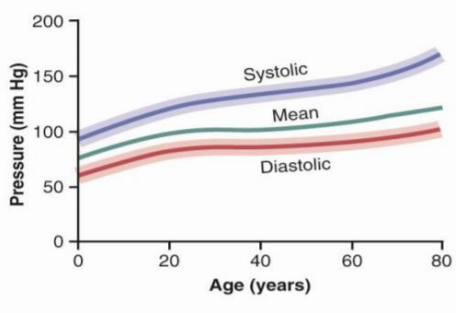
Subject | Physiology
Done by | Tala Saleh
Corrected by | Basil Al-Ramahi
Doctor | Faisal

Mean Arterial Pressure

- Recall, mean arterial pressure (MAP) is the **average** pressure in a patient's arteries during **one** cardiac cycle.

  ⇨ **MAP** = 1/3 Systolic pressure + 2/3 Diastolic pressure
  ⇨ **Pulse pressure** = Systolic pressure – Diastolic pressure
  ⇨ **MAP** = Diastolic pressure + 1/3 Pulse pressure



- Factors mainly affecting the pressure are **cardiac output** and **total peripheral resistance** according to the formula: **MAP = CO x TPR.**

  ⇨ Arterial Pressure can be **increased** by:

  1- **Constricting** almost all **arterioles** of the body which **increases** total peripheral resistance.
  2- **Constricting** large vessels of the circulation thereby **increasing** venous return and cardiac output.
  3- **Directly increasing** cardiac output by **increasing** heart rate and contractility.

Measuring Blood Pressure (Auscultatory method):

- A **sphygmomanometer** (mercury/electronic manometer) is a device used to measure blood pressure, composed of an inflatable **cuff** to collapse and then release the artery under the cuff in a **controlled** manner.

1- The cuff is placed around the **upper arm** above the cubital fossa, with the stethoscope on the **antecubital artery** (not the vein because veins have a very weak pulsation).

2- Pressure in the cuff is **raised above** the **systolic** pressure **stopping** blood flow, thus **no pulsation** will be heard.

3- We start **decreasing** the pressure of the cuff. As the pressure decreases, a pounding **sound** is heard when the blood flow **starts** again in the artery. The pressure at which this sound began is noted and recorded as the **systolic blood pressure**.

   ⇨ The sound indicates that there is **blood flow** during **systole**; because the manometer pressure now is equal to or less than the systolic pressure → The sound heard is caused by the **turbulent** flow of blood → The turbulent flow is caused by arterial **constriction** which is caused by the manometer cuff.

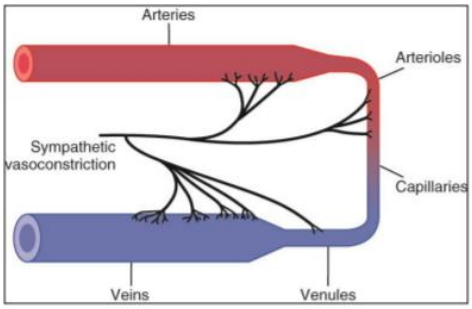
4- The cuff pressure is further released until the sound can **no longer be heard;** because the flow becomes **laminar,** not turbulent. The pressure at which the sound disappears is equal to the **diastolic pressure**.

- **In conclusion:** the **beginning** of sound marks the **systolic** pressure and the **disappearance** of the sound marks the **diastolic** pressure.

> ***Note:*** ***Auscultatory method*** *is the* ***most*** *commonly used method for measuring both* ***diastolic*** *and* ***systolic*** *pressure. In the* ***palpatory*** *method, only* ***systolic*** *pressure is measured.*

Blood Vessels and the Autonomic Nervous System

- **The sympathetic** nervous system is important in **controlling the circulation**, while the **parasympathetic** is mainly important in regulating **heart function** via the vagus nerve.
- **Parasympathetic** nerves **do not** supply blood vessels. **Almost** all blood vessels are **only** innervated by the **sympathetic** nervous system **except capillaries** *(since they lack smooth muscles)*, **precapillary sphincters**, and some **metarterioles**.



- Normally without any stimulation, there is a **basal tone** of the vessels; in which there is basal **sympathetic** outflow causing the vessels to be **semi-constricted**. Therefore, **increasing** the sympathetic outflow leads to **vasoconstriction**, and **reducing** the sympathetic outflow leads to **vasodilation**.

