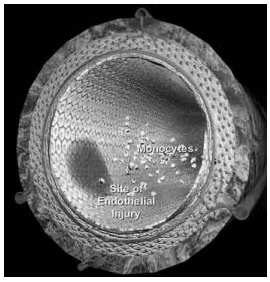
CARDIOVASCULAR PHYSIOLOGY
BLOOD PRESSURE AND ITS REGULATION



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OBJECTIVES

At the end of this lecture you should be able to

- **Define blood pressure and Mean Arterial Pressure (MAP)**
- **List the factors affecting MAP**
- **Describe Short term and long term control of Blood Pressure**

Systolic blood pressure

Maximum pressure exerted in the arteries

when blood is ejected into them

during systole

120 mm Hg

(120 mm Hg)

Diastolic blood pressure

Minimum pressure within the arteries

when blood is drained off from them

during diastole

80 mm Hg

(80 mm Hg)

Pulse pressure

The difference between systolic and diastolic pressures

40 mm Hg

(120 - 80 = 40 mm Hg)

Mean Arterial Pressure

Average pressure which drives blood forward into the tissues

93 mm Hg

diastolic pressure + (1/3 × (systolic - diastolic pressure)

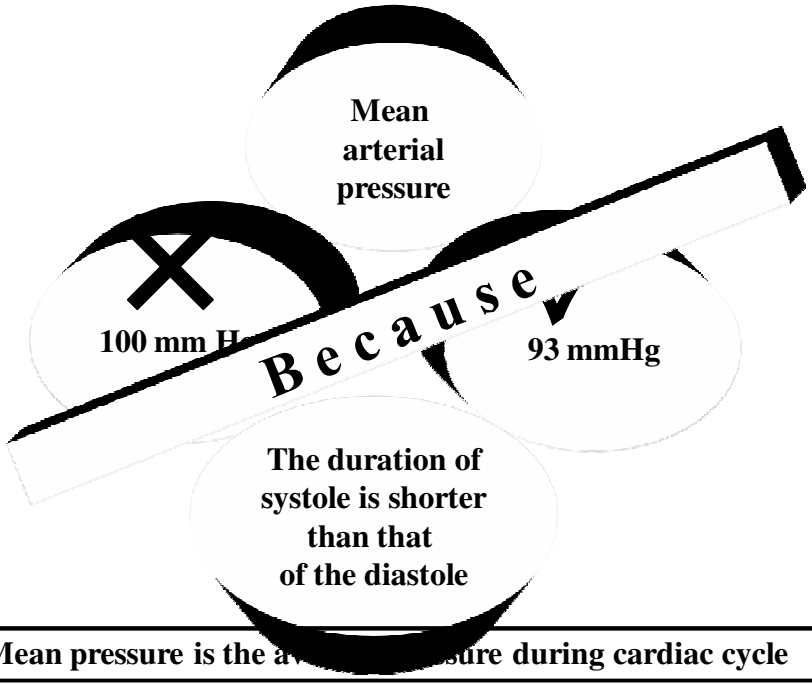
80 + 13 = 93 mm Hg

Arterial blood pressure

Blood pressure is the force the blood exerts against the walls of the blood vessels

Systolic pressure	**Maximum pressure during systole**	**120mmHg**
Diastolic pressure	**Minimum pressure during diastole**	**80 mmHg**
Pulse pressure	**Systolic pressure – diastolic pressure**	**40 mmHg**
Mean pressure	**Diastolic pressure + (1/3 pulse pressure)**	**93 mmHg**

Mean arterial pressure is the main driving force for blood flow



Mean pressure is the a sure during cardiac cycle

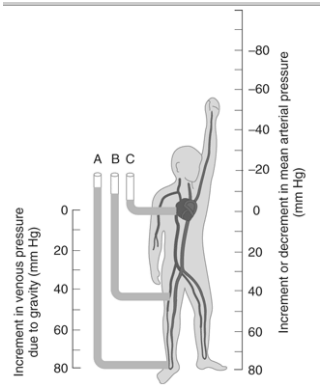
Normal Variations

- **Age**
- **Sleep**
- **Posture**
- **Exercise SBP increases and DBP is mantained in mild to moderate. (Therefore DBP is more imp)**
- **Gravity**

Effect of Gravity

•The pressure in any vessel below heart level is increased and above heart level is decreased by the effect of gravity.
•The magnitude of the gravitational effect is 0.77 mm Hg/cm of vertical distance above or below the heart at the density of normal blood.

•In an adult human in the upright position, when the mean arterial pressure at heart level is 100 mm Hg, the mean pressure in a large artery in the head (50 cm above the heart) is 62 mm Hg (100 – [0.77 x 50])
• and the pressure in a large artery in the foot (105 cm below the heart) is 180 mm Hg (100 + [0.77 x 105]).



Factors Determining Blood Pressure

Ohm's Law

$$F = \frac{\triangle P}{R}$$

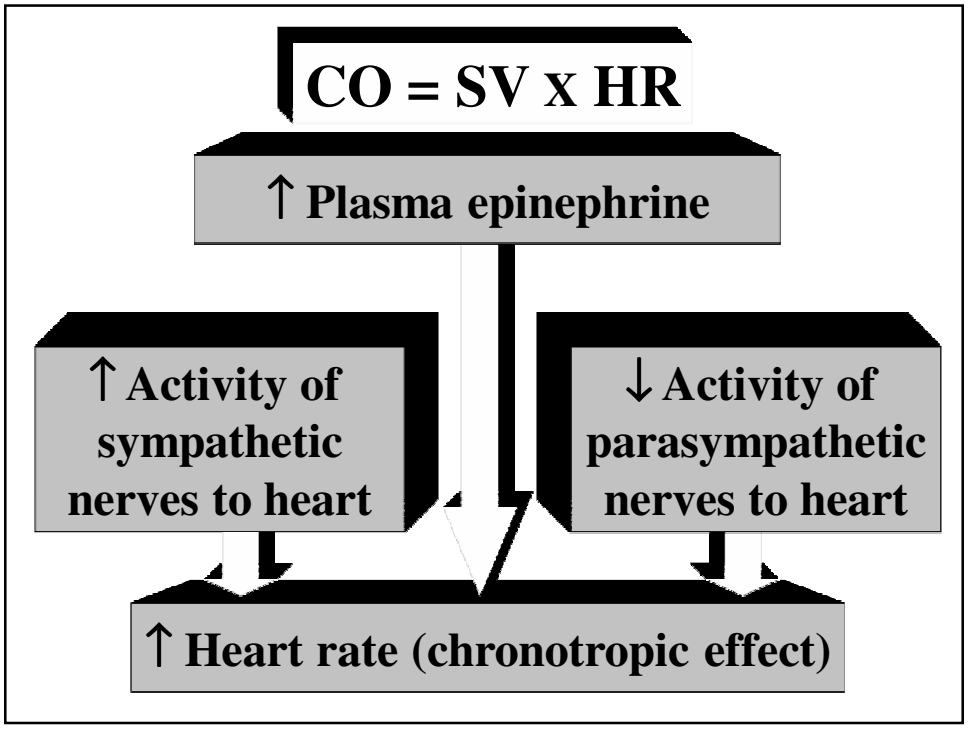
F = Cardiac output (CO)

