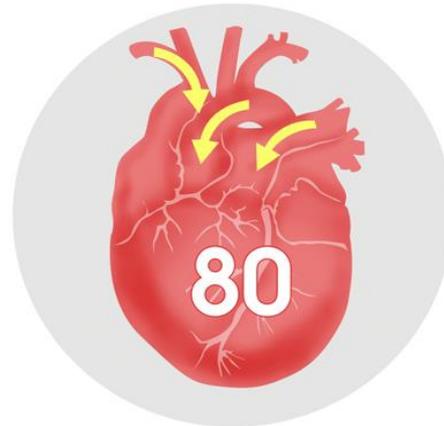


Regulation Of Arterial **Blood Pressure**



Systolic | Diastolic

The highest, normal blood pressure reading: 120/80

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1. Rapidly acting mechanism

(Nervous)

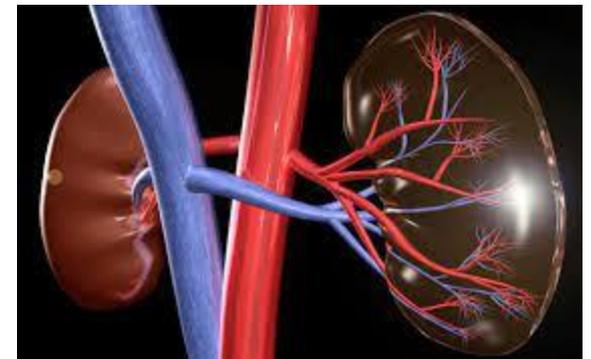


2. Intermediately acting mechanism



3. Slowly acting mechanism

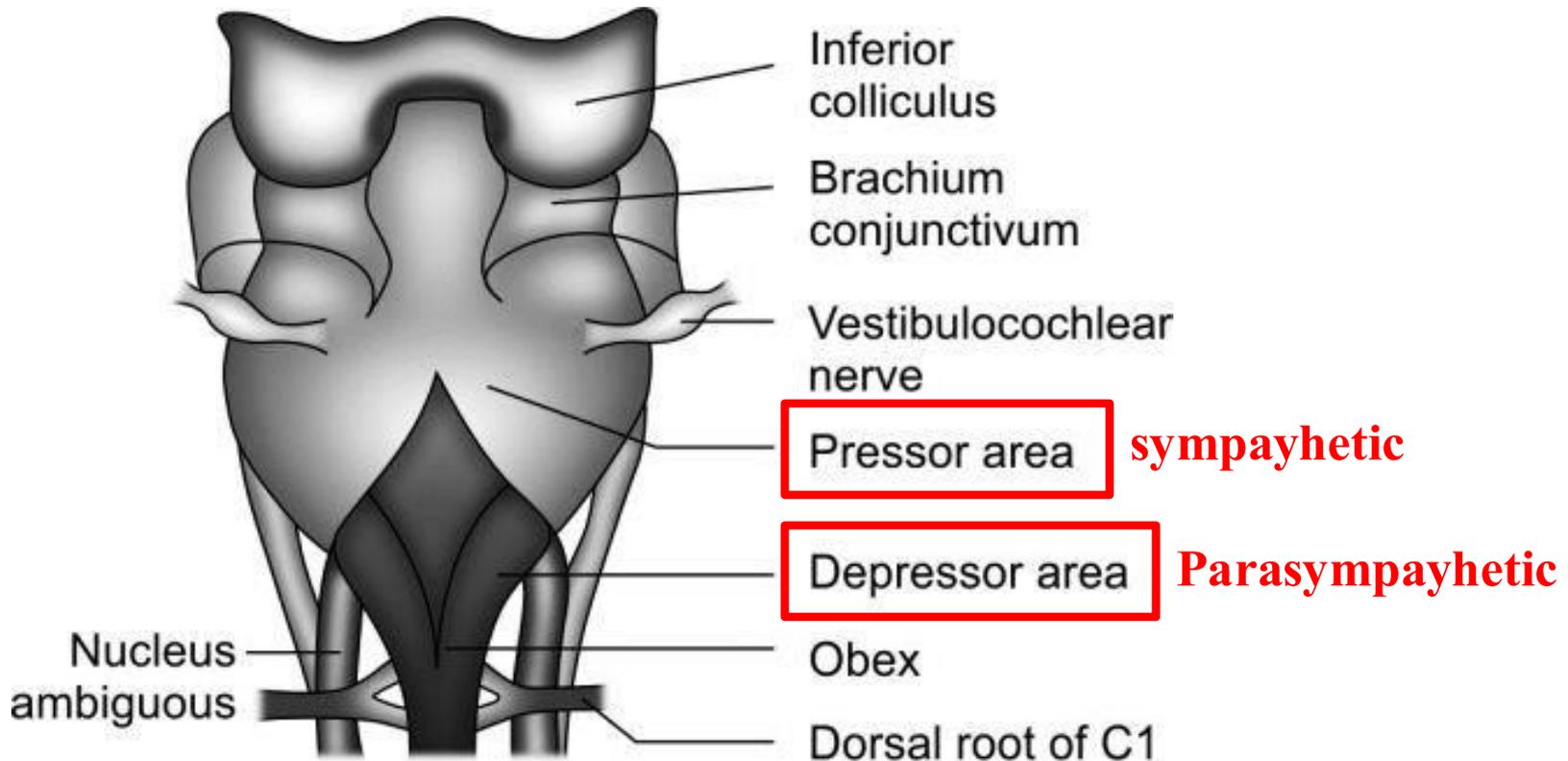
(Hormonal)



