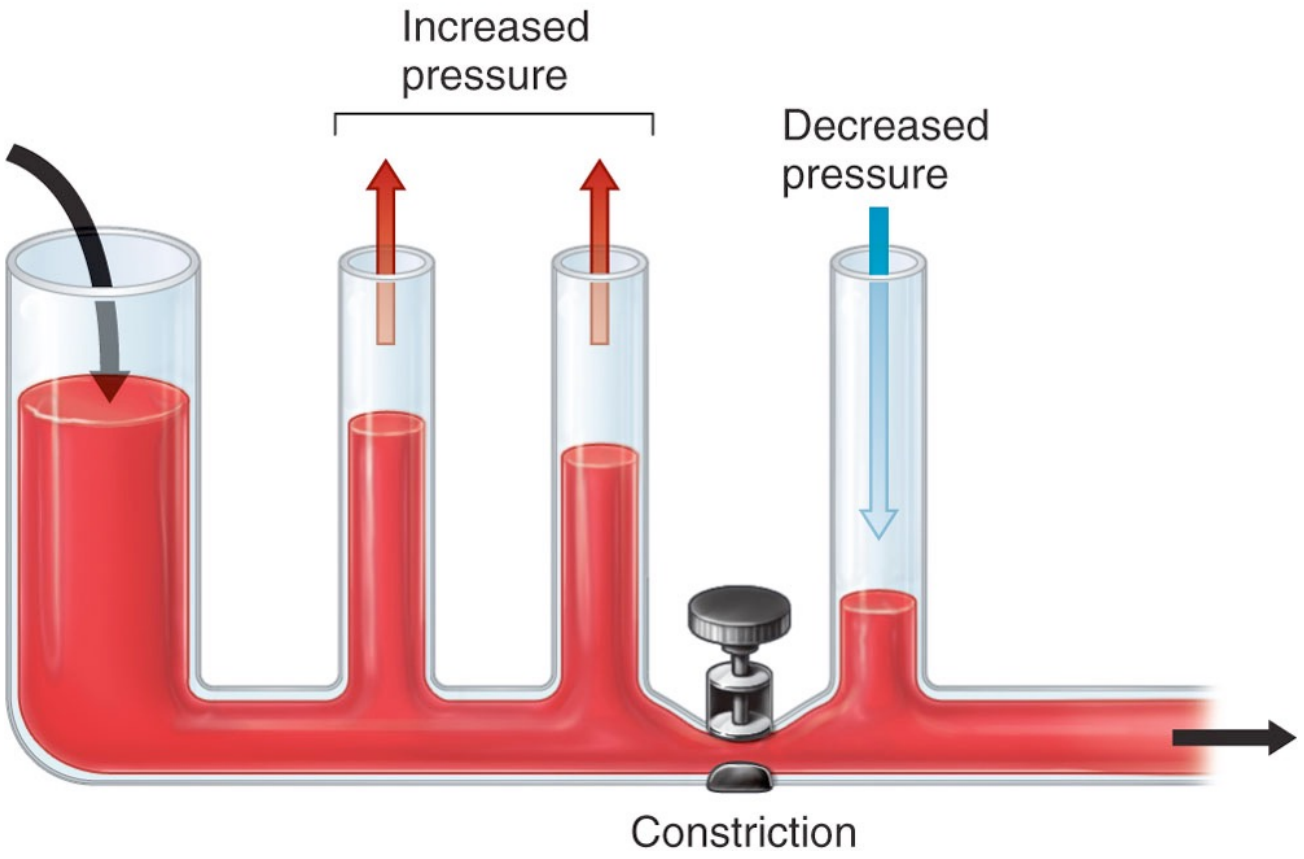
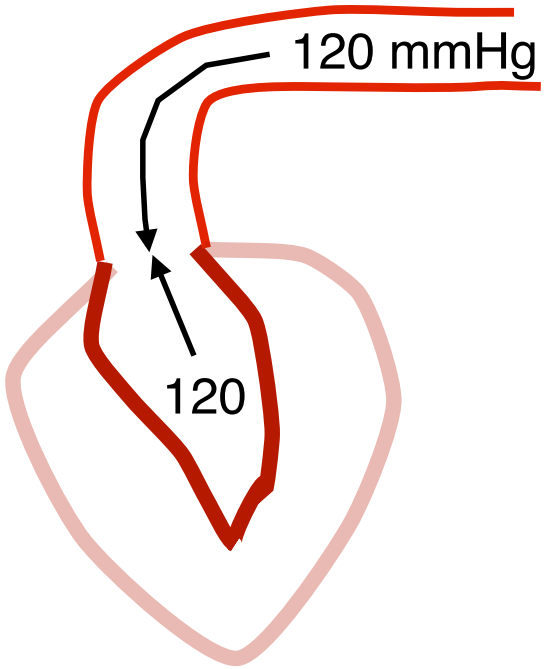
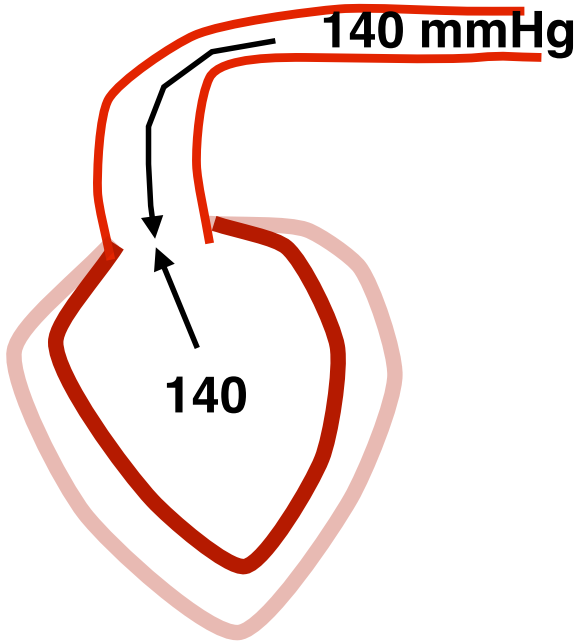


Figure 14.24

Total peripheral resistance resists cardiac output



vasoconstriction raises
total peripheral resistance



because of higher pressure,
ventricle can't pump as much blood out

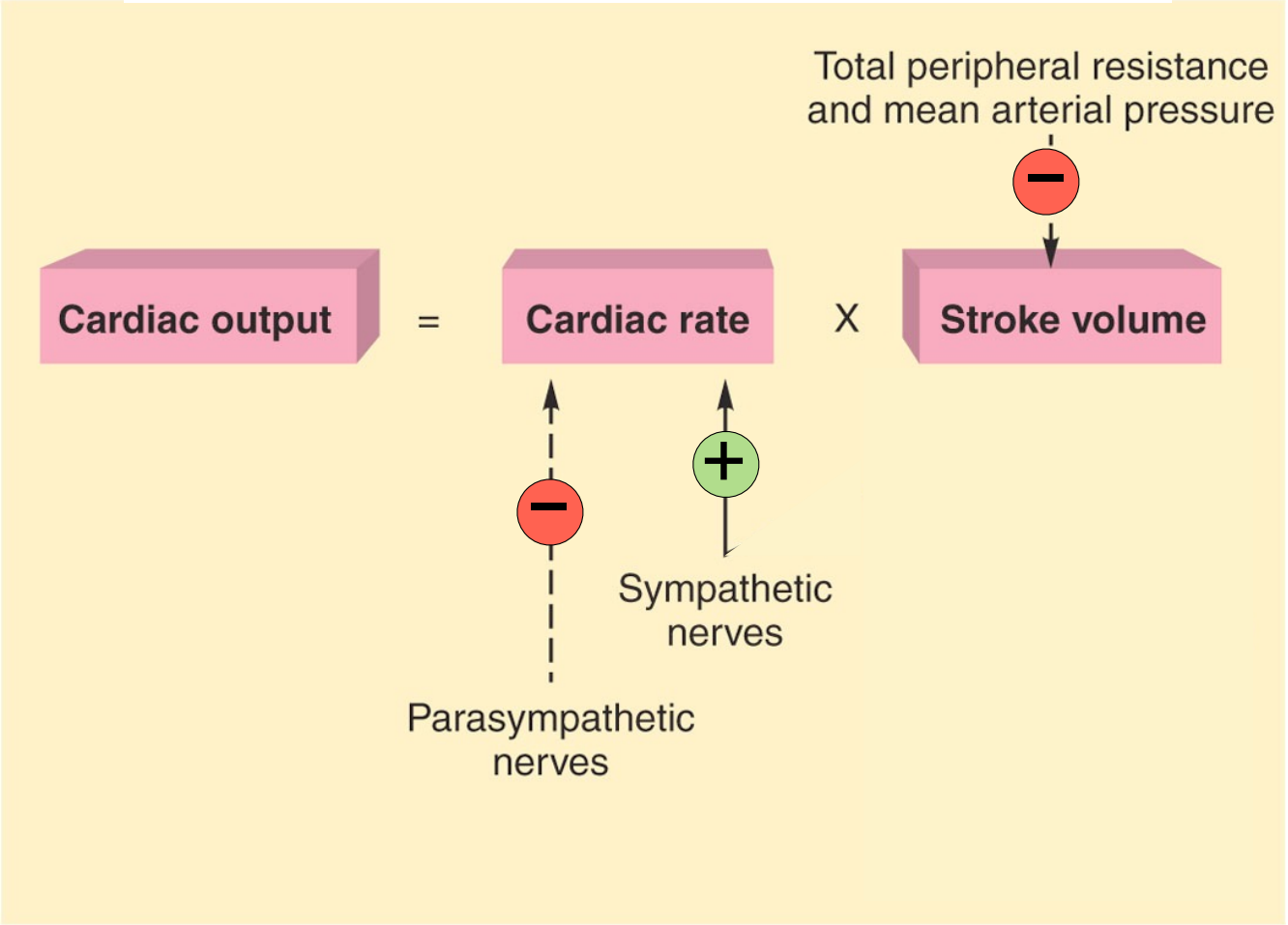

Total peripheral resistance
and mean arterial pressure
Cardiac output
=
Cardiac rate
X
Stroke volume
Sympathetic
nerves
Parasympathetic
nerves

Figure 14.5

Frank Starling Law of the Heart

strength of contraction varies directly with end-diastolic volume (EDV)