Blood Pressure Regulation

Blood pressure can be regulated through:

1- **Short-term regulators:** these regulate pressure through neural mechanisms acutely; in minutes or seconds. **Example:**
   - **a-** High-pressure baroreceptors
   - **b-** Low-pressure baroreceptors
   - **c-** Chemoreceptors

2- **Intermediate regulators:** work in terms of hours to days through chemicals.

3- **Long-term regulators:** they regulate pressure in long periods (chronic) through chemicals; in terms of days, weeks, or months. They usually work through the renal system and body fluid.
   Example: Renin-Angiotensin Aldosterone system.

'In this sheet, we will discuss short-term regulation'

1- High-pressure Baroreceptors

- They are called high-pressure baroreceptors since they are sensors located in high-pressure areas:
  1- **The carotid sinus**; at the bifurcation of external and internal carotids.
  2- **The aortic arch.**

- Baroreceptors are **stretch-regulated receptors** (mechanoreceptors / pressoreceptorts). They **sense** changes in pressure according to **change** in the **tension** (stretching) of the arterial wall; **increased pressure** causes **more stretching** and vice versa. Signals are then relayed to the brain so that proper blood pressure can be **maintained**. This baroreflex mechanism is a **fast** response to changes in blood pressure.

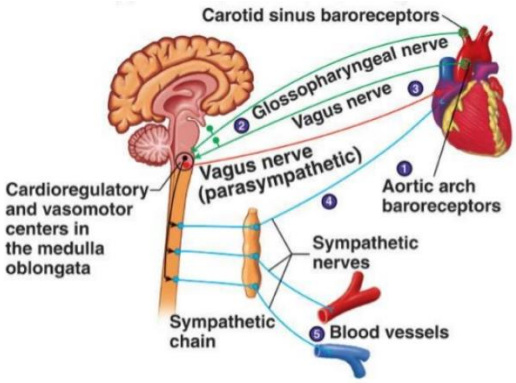
  ⇨ More stretch → Increased firing rate

  ⇨ Less stretch → Decreased firing rate

- **Afferent nerves** carry impulses to relay the information of blood pressure changes from the baroreceptors to the brain:

  1- **Aortic Baroreceptors**: supplied by a branch from the **vagus nerve** (cranial nerve X).

  2- **Carotid Baroreceptors**: supplied by **Hering's nerve;** a branch from the **glossopharyngeal nerve** (cranial nerve IX).



- Impulses carried through the **afferent nerves** specifically reach the **'Solitary Tract'** in the **medulla oblongata** of the brain stem. The Solitary tract is a **sensory** nucleus that receives input from the baroreceptors, it regulates:

  1- **Cardio-acceleratory center:** sends impulses to the heart through **sympathetic** fibers.

  2- **Cardio-inhibitory center:** sends impulses to the heart through **parasympathetic** fibers.

  3- **Vasomotor center (VMC)**: transmits impulses downward through the spinal cord to **almost all** blood vessels. It is located bilaterally in the reticular substance of the medulla and the lower third of the pons. The VMC is composed of 3 areas:

     a- Sensory area: impulses go to the sensory area, then either to the vasoconstrictor area or the vasodilator area.

     b- Vasoconstrictor area sends impulses to the vessels through **sympathetic fibers**.

     c- Vasodilator area: sends its impulses upward **inhibiting** the **vasoconstrictor area** to cause **vasodilation** rather than sending impulses directly to vessels.

⇨ Solitary Tract upon **stimulation** (increased firing rates in case of increased BP) causes:

1- **Activation** of the Cardo-inhibitory center and the vasodilator area.

2- **Inhibition** of the Cardio-acceleratory center and vasoconstrictor area.

⇨ The opposite occurs in a **reduced** firing rate due to **decreased BP**.

Baroreflex Mechanism:

Recall: factors affecting the pressure mainly are **cardiac output** and **total peripheral resistance** according to the formula: **MAP = CO x TPR**

A- In the case of increased Blood Pressure:

Arterial **stretching increase** due to **increased** BP → **increased** afferent **firing** rates from baroreceptors → **stimulates** the 'Solitary Tract' leading to:

1- **Activating** cardio-inhibitory center while **inhibiting** the cardio-acceleratory center:

In other words: increased PNS with decreased SNS effects.