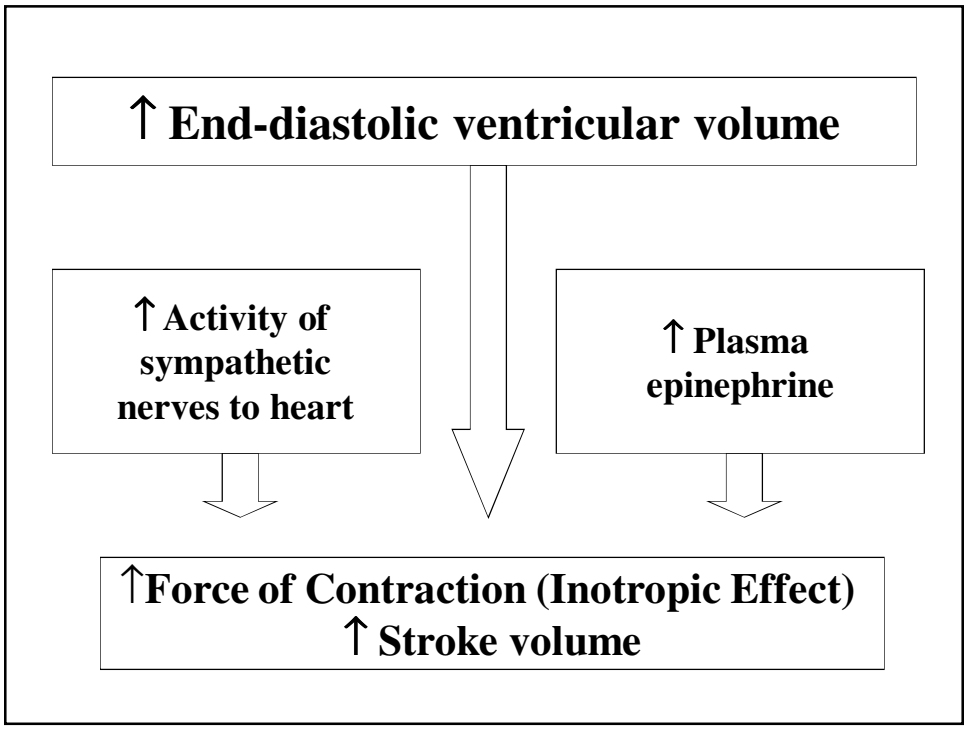
$\triangle$ P = Mean arterial pressure (MAP)

R = Total peripheral resistance (TPR)

$$\therefore CO = \frac{MAP}{TPR}$$

$$\therefore MAP = CO \times TPR$$


CO = SV x HR
↑ Plasma epinephrine
↑ Activity of sympathetic nerves to heart
↓ Activity of parasympathetic nerves to heart
↑ Heart rate (chronotropic effect)


↑ End-diastolic ventricular volume
↑ Activity of sympathetic nerves to heart
↑ Plasma epinephrine
↑Force of Contraction (Inotropic Effect)
↑ Stroke volume

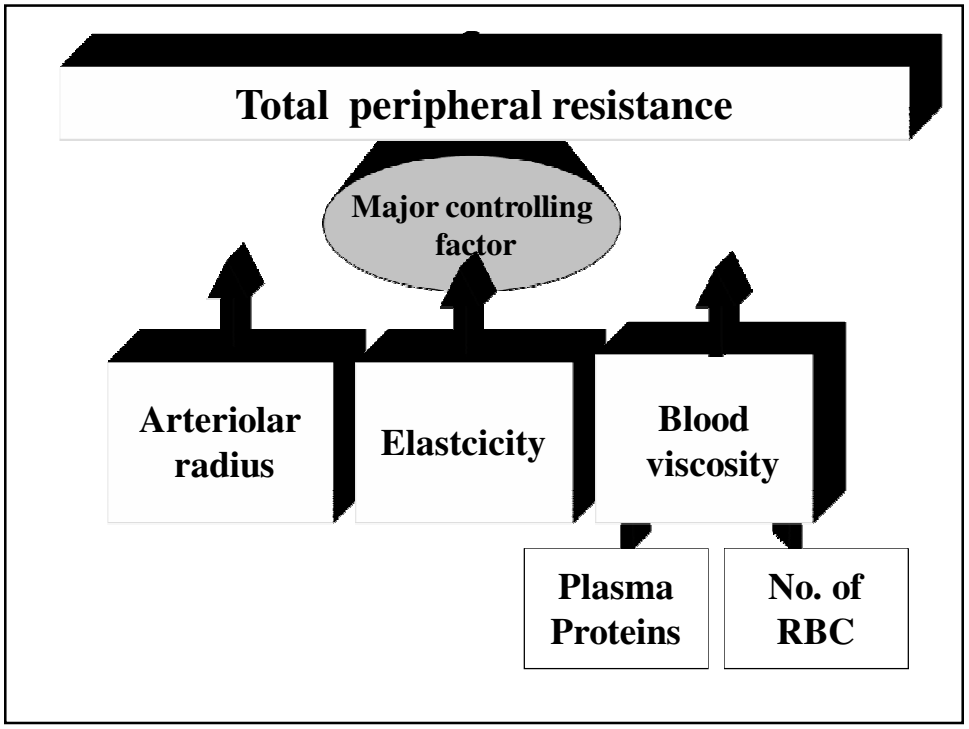
Poiseuille's Law

$$Q = \frac{\pi \triangle P r^4}{8 \eta L}$$

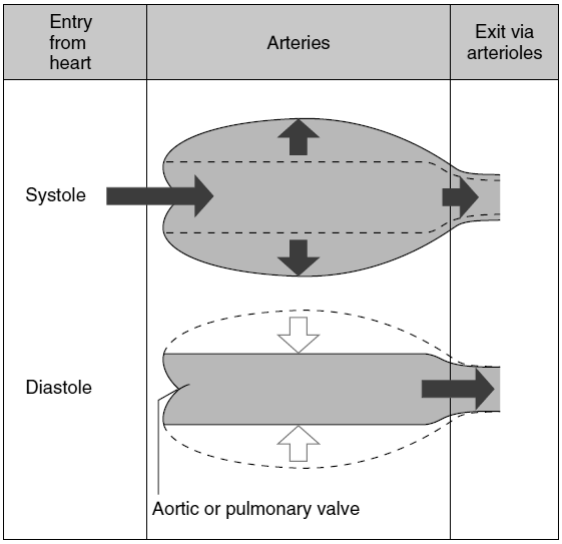
Q = Flow
$\triangle$ P = Pressure gradient
r = Radius
η = Viscosity
L = Length of tube
$\pi/8$ = Constant

Length of the blood vessels remains unchanged

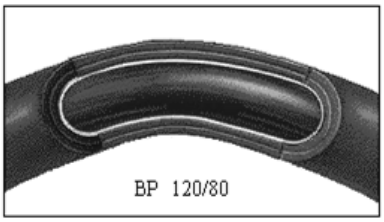
Viscosity of blood usually varies little



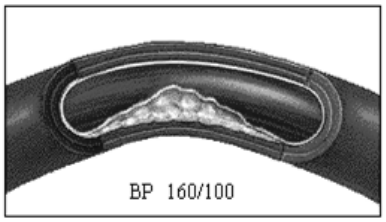
Elasticity depends on kinetic energy and PE. KE is responsible for expansion of Arterial Wall While PE is responsible for elastic recoil.



VESSEL ELASTICITY AFFECTS BLOOD PRESSURE



Healthy **elastic artery** expands, absorbing shock of **systolic pressure**.



Arteriosclerosis: calcified and rigid artery cannot expand; artery walls experience higher pressures.

The elasticity of the large arteries serves as a "shock absorber" to reduce the sudden sharp increase in systolic pressure. The elastic recoil of the vessel then serves to maintain the continued flow during **diastole**.

The major determinant of resistance and blood flow is

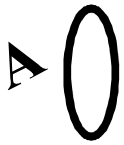
the 4th power of the

Radius of the blood vessel

$$R \propto \frac{1}{r^4}$$

Resistance varies inversely with the caliber of the blood vessel

$$Q \propto \triangle P$$



50 mm Hg 10 mm Hg

$\triangle P = 40$ mm Hg

90 mm Hg 10 mm Hg

$\triangle P = 80$ mm Hg

Flow in vessel B is two times the flow in vessel A because the $\triangle P$ is two times more in vessel B

Blood vessel - 1

Flow = 1 ml / min.