Rapidly Acting Mechanism

Nervous regulation

Medullary cardiovascular centers



Medullary Cardiovascular Centers

- **[A] The pressor area (vasomotor center - VMC)**

- Site: located in the ventrolateral part of medulla.
- Its neurons project downward and synapse with the sympathetic pre ganglionic neurons of the lateral horn cells of spinal cord.
- It is composed of

- (1) Vasoconstrictor center (VCC)**

During rest, it discharges continuously at moderate rate to the blood vessels via the sympathetic fibers, this is called
(vasoconstrictor tone)

Its stimulation produces:



(a) Vasoconstriction (VC) of the arterioles which increase the peripheral resistance (PR). So, increases the arterial blood pressure (ABP).

(b) Veno-constriction that leads to increase the venous return (VR), so that the cardiac output and arterial blood pressure are elevated .

(2) Cardiac stimulatory center (CSC)

- It discharges during rest but at low tone via the sympathetic nerve fibers to the heart this is called the sympathetic tone.
- its stimulation produces. ● Increase the heart rate. ● Increase the force of contraction which increase the stroke volume. ● Increased HR and SV lead to increase COP and ABP.



Medullary Cardiovascular Centers

- **[B] The depressor area**

- Site: It is located central and dorsal to the pressor area.
- It is composed of two centers.

(1) Vasodilator center (VDC)

- It sends inhibitory impulses to inhibit vasoconstrictor center (VCC) ⇒ VD
⇒ ↓ peripheral resistance ⇒ ↓ ABP.

- **(2) Cardiac inhibitory center (CIC)**

- It discharges continuously at moderate rate via vagal fibers to the heart
- This is called **vagal tone on SAN**.

Functions:

1. It decreases the heart rate.

2. Stimulation of the **depressor area** leads to **decrease** of **arterial blood pressure** due to:

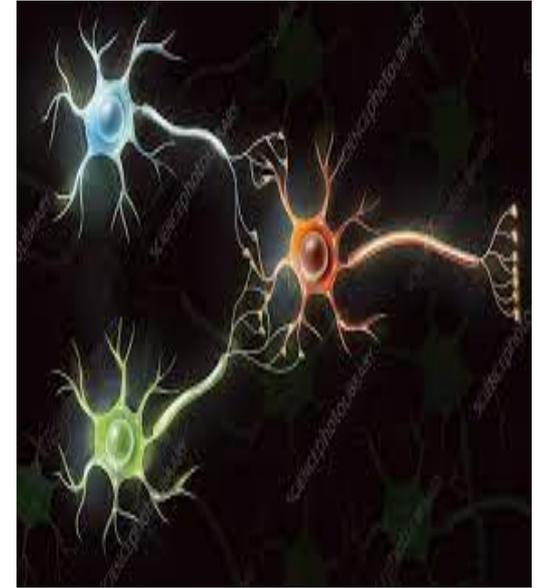
- (a) Decrease the peripheral resistance as a result of vasodilatation.
- (b) Decrease in the cardiac output as a result of decreased heart rate.