- More blood in the ventricle stretches the relaxed cardiac muscle more.
- Less overlap of actin and myosin fibers -> allows formation of more cross bridges -> greater contractile strength

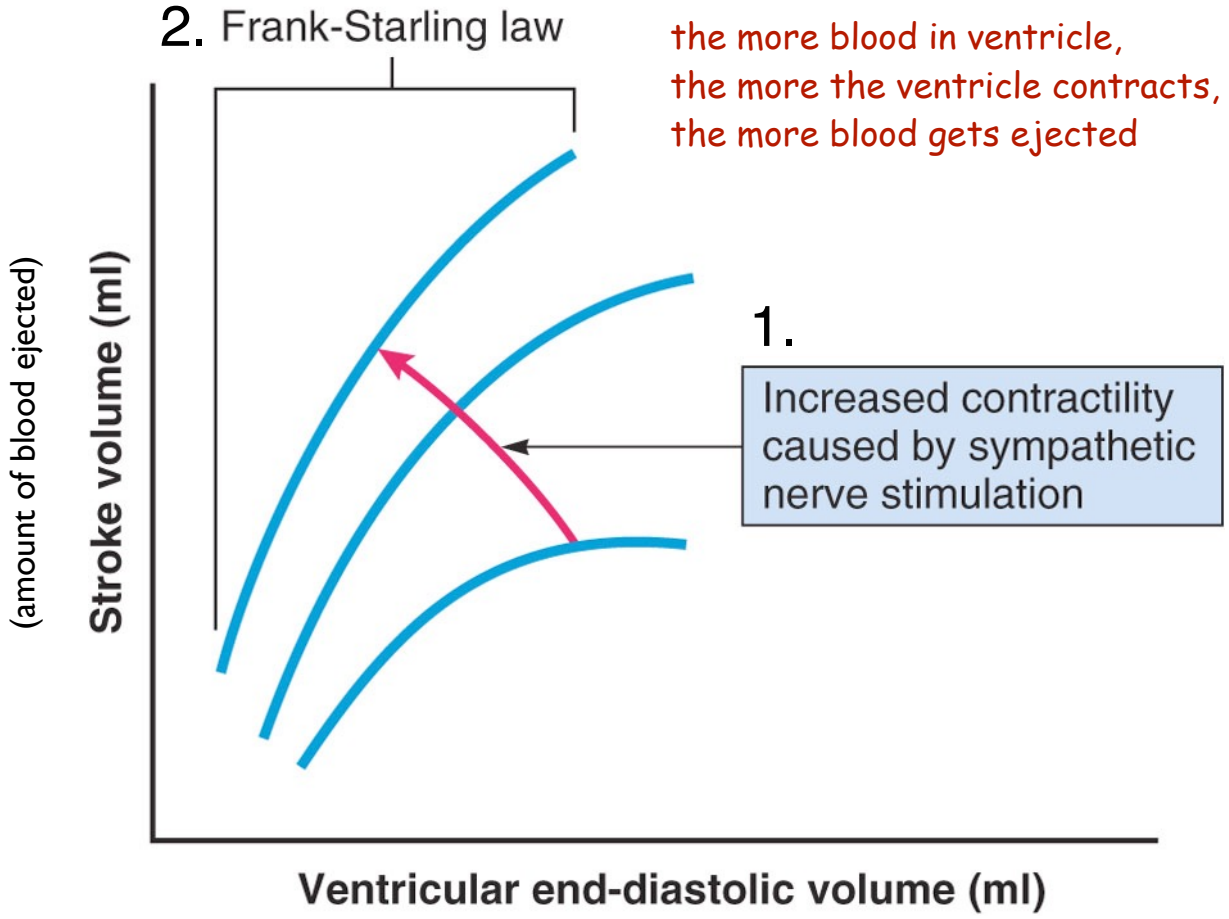
Ejection fraction in healthy heart is maintained at 60%.

At rest, EDV is 130 ml/ventricle, and stroke volume is 80 ml/beat, i.e., 60% of EDV is ejected from the ventricle into aorta.

During exercise, EDV goes up, but ejection fraction remains at 60%, thus **contractility must increase** to pump higher volume.

Figure 14.2

Two mechanisms increase contractility:



More blood in the ventricle stretches the cardiac muscle, resulting in greater strength during contraction