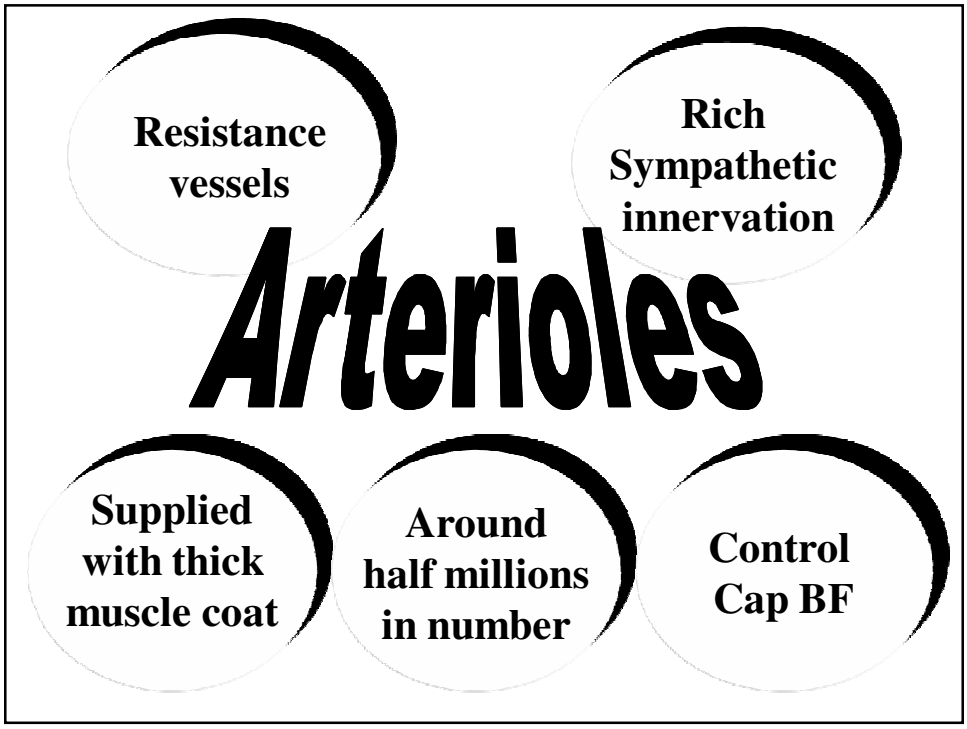
Blood vessel - 2
(radius × 2 the radius of vessel - 1

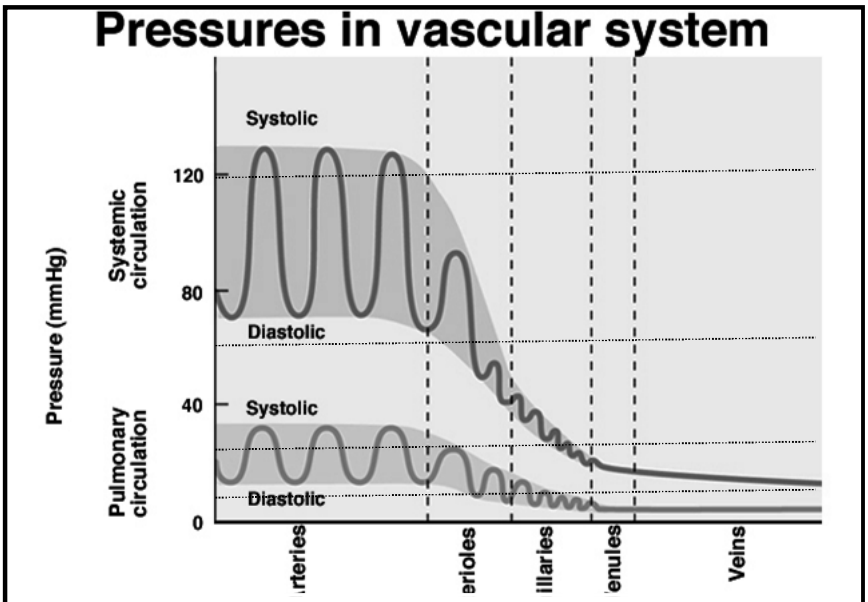
Flow = (2 × 2 × 2 × 2) = 16 ml / min.

Blood vessel - 3
(radius × 3 the radius of vessel - 1

Flow = (3 × 3 × 3 × 3) = 81 ml / min.

Flow varies directly and resistance inversely with the 4th power of the radius



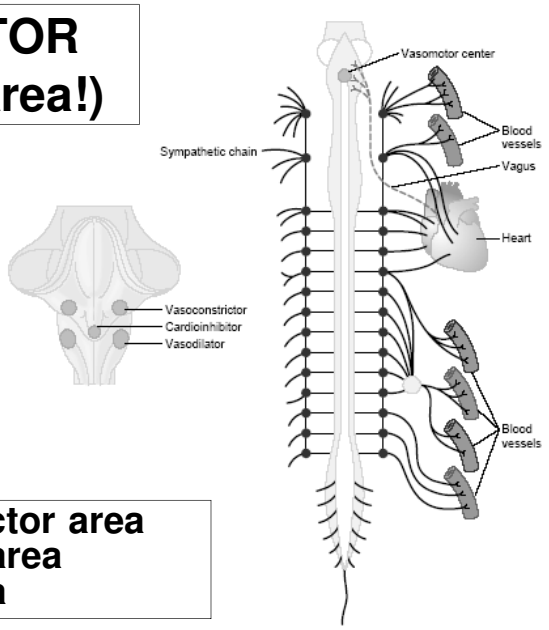


The magnitude of this pressure drop depends upon the degree of arteriolar constriction or dilatation

BLOOD PRESSURE REGULATING MECHANISMS

1. **Short Term (Within few seconds)**
2. **Intermediate (Within few hours)**
3. **Long Term (Within few days)**

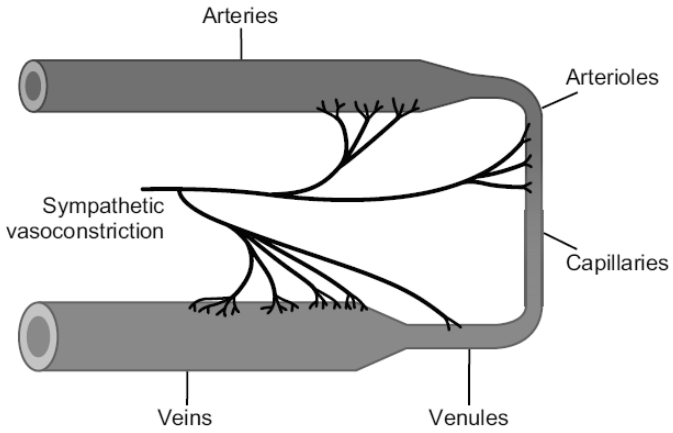
VASOMOTOR CENTER (Area!)



1. **Vasoconstrictor area**
2. **Vasodilator area**
3. **Sensory area**

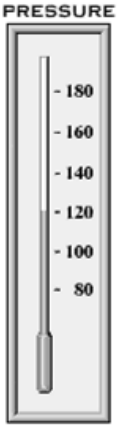
VASOMOTOR CENTER (Area!)

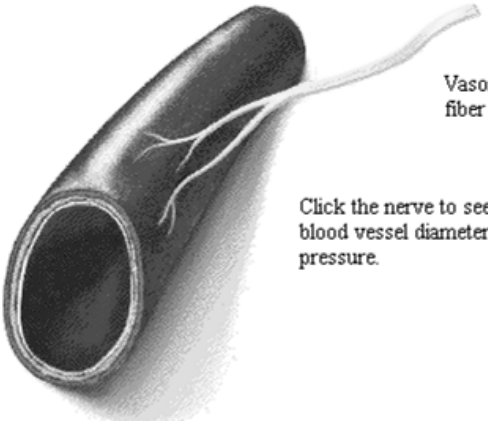
1. **A vasoconstrictor area located bilaterally in the anterolateral portions of the upper medulla. exite vasoconstrictor neurons of the sympathetic nervous system.**
2. **A vasodilator area located bilaterally in the anterolateral portions of the lower half of the medulla. inhibit the vasoconstrictor area, thus causing vasodilation.**
3. **A sensory area located bilaterally posterolateral portions of the medulla and lower pons (tractus solitarius). Receive sensory nerve signals by vagus and glossopharyngeal nerves and output control activities of both the vasoconstrictor and vasodilator areas An example is the baroreceptor reflex**



Continuous Partial Constriction of the Blood Vessels Is Normally Caused by Sympathetic Vasoconstrictor Tone.

- Constriction of blood vessels raises **blood pressure**.
- Vessel diameter is actively regulated by **vasomotor fibers**, sympathetic nerve fibers that innervate the vessel's smooth muscle layer.
- Vasomotor fibers release **norepinephrine**, a powerful **vasoconstrictor**.





Click the nerve to see its effect on blood vessel diameter and blood pressure.

