Physiology Team 431

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Arterial Blood Pressure

- ## Physiological variation in arterial blood pressure:

BP range: 90-140/60-90 mmHg.

1. **Age:**
    a. **At birth: 50/30**
    b. **Adult : 120/80**
    c. **Old age 170/90**
2. **Sex: males have higher BP than F before menopause.**
3. **Body built: increase in obese.**
4. **Emotions (↑ BP)**
5. **Exercise (↑ BP)**
6. **Meals. (↑ BP)**
7. **Sleep (↓ BP)**
8. **Gravity:**

****The gravity increases as it goes down and it decrees as it goes up. And so on the pressure.**

So if the pressure at the heart level is 100mmhg

Above that level the pressure in any vessel will decrease and below it the pressure will increase due to effect of Gravity.

And for every 50 cm above the heart level or either below it there is a change in the pressure equals 0.77 mmHg/cm at the density of normal blood.

In adult human in upright position, if mean BP at heart level = 100 mmHg, the mean pressure

1. **in an artery at the head (50 cm above heart) = 100-[0.77X 50] = 62 mmHg**
2. **in an artery (50 cm below the heart)= 100+(0.77 * 50)= 138.5mmhg**

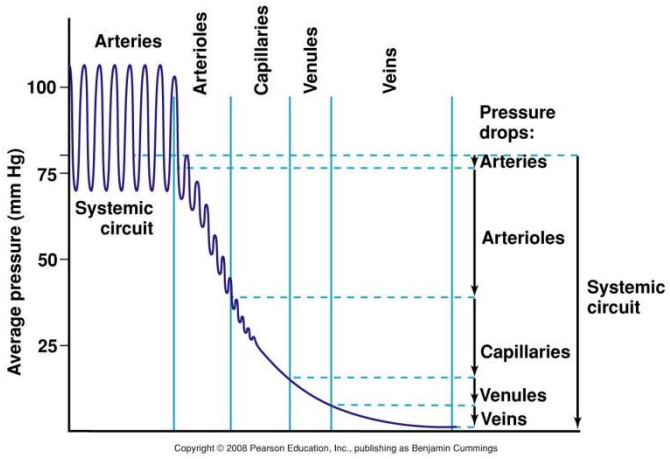
Blood pressure:

Some rules:

- **Pulse pressure:**

PP = SP-DP

- **Mean arterial blood pressure (MABP)**



MABP = Diastolic + PP/3

CO = ABP

TPR

ABP = CO X TPR

Determinants of arterial blood pressure:

1-Cardiac output:

ABP = CO X TPR Increase in CO lead to an increase in ABP

Decrease in CO decrease ABP

- **CO= HR X SV**

ABP= HRxSVxTPR

Heart rate, stroke volume and peripheral resistance affect MABP

**** the ABP can be affected by the heart rate only if there is extreme changes in heart rate, because it will lead to decrease in CO**

****during excursive there is an increase in HR and stroke volume**

**** When the ABP decrease, the heart compensate increase the BP by increase in the heart rate to pump more blood in order.**

2- Elasticity of blood vessels: Changes in great vessels elasticity affect BP.

Lose of the elasticity means that the artery can't recoil during diastole or to distend during systole.

Atherosclerosis makes blood vessel like a tube, so during systole as blood is ejected into the arteries, they don't distend and pressure increases significantly. And during the diastole the pressure will decrease significantly because they can't recoil.

3- Blood volume:

An increase in blood volume → ↑CO → increase ABP.

A decrease in blood volume as in Hege, dehydration → decrease VR → decrease CO decrease ABP.

4-Total peripheral resistance:

APB is directly proportional to TPR

TPR is determined by:

1. Diameter of blood vessel (r).

2. Blood viscosity:

a. Red cells

Polycythemia increases viscosity.

b. Plasma proteins:

Hypoprotenimia decreases viscosity.