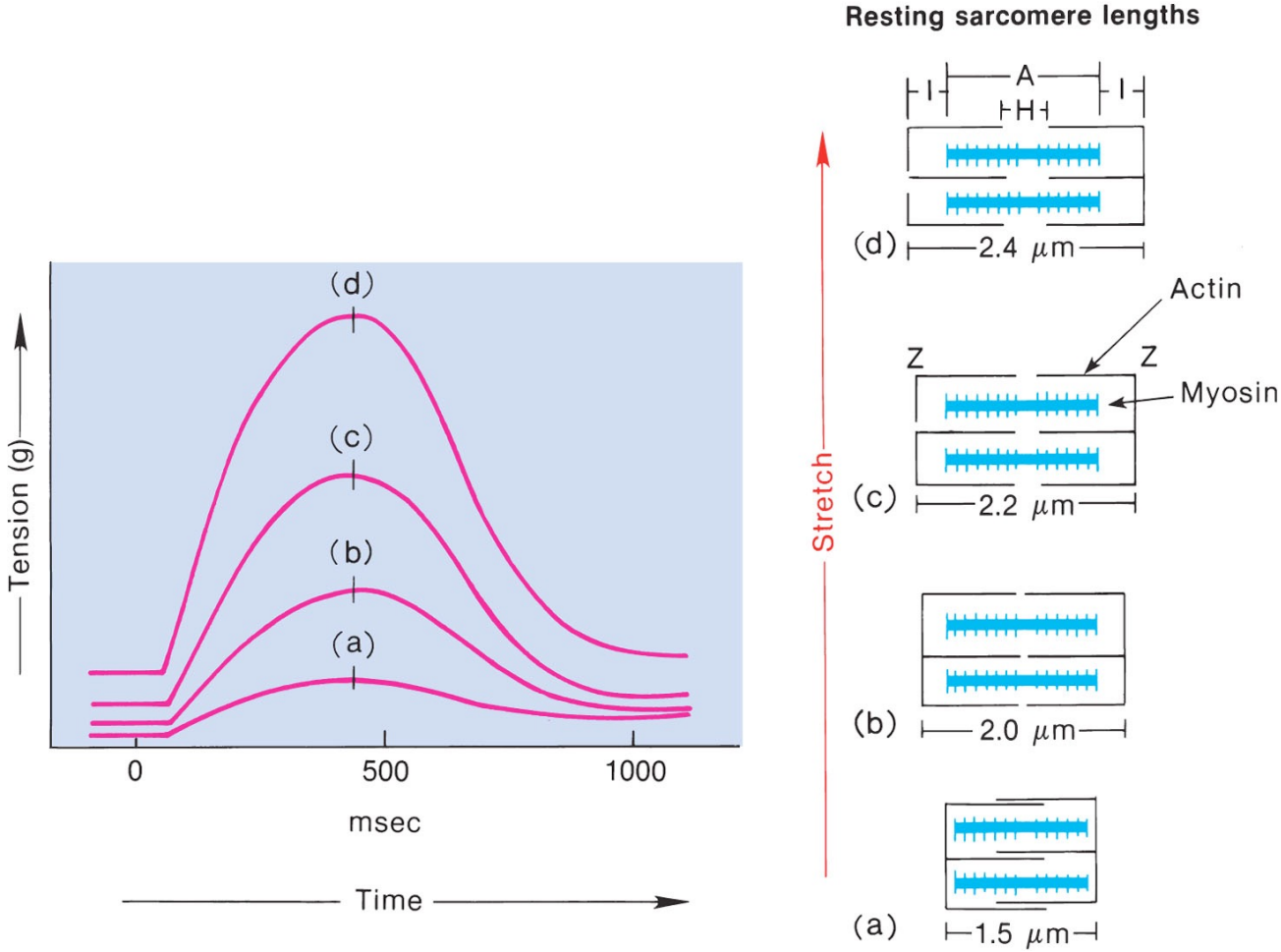


Figure 14.3

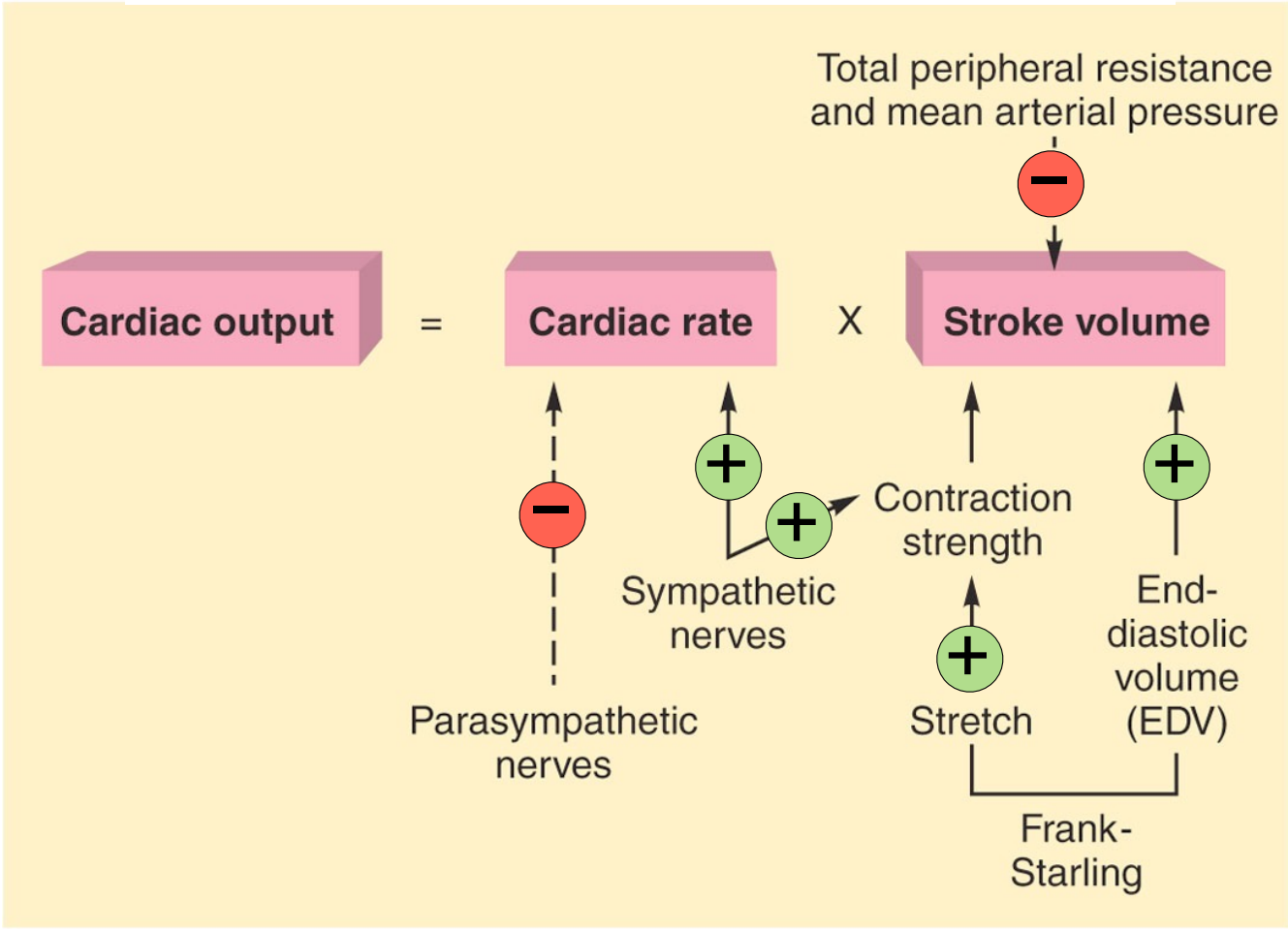

Total peripheral resistance and mean arterial pressure
Cardiac output
=
Cardiac rate
X
Stroke volume
Contraction strength
Sympathetic nerves
Parasympathetic nerves
End-diastolic volume (EDV)
Stretch
Frank-Starling

Figure 14.5

Venous Return

Return of blood to the heart via the veins.

Capacitance Vessels: Veins have higher **compliance** (floppy, can expand to hold more blood)

Two-thirds of total blood is in the veins, but venous pressure only 2 mmHg. Right atrium is at 0 mmHg.

Venous blood returns to heart by:

- pressure difference (2 vs 0 mmHg)
- sympathetic nerve activity contracts smooth muscle around veins
- skeletal muscle contractions squeezes blood through veins
- breathing causes negative thoracic/positive abdominal pressure to suck blood into chest

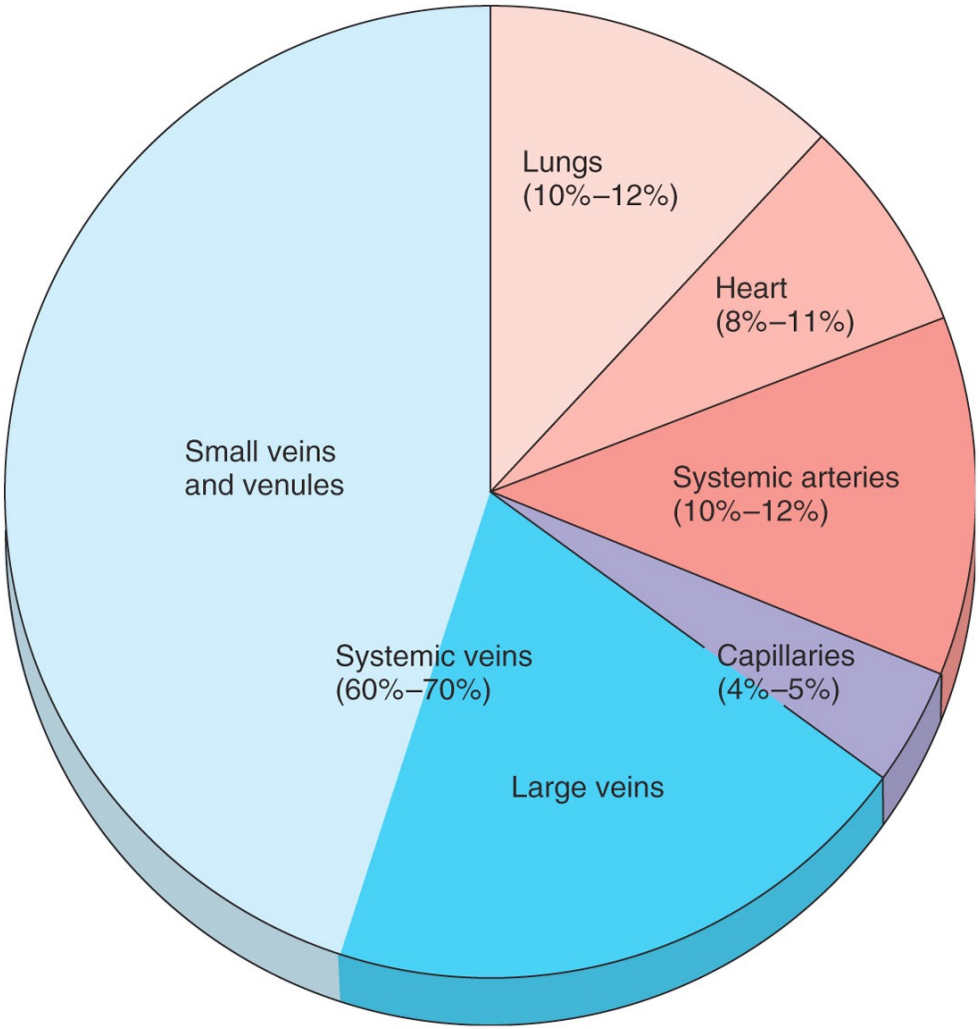

Lungs
(10%–12%)
Heart
(8%–11%)
Systemic arteries
(10%–12%)
Capillaries
(4%–5%)
Large veins
Systemic veins
(60%–70%)
Small veins
and venules

Figure 14.6

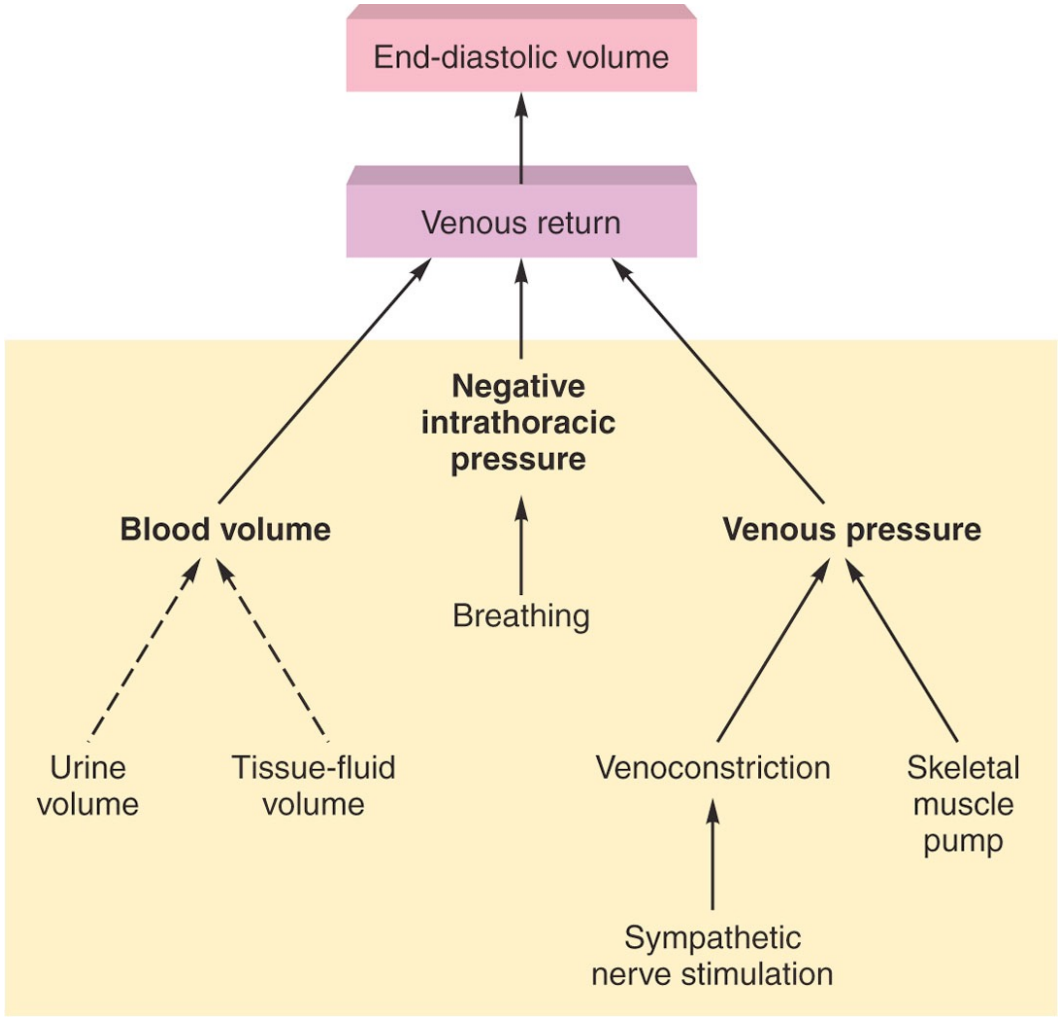

End-diastolic volume
Venous return
Negative intrathoracic pressure
Blood volume
Venous pressure
Breathing
Urine volume
Tissue-fluid volume
Venoconstriction
Skeletal muscle pump
Sympathetic nerve stimulation