CONTROL OF VMC

Reticular Substance of Brain Stem

Hypothalamus

Posterolateral portions Cause Excitation. Anterior part can cause Excitation or Inhibition

Cerebral Cortex

Motor Cortex Cause Excitation

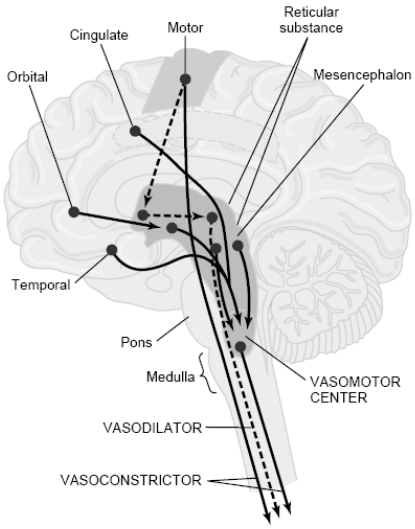
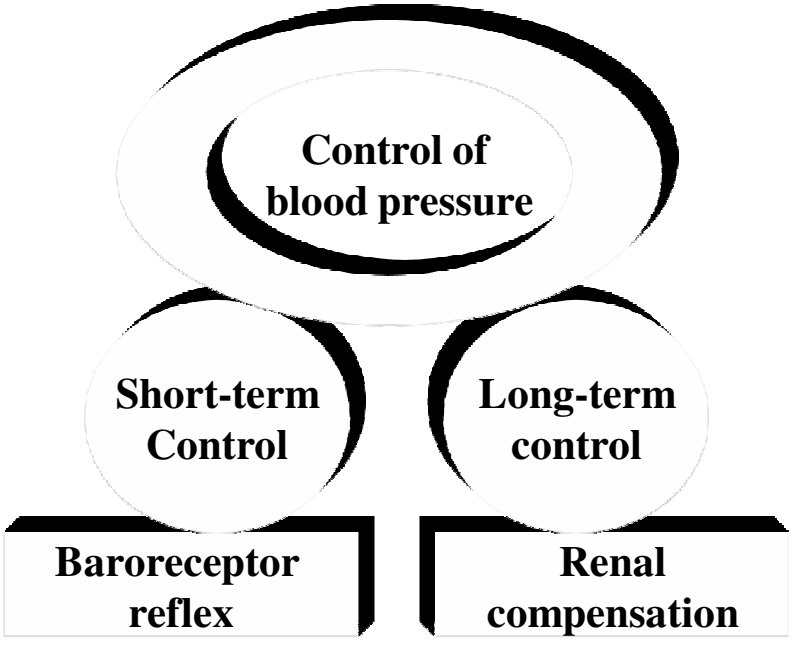
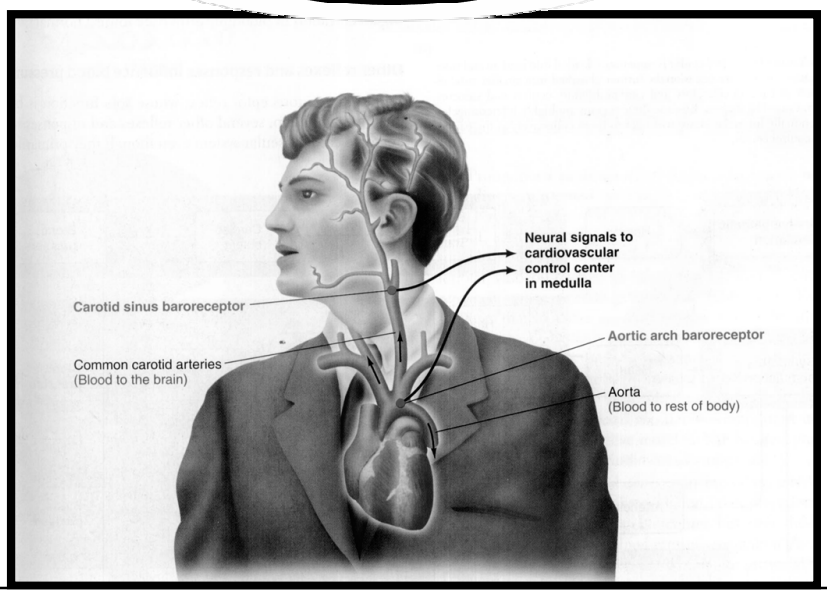


Figure 18–3



Baroreceptors



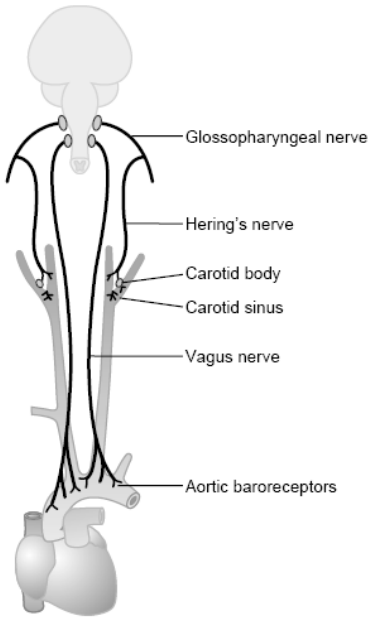
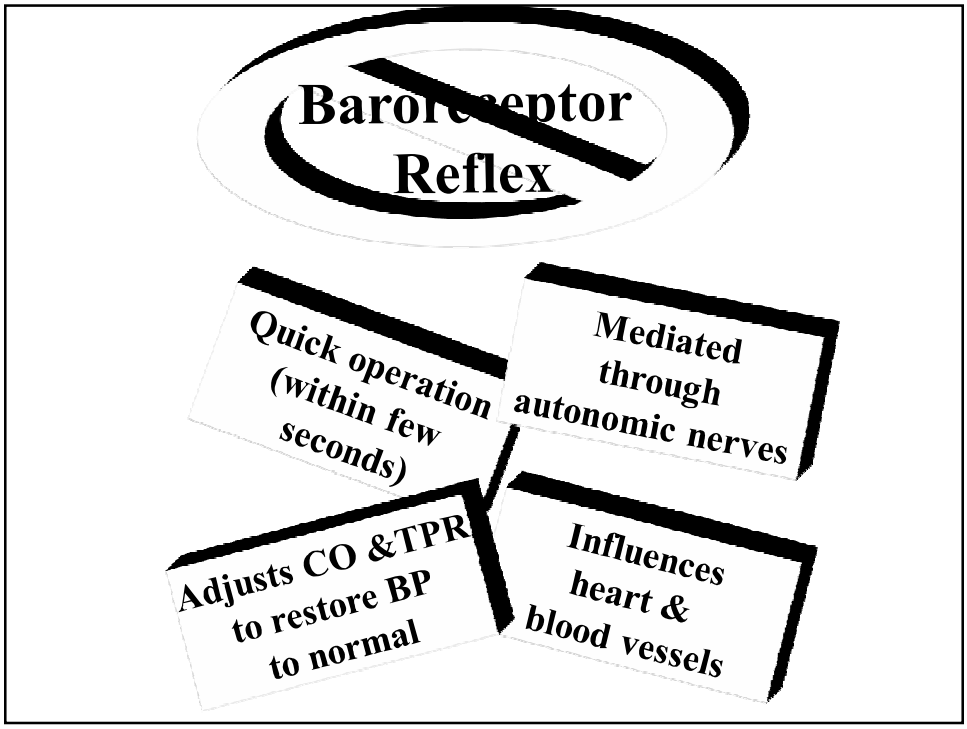
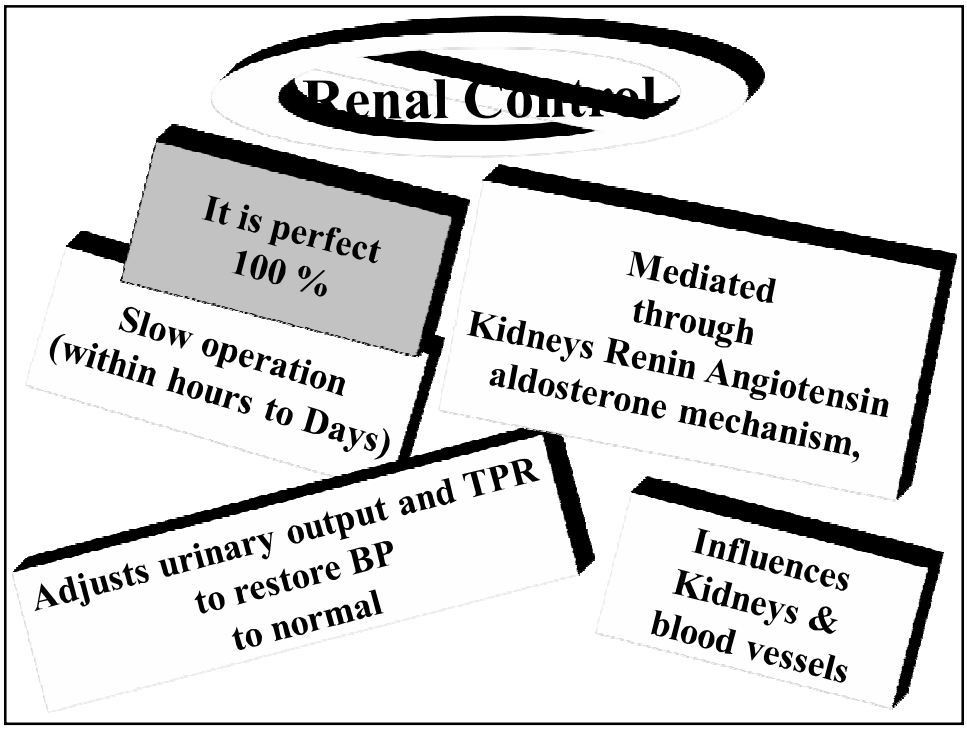