- There is a reciprocal innervation between the pressor and the depressor areas.
- The dominant centers are VCC & CIC.
- Impulses which stimulate the pressor area, also stimulate the medullary respiratory area and vice versa.

**The activity of cardio-vascular centers
is modified by afferent impulses from:**



- I. Receptors in the cardiovascular system
- II. Receptors outside the cardiovascular system
- III. Higher centers
- IV. Blood gases

Impulses From Cardiovascular Receptors

Cardiovascular receptors

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graph TD; A[Cardiovascular receptors] --> B[Baroreceptors]; A --> C[Chemoreceptors]; B --> D[These Receptors Are Stimulated By Blood Pressure Variations & Are Divided into:]; D --> E[High P. Arterial]; D --> F[Low P. Atrial]; D --> G[Ventricular]; C --> H[Peripheral]; H --> I[Coronary chemo-reflex (Bezold-Jarish reflex)]; H --> J[Pulmonary (juxtacapillary) receptors (j-receptors)];
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Baroreceptors

These Receptors Are Stimulated By Blood Pressure Variations & Are Divided into:

High P.

Arterial

Low P.

Atrial

Ventricular

Chemoreceptors

Peripheral

Coronary chemo-reflex
(**Bezold-Jarish reflex**)

Pulmonary (juxtacapillary)
receptors (**j-receptors**)

[A] Baroreceptors (pressor-receptors) (mechano-receptors)

1) Arterial baroreceptors

- Site: **Aortic arch & carotid sinus**
- Nerve connection: **Buffer Nerves**
- Stimulus is stretch of arterial wall by the blood pressure changes

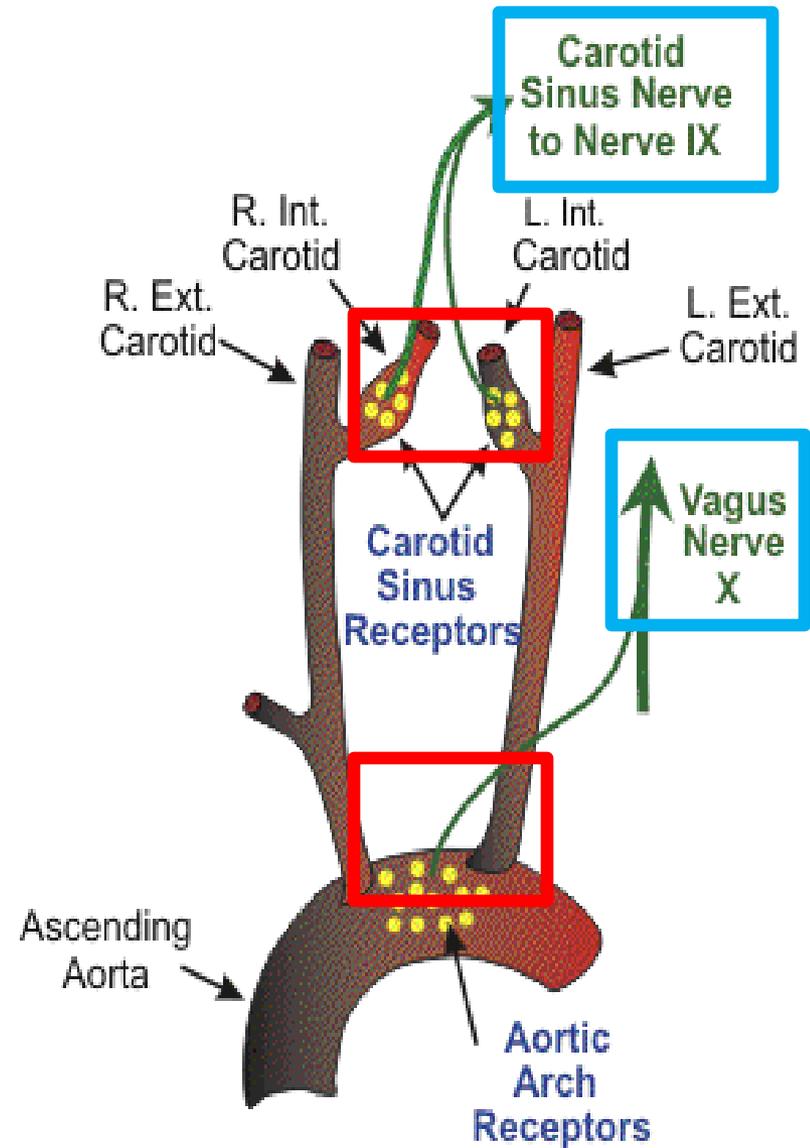


Figure 1. Location and innervation of arterial baroreceptors.