Figure 14.7

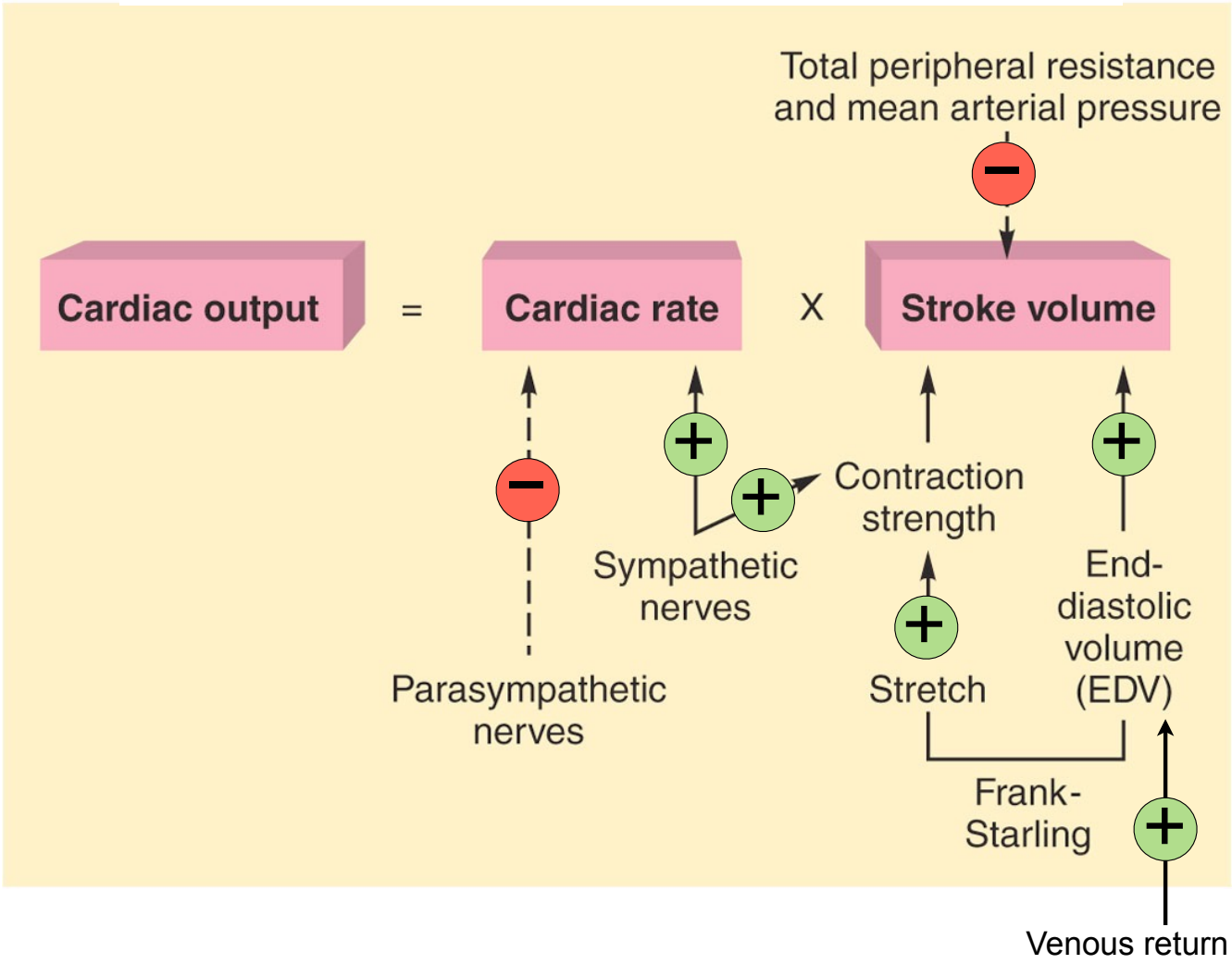

Total peripheral resistance and mean arterial pressure
Cardiac output
=
Cardiac rate
X
Stroke volume
Contraction strength
Sympathetic nerves
Parasympathetic nerves
End-diastolic volume (EDV)
Stretch
Frank-Starling
Venous return

Figure 14.5

Cardiac Output and Blood Pressure

How to Measure Blood Pressure
Contribution of vascular resistance to blood pressure

Cardiovascular Response to Exercise

Changes in Blood Flow
Increase Cardiac Output to
maintain BP, increase heart rate, increase stroke volume

Cardiac Output and Blood Pressure

Blood flow = ΔP / resistance
cardiac output = MAP / TPR
MAP = mean arterial pressure; TPR = total peripheral resistance.

Changes in cardiovascular function must be caused by changes in these 3 variables.

Note the similarity to electrical current equation: I = V / R

Arterial Blood Pressure is measured with a pressure cuff and **sphygmomanometer**.
1st Korotkoff sound occurs at systolic pressure, 2nd sound at diasystolic pressure.

Blood pressure is highest in aorta and large arteries; drops off in smaller arteries and capillaries because of increased resistance to blood flow.
(high on one side of resistance, low on the other side)

Blood flow = ΔP / resistance

cardiac output = MAP / TPR

(I = V/R)

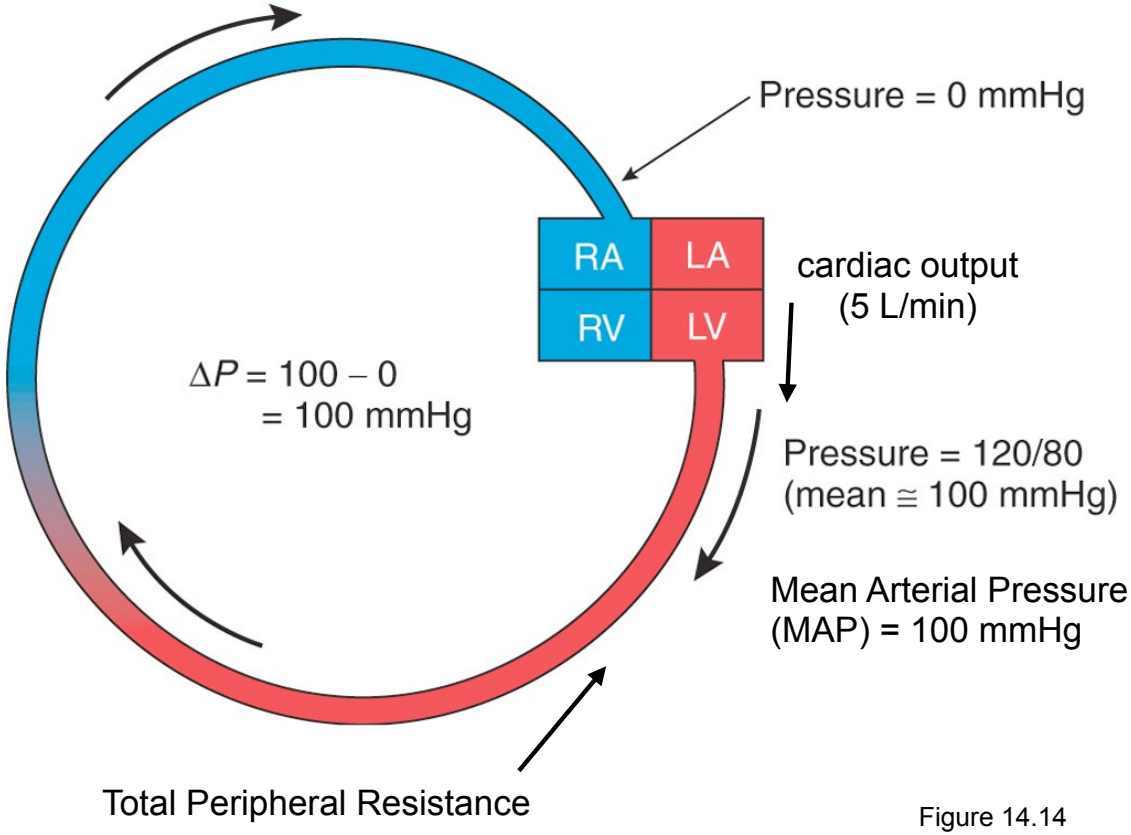


Figure 14.14

Resistance of Blood Vessels

resistance = (length of vessel) x (viscosity of blood) / (radius of vessel)^4

note power of 4!

Smaller, longer vessels have greater resistance.

Wider vessels have lower resistance.

Vasoconstriction:
Regulation of radius of blood vessels occurs in small arteries & arterioles.
Vasoconstriction will decrease radius, raising resistance.
Vasodilation will increase radius, lowering resistance.

Capillaries
Capillaries have very high surface area & very small radius, thus have high resistance. Blood pressures drops precipitously across capillary beds.

Blood flow to different capillary beds in different organs is regulated by vasoconstriction/dilation of arterioles. Blood is **shunted** through **arteriovenous anastomoses** (short circuit from artery to veins, bypassing capillaries).

Blood flow = ΔP / resistance

cardiac output = MAP / TPR

(I = V/R)