⇨ ↓ Heart rate and Stroke volume → ↓ Cardiac output → ↓ BP

2- **Inhibiting** vasoconstrictor area while **activating** the vasodilator area:

⇨ Suppression of the vasoconstrictor area → vasodilation → ↓ TPR → ↓ BP

B- In the case of decreased Blood Pressure: *'the opposite happens'*

Arterial **stretching decrease** due to **decreased** BP → **decreased** afferent **firing** rates from baroreceptors → **inhibits** the Solitary Tract leading to:

1- **Inhibiting** cardio-inhibitory center while **activating** the cardio-acceleratory center:

⇨ ↑ Heart rate and Stroke volume → ↑ Cardiac output → ↑ BP

2- **Activating** vasoconstrictor area while **inhibiting** the vasodilator area:

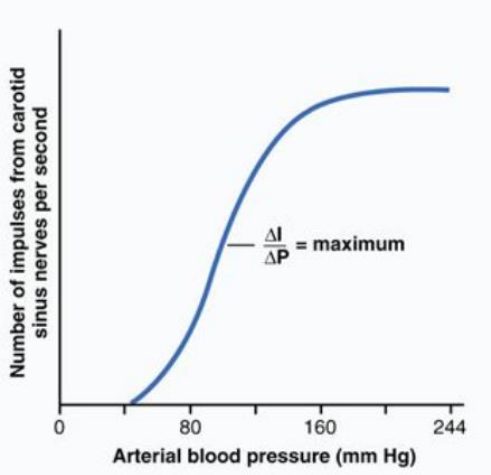
⇨ No suppression of the vasoconstrictor area → vasoconstriction → ↑TPR → ↑BP

Response of the Baroreceptors to Arterial Pressure Curve:

- This figure plots **arterial pressure** vs. the **number of impulses** from the carotid sinus baroreceptors.

  ΔI = change in impulse

  ΔP = change in pressure



- Baroreceptors respond to pressure changes in the range from **60-180mmHg**. They work **best** (most sensitive) around the **normal MAP** (almost 100mmHg).
  - ⇨ Any small change **above or below** 100mmHg leads to a **large change** in the number of impulses to effectively maintain normal BP.
  - ⇨ High-pressure baroreceptors are **not sensitive** to **very low BP** (<60 mmHg). Other receptors *(low-pressure baroreceptors)* can work at <60mmHg which will be discussed later.
  - ⇨ Baroreflex response to **very high BP** (>180 mmHg) is **diminished**; i.e. there isn't that much change in the number of impulses.

Side Notes:

1- *The previous curve looks like a buffer curve. A buffer resists any change in pH and brings it back to normal. Any buffer works best around its pKa. We consider baroreceptors buffers for blood pressure; they resist any change in pressure and work best around* ***≈100mmHg****.*

2- *Baroreceptors in the aortic arch are* ***less*** *sensitive than in the Carotid sinus.*
Carotid sinus baroreceptors are responsive to ***both*** *increased or decreased in pressure, while* ***aortic arch*** *baroreceptors are mostly responsive to* ***increased*** *pressure only.*

Functions of the Baroreceptors:

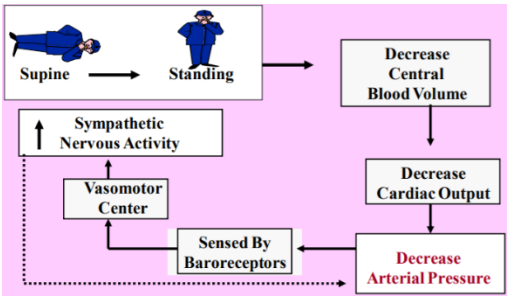
1- They oppose either increases or decreases in arterial pressure thereby **reducing daily variations in arterial pressure** maintaining the BP around 100mmHg most of the time.

When we denervate baroreceptors (i.e. cut their afferent nerves), variations in pressure will be **more exaggerated**, a lot less or a lot more than normal, since the baroreflex responses are lost.