a. Arterial blood pressure

- They are stimulated by changes in blood pressure ranges from **60-180** mmHg.
- Below 60 mmHg → no discharge
- At 180 mmHg → maximal discharge.
- above 180 mmHg → no further increase in the rate of discharge.

b. Pulse pressure (systolic pressure-diastolic pressure)

- They respond to the pulse pressure changes, so their rate of discharge increases during systolic rise of the blood pressure & decreases during the diastolic fall of blood pressure.

Functions:

1. **Discharge continuous inhibitory impulses during rest.**
2. **Buffering action on the ABP** (i.e. if blood pressure increases or decreases, they act to return it to the normal level)

- **If ABP increases:**

↑ **inhibitory signals to the pressor area leading to:**

(a) Arteriolar dilatation → ↓ peripheral resistance → ABP to normal.

(b) Veno-dilatation → decreased VR → decreased COP → decreased ABP.

(c) ↓ heart rate leading to decrease COP and ABP.

- **Marey's reflex (Marey's law)**

A rise of the arterial blood pressure (ABP) leads to a decrease of the heart rate and vice versa, provided other factors affecting the heart rate remain constant".

It is initiated by stimulation of the **arterial baroreceptor**

Effects of stimulation of arterial baroreceptors:

- (1) Stimulation of both the CIC (resulting in reflex bradycardia) as well as the VDC (resulting in generalized V. D. and hypotension).
- (2) Inhibition of the respiratory center (resulting in temporary apnea).

➤ **Carotid sinus syndrome:**

In some people the carotid sinus reflex is very sensitive so that a slight pressure on the carotid sinus leads to great drop in the ABP and fainting as a result of cerebral ischemia. It is treated by denervation of the carotid sinus.

➤ **Nervous mechanism of essential hypertension:**

Resetting of the arterial baroreceptors to a new higher level may be the cause of essential hypertension.