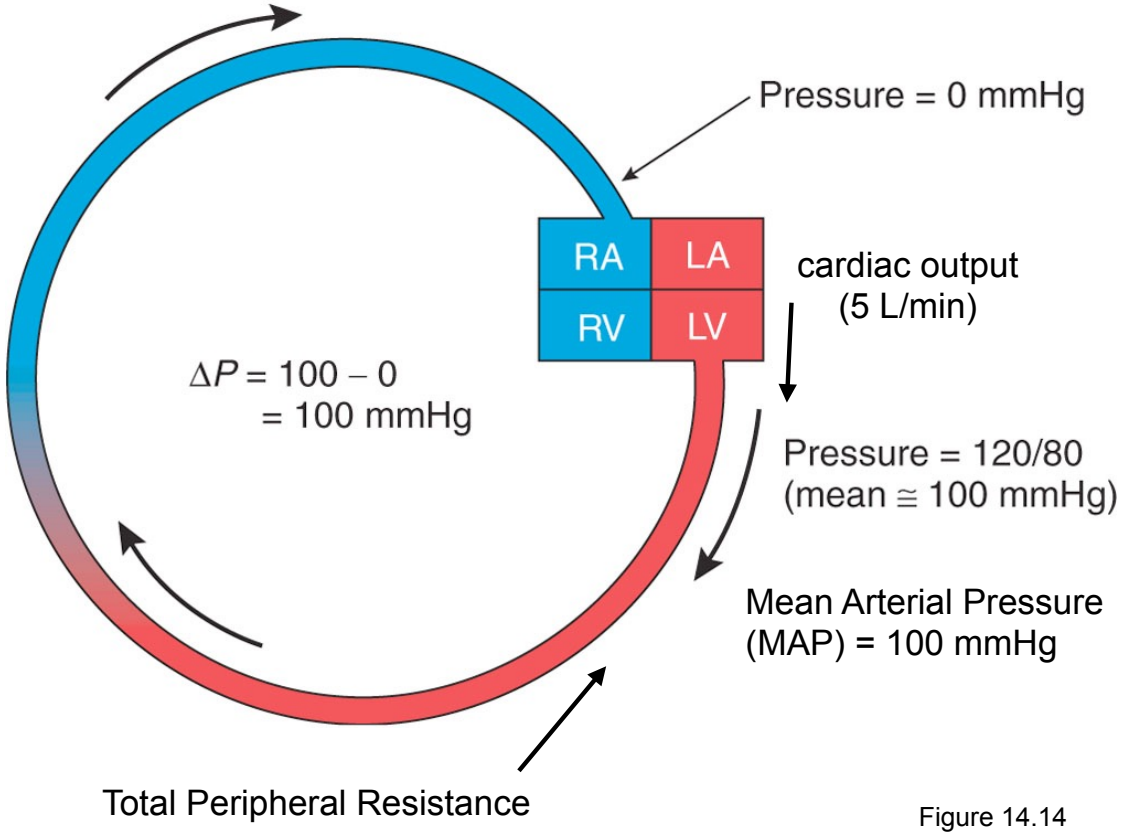
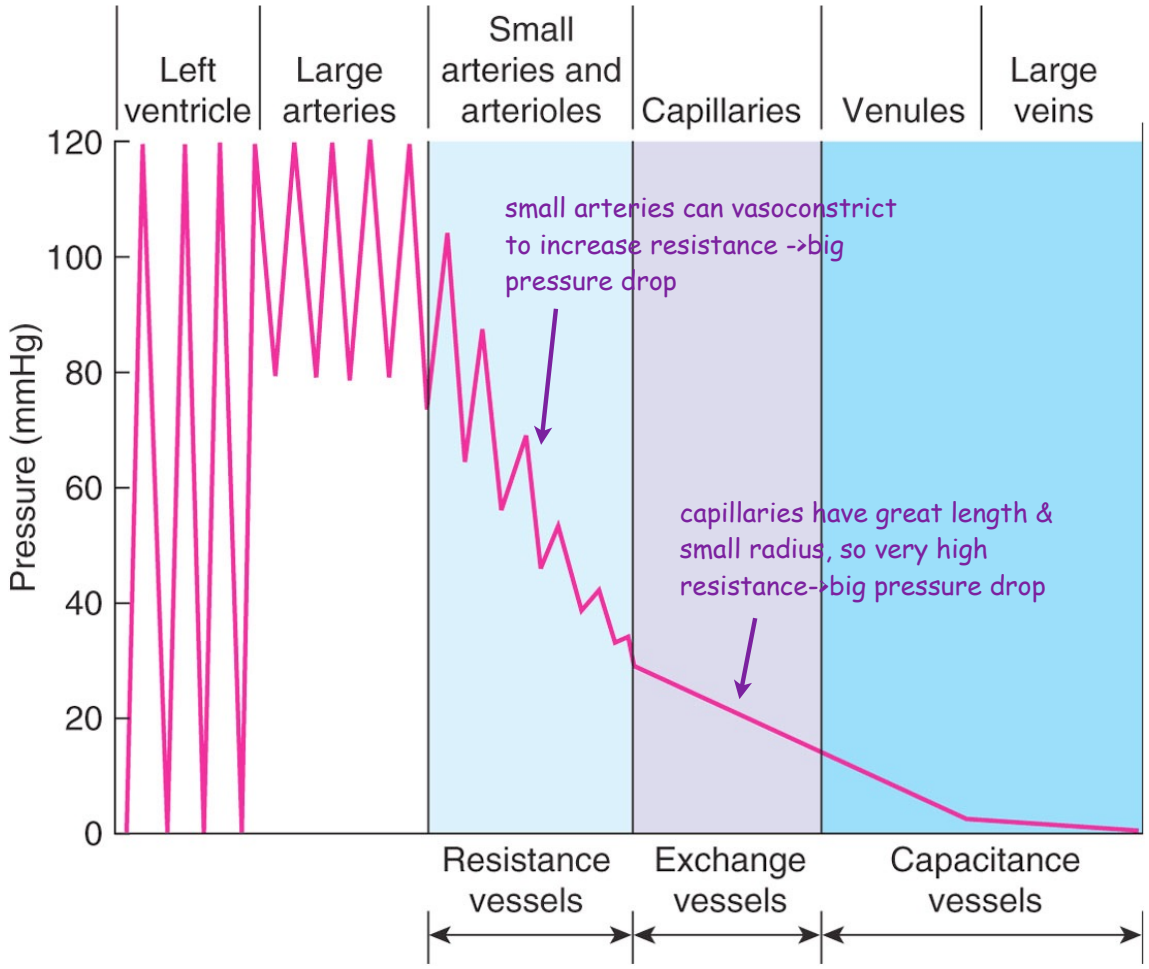


Figure 14.14

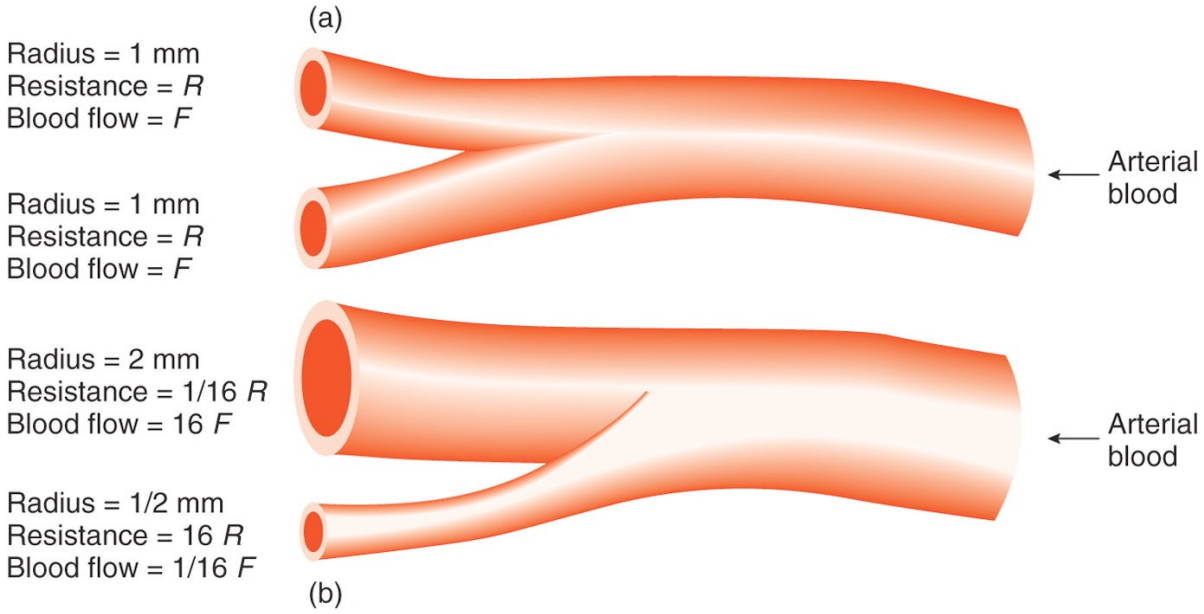
Figure 14.16


Left ventricle
Large arteries
Small arteries and arterioles
Capillaries
Venules
Large veins
120
100
80
60
40
20
0
Pressure (mmHg)
small arteries can vasoconstrict to increase resistance ->big pressure drop
capillaries have great length & small radius, so very high resistance->big pressure drop
Resistance vessels
Exchange vessels
Capacitance vessels

Smaller, longer vessels have greater resistance

$$\text{resistance} = \frac{\text{length x viscosity}}{\text{radius}^4}$$

So if a vessel is 1/2 the radius, the resistance increases by $(1/2)^4 = 16$ fold.
Therefore capillaries have greater resistance, and vasoconstriction increases resistance.



(length of vessels & viscosity of blood are constant,
but radius of vessels can be altered by vasodilation or vasoconstriction)

Figure 14.15

Vasoconstriction elevates blood pressure

Blood flow = ΔP / resistance

if resistance increases, ΔP has to increase to keep blood flow constant

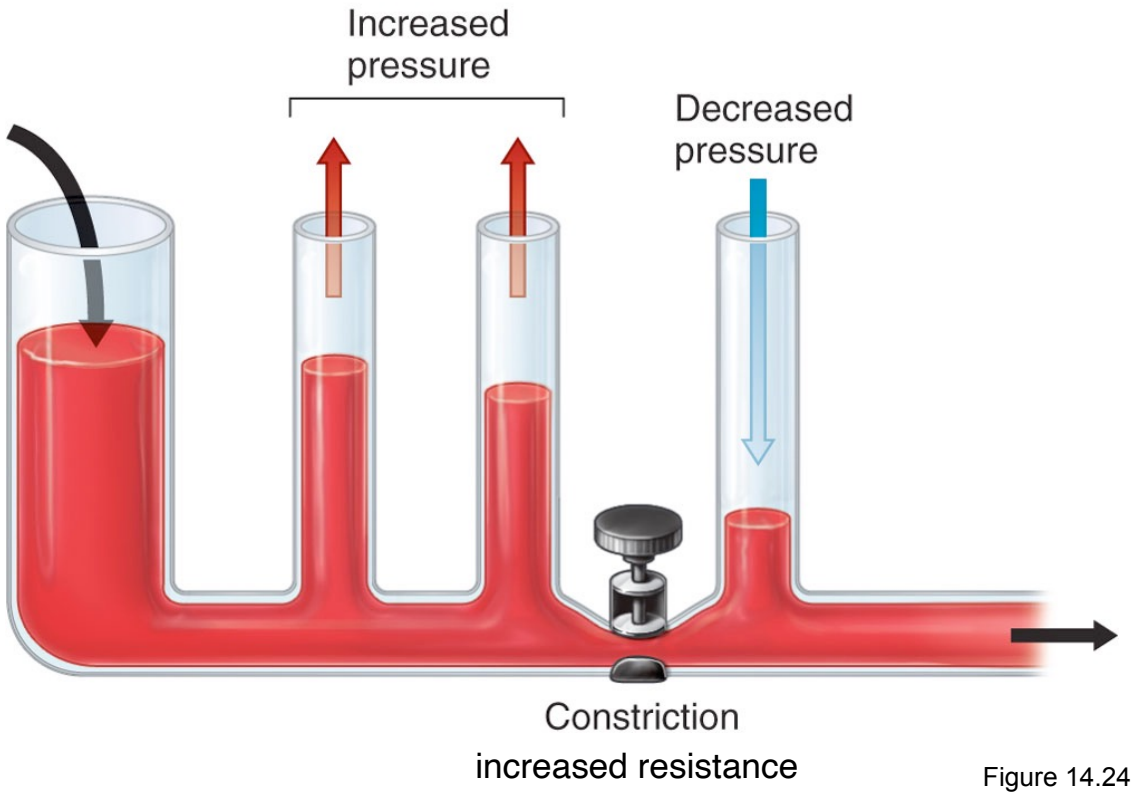


Figure 14.24

Can change Blood Pressure by changing Resistance

cardiac output = MAP / TPR

MAP = cardiac output x TPR

⬆resistance -> ⬆ Mean Arterial Pressure

⬇resistance -> ⬇ Mean Arterial Pressure

resistance = (length of vessel) x (viscosity of blood) / (radius of vessel)^4

length of vessels & viscosity of blood are constant, so can't change MAP that way.