Figure 18–5


Baroreceptor Reflex
Quick operation (within few seconds)
Mediated through autonomic nerves
Adjusts CO &TPR to restore BP to normal
Influences heart & blood vessels

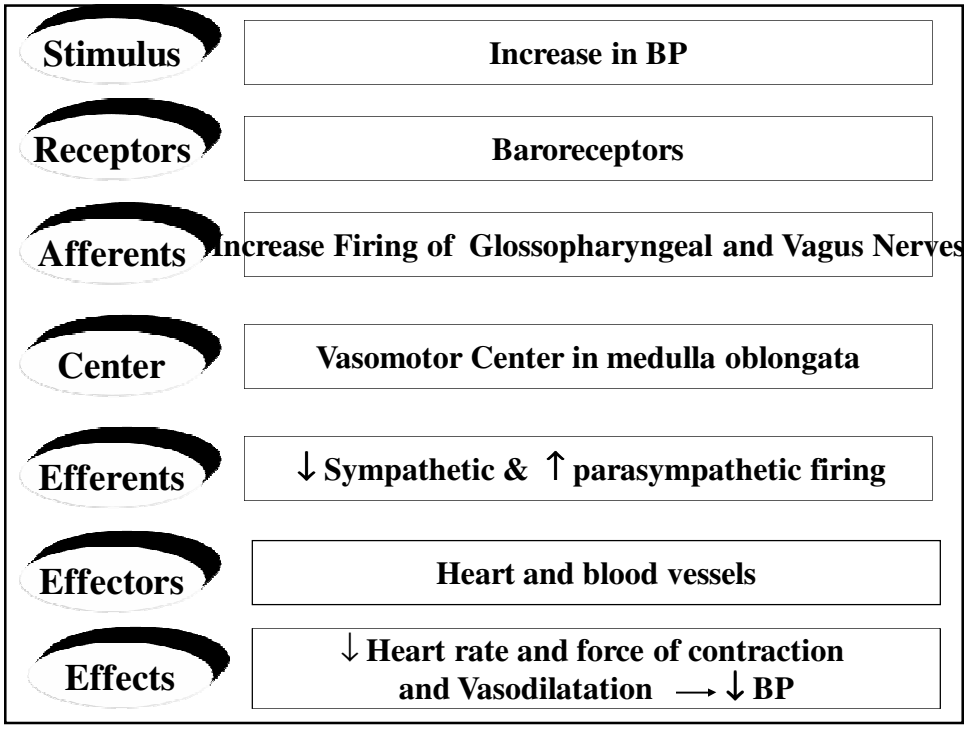

Renal Control
It is perfect 100 %
Slow operation (within hours to Days)
Mediated through Kidneys Renin Angiotensin aldosterone mechanism,
Adjusts urinary output and TPR to restore BP to normal
Influences Kidneys & blood vessels

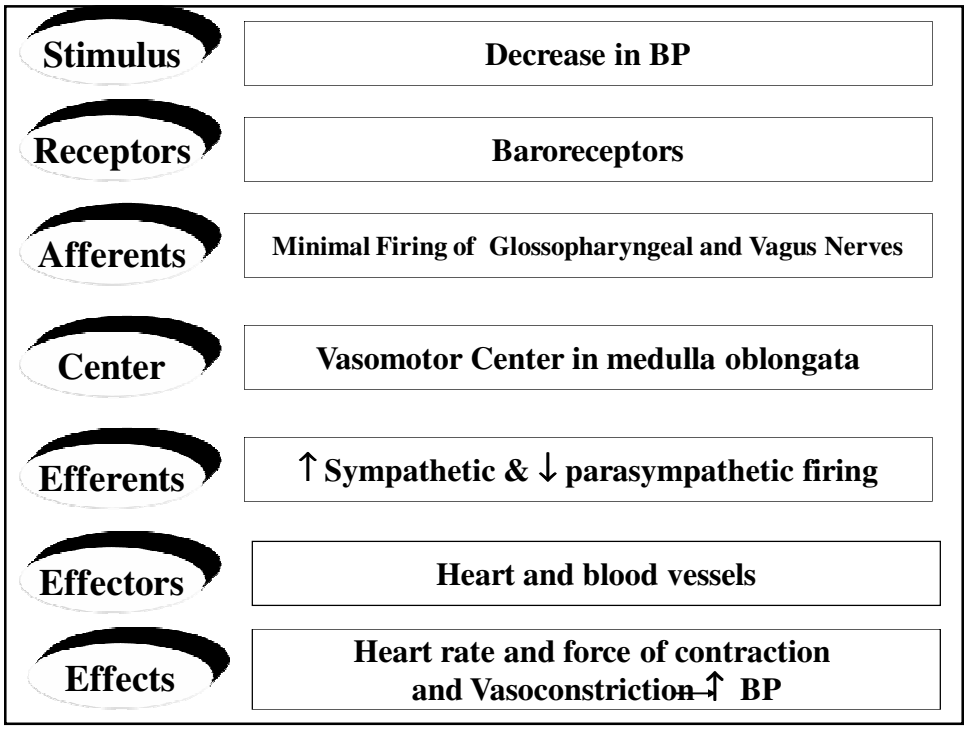
Components Of Baroreceptor Reflex Arc

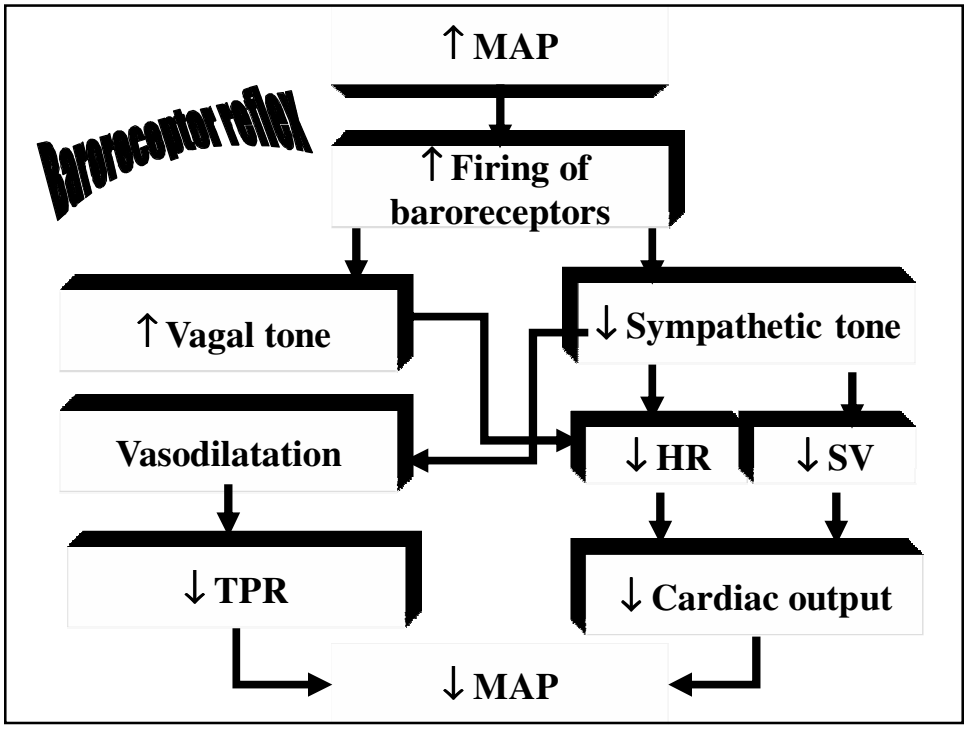
Receptors	Baroreceptors in carotid sinuses & arch of aorta
Afferents	Carotid sinus nerves & nerve from arch of aorta
Center	Vasomotor Center in medulla oblongata
Efferents	Sympathetic & parasympathetic nerves
Effectors	Heart and blood vessels