They are unimportant in **long** term control of arterial pressure because the baroreceptors **adapt** and have **a low gain** *(gain corresponds to the effectiveness with which the baroreflex is able to maintain constant conditions;* $Gain = \frac{Correction}{Error}$ *).*

2- Maintain relatively constant pressure despite changes in **body posture**.

Upon changing positions from lying/sitting down to standing up, blood pools in the lower limb due to gravity causing a **drop in BP**. This sudden drop in blood pressure can cause dizziness or lightheadedness, and maybe even fainting.



If baroreceptors are working normally, they sense this change within seconds and raise the pressure through **sympathetic** responses to prevent sudden hypotension.

This fast baroreflex response can be diminished with medications that **block** sympathetic responses (e.g. β-blockers) and in **older** people.

> ***Note:*** *elders feel dizzier after getting up than young people can be because of* ***atherosclerosis*** *of arteries. Atherosclerotic arteries are* ***less*** *stretchable (less compliant)* ***reducing*** *baroreceptors reflex to changes in pressure.*

2- Low-Pressure Baroreceptors

- Low-pressure receptors are baroreceptors located in **low**-pressure areas: large systemic veins, in the pulmonary arteries, in the walls of the Rt. atrium, and Rt. ventricles.
- They are also called **volume receptors**; because these receptors respond to changes in the wall tension (stretch), which is **proportional** to the **filling** state of the low-pressure side of circulation (<60mmHg). Thus, they respond mostly to changes in **volume** rather than pressure and are involved with the **regulation of blood volume**.

Low-Pressure Baroreflexes:

Atrial volume receptors are baroreceptors found in the **atria** that are **sensitive** to **low** pressure. **Increased** blood volume **stretches** the atria causing **activation** of Atrial volume receptors which causes:

1- **Decreased ADH** (vasopressin) production.

2- **Decreased sympathetic** drive to the kidneys.

> ***Note:*** *ADH causes* ***vasoconstriction*** *and* ***increased water reabsorption****.*

A- Atrial-hypothalamic reflex

In the case of increased volume:

Atrial volume receptors are **activated** when detecting **increased** blood volume (increased pressure) in the atria. Signals are transmitted from the receptors to the **hypothalamus inhibiting ADH production** causing:

a- **Decreased** renal water reabsorption.
b- Peripheral **vasodilation**.

⇨ This **decreases** the blood volume and blood pressure back to normal.

In the case of decreased volume:

The **hypothalamus**, in turn, **increases** the production of ADH (vasopressin) causing:

a- **Increased** renal water reabsorption.
b- Peripheral **vasoconstriction**.

⇨ This **increases** the blood volume and blood pressure back to normal.

B- Atrial-renal reflex

In the case of increased blood volume:

Atrial volume receptors upon stimulation can also cause **decreased sympathetic** drive to the kidneys:

⇨ **Afferent** renal arterioles **vasodilation** → **increased** glomerular filtration rate → **increased** urine formation → **decreased** blood volume (towards normal).

In the case of decreased blood volume:

Increased sympathetic drive to the kidneys:

⇨ **Afferent** renal arterioles **vasoconstriction** → **decreased** glomerular filtration rate → **decreased** urine formation → **increased** blood volume (towards normal).

3- Chemoreceptors

- Chemoreceptors are **sensors** that detect CO_2, O_2, and pH **concentration changes** in the blood. They are important for **maintaining** arterial blood pO_2, pCO_2, and pH within **appropriate** physiological ranges.

- They are **highly vascularized** (they have very high blood flow), and sensitive to O_2, CO_2 and H^+ concentrations in the interstitial fluid around them, but because they are highly vascularized, we can say that they are sensitive to **arterial concentrations** as well.

- There are **two types** of Chemoreceptors according to their **location**:

  1- Central (medulla): sensitive mainly to changes in pH and pCO_2.

  2- Peripheral (in the carotid body ear the carotid bifurcation and aortic arch): sensitive mainly to changes in pO_2. *'which we will discuss mainly'*

- From the formula: **Flow** $= \frac{\Delta \text{ Pressure}}{\Delta \text{ Resistance}}$, **flow** is directly **proportional** to the **pressure** with **constant** resistance.

- **Peripheral chemoreceptors** are **sensitive** to oxygen lack, CO_2 excess, or H^+ ion excess which usually happens due to **decreased** blood flow (decreased BP). **Activation** of chemoreceptors receptors results in **stimulating** the **vasoconstrictor area**; however, chemoreceptors are **not** stimulated until the pressure falls **below** 80mmHg.

- **Decreased BP** (decreased blood flow) causes **decreased O_2** concentration and **increased CO_2** and **H^+** concentrations (↓ pH). This **stimulates** chemoreceptors causing:

  a- **Stimulated** cardio-acceleratory center and **inhibited** cardio-inhibitory center.

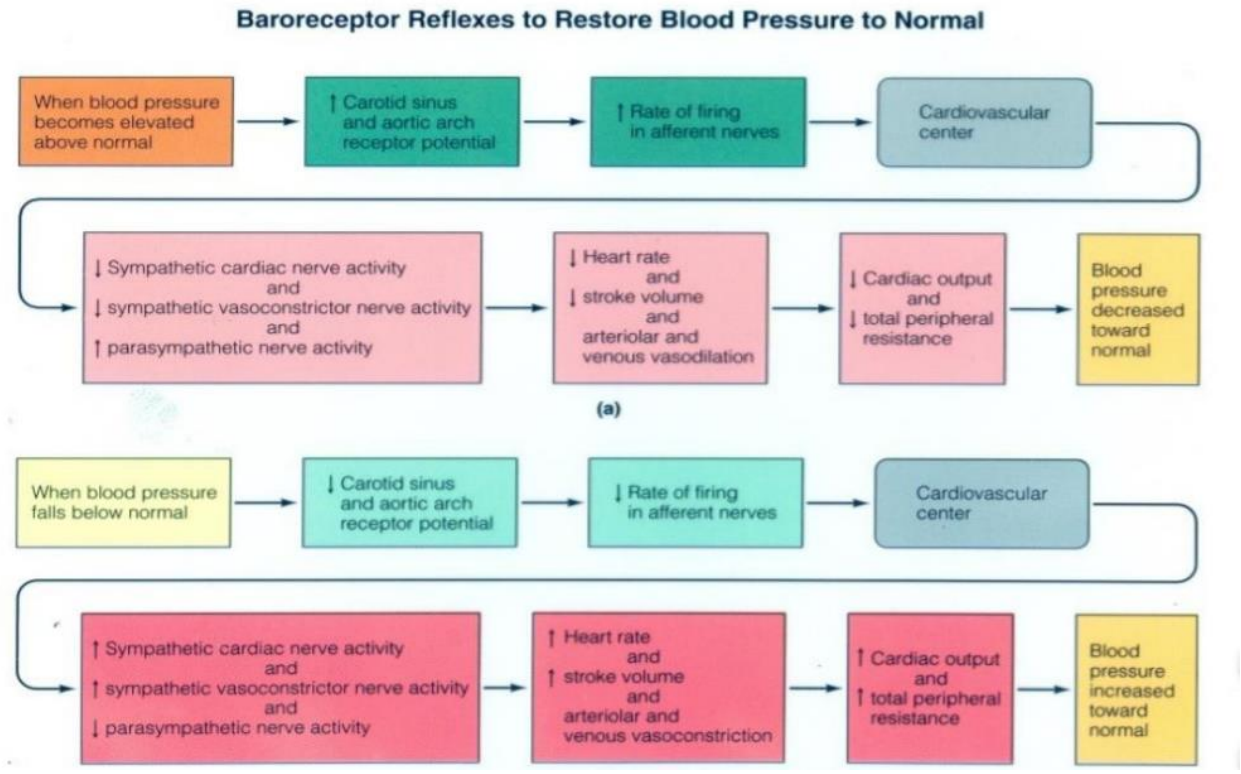
  b- **Stimulated** vasoconstrictor area.

  ⇨ **Increased** cardiac output (↑ HR and contractility) and blood pressure, restoring the blood's flow and chemicals' concentration back to normal.

- **Increased BP** (increased blood flow) causes **increased O_2** concentration and **decreased CO_2** and **H^+** concentrations (↑ pH). This causes the **opposite** in the cardiovascular centers and vasoconstrictor area leading to:

  ⇨ **Decreased** cardiac output and pressure.

Additional picture from the slides which summarizes baroreceptors reflexes:



Good Luck