but radius of vessels can be altered by **vasodilation** or **vasoconstriction**

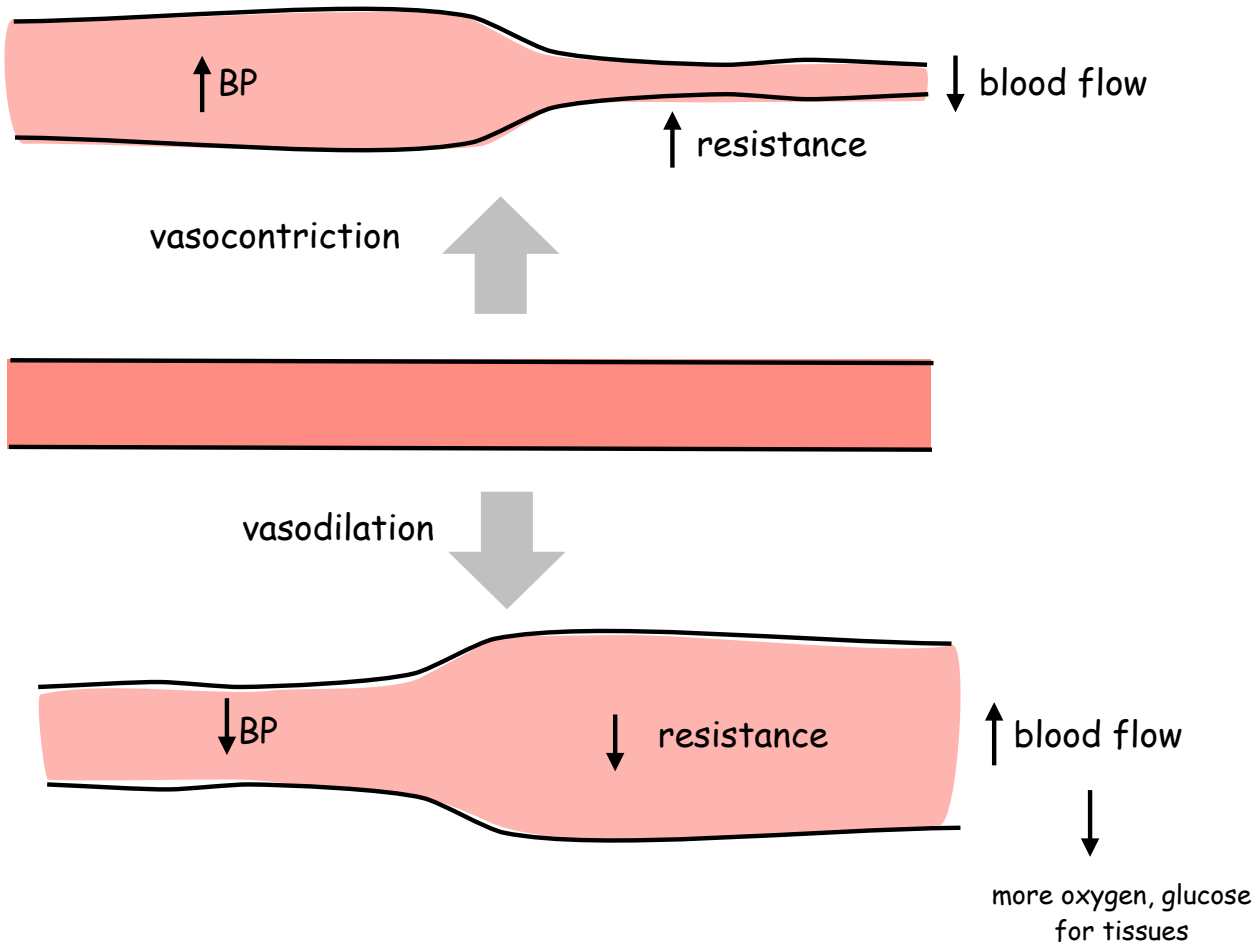
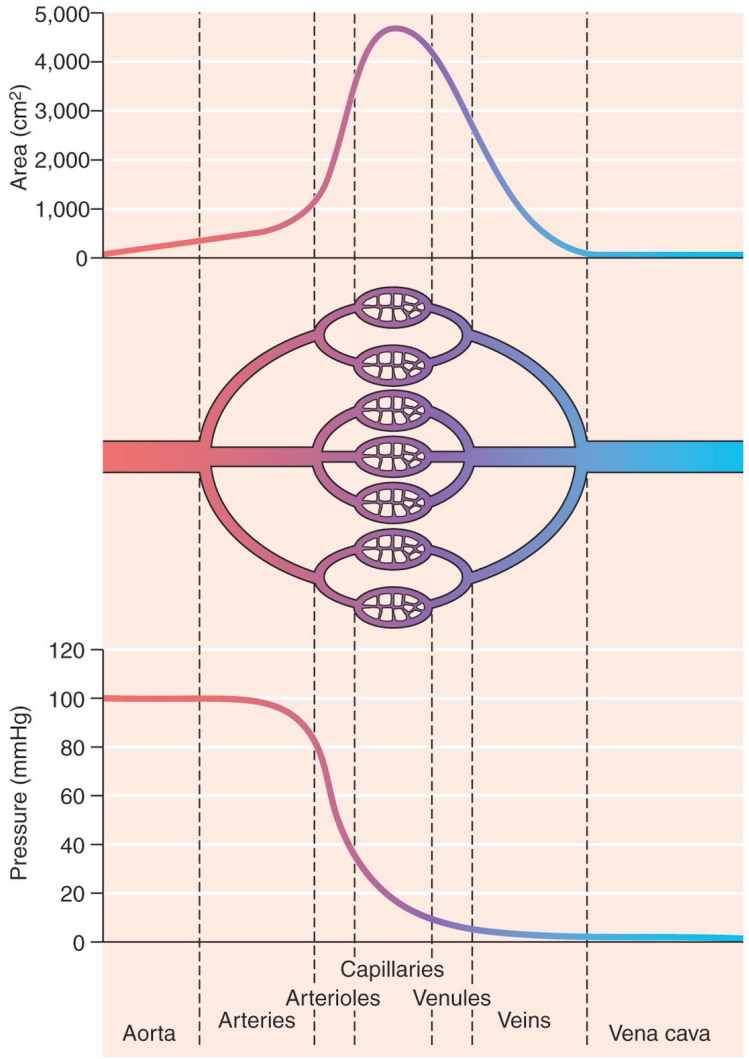

BP
blood flow
resistance
vasocontriction
vasodilation
BP
resistance
blood flow
more oxygen, glucose
for tissues

Figure 14.25

Increased area of capillaries
-> long length, small radius
-> large resistance
-> drop in pressure