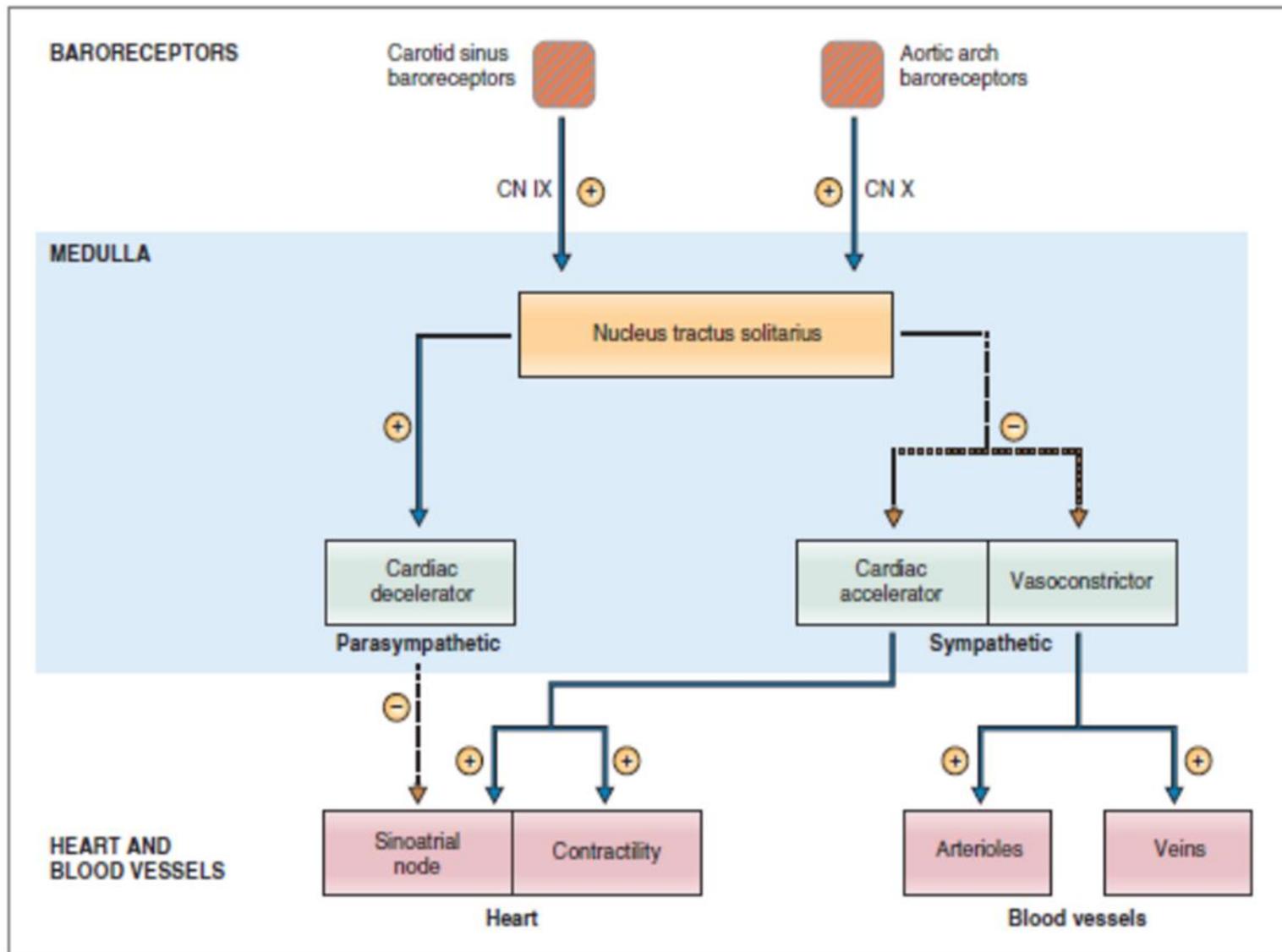


Fig. 4.31 Response of baroreceptor reflex to increased arterial pressure. The + symbol shows increases in activity; the - symbol shows decreases in activity; the dashed lines show inhibitory pathways. CN, Cranial nerve.

