Arterial Blood Pressure

Definition:

Is the pressure of the blood on the lateral walls of the arteries.

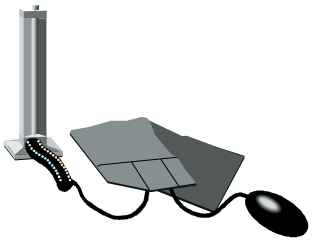
- ***Systolic blood pressure (SBP):*** is the maximal pressure reached in arteries during systole (90-130 mmHg)
- ***Diastolic blood pressure (DBP):*** is the minimal pressure reached in arteries during diastole (60-90 mmHg)
- ***Pulse Pressure (P.P)*** = SBP – DBP
- ***Mean arterial blood pressure*** = DBP + ⅓ P.P

Measurement of ABP:

I. Direct Method:

- *In experimental animal:* by cannula inserted into an artery & connected to recording apparatus.
- *In Human*: by cardiac catheterization.

II. Indirect Method:



1. Palpatory method:

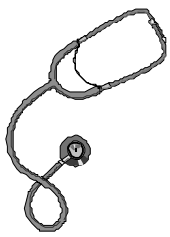
- Place the subject in sitting position with the forearm at the level of the heart.
- Apply completely deflated cuff of sphygmomanometer around the arm (with the rubber bag over the anterolateral aspect).
- Feel radial pulse.
- Raise pressure gradually → till radial pulse disappears (**A**).
- Release the pressure slowly till radial pulse is felt again (**B**).
- *Systolic blood pressure* = $\frac{\mathbf{A + B}}{\mathbf{2}}$

N.B.: Importance of palpatory method:

- Give rough idea about systolic blood pressure
- Avoid auscultatory gap.

2. Auscaltatory method:

- Deflate the sphygmomanometer cuff & apply as before.
- Place the stethoscope bell over the brachial artery (not in contact with applied cuff).
- Inflate the cuff to at least 30 mmHg above systolic pressure obtained by palpatory method.

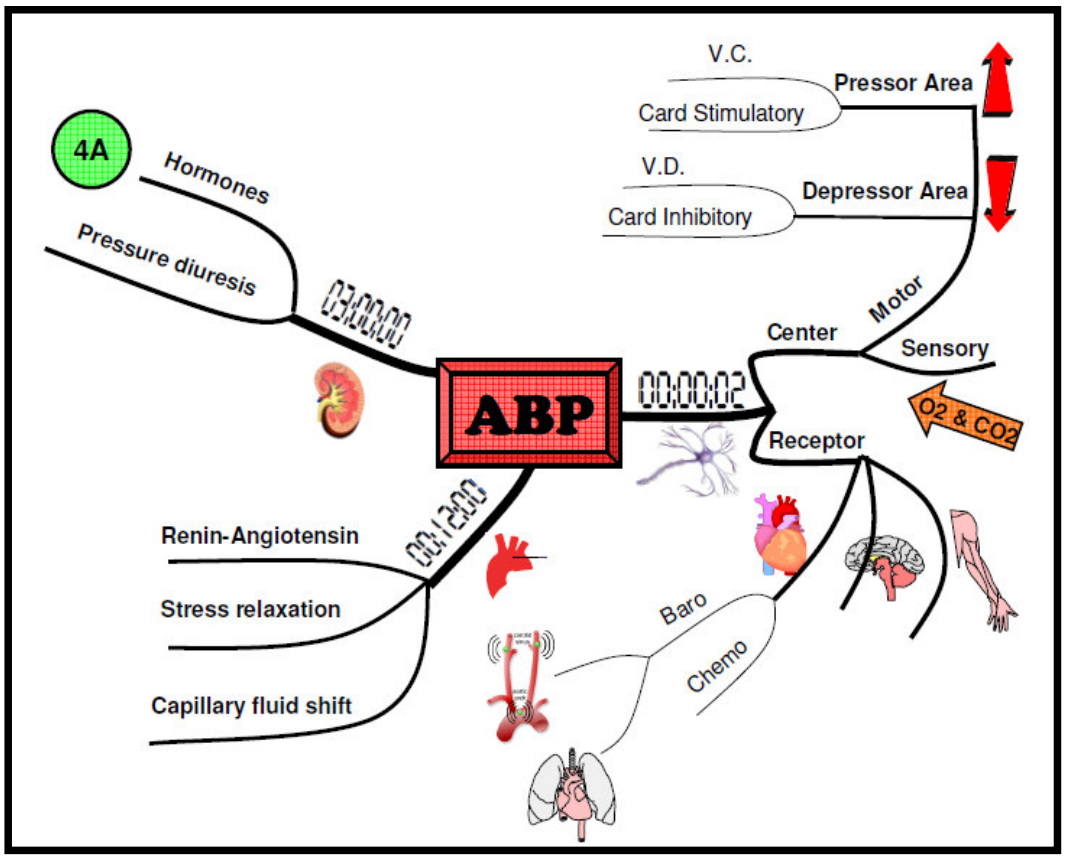


- Lower the pressure gradually → ***Korotkoff's sounds:***
    1) Moderate loud sound → Systolic pressure.
    2) Sounds with added murmur.
    3) Loud sound again.
    4) Sudden lowering of sound → Diastolic pressure.
    5) Silence.

Physiological variation in ABP .

1) **Age:**
    - In newborn → ABP average = 80/40 mmHg
    - At 20 years → ABP average = 120/80 mmHg
    - By age → ↓ elasticity of blood vessel → ↑ABP.
2) **Sex:**
    - Estrogen hormone in female lower blood pressure
    - After age of 45 in female → ↓ estrogen hormone → ABP rise more rapidly in female than male.
3) **Race:**
    - ABP higher in Europeans & Americans due to high cholesterol in diet & more stress in work.
4) **Emotion:**
    - Emotion → ↑ C.O → ↑ ABP.
5) **Exercise:**
    - ↑ C.O. → ↑ SBP.
    - Peripheral vasodilatation → ↓ peripheral resistance → ↓DBP.
6) **Gravity:**
    - During standing → ↑ blood pressure in vessels below the heart & ↓ above the level of the heart.
    - The amount of rise or decrease = weight of blood column.
    - ***Weight of blood column*** = *distance from heart to the vessel (cm)* X *density of blood* X *acceleration due to gravity*.
7) **Respiratory movement:**
    - During inspiration → ↑ intrathorasic –ve pressure → dilatation of pulmonary circulation → accommodate more blood → ↓ blood return to Lt. side of the heart → ↓ S.V. → ↓C.O →↓ ABP
    - During expiration → the reverse occurs → ↑ ABP
    - This changes are called (***Traube-Hering waves)***

Regulation Of ABP .



Vasomotor Centre (VMC):

- Located in reticular formation in medulla & lower pons
- It is formed of ***sensory*** & ***motor*** areas.

I. Sensory area:

- Located bilaterally in postero-lateral portion of RF
- Receive sensory impulses from vagus & glossopharyngeal n.
- Send impulses to control motor area in VMC.

II. Motor area:

- Formed of 4 centers:
  1) Vasoconstrictor center
  2) Cardiac stimulatory center.

  ] Pressor area
  3) Vasodilator center
  4) Cardiac inhibitory center

  ] Depressor area

	Vasoconstrictor area (Area C-1)	Vasodilator area (Area A-1)
Location	Bilateral in anterolateral portion of *upper* medulla	Bilateral in anterolateral portion of *lower* medulla
Discharge to	Blood vessels along sympathetic n → vasoconstrictor tone	Vasoconstrictor center → inhibition
Effect of stimulation	• V.C. of arterioles → ↑ peripheral resistane →↑ ABP • V.C. of veins → ↑Psf → ↑ venous return → ↑ C.O →↑ABP	• V.D. of arterioles →↓ peripheral resistance →↓ABP • V.D. of veins → ↓ Psf →↓venous return →↓ABP

	Cardiac stimulatory center CSC	Cardiac inhibitory center CIC
Location	Lateral in VMC	Medial in VMC
Discharge to	Heart along sympathetic n supply	Heart along vagus n.
Effect of stimulation	• ↑ heart rate • ↑ contractility of the heart • These lead to ↑ ABP	• ↓ heart rate → ↓C.O → ↓ ABP

Modification of VMC activity :

- Impulses from receptors in (***cardiovascular system - higher brain center - peripheral sensory receptors***)
- Direct effect of blood gases.

1. Cardiovascular receptors

Baroreceptors & Chemoreceptors

A. Baroreceptors:

- They are stretch receptors in the wall of the heart & great blood vessels
- they are either:
  1) ***Arterial baroreceptors.***
  2) ***Cardiopulmonary receptors.***

1) Arterial baroreceptors .

- ***Definition:***
  - are stretch receptors present in the wall of the carotid sinus & aortic arch.
- ***Nervous connection:***