Vert Phys PCB3743

# Blood Pressure
# Fox Chapter 14 part 2





**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

**Control of Blood Volume and Pressure**

Baroreceptors
Osmoreceptors
Endocrine Responses: ADH, Angiotensin II, ANP



## 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 )

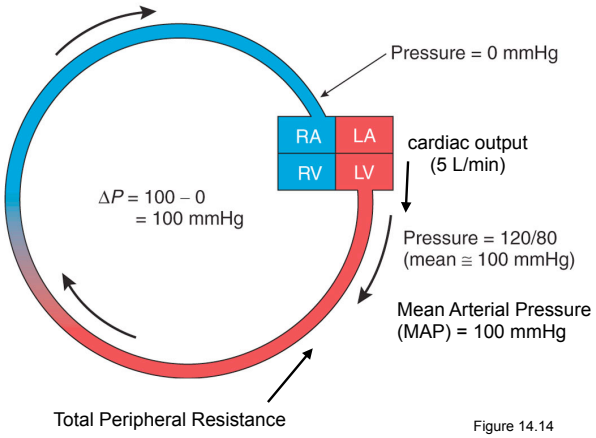


Figure 14.14

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Figure 14.29

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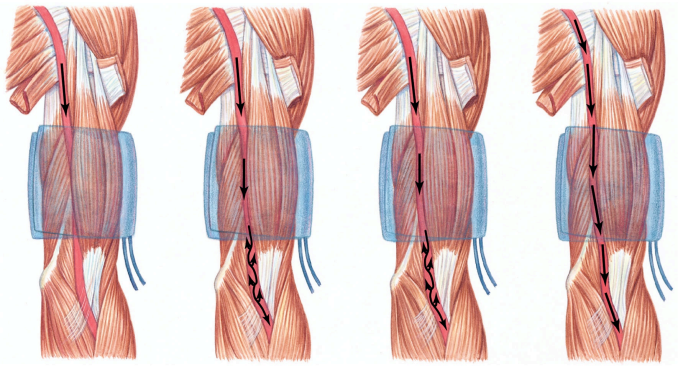
Pressure at which blood can **first** get past cuff (i.e. when blood is at highest pressure) gives systolic pressure

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

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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

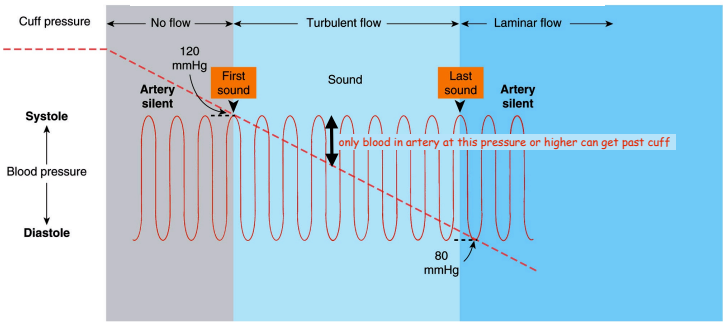


Figure 14.31

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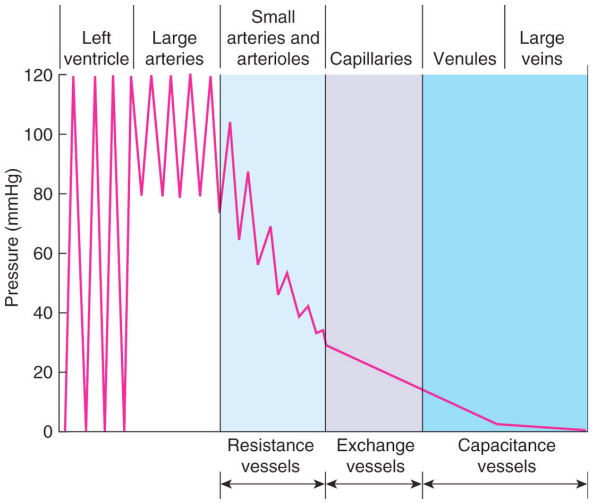
Figure 14.16

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## 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).