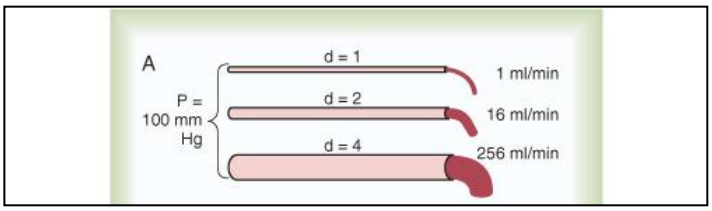
Total peripheral resistance:

- **Change in blood vessels' diameter by increase or decrease will affect blood pressure.**
- **TPR Is inversely proportional to blood vessel diameter (r) :**
- **R α 1/r4 (it means that the resistance will increase 4 times if the diameter decreased.**
- **If r is doubled, TPR is reduced by 16, and so on……..**
- **Factors affecting diameter of arterioles (check previous lecture)**

If the diameter decreases → the TPR increase → the ABP increase fourth times as the diameter.

If the diameter increases → the TPR decrease → the ABP decrease fourth times as the diameter.

TPR and vessel diameter



- **Slight changes in the diameter of a vessel cause tremendous changes in the vessel's ability to conduct blood when the blood flow is streamlined**
- **Although the diameters of these vessels increase only fourfold, the respective flows are 1, 16, and 256 ml/mm, which is a 256-fold increase in flow. Thus, the conductance of the vessel increases in proportion to the *fourth power of the diameter***

Factors affecting vessel diameter:

⊡ Vasodilator agents:

- **Nitric oxide.**
- **Histamine.**
- **Atrial natriuretic peptide (ANP).**
- **Prostacyclin**

⊡ Vasoconstrictor agents:

- **Norepinephrine.**
- **Angiotensin II.**
- **Vasopressin.**
- **Endothelin-1**
- **Thromboxane A.**

Why is it important to control blood pressure?

Blood pressure is a key factor for providing blood (thus oxygen and energy) to organs.

SBP must be a minimum of 70 to sustain kidney filtration and adequate blood flow to the brain

Neural control medullary CVCs

The vasomotor center integrates all these information

The vasomotor sends decision to the ANS center:

- **Both parasympathetic and sympathetic innervate the S/A node → can accelerate or slow down the heart rate**
- **The sympathetic NS innervates the myocardium and the smooth muscle of the arteries and veins → promotes vasoconstriction**

Information like decrease in ABP will be send to the vasomotor center via the glossopharyngeal and vagus nerves → vasomotor center sends decisions to the ANS center → parasympathetic and sympathetic nerves start to take actions.

Regulation of blood pressure

Short term | **intermediate** | **long term**

- a. Baroreceptor reflex.
- b. Chemoreceptor reflex.
- c. CNS ischemic response.
- d. Atrial reflexes.

Seconds to minutes

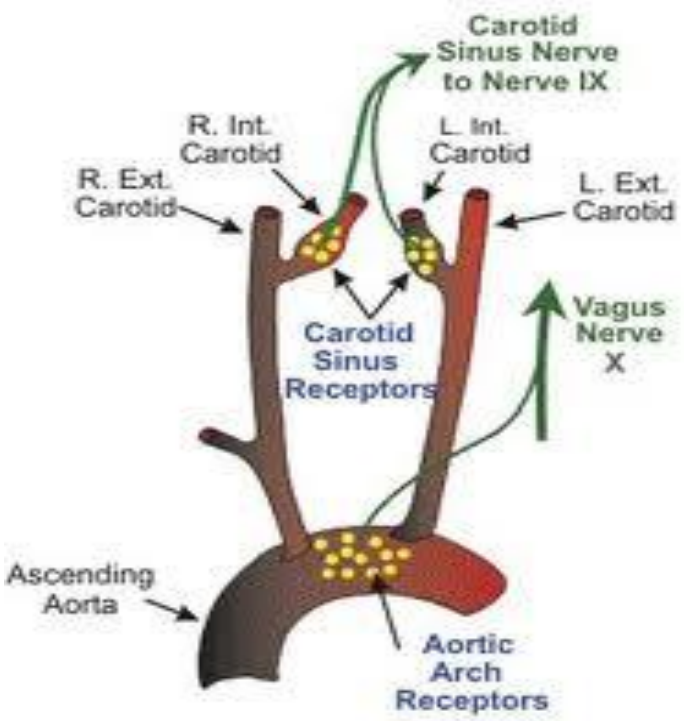
a. Baroreceptor reflexes:

- Baroreceptors: Stretch receptors.
  Located in: Carotid sinus and aortic arch
- They sense the blood pressure in the aortic arch and internal carotid → send signal to the vasomotor center in the medulla oblongata along vagus and glossopharyngeal Ns.
- They respond to a rapidly changing BP. In the range 60-180 mmHg.