↑ surface area of capillaries helps promote diffusion & exchange with tissues



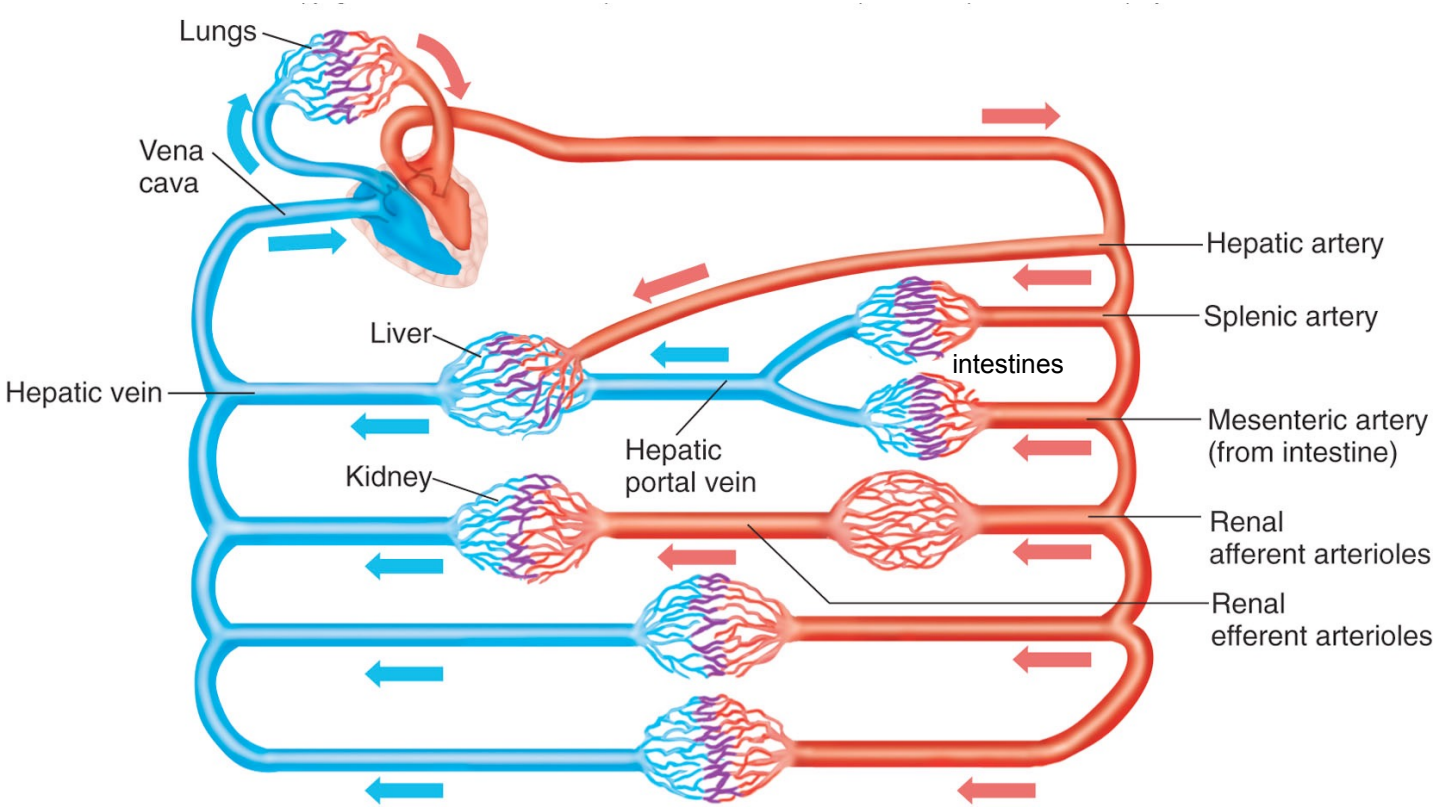
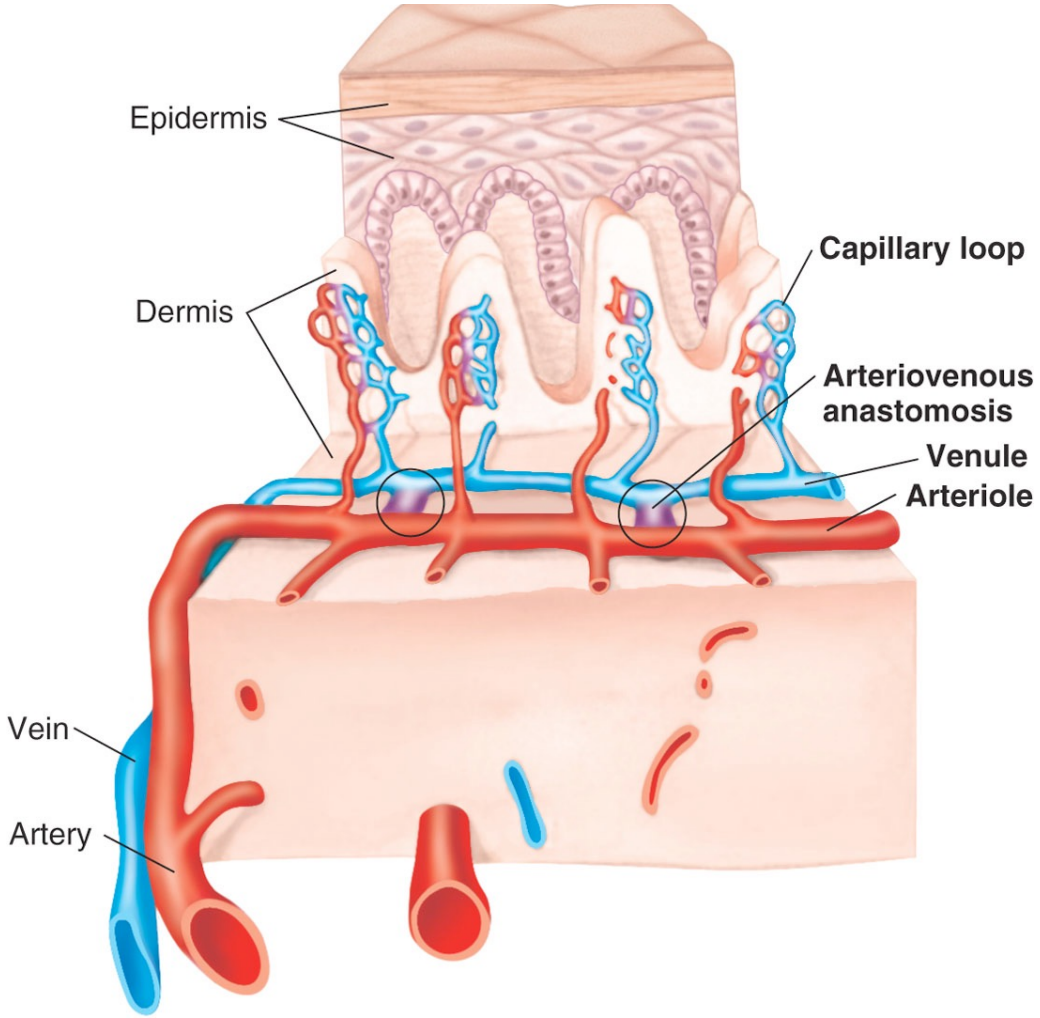

Lungs
Vena cava
Hepatic artery
Splenic artery
Liver
intestines
Hepatic vein
Mesenteric artery (from intestine)
Hepatic portal vein
Kidney
Renal afferent arterioles
Renal efferent arterioles
other capillaries, e.g. skeletal muscle

Figure 14.17

Figure 14.23


Epidermis
Dermis
Capillary loop
Arteriovenous anastomosis
Venule
Arteriole
Vein
Artery

Cardiovascular Responses to Exercise:

Problem:
Need to increase delivery of oxygen, glucose to skeletal muscle during exercise.

Solution:
1) Increase cardiac output so more oxygen can be delivered
2) re-route blood to target heart & skeletal muscle

Implementation:
- decrease blood flow to some organs by vasoconstriction
- increase blood flow to heart & skeletal muscle by vasodilation
- increase cardiac output by increasing heart rate & stroke volume

Regulatory Mechanisms:
- Decreased parasympathetic, increased sympathetic outflow -> heart, arterioles
- Epinephrine release from adrenal medulla -> heart, arterioles
- Metabolites released from muscle -> vasodilation (intrinsic factors).
- Increased breathing, muscle movements -> increased venous return.

Table 14.3

Table 14.3 | Estimated Distribution of the Cardiac Output at Rest

Organs	Blood Flow	
	Milliliters per Minute	Percent Total
Gastrointestinal tract and liver	1,400	24
Kidneys	1,100	19
Brain	750	13
Heart	250	4
Skeletal muscles	1,200	21
Skin	500	9
Other organs	600	10
Total organs	5,800	100

What is cardiac output (ml/min) through lungs at rest?

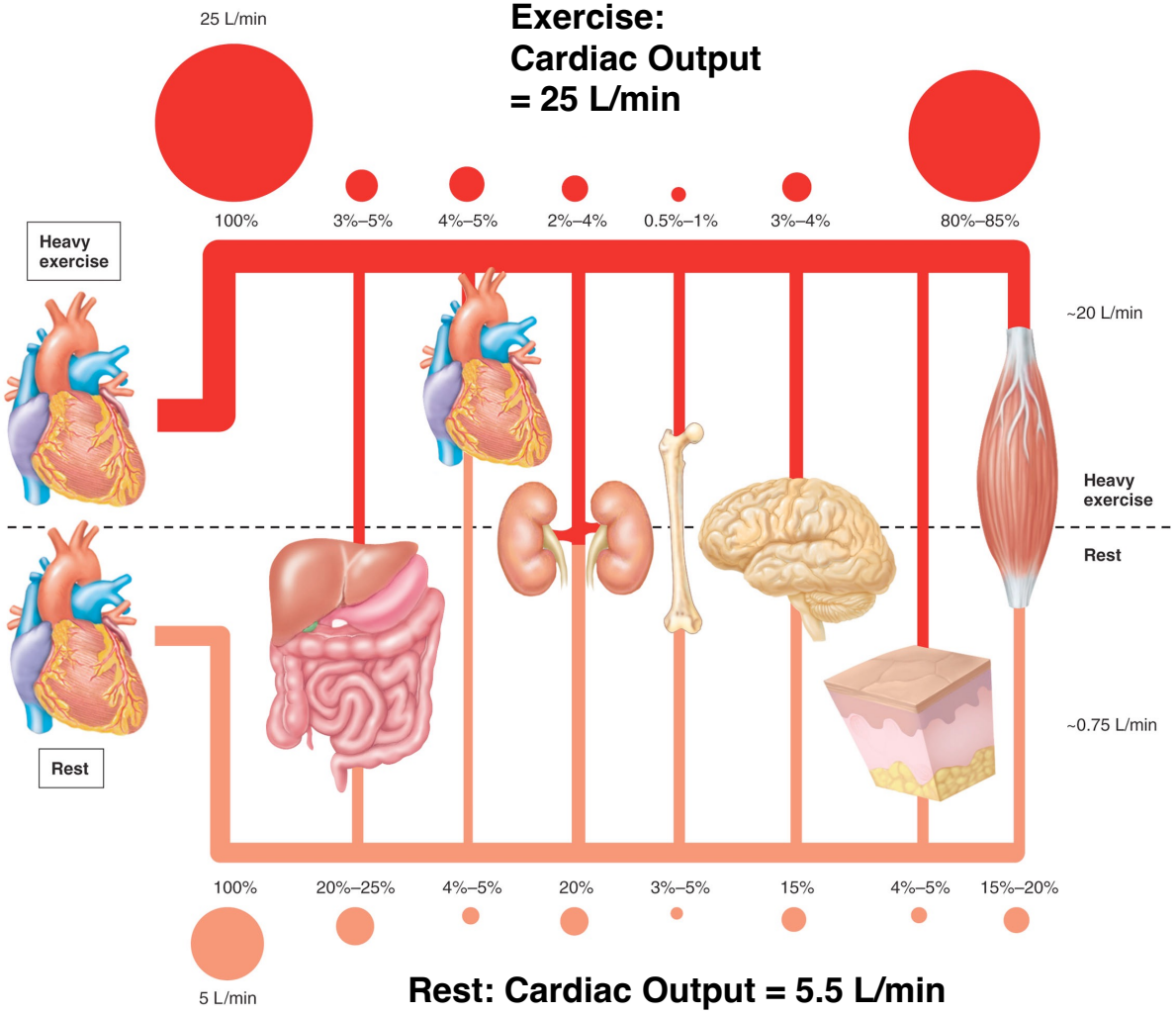

Exercise:
Cardiac Output
= 25 L/min
25 L/min
100%
3%–5%
4%–5%
2%–4%
0.5%–1%
3%–4%
80%–85%
Heavy exercise
~20 L/min
Heavy exercise
Rest
~0.75 L/min
Rest
100%
20%–25%
4%–5%
20%
3%–5%
15%
4%–5%
15%–20%
5 L/min
Rest: Cardiac Output = 5.5 L/min

Figure 14.20

Exercise increases **cardiac output** and causes **redistribution** of blood flow