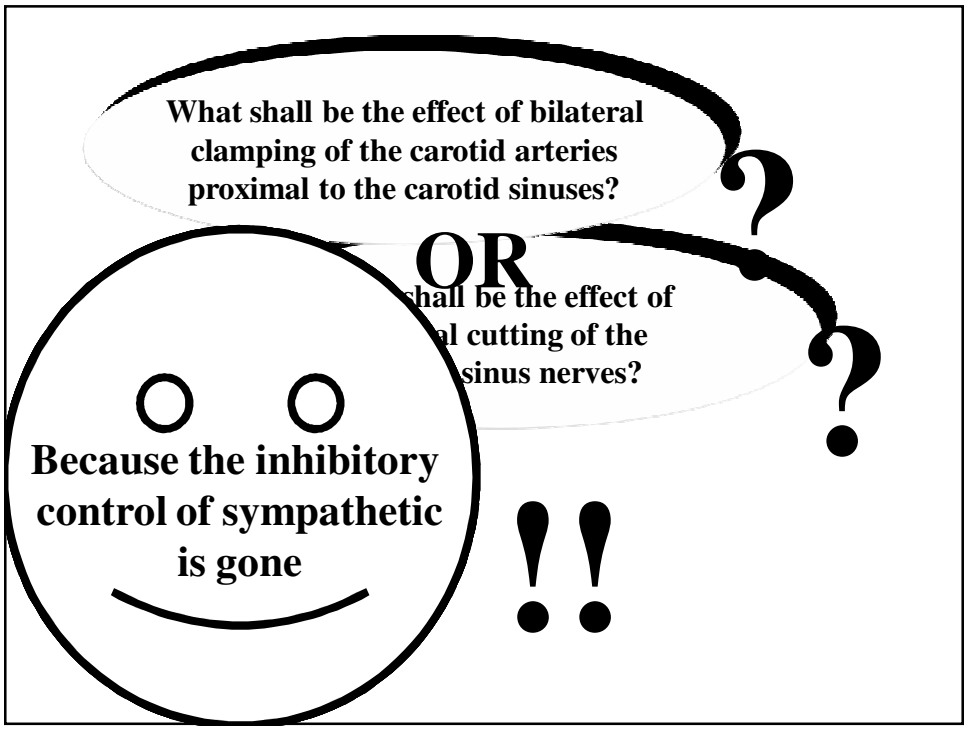
Carotid sinus nerve runs along with glossopharyngeal nerve
Aortic nerve runs along with vagus nerve

COMPONENTS OF BARORECEPTOR REFLEX ARC


Stimulus
Increase in BP
Receptors
Baroreceptors
Afferents
Increase Firing of Glossopharyngeal and Vagus Nerves
Center
Vasomotor Center in medulla oblongata
Efferents
↓ Sympathetic & ↑ parasympathetic firing
Effectors
Heart and blood vessels
Effects
↓ Heart rate and force of contraction
and Vasodilatation ⟶ ↓ BP


Stimulus
Decrease in BP
Receptors
Baroreceptors
Afferents
Minimal Firing of Glossopharyngeal and Vagus Nerves
Center
Vasomotor Center in medulla oblongata
Efferents
↑ Sympathetic & ↓ parasympathetic firing
Effectors
Heart and blood vessels
Effects
Heart rate and force of contraction
and Vasoconstriction ⟶ ↑ BP


↑ MAP
Baroreceptor reflex
↑ Firing of baroreceptors
↑ Vagal tone
↓ Sympathetic tone
Vasodilatation
↓ HR
↓ SV
↓ TPR
↓ Cardiac output
↓ MAP


What shall be the effect of bilateral clamping of the carotid arteries proximal to the carotid sinuses?
OR
shall be the effect of
l cutting of the
sinus nerves?
Because the inhibitory control of sympathetic is gone
!!

Pressure on the carotid sinus, produced, for example by the tight collar or carotid massage

can cause

marked bradycardia

vasodilatation

Fainting or syncope

Syncope

Transient loss of consciousness

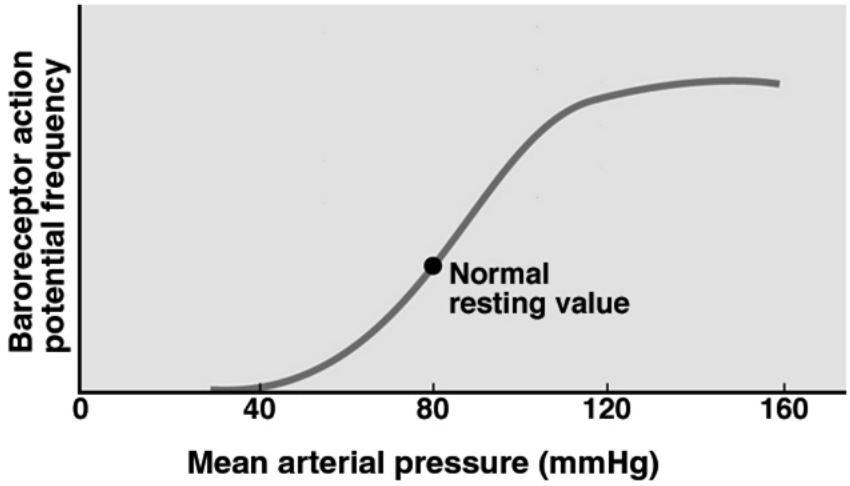
Associated with

Abrupt vasodilatation

Inadequate cerebral blood flow

Hypotension and bradycardia

Mean arterial pressure (MAP)



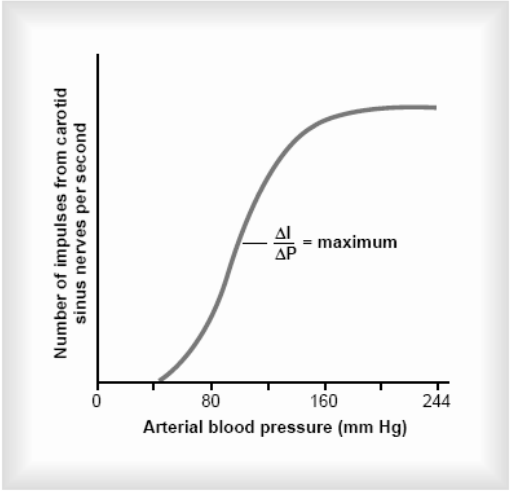
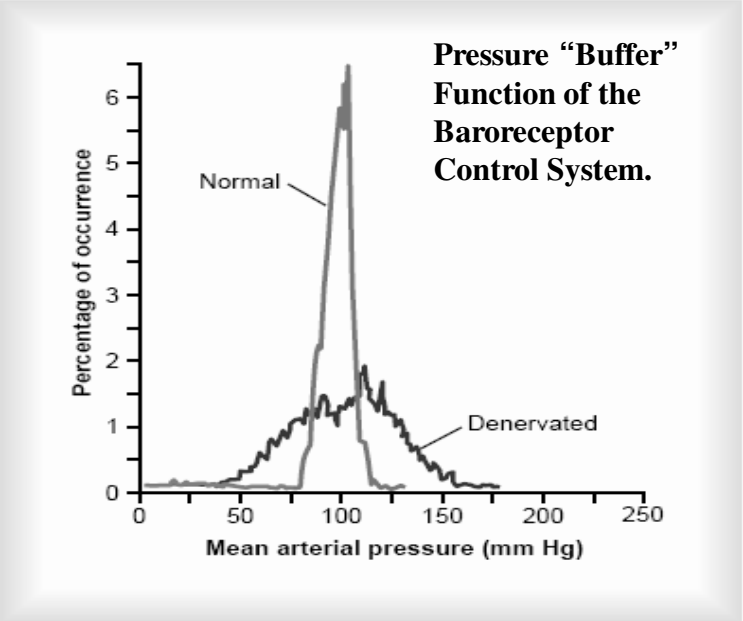