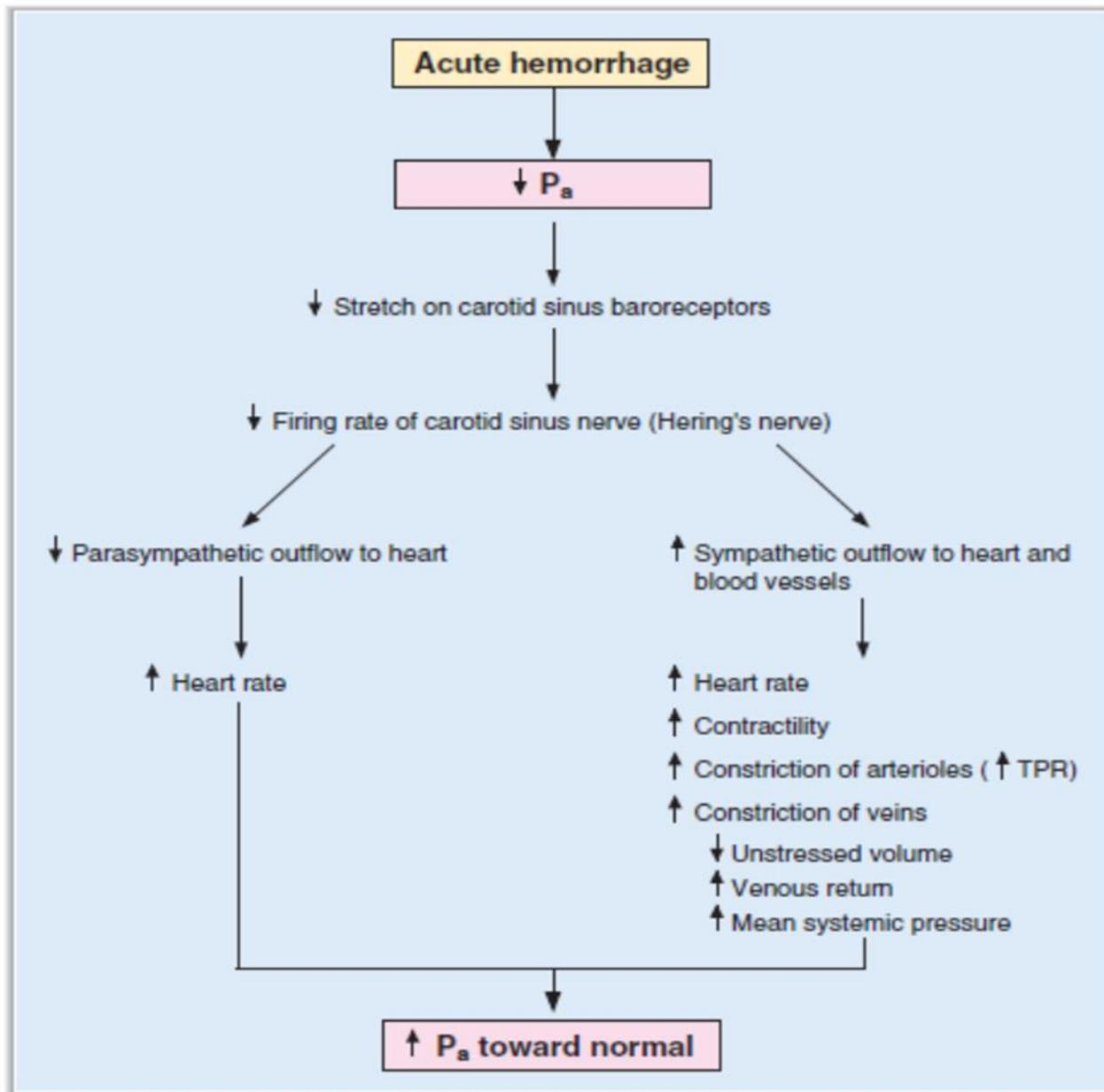


FIGURE 3.16 Role of the baroreceptor reflex in the cardiovascular response to hemorrhage. P_a = mean arterial pressure; TPR = total peripheral resistance.

2) Atrial baroreceptors

- Site In the wall of both atria near the venous openings.
- Nervous connection: Vagus nerve.
- **Types:**
 - Type A :** Discharges during atrial systole.
 - Type B :** Discharges late in diastole (by atrial filling)
- Stimulus: by ↑ **CVP** (so also called volume receptors)

- **Functions:**

↑ VR → ↑ CVP → ↑ discharge from atrial receptors to:

a. Inhibition of VCC leading to:

- Arteriolar dilatation (↓ PR) → ↓ ABP

-Veno-dilatation → ↓ VR → ↓ central venous pressure → ↓ COP → ↓ ABP

b. Inhibition of secretion of ADH and aldosterone (+↑ ANP) →

increased excretion of Na⁺ and water in urine decreased blood volume → ↓

central venous pressure → ↓ VR → ↓ COP → ↓ ABP

Bainbridge reflex

Bainbridge effect

$\uparrow \text{CVP} \rightarrow \uparrow \text{HR}$



Bainbridge reflex:

$\uparrow \text{RAP} \Rightarrow \uparrow \text{HR}$.

Mechanism:

$\uparrow \text{RAP} \Rightarrow$ stimulates atrial baroreceptors \Rightarrow sends impulses via vagus nerve to stimulate the CSC \Rightarrow tachycardia ($\uparrow \text{HR}$).

Bainbridge effect:

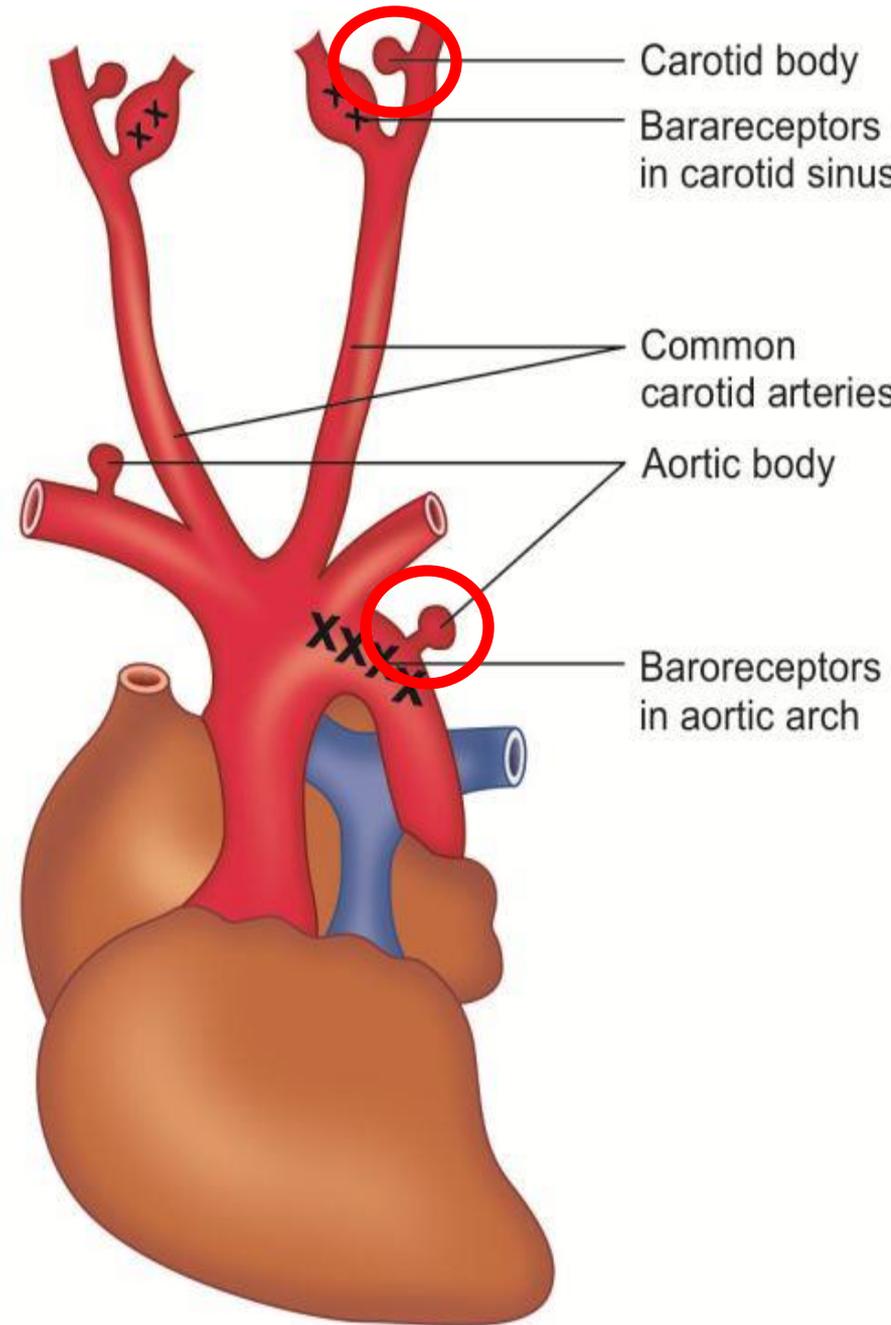
Some authors believe that the tachycardia occurring during the increased right atrial pressure is resulting from local stretch of SAN.

So, $\uparrow \text{VR} \Rightarrow \uparrow \text{HR}$ (tachycardia)

[B] Chemoreceptors:

1) Peripheral chemoreceptors:

- **Site: Aortic body & Carotid body**
- **Nervous connection : The buffer nerves**
- **Stimuli:**
 - - Hypoxia (the main stimulus)
 - - Hypercapnia
 - - Acidosis



Function

-It sends excitatory impulses to stimulate the pressor area and inhibit the depressor area leading to increased ABP.

-Its main function is stimulate the **respiratory centers**.

2) The pulmonary chemo-reflex

Distension of the pulmonary vessels as in pulmonary embolism or congestion → **reflex** hypotension, bradycardia and apnea.

3) Coronary chemo-reflex: (Bezold-Jarish reflex)