Organs	Blood Flow (ml/min)		
	At Rest	Exercising	
Gastrointestinal tract and liver	1,400	750	constriction
Kidneys	1,100	500	constriction
Brain	750	750	
Heart	250	1000	dilation
Skeletal muscles	1,200	21,250	dilation
Skin	500	0	constriction
Other organs	600	750	
Total organs	5,800	25,000	
Cardiac Output:	5.8 L/min	25 L/min	

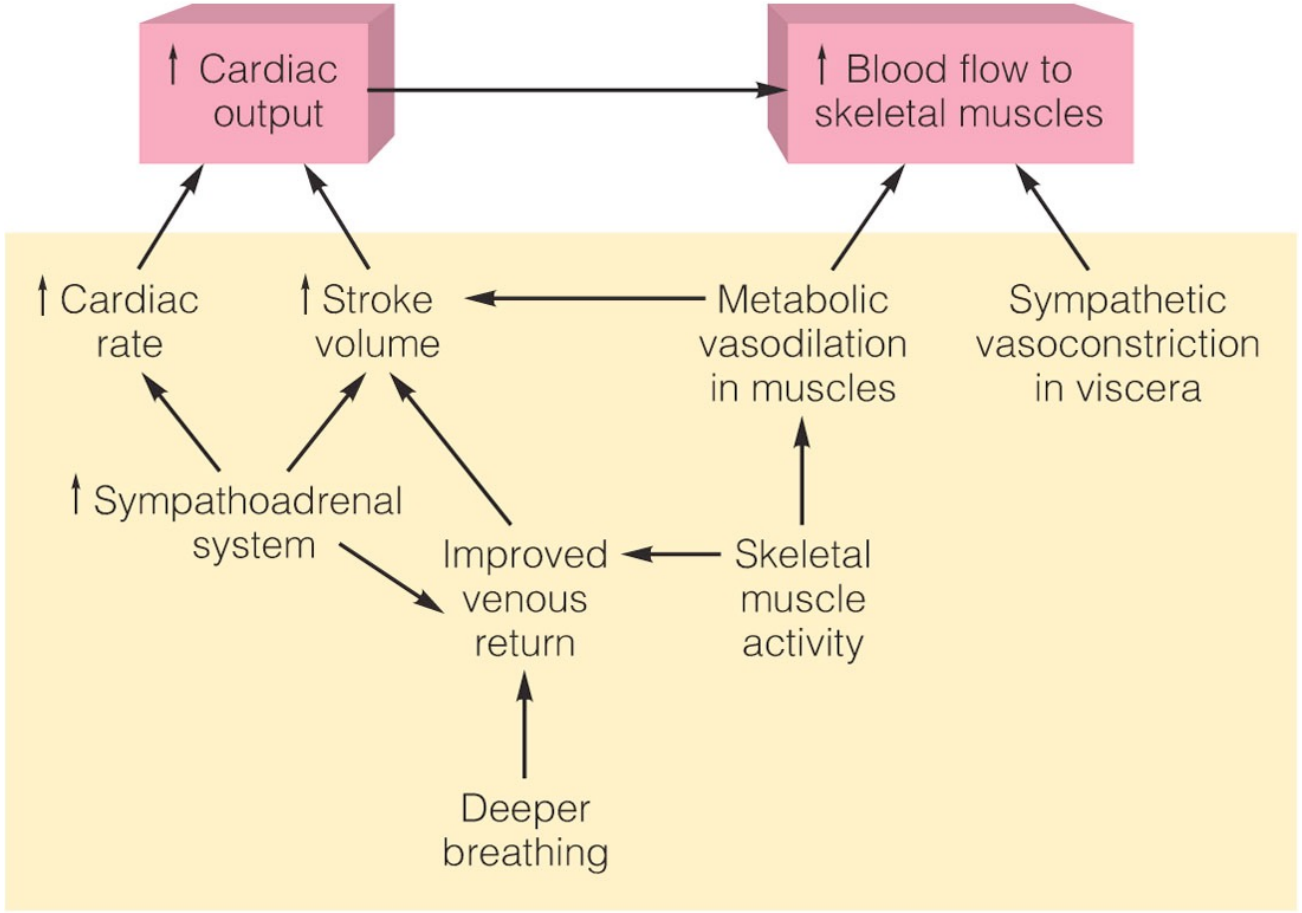
Blood flow = ΔP / resistance

to increase blood flow: increase ΔP and/or decrease resistance

↑ cardiac output at heart

↑ vasodilation at skeletal muscles

Figure 14.21


↑ Cardiac output
↑ Blood flow to skeletal muscles
↑ Cardiac rate
↑ Stroke volume
Metabolic vasodilation in muscles
Sympathetic vasoconstriction in viscera
↑ Sympathoadrenal system
Improved venous return
Skeletal muscle activity
Deeper breathing

Vasodilation of Arterioles in Skeletal muscle during exercise

Table 14.5 | Changes in Skeletal Muscle Blood Flow Under Conditions of Rest and Exercise

Condition	Blood Flow (ml/min)	Mechanism
Rest	1,000	High adrenergic sympathetic stimulation of vascular alpha receptors, causing vasoconstriction
Beginning exercise	Increased	Dilation of arterioles in skeletal muscles due to cholinergic sympathetic nerve activity and stimulation of beta-adrenergic receptors by the hormone epinephrine
Heavy exercise	20,000	Fall in alpha-adrenergic activity Increased cholinergic sympathetic activity Increased metabolic rate of exercising muscles, producing intrinsic vasodilation

Table 14.5

Increase of Cardiac Output with Exercise

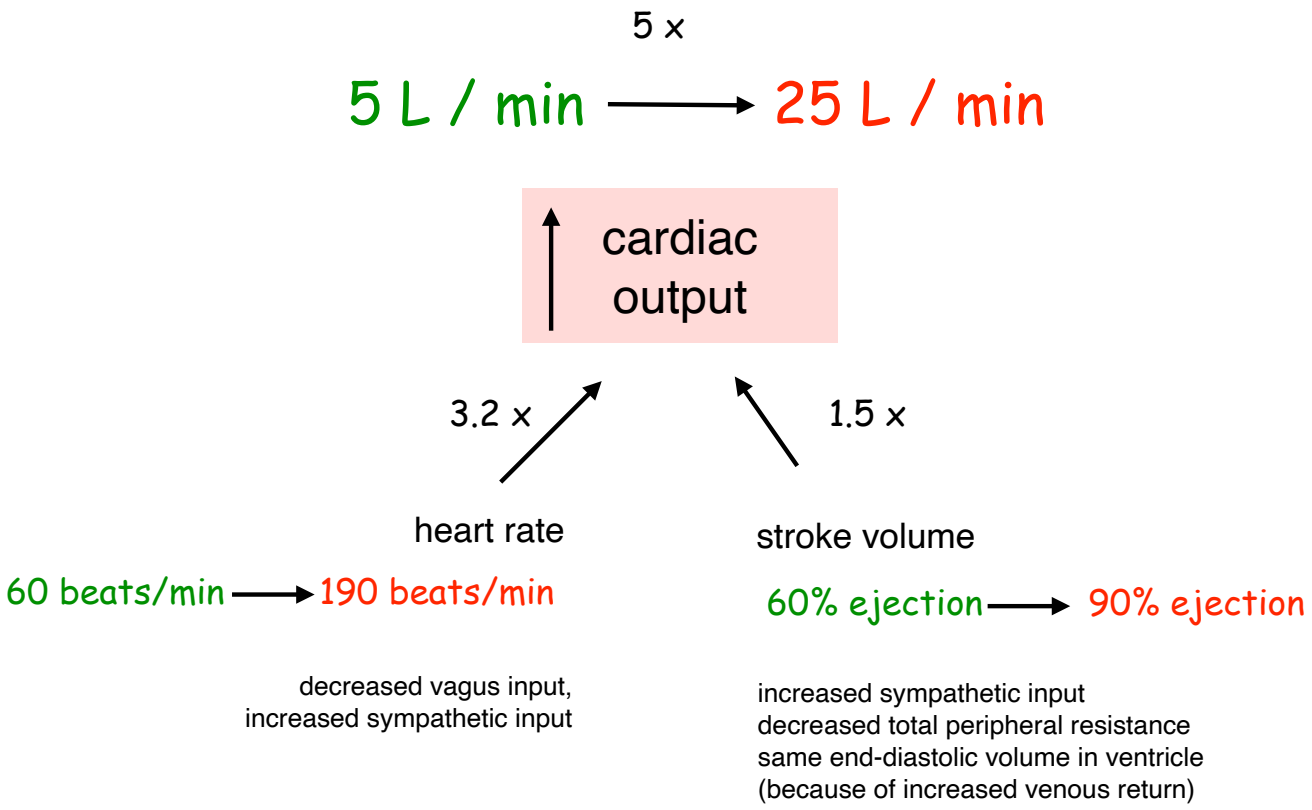


Table 14.7 | Cardiovascular Changes During Moderate Exercise

Variable	Change	Mechanisms
Cardiac output	Increased	Increased cardiac rate and stroke volume
Cardiac rate	Increased	Increased sympathetic nerve activity; decreased activity of the vagus nerve
Stroke volume	Increased	Increased myocardial contractility due to stimulation by sympathoadrenal system; decreased total peripheral resistance
Total peripheral resistance	Decreased	Vasodilation of arterioles in skeletal muscles (and in skin when thermoregulatory adjustments are needed)
Arterial blood pressure	Increased	Increased systolic and pulse pressure due primarily to increased cardiac output; diastolic pressure rises less due to decreased total peripheral resistance
End-diastolic volume	Unchanged	Decreased filling time at high cardiac rates is compensated for by increased venous pressure, increased activity of the skeletal muscle pump, and decreased intrathoracic pressure aiding the venous return
Blood flow to heart and muscles	Increased	Increased muscle metabolism produces intrinsic vasodilation; aided by increased cardiac output and increased vascular resistance in visceral organs
Blood flow to visceral organs	Decreased	Vasoconstriction in digestive tract, liver, and kidneys due to sympathetic nerve stimulation
Blood flow to skin	Increased	Metabolic heat produced by exercising muscles produces reflex (involving hypothalamus) that reduces sympathetic constriction of arteriovenous shunts and arterioles
Blood flow to brain	Unchanged	Autoregulation of cerebral vessels, which maintains constant cerebral blood flow despite increased arterial blood pressure