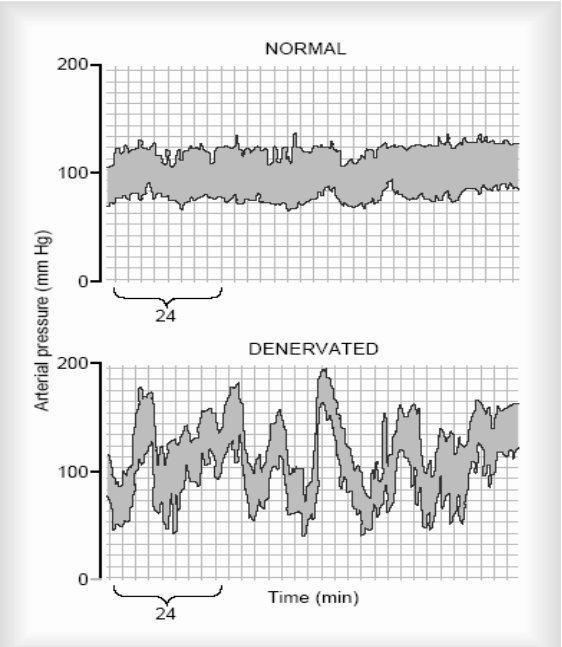
Figure 18–6

Activation of the baroreceptors at different levels of arterial pressure. ΔI, change in carotid sinus nerve impulses per second; ΔP, change in arterial blood pressure in mm Hg.

Pressure "Buffer" Function of the Baroreceptor Control System.

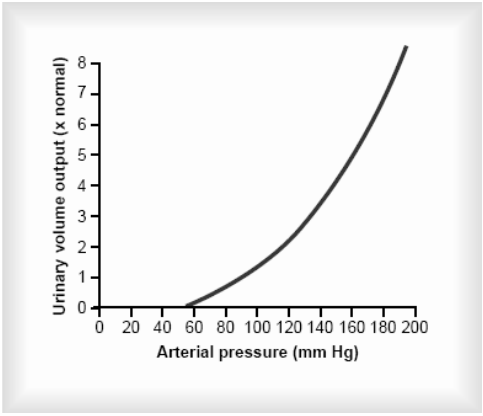


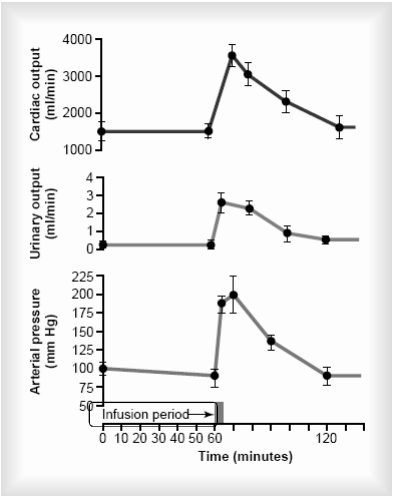
Pressure "Buffer" Function of the Baroreceptor Control System.



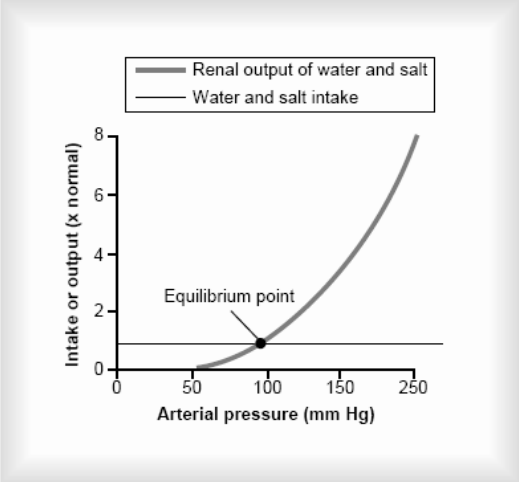
COTROL OF ARTERIAL PRESSURE IS ALSO BY

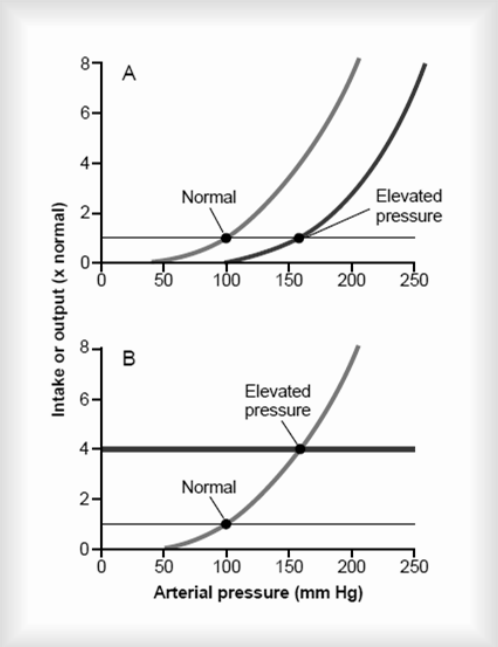
Chemoreceptors
(Carotid and Aortic Bodies)
Atrial and Pulmonary Artery Reflexes
(Low Pressure Receptors)
CNS Ischemic Response



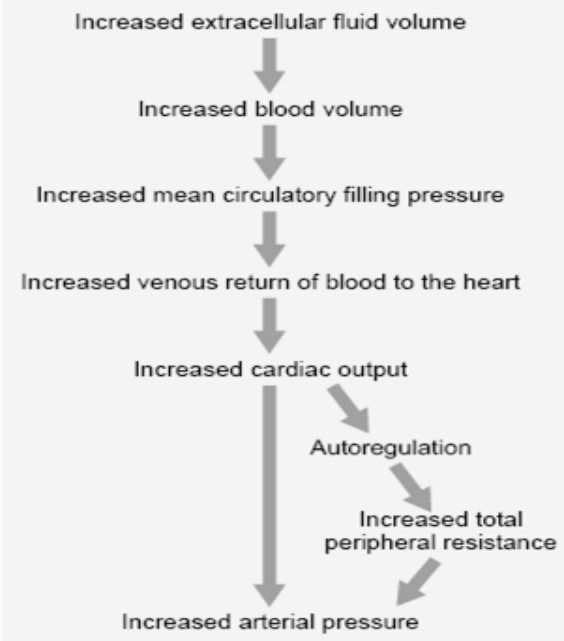


Pressure Natriuresis and Pressure Diuresis

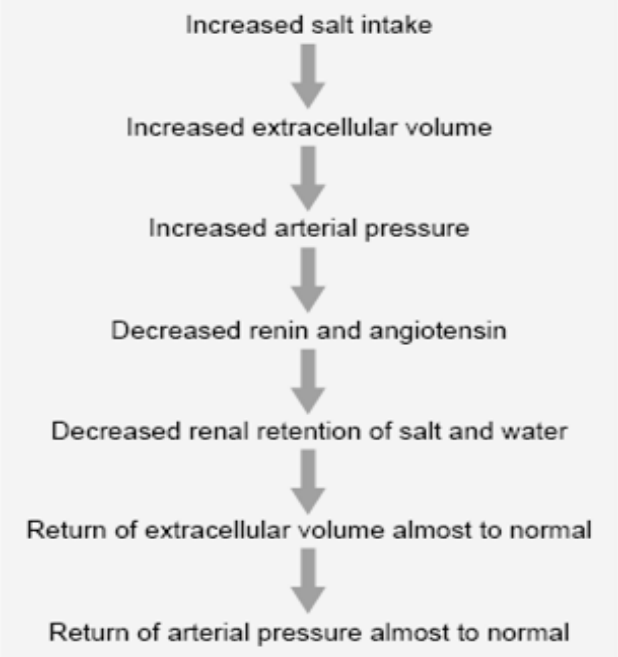




Increased Fluid Volume Can Elevate Arterial Pressure by Increasing Cardiac Output or Total Peripheral Resistance



Salt (NaCl) intake & Arterial Pressure Regulation



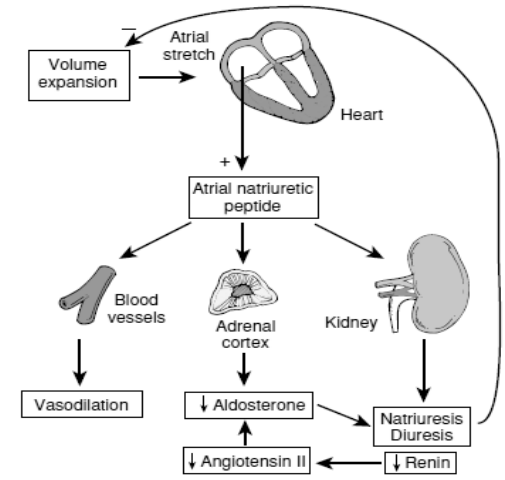


FIGURE 24.10 **Atrial natriuretic peptide and its actions.** ANP release from the cardiac atria is stimulated by blood volume expansion, which stretches the atria. ANP produces effects that bring blood volume back toward normal, such as increased Na^+ excretion.