(They only work when the pressure is in this range)

Stretch receptors are mechanoreceptors responsive to distention of various organs and muscles, and are neurologically linked to the medulla.

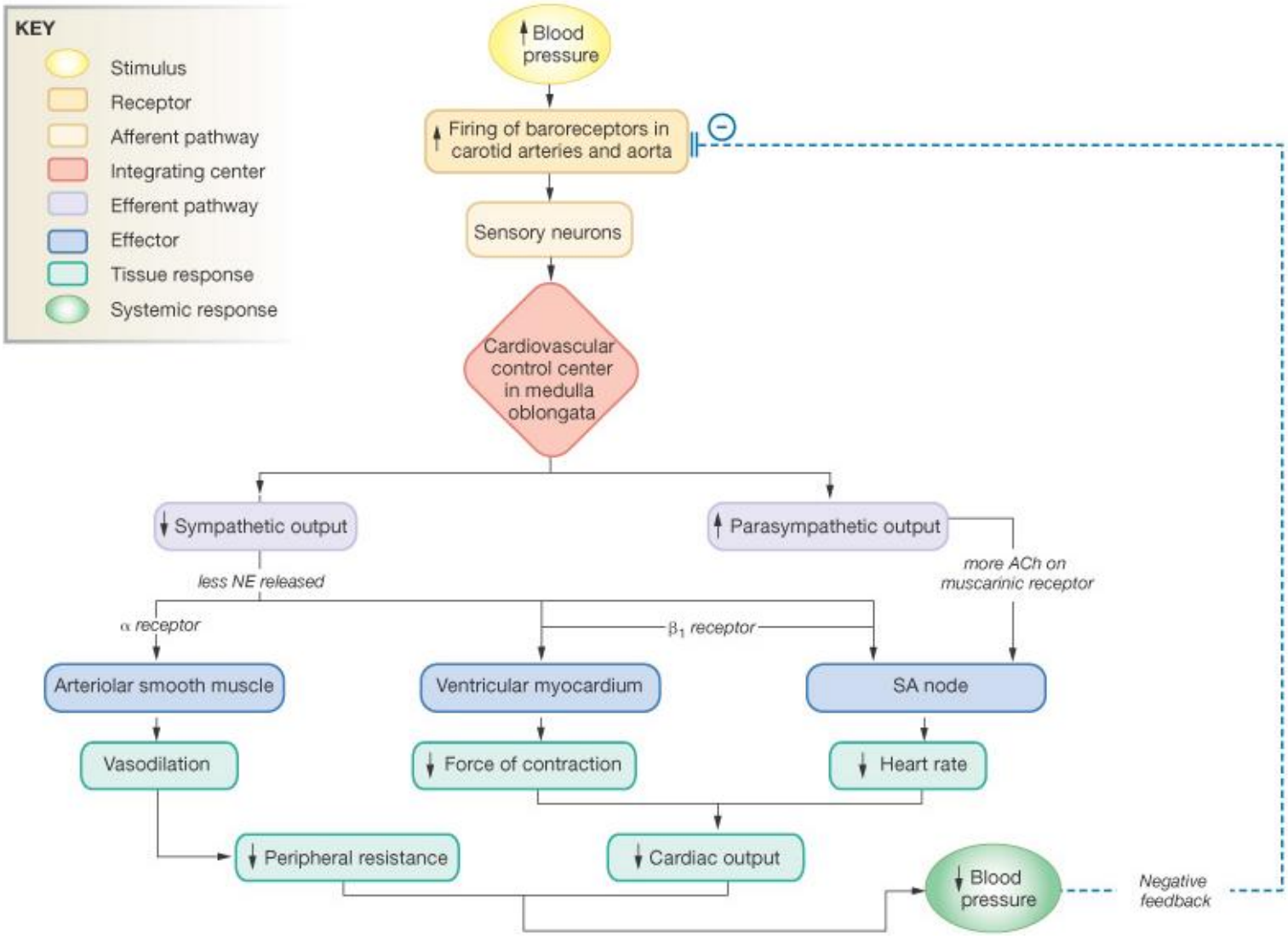
Baroreceptors sense the stretching in the vessel when the blood flow is increased (increased BP)(They themselves are stretching due to increase BP)