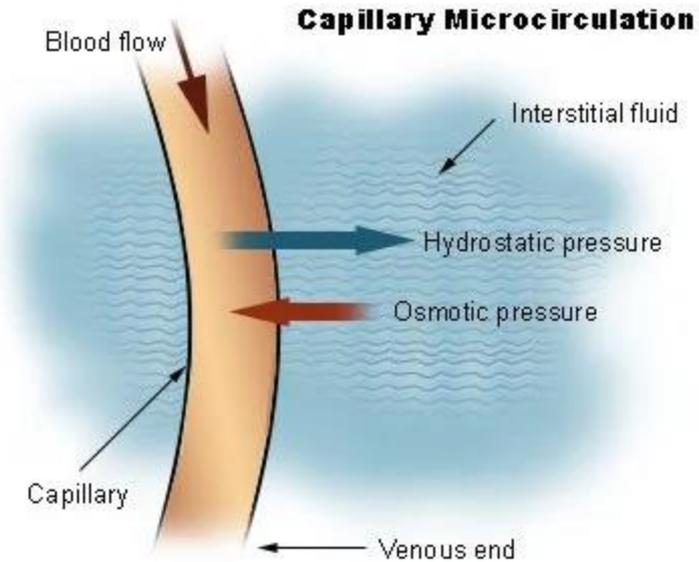
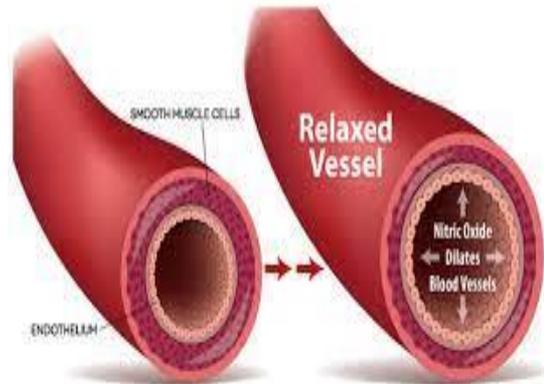
- **Site :** Coronary vessels
- **Afferent:** Vagus nerve
- **Stimuli:** Chemicals released from myocardial infarction.
Injection of serotonin (VC of coronaries)
- **Response : Reflex**
 - ↓ ABP.
 - ↓ HR.
 - ↓ Respiration(apnea)

Intermediate mechanisms

1-Capillary fluid shift mechanism

2-Stress and reverse-stress

relaxation



3-Thirst sensation



1. Capillary fluid shift mechanism

-The intermediate mechanisms begin to act within a few minutes, and reaching full function within a few hours.

-Any changes in the arterial pressure lead to similar changes in the capillary hydrostatic pressure which in turn affects the rate of filtration and reabsorption.

(1) \uparrow ABP \rightarrow \uparrow capillary hydrostatic pressure \rightarrow \uparrow fluid filtration \rightarrow \downarrow blood volume \rightarrow \downarrow CVP \rightarrow \downarrow venous return \rightarrow \downarrow cardiac output \rightarrow the blood pressure return to normal.

(2) \downarrow ABP produces shift of fluid from tissue fluid to plasma so tissue fluid acts as a reservoir for the plasma.

2. Stress and reverse-stress relaxation

-Pressure change causes the vessels gradually to adapt to a new size, thereby accommodating the available amount. The phenomenon is called stress relaxation or reverse stress relaxation.

-Massive transfusion leading to increase in blood pressure at first but because of relaxation of the circulation during the next ten minutes to an hour return nearly to the normal even if the blood volume is 30% above normal.

-Reverse stress relaxation occurs when there is a blood loss (it's limit is only 15% blood loss).

3- Thirst sensation

-In cases of fluid or blood loss volume receptors in the right atrium stimulate thirst center in the hypothalamus → ↑ water intake → ↑ blood volume → restoration of blood volume and ABP.

Slowly Acting Mechanisms (Role of kidney)

1- Pressure Diuresis

Increased ABP → increased filtration force in the kidney → more urine excretion → decreased blood volume → ABP decreased back to normal.

Decreased ABP as in hemorrhage and shock → decreased urine formation → preservation of blood volume and blood pressure.

2- Renin- angiotensin system

Decreased blood pressure (dehydration, hemorrhage) → ↓renal blood flow → renal ischemia → juxtaglomerular apparatus secret renin which act on **alpha globulin** in the plasma called **angiotensinogen** producing **angiotensin I** which is converted to **angiotensin II** by the angiotensin convertase enzyme in the lung.

Angiotensin II has the following effects:

1-Strong arteriolar VC (50 times as noradrenaline) leading to increased peripheral resistance and blood pressure.

2-Stimulation of aldosterone release from the suprarenal gland → salt and water retention→ increase blood volume, COP and blood pressure.

3-Stimulation of ADH (vasopressin) secretion from the pituitary gland → water retention and increase blood pressure .

4-Stimulate NA release from postganglionic sympathetic fibers.

5-Stimulate thirst sensation→↑ water intake→ ↑Blood volume →↑ ABP.

6-Stimulate salt and water retention by the kidney → Increase blood volume and blood pressure.

THANK YOU