Renal control

↓ MAP

↑ Renin secretion

Angiotensinogen → Angiotensin I

ACE in Lungs

Angiotensin II

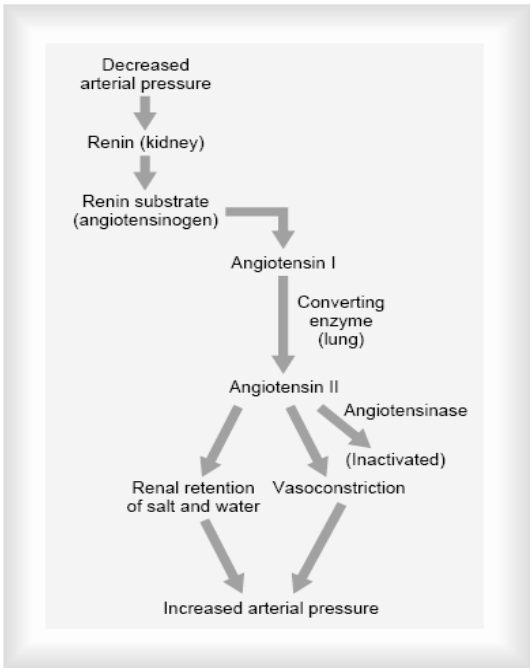
Vasoconstriction

↑ TPR

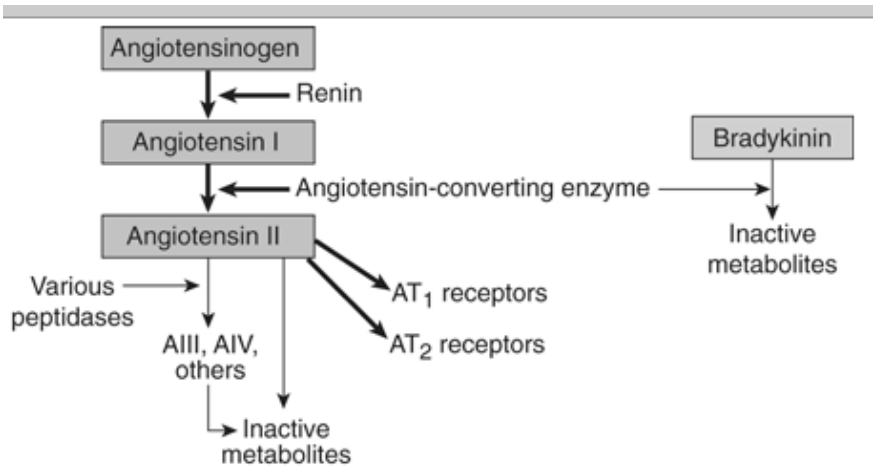
Salt & water Retention

↑ ECF Volume

↑ MAP



ACE synthesize Ang II (Vascoconstrictor) and Inactivates Bradykinin (Vasodilator)



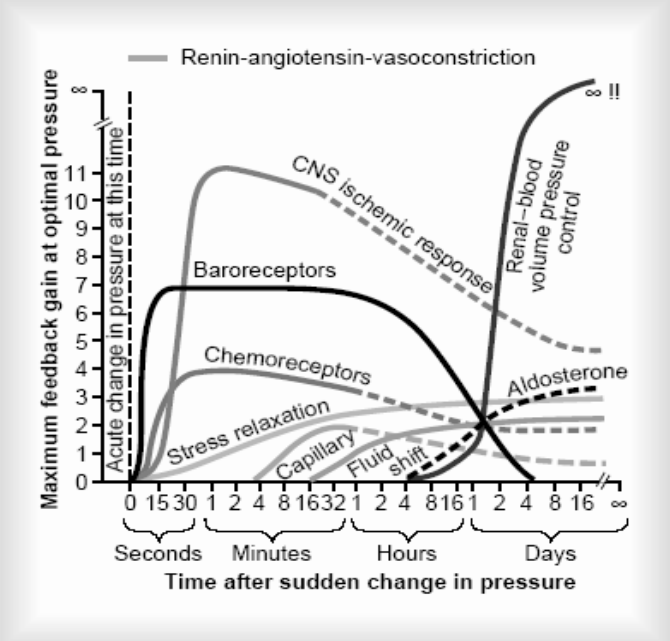


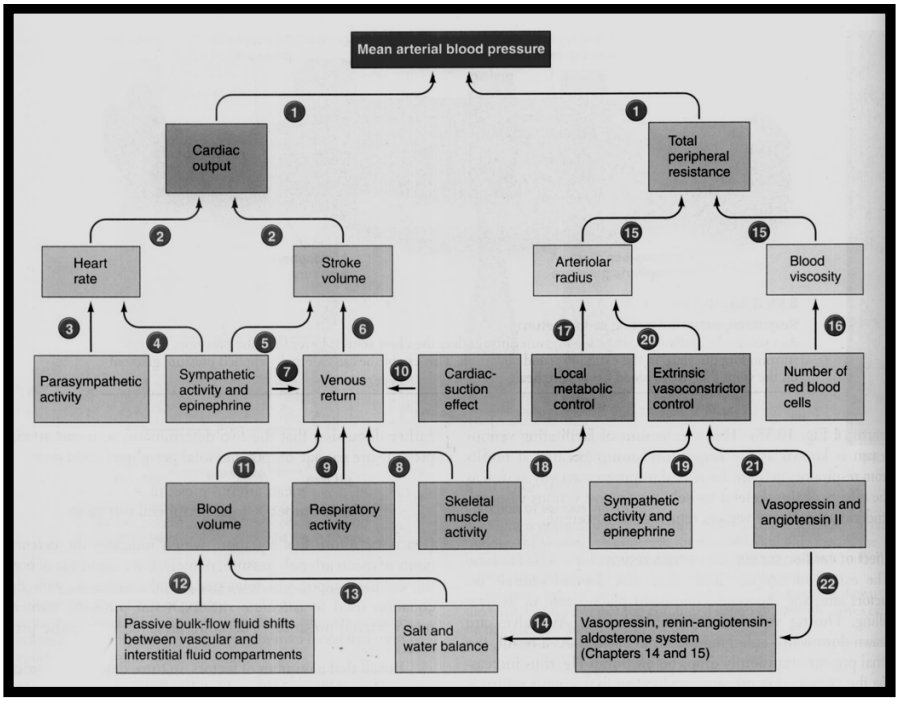
TABLE 21.2

Blood Pressure Regulation by Hormones

FACTOR INFLUENCING BLOOD PRESSURE	HORMONE	EFFECT ON BLOOD PRESSURE
CARDIAC OUTPUT		
Increased heart rate and contractility	Norepinephrine Epinephrine	Increase
SYSTEMIC VASCULAR RESISTANCE		
Vasoconstriction	Angiotensin II Antidiuretic hormone (vasopressin) Norepinephrine* Epinephrine†	Increase
Vasodilation	Atrial natriuretic peptide Epinephrine† Nitric oxide	Decrease
BLOOD VOLUME		
Blood volume increase	Aldosterone Antidiuretic hormone	Increase
Blood volume decrease	Atrial natriuretic peptide	Decrease

*Acts at α_1 receptors in arterioles of abdomen and skin.

†Acts at β_2 receptors in arterioles of cardiac and skeletal muscle; norepinephrine has a much smaller vasodilating effect.


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)

THANKS