Reflexes initiated by baroreceptors:

- ↑ABP → Stretch of receptors ↑ rate of firing and impulses travel along vagus & glossopharyngeal to the medullary Cardiovascular centers (CVCs) :
  - a. (+) vagal center : ↓ HR. (Parasympathetic)
  - b. (-) vasoconstrictor center: Vasodilation (sympathetic inhibition)
- ↓ABP → inhibitory impulse discharge from baroreceptors → vasomotor center is released from inhibition resulting in:
  - a. (+) Heart: HR & contractility.
  - b. (↑) Sympathetic VC tone: Vasoconstriction

Baroreceptors have inhibitory response to sympathetic system



- Baroreceptors are important in maintaining ABP constant during changes in body posture:
- When you change your posture from superior (supine) to erect, a drop in ABP in the head and upper part of the body will occur.
- As baroreceptor reflex becomes activated, strong sympathetic impulses lead to VC and minimize the decrease in BP. That's why we don't faint

Resetting of baroreceptors (adaptation):

- This property makes baroreceptors not suitable for long-term regulation of ABP, as they are rapidly reset to the new pressure. They adapt rapidly to the new pressure level.
- Adaptation of a receptor means decrease in impulse discharge from the receptor despite persistence of the stimulus.

This means that: baroreceptors only respond to rapid changes, but if the BP is high and stays high for a long amount of time ,then baroreceptors will not be able to control this increase ; so we need another solution.

What is the effect of denervation (cutting the vagal and glossopharyngeal nerves) of baroreceptors?

↑ BP, because the baroreceptors' impulses will not reach the CVCs

b. Chemoreceptor reflex: (emergency reflex)

Chemo sensitive cells, stimulated in response to: O2 lack, CO2 excess, H+ excess.

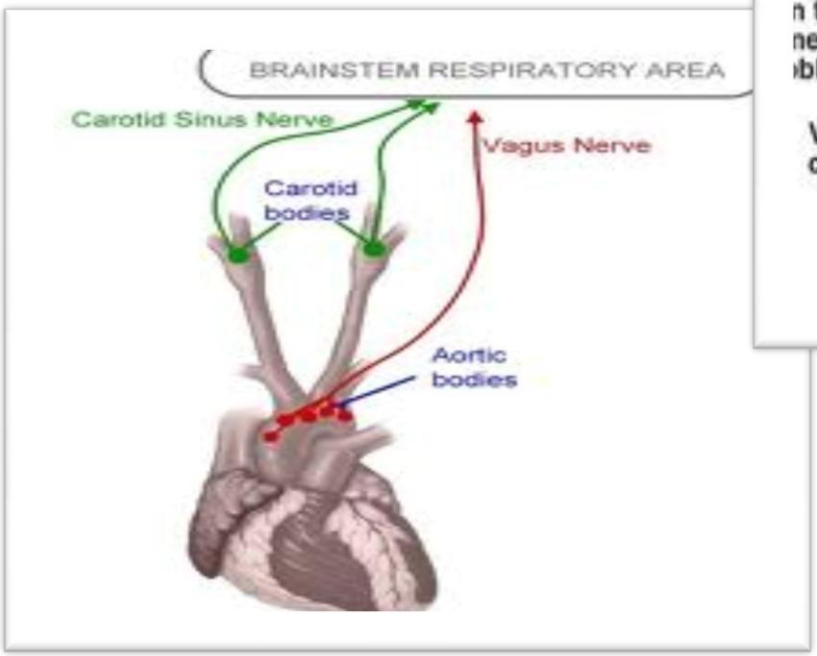
Located in carotid bodies & aortic bodies.

