Vert Phys PCB3743

Cardiac Output 1
Fox Chapter 14 part 1

© T. Houpt, Ph.D.

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

- **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)

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

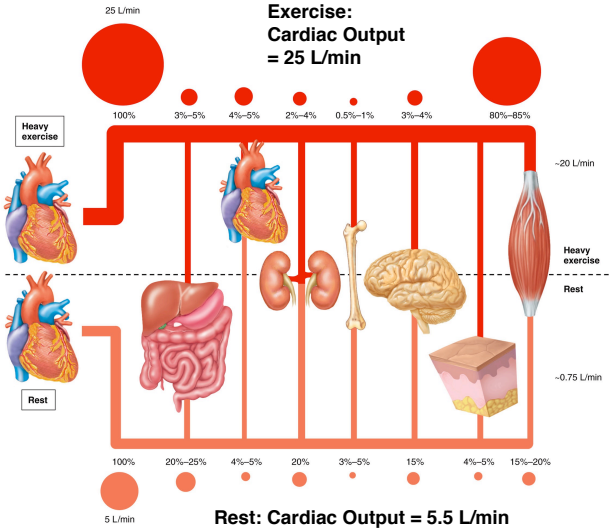


Figure 14.20

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.

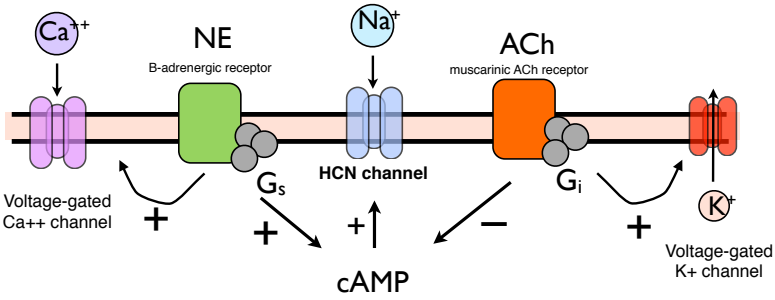
Regulation of Cardiac Rate

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?

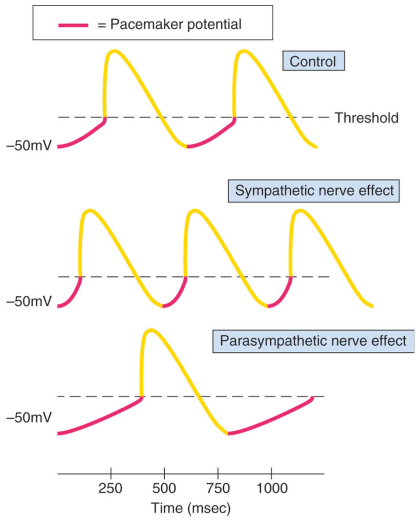


Figure 14.1

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

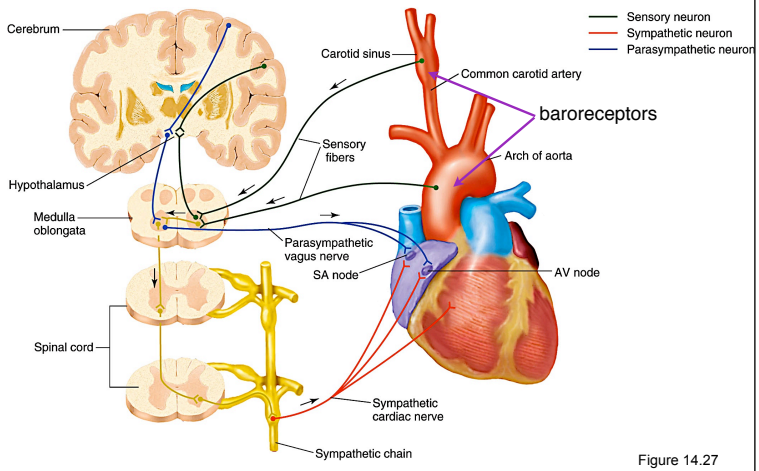
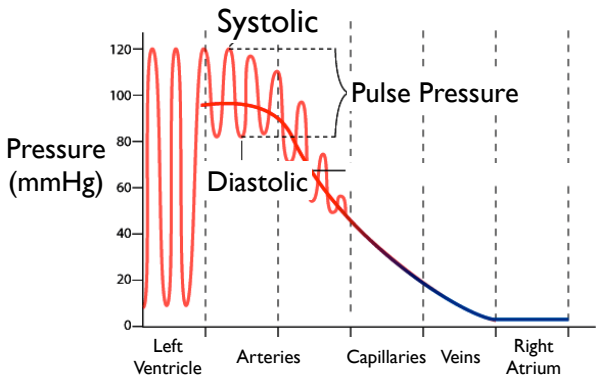


Figure 14.27

Blood Pressure:

Arterial Pressure > Venous Pressure



http://www.adinstruments.com

Baroreceptor Sensory Neurons report Blood Pressure

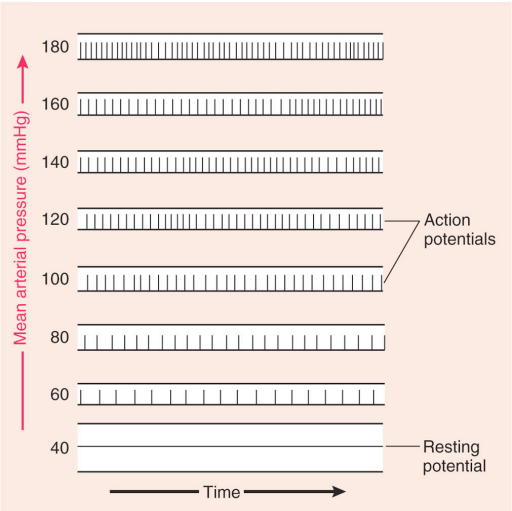


Figure 14.26

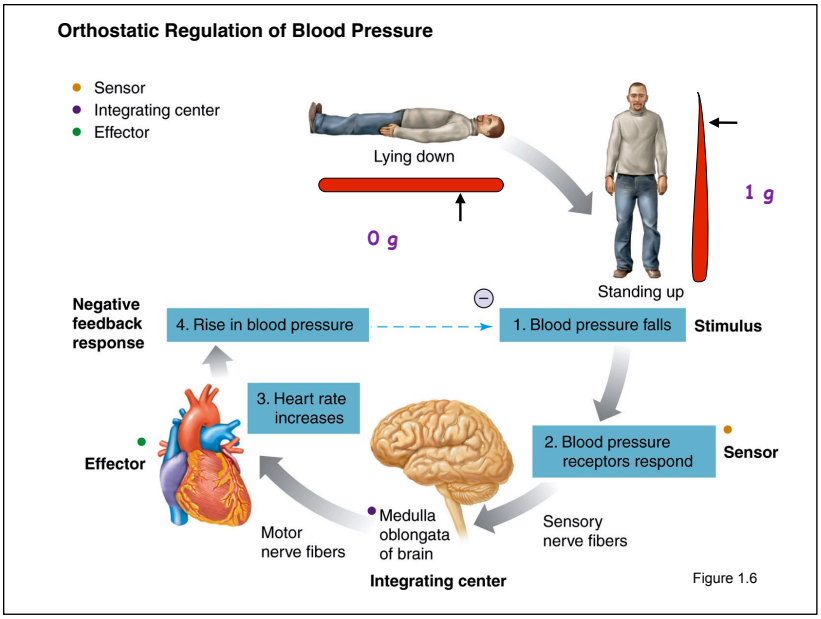


Figure 1.6

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

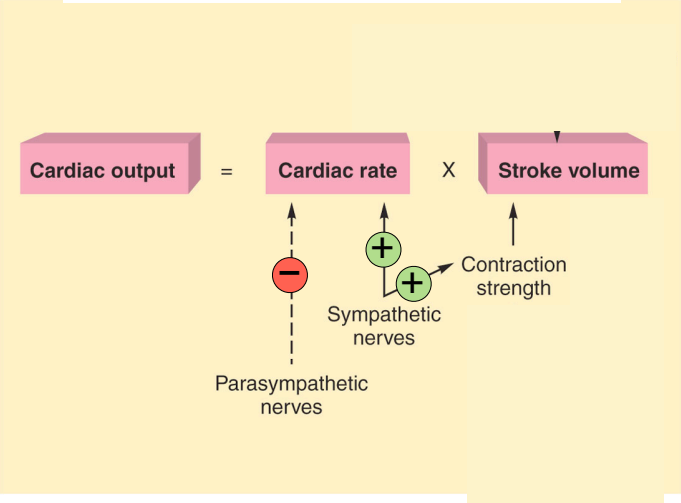


Figure 14.5

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**

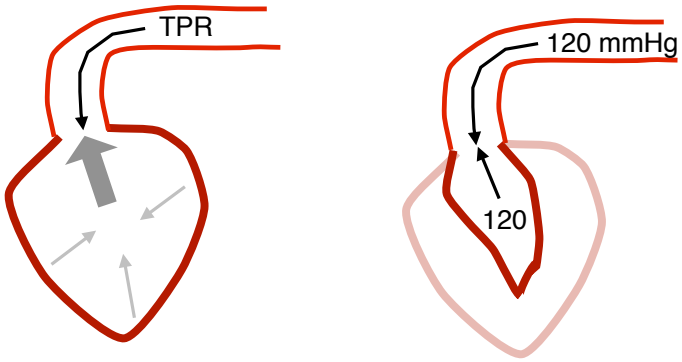
(ml/beat) how much blood is ejected from each ventricle with each beat

total peripheral resistance

resistance of arteries to increased blood flow

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

Total peripheral resistance resists cardiac output



Vasoconstriction elevates blood pressure

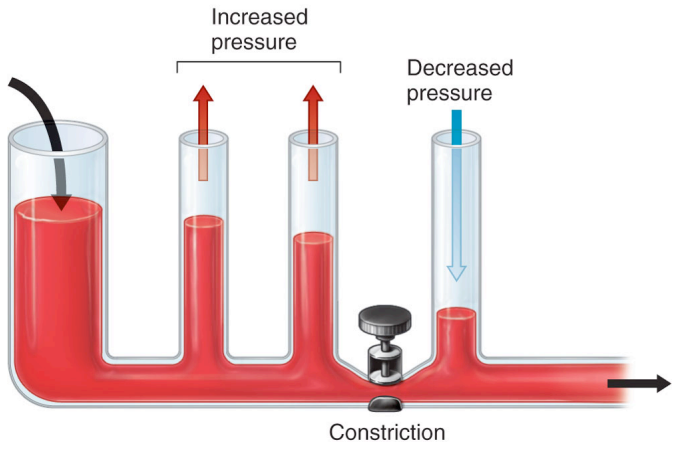
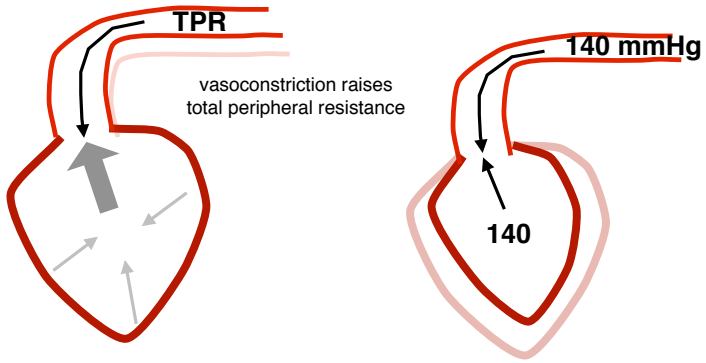


Figure 14.24

Total peripheral resistance resists cardiac output



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

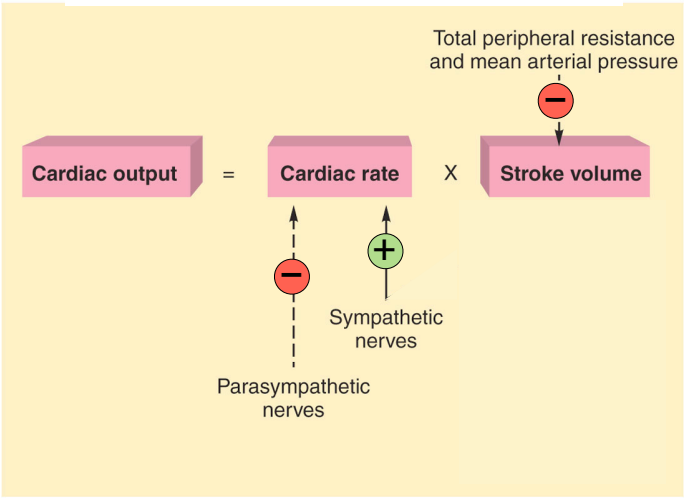


Figure 14.5

Frank Starling Law of the Heart

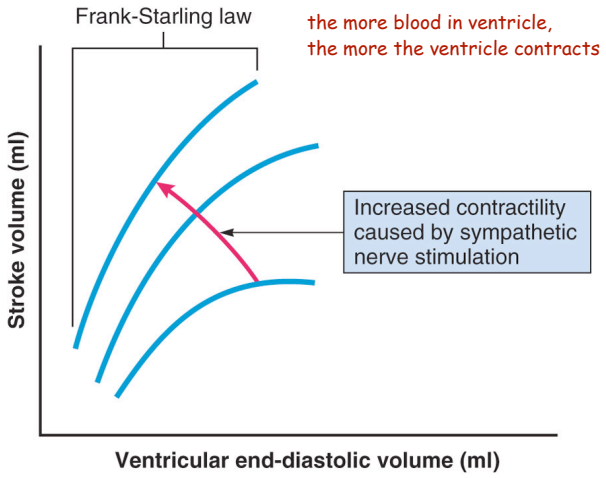
strength of contraction varies directly with end-diastolic volume (EDV)
- More blood in the ventricle stretches the relaxed cardiac muscle more.
- Less overlap of actin and myosin fibers -> allows formation of more cross bridges -> greater contractile strength

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



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

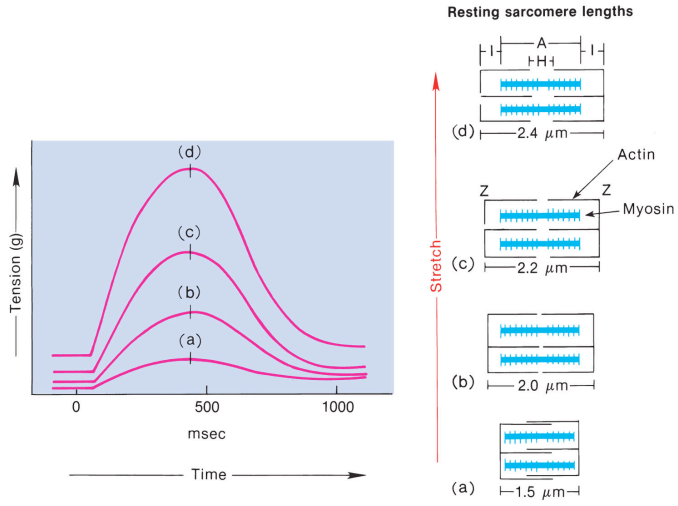


Figure 14.3

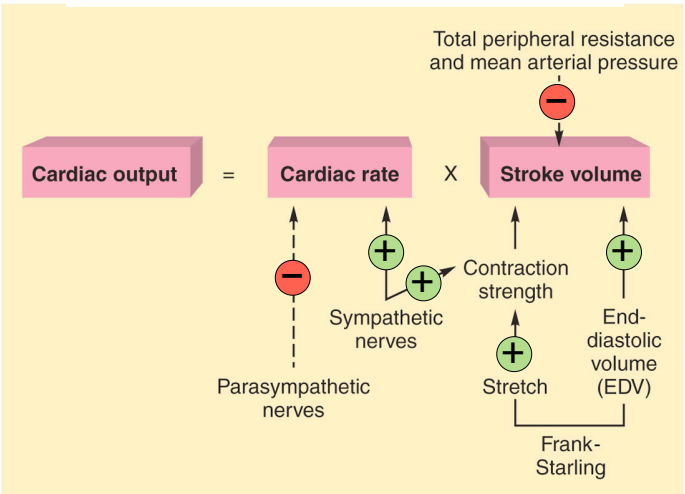


Figure 14.5

Venous Return

Return of blood to the heart via the veins.

Capacitance Vessels: Veins have highler **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

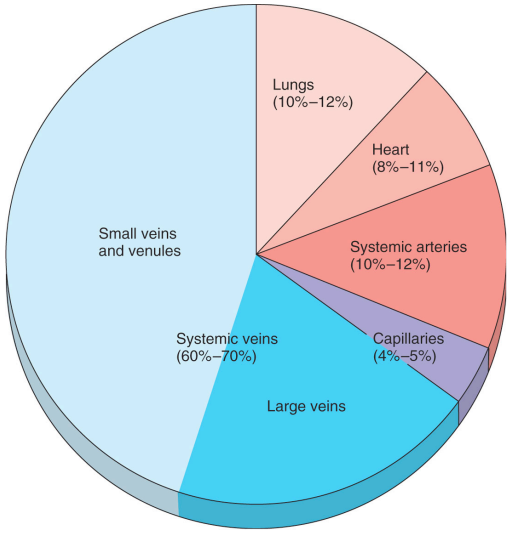


Figure 14.6

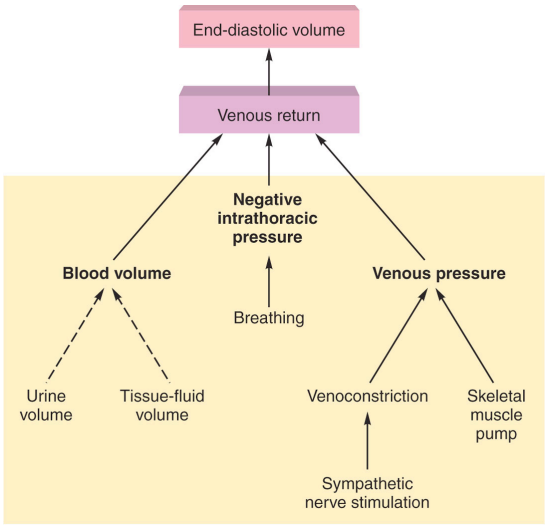


Figure 14.7

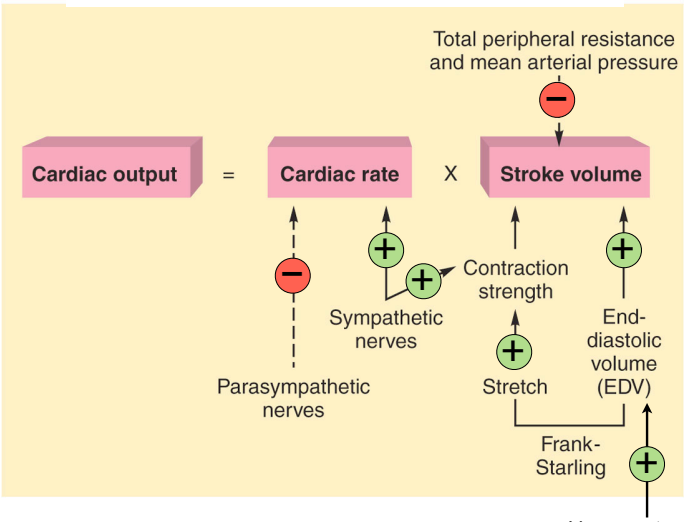


Figure 14.5

Capillary Exchange and Edema

Starling Forces

P_c = hydrostatic pressure in the capillary (pushing out of capillary)

π_i = osmotic pressure of interstitial fluid (pulling into interstitial fluid)

P_i = hydrostratic pressure of interstitial fluid (pushing out of intersitial fluid)

π_p = osmotic pressure of plasma (pulling into capillary)

Fluid Movement out of capillary = $(P_c + \pi_i) - (P_i + \pi_p)$

Filtration Pressue = $P_c - P_i$

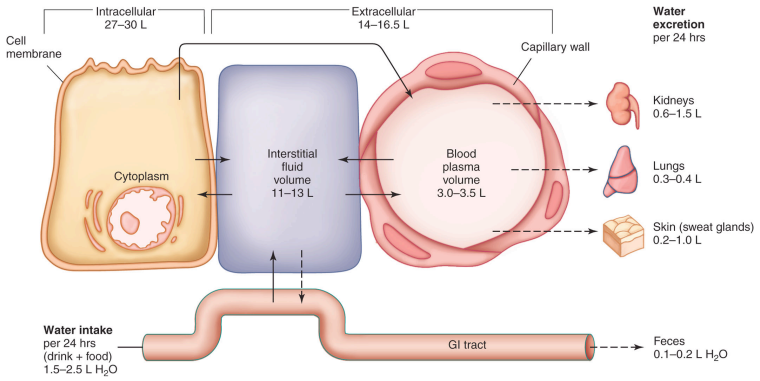
due to blood pressure in capillaries (37 - 17 mmHg) and hydrostatic pressure of intersitial fluid (varies by tissue); forces fluid through capillary pores and fenestra, carrying small molecules with the fluid (e.g. glucose).

Oncotic Pressure = $\pi_i - \pi_p$

due to osmotic pressure exerted by large proteins in the interstitial fluid (π_i) and large proteins in the plasma (π_p) that cannot pass through pores.

Only 85% of capillary fluid returns to capillary; remaining 15% of fluid returned by lymphatic vessels.

Figure 14.8



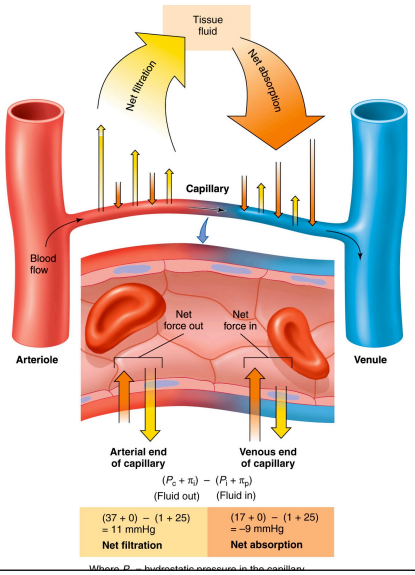


Figure 14.9

Lymphatic System (Chapter 13.8)

Parallel system of vessels filled with **lymph** that
- transport interstitial fluid back to veins
- transports absorbed fat from intestine to the blood
- provides immune cells (lymphocytes)

Lymphatic capillaries are closed end but very porous vessels in interstitial space. Pick up interstitial fluid, proteins, fats, white blood cells, microorganisms.

Capillaries merge to form **lymph ducts**, similar to veins with valves. Lymph is moved by **peristaltic contractions** of smooth muscle of lymph ducts.

Lymph filtered through l**ymph nodes**; immune cells phagocytose pathogens or respond to antigens.

Lymph is dumped into subclavian veins via **thoracic duct** and **right lymphatic duct**.