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**Blood flow = ΔP / resistance**

**cardiac output = MAP / TPR**

( I = V/R )

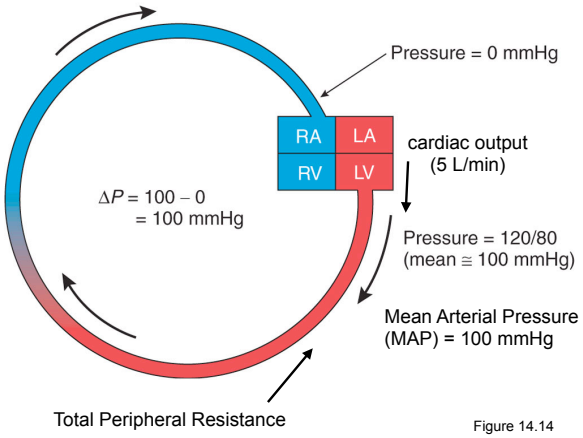


Figure 14.14

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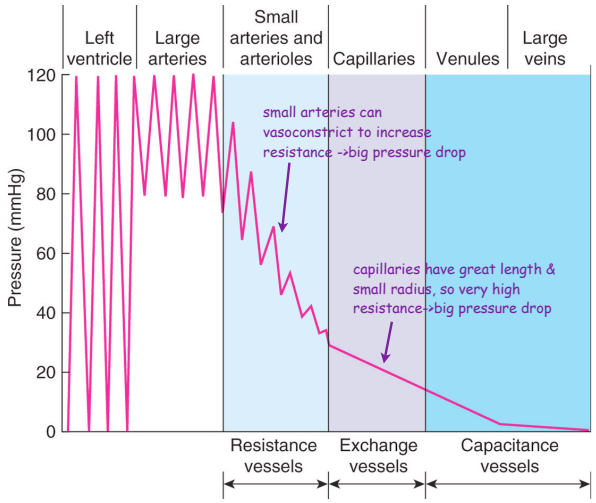
Figure 14.16

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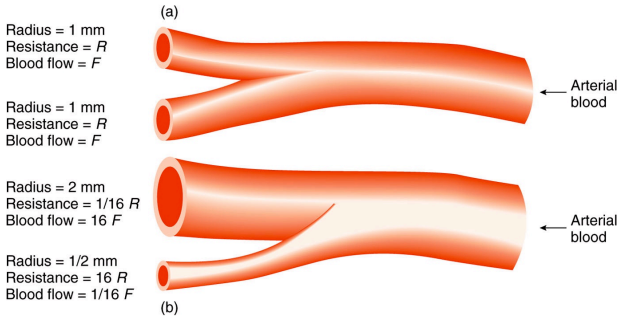
**Smaller, longer vessels have greater resistance**

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

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

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## Vasoconstriction elevates blood pressure

Blood flow = ΔP / resistance

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

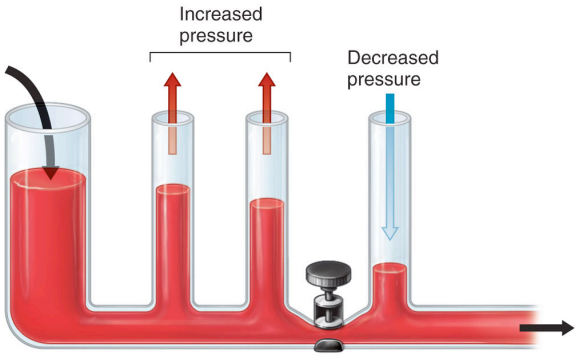


Figure 14.24

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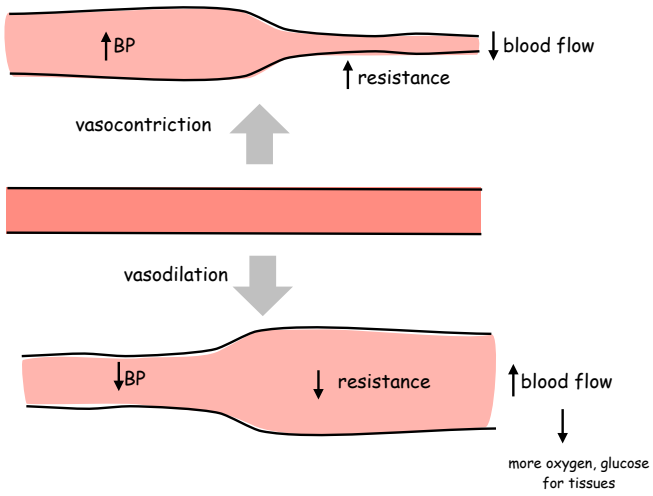


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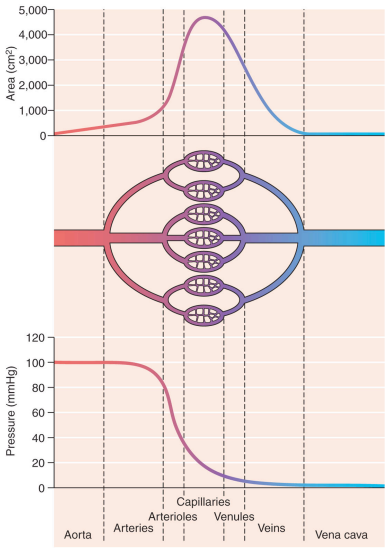
Figure 14.25