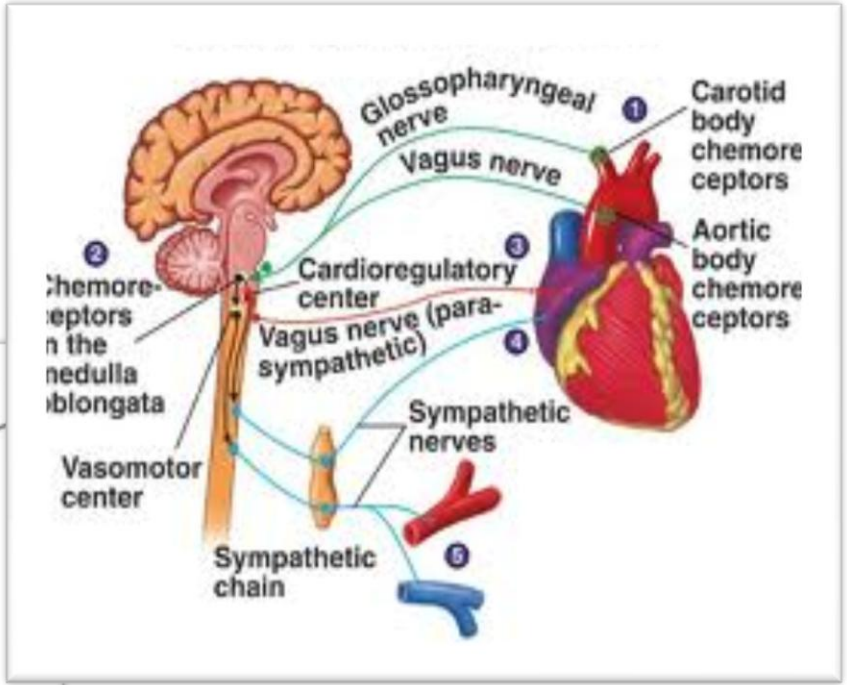
They have a very high blood flow (1200 ml/min/g tissue). This makes it easy for these cells to detect changes in O2, CO2, and H+.

Become activated when ABP becomes less than 60 mmHg. So, they are not involved in ABP control at normal range. When blood flow to chemoreceptors decreases it leads to ↓O2, ↑ CO2, ↑ H+ → (+) chemo. Signals (+) CVS → VC (+ sympathetic & - parasympathetic)

Chemoreceptors can work when there is a change in one of the chemicals even in normal BP.

e.g. ↑ H+ (acidosis) without changing in blood pressure → chemoreceptors respond. But when BP is < 60 then ↓ O2 , ↑ CO2, ↑ H+ and chemoreceptors will respond → tachycardia, tachypnea and vasoconstriction.





c. CNS ischemic response:

- It operates as an emergency arterial pressure control system that acts rapidly and powerfully to prevent further decrease in ABP whenever blood flow to the brain decreases to lethal level.
- It is one of the most powerful activators of the sympathetic vasoconstrictor system.
- When BP < 20 mmHg (in shock) → cerebral ischemia of vasomotor center → strong excitation of vasomotor center (due to accumulation of CO_2, lactic acid,….) → strong VC of blood vessels including the kidney.

If ↑perfusion of the brain → CVCs ischemia → CVCs excitation → sympathetic stimulation → VC and tachycardia

d. Atrial Reflexes:

- Receptors: Low pressure receptors especially in the Right Atrium.
- Respond to changes in blood volume.
- What happen if blood volume is increased? E.g. infusing 500 ml into a person

blood volume → stretch of the atria leading to:

a. (+) ANP release → VD of renal vessels, diuresis, natriuretic (Na &H_2O excretion)

b. Hypothalamus:

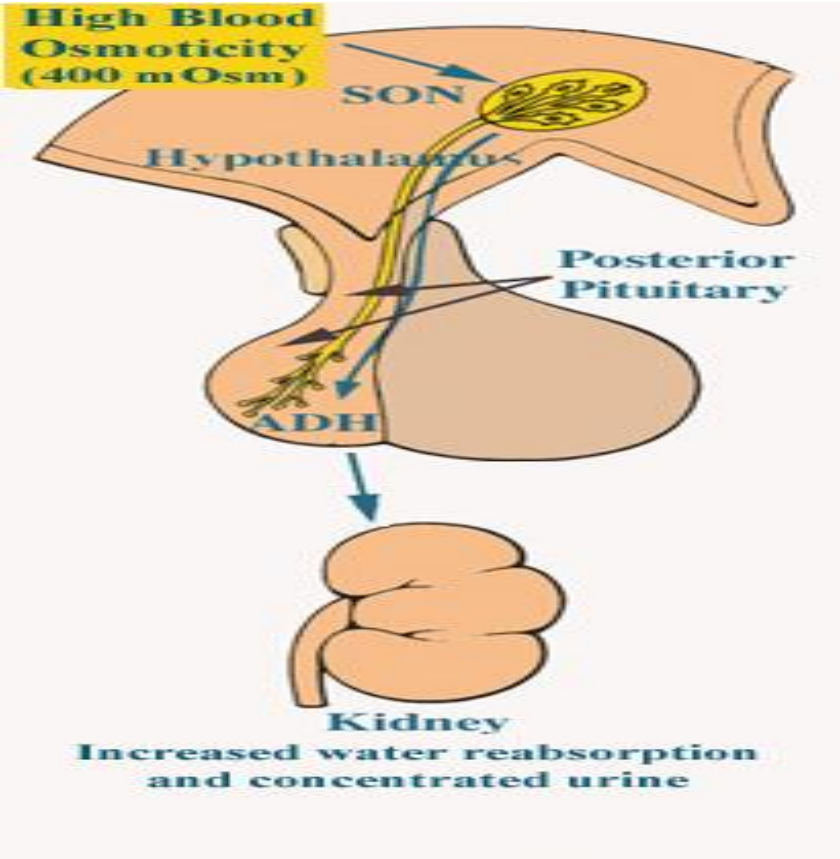
1. (-) ADH → water diuresis.
2. (-) Sympathetic discharge → VD of renal vessels

c. stretch SAN and increase HR

to understand:

Anti-diuretic hormone = ADH

- **Secreted by the posterior pituitary in response to ↑blood osmolarity (often due to dehydration)**
- **Promote water reabsorption by the kidney tubules → H2O moves back into the blood → less urine formed**



What happens if there is sudden loss of blood volume by 800 ml?

The opposite of the above???????????

The body will stimulate releasing of ADH to remaintain blood volume.

Intermediate regulation of ABP

The Capillary fluid shift mechanism:

refers to the exchange of fluid that occurs across the capillary membrane between the blood and the interstitial fluid. This fluid movement is controlled by the capillary blood pressure, the interstitial fluid pressure and the colloid osmotic pressure of the plasma.

Low blood pressure results in fluid moving from the interstitial space into the circulation helping to restore blood volume and blood pressure & vice versa.

Long-term regulation of ABP

role of the kidney

- **The kidneys helps to regulate the blood pressure by:**
- **-Pressure diuresis.**
- **- Pressure natriuretsis.**
- **- Renin-angiotensin system.**

It regulates blood volume increasing or decreasing the blood volume and also by the renin-

When there is sympathetic stimulation or hypotension or decrease Na delivery the kidney will synthesize renin. Renin will produce eventually angiotensin II renal Na & fluid retention

Aldosterone secretion (works on Na), thirst

Systemic vasoconstriction

If there is increase in Na

vasodilation (to get rid of excess Na and fluid)

0

angiotensin system.

They are the most important organs for the long-term control of blood pressure.

**SBP is dependant on CO and DBP is dependant on TPR. TPR is responsible for DBP and forward flow of blood

**Max is in arterioles because of max smooth Ms and rich innervation by sympathetic

**RBC, PLASMA PROTEINS (Albumin), Eg Polycythemia viscosit is high so high DBP and in Anemia SBP is high

**The Arteries and Arterioles are the Pulsatile vessels. The drop in the arteriolar pressure depends on 1-dilatation: if the arterioles are dilated so there is high flow of blood to them from arteries so reduce the drop in pressure (Min.Drop) but If 2- Constriction so no significant amount of blood flowing in the arteriol so will contribute in increasing the drop (Max. Drop).