Figure 13.36

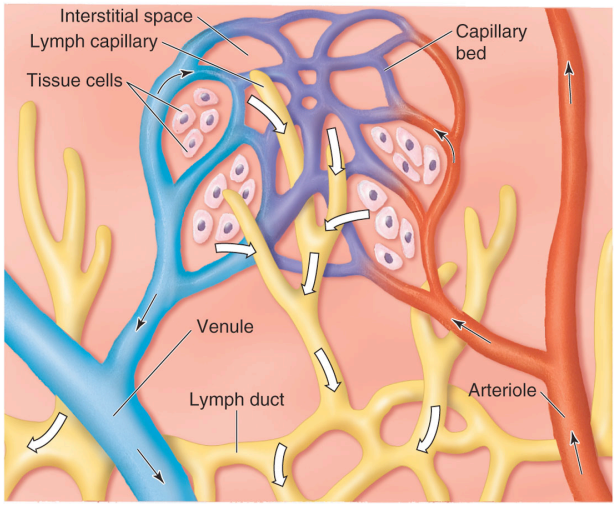
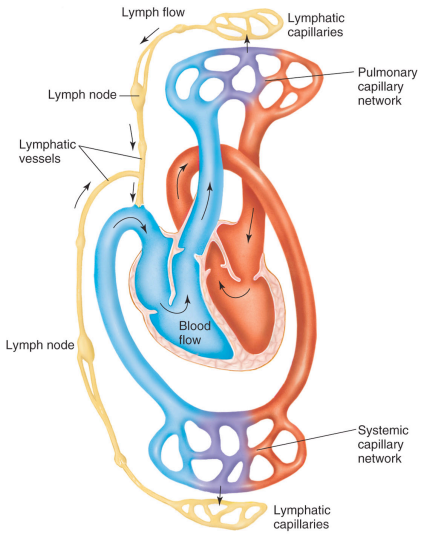


Figure 13.37



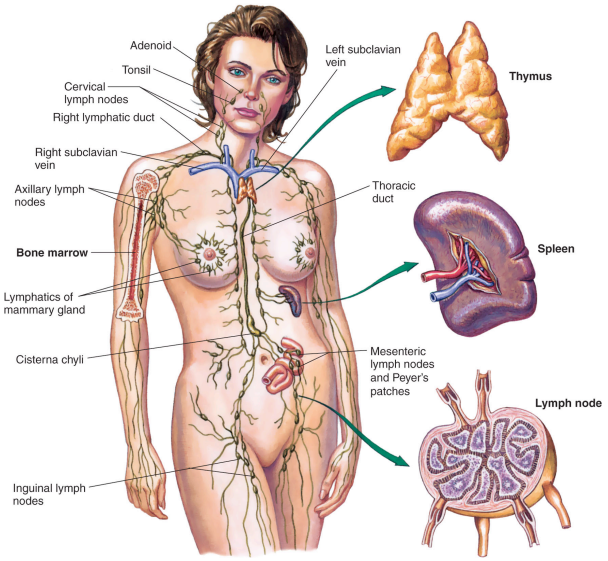


Figure 13.38

Edema = excessive interstitial fluid

due to imbalance of Starling Forces or lymphatic obstruction.

1. High arterial blood pressure -> $\mathbf{P_c}$ elevated -> increased filtration

2. Venous obstruction -> $\mathbf{P_c}$ elevated -> increased filtration
   e.g. phlebitis (clot in vein) or compression of veins

3. Leakage of plasma proteins into interstitial fluid -> $\boldsymbol{\pi_i}$ increased -> less osmotic flow back into capillaries
   e.g. inflammation & allergic reactions open up capillaries

4. decreased plasma proteins -> $\boldsymbol{\pi_p}$ decreased -> less osmotic flow back into capillaries
   e.g. liver disease (synthesis of proteins) or kidney disease (excretion of proteins)

5. Obstruction of lymphatic drainage -> excess intersititial fluid does not drain into lymph and veins
   e.g. parasite in elephantiasis blocks lymph vessels

Figure 14.10

From E.K. Markell and M. Vogue, Medical Parasitology, 6th edition, 1986, W.B. Saunders Company