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

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

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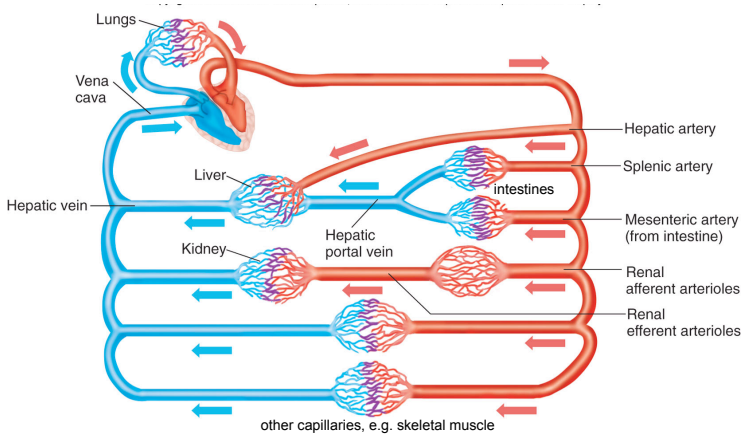


Figure 14.17

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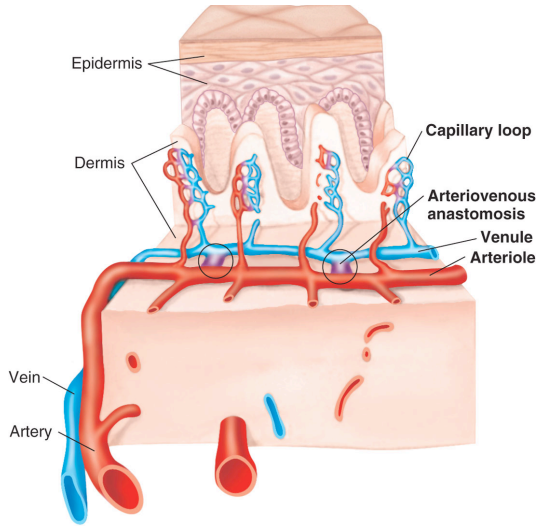
Figure 14.23

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## 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.

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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?

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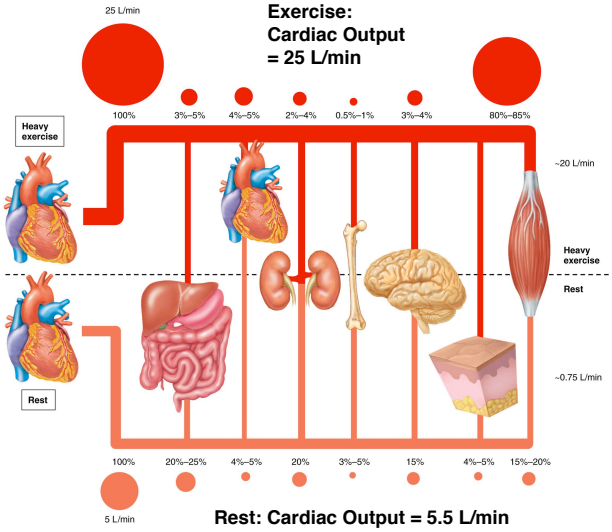


Figure 14.20

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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

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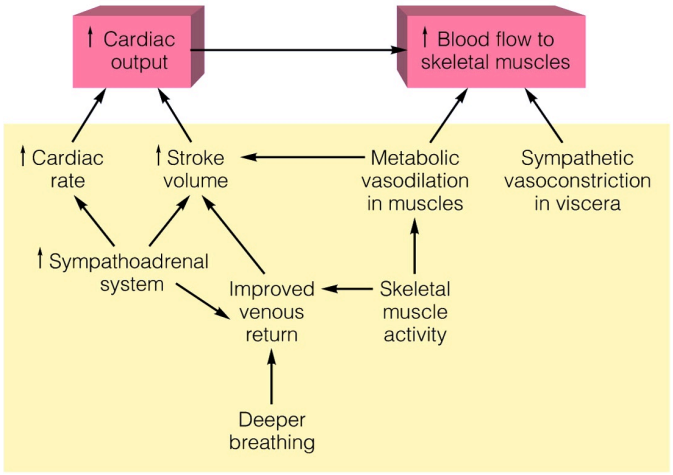
Figure 14.21

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## 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<br>Increased cholinergic sympathetic activity<br>Increased metabolic rate of exercising muscles, producing intrinsic vasodilation |

Table 14.5

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## Increase of Cardiac Output with Exercise

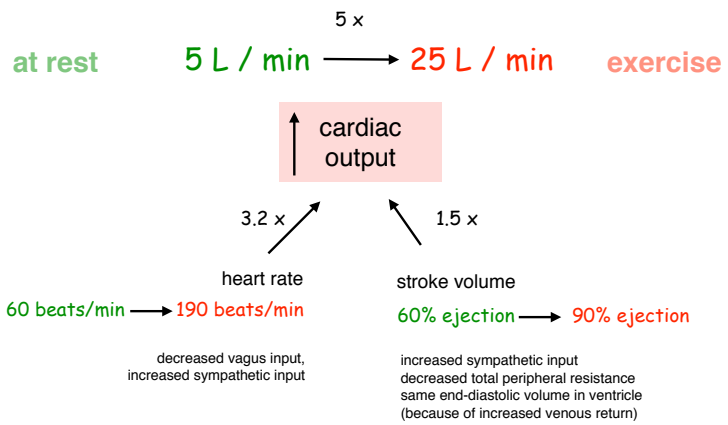


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**Table 14.7 | Cardiovascular Changes During Moderate Exercise**

| 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 |

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# Control of Blood Pressure & Blood Volume

## Rapid neural responses:

**1. Baroreflex**
change in baroreceptor sensory afferent fibers causes change in autonomic nerve activity
e.g. decreased vagus activity, increase sympathetic -> increased cardiac output & vasocontriction -> increase blood pressure

**2. Osmoreceptors**
Hypothalamic neurons respond to increase in osmolality of blood to induce thirst & drinking -> dilute blood back to 300 mOsm.

## Endocrine Responses:

**1. Antidiuretic hormone (ADH)**
Increased osmolality causes release of **antidiuretic hormone (ADH)** from hypothalamic axons in the posterior pituitary which causes kidney to retain more water

**2.Renin-Angiotensin II-Aldosterone**
Decreased blood flow & pressure in kidneys -> release of renin, which cleaves angiotensinogen to angiotensin I -> converted to **angiotensin II** by angiotensin converting enzyme (**ACE**) in lungs. Ang II causes vasoconstriction-> increased blood pressure.
(Ang II also stimulates aldosterone synthesis in adrenal cortex leading to Na retention by kidneys )

**3. Atrial Natriuretic Peptide (ANP)**
Increased blood volume -> stretch of left atrium -> release of ANP -> increased NaCl and H20 excretion by kidney in urine -> decreased blood volume

*diurectic* - increases urine production *(uresis = urine)*
*angio*- little vessel, i.e. related to blood vessels
*natriuretic* - to increase salt (*natrium*) in the urine.

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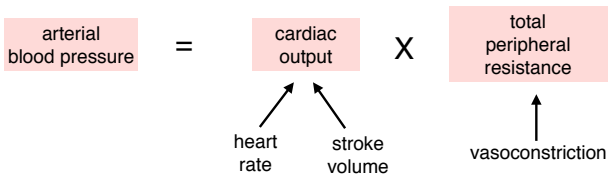
**Normal:**
adjust cardiac output & TPR & blood volume to maintain 120/80 mmHg

**Essential Hypertension:**
increase in cardiac output or TPR or both leads to high blood pressure of 140/90 mmHg -> increased risk of heart disease, stroke, kidney failure.




$V = I \times R$

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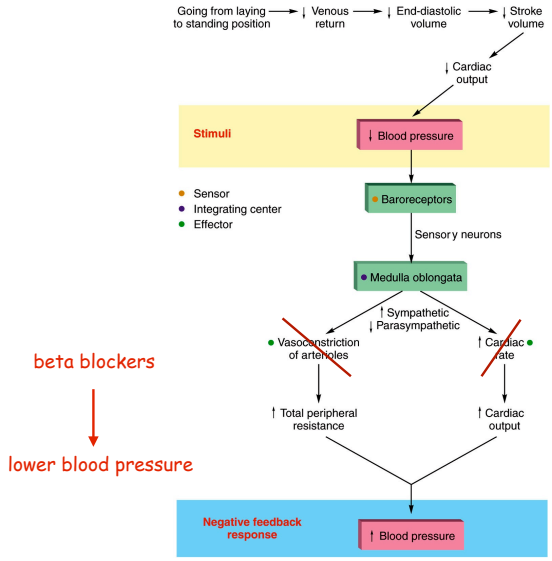
Figure 14.28

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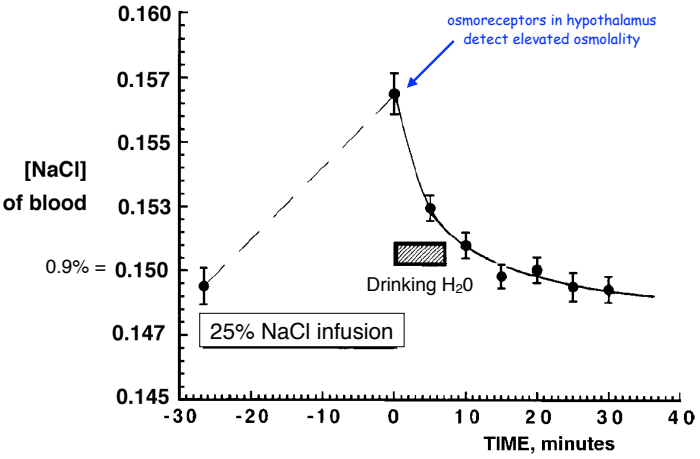
## Elevated [NaCl] in blood above 0.15 M causes drinking in pigs

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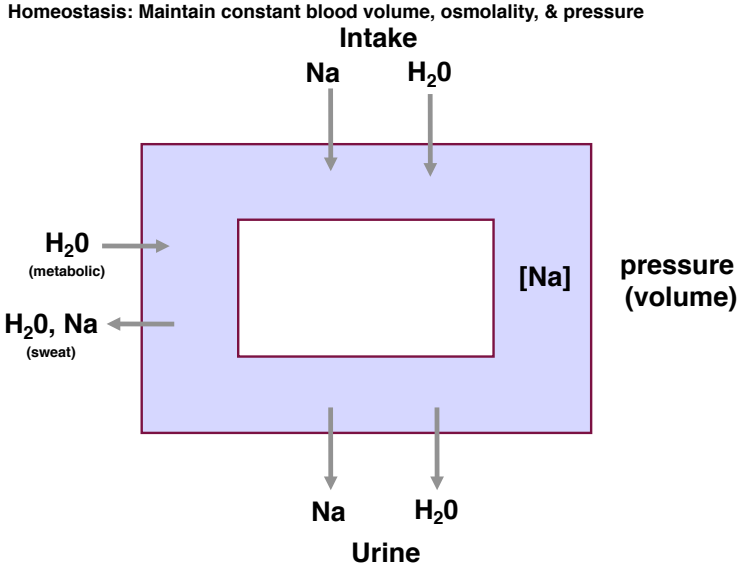
**Homeostasis: Maintain constant blood volume, osmolality, & pressure**

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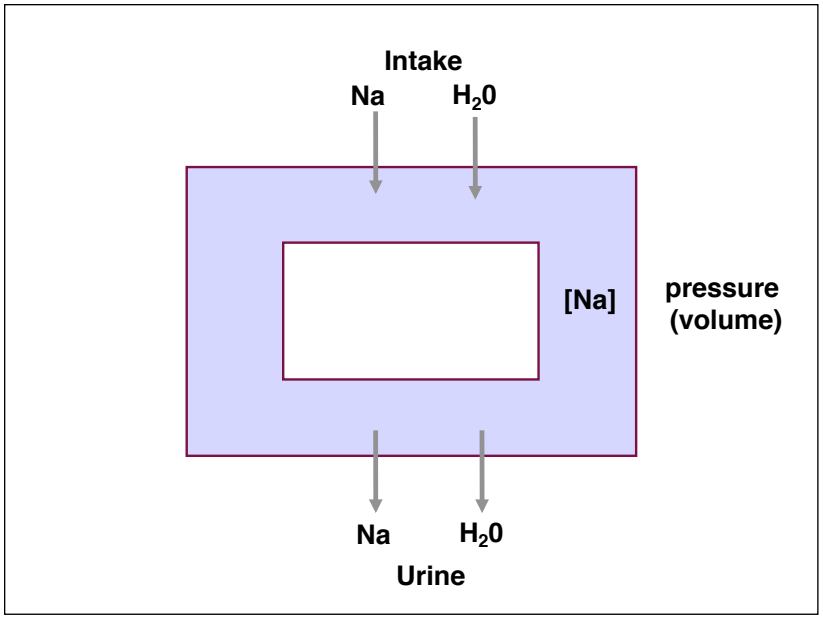


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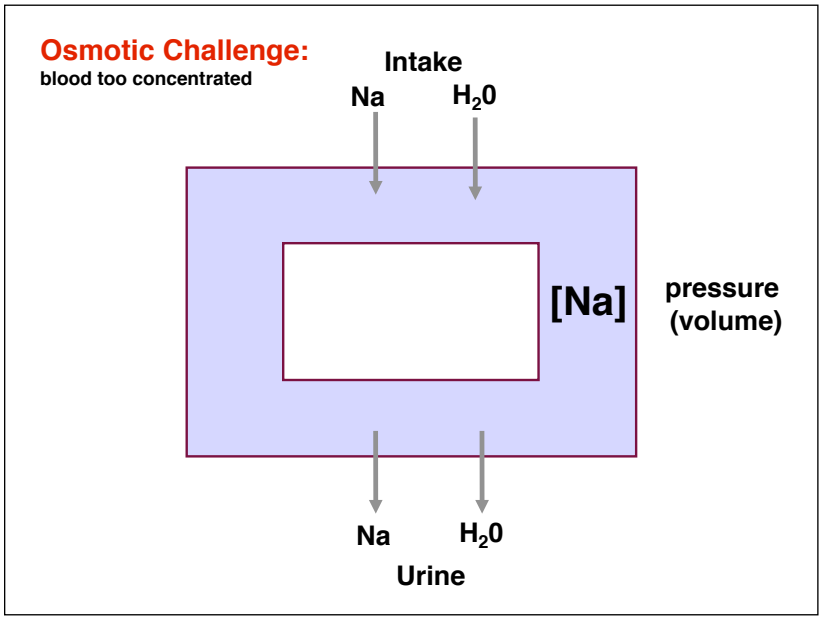
**Osmotic Challenge:**
blood too concentrated

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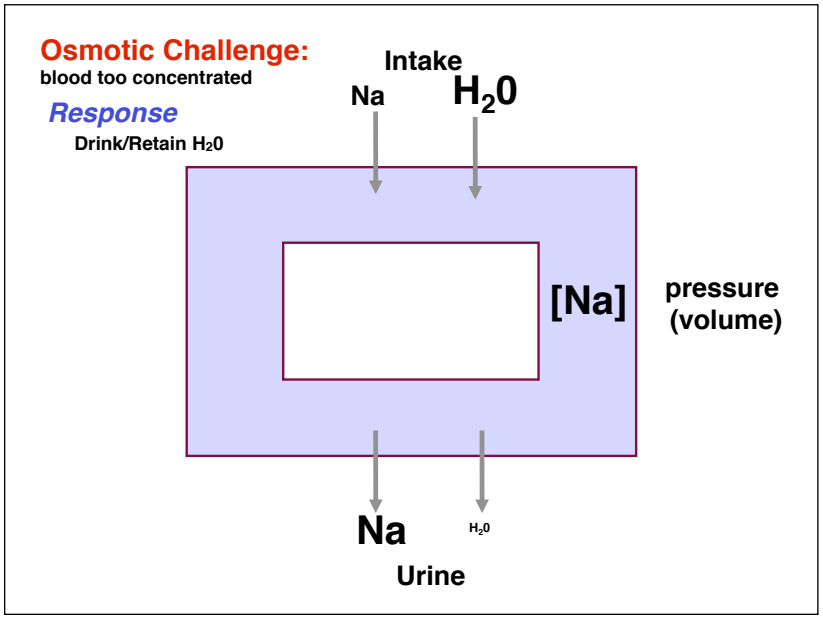
**Osmotic Challenge:**
blood too concentrated

***Response***
Drink/Retain $H_20$

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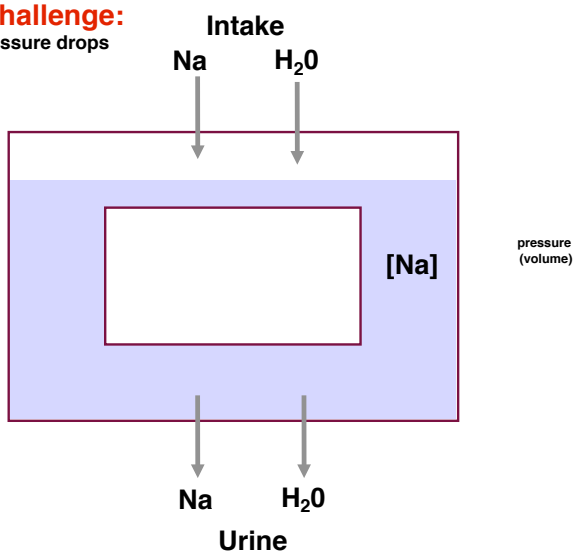
## Volemic Challenge:

Blood loss, pressure drops

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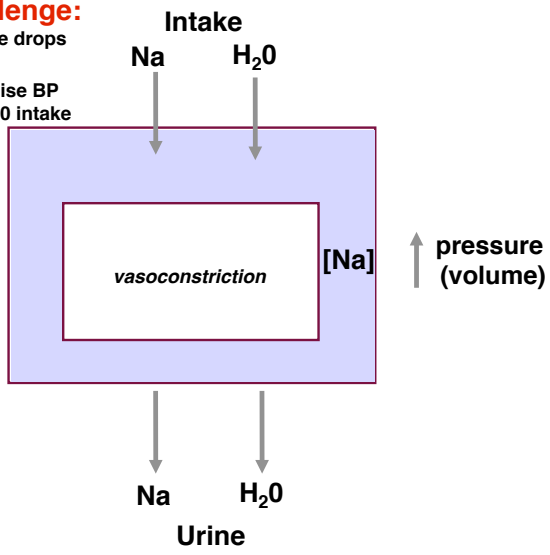
## Volemic Challenge:

Blood loss, pressure drops

***Response***

vasoconstrict to raise BP

drink, retain Na, $H_20$ intake

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## Osmoreceptors

Cells that respond to changes in plasma osmolality.

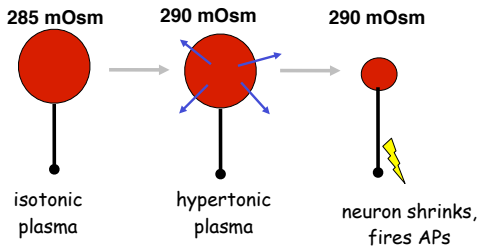


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Figure 14.11

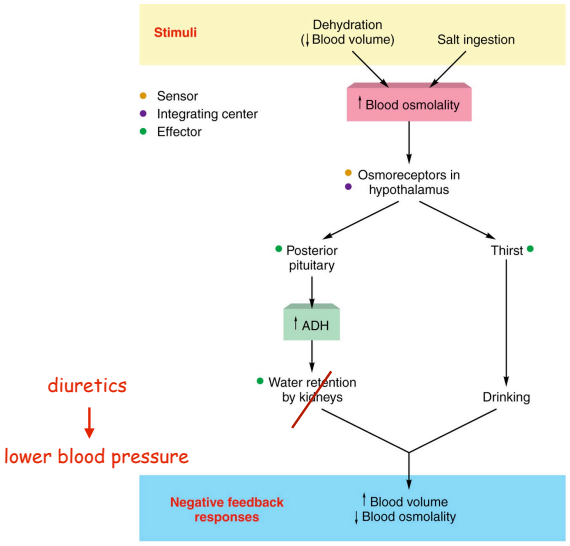


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Figure 14.12

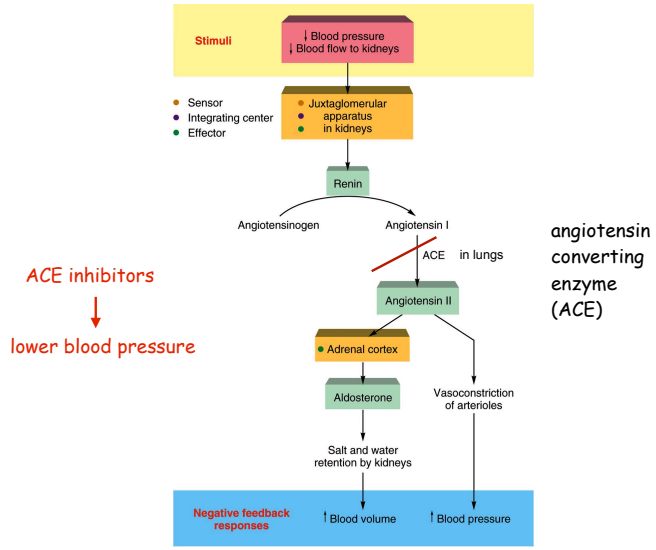


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Figure 14.13

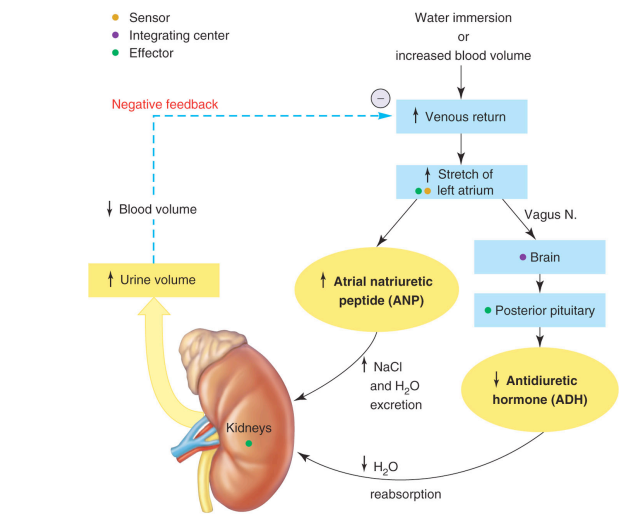


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Table 14.4

**Table 14.4 | Extrinsic Control of Vascular Resistance and Blood Flow**

| Extrinsic Agent | | Effect | Comments |
|---|---|---|---|
| Sympathetic nerves | neural | | |
| Alpha-adrenergic | neural | Vasoconstriction | Vasoconstriction is the dominant effect of sympathetic nerve stimulation on the vascular system, and it occurs throughout the body. |
| Beta-adrenergic | neural | Vasodilation | There is some activity in arterioles in skeletal muscles and in coronary vessels, but effects are masked by dominant alpha-receptor-mediated constriction. |
| Cholinergic | neural | Vasodilation | Effects are localized to arterioles in skeletal muscles and are produced only during defense (fight-or-flight) reactions. |
| Parasympathetic nerves | neural | Vasodilation | Effects are restricted primarily to the gastrointestinal tract, external genitalia, and salivary glands and have little effect on total peripheral resistance. |
| Angiotensin II | endocrine | Vasoconstriction | A powerful vasoconstrictor produced as a result of secretion of renin from the kidneys; it may function to help maintain adequate filtration pressure in the kidneys when systemic blood flow and pressure are reduced. |
| ADH (vasopressin) | endocrine | Vasoconstriction | Although the effects of this hormone on vascular resistance and blood pressure in anesthetized animals are well documented, the importance of these effects in conscious humans is controversial. |
| Histamine | paracrine | Vasodilation | Histamine promotes localized vasodilation during inflammation and allergic reactions. |
| Bradykinins | paracrine | Vasodilation | Bradykinins are polypeptides secreted by sweat glands and by the endothelium of blood vessels; they promote local vasodilation. |
| Prostaglandins | paracrine | Vasodilation or vasoconstriction | Prostaglandins are cyclic fatty acids that can be produced by most tissues, including blood vessel walls. Prostaglandin $I_2$ is a vasodilator, whereas thromboxane $A_2$ is a vasoconstrictor. The physiological significance of these effects is presently controversial. |

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Table 14.10

**Table 14.10 | Mechanisms of Action of Selected Antihypertensive Drugs**

| Category of Drugs | Examples | Mechanisms |
|---|---|---|
| Diuretics | Thiazide; furosemide | Increase volume of urine excreted, thus lowering blood volume |
| Sympathoadrenal system inhibitors | Clonidine; alpha-methyldopa | Act to decrease sympathoadrenal stimulation by bonding to $\alpha_2$-adrenergic receptors in the brain |
| | Guanethidine; reserpine | Deplete norepinephrine from sympathetic nerve endings |
| | Atenolol | Blocks beta-adrenergic receptors, decreasing cardiac output and/or renin secretion |
| | Phentolamine | Blocks alpha-adrenergic receptors, decreasing sympathetic vasoconstriction |
| Direct vasodilators | Hydralazine; minoxidil sodium nitroprusside | Cause vasodilation by acting directly on vascular smooth muscle |
| Calcium channel blockers | Verapamil; diltiazem | Inhibit diffusion of $Ca^{2+}$ into vascular smooth muscle cells, causing vasodilation and reduced peripheral resistance |
| Angiotensin-converting enzyme (ACE) inhibitors | Captopril; enalapril | Inhibit the conversion of angiotensin I into angiotensin II |
| Angiotensin II–receptor antagonists | Losartan | Blocks the binding of angiotensin II to its receptor |

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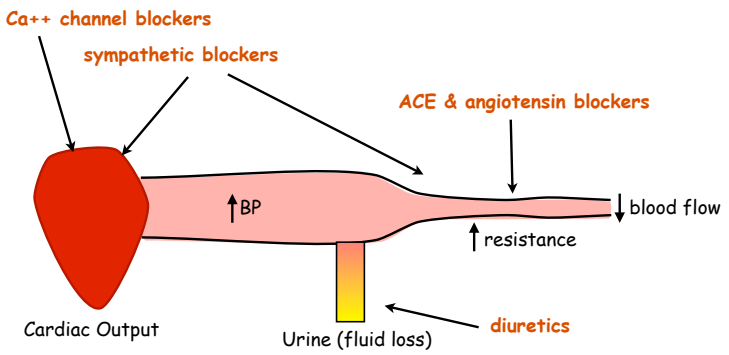


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