**Nervous Regulation of Circulation The Autonomic Nervous SystemSympathetic Vasoconstrictor Tone (Vasomotor Tone)Control of Vasomotor CenterBaroreceptor Control System
in most tissues all the vessels *except the capillaries, precapillary sphincters, and metarterioles* are **innervated by sympathetic nerves and No Parasympathetic**

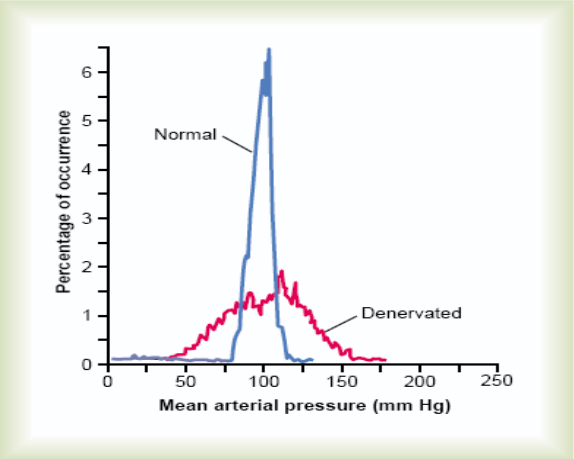
*** The short term regulation is done by mean of nervous control of BP which is the fastest control mechanism in the body.

**Baroreceptors are located in the Aortic sinus in arch of aorta and in carotid sinus in the internal carotid arteries slightly above common carotid bifurcation. It is important to know that the baroreceptors are responding in pressure changes from 60 mmHg – 180 mmHg and lower this level they will not work and also above this level. Even more they will readjust the ABP if it is slightly above or lower the normal range rapidly.

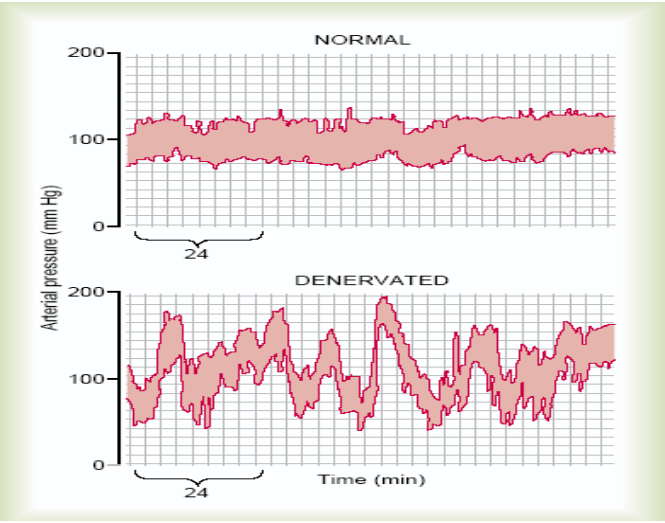
*** This figure shows that signals from the "carotid baroreceptors" are transmitted through very small *Hering's nerves to the glossopharyngeal nerves in* the high neck, and then to the *tractus solitarius(sensor area) in the* medullary area of the brain stem. Signals from the"aortic baroreceptors" in the arch of the aorta are transmitted through the *vagus nerves also to the same* tractus solitarius of the medulla.

*** the sympathetic NS is continuously innervating the Blood vessels, so if there is no sensor for blood vessles constricting as baroreceptors so no inhibition of sympathetic activity so raising in BP.

**Pressure on the carotid sinus, produced, for example by the tight collar or carotid massage can cause vasodilatation, marked bradychardia, and fainting. Because they will sense an increase in the pressure but not a blood pressure actually so they will stretch causing inhibition for symp.system (Feedback).



*** Note that when the baroreceptors were functioning normally the mean arterial pressure remained throughout the day within a narrow range between 85 and 115 mm Hg—indeed, during most of the day at almost exactly 100 mm Hg. Conversely, after denervation of the baroreceptors, the frequency distribution curve became the broad, low curve of the figure, showing that the pressure range increased 2.5-fold, frequently falling to as low as 50 mm Hg or rising to over 160 mm Hg. Thus, one can see the extreme variability of pressure in the absence of the arterial baroreceptor system.



<u>buffer function**</u> of the baroreceptors. The upper record in this figure shows an arterial pressure recording for 2 hours , and the lower record shows an arterial pressure recording from baroreceptor nerves from both the carotid sinuses and the aorta had been removed. Note the extreme variability of pressure in the denervated dog caused by simple events of the day, such as lying down, standing, excitement, eating, defecation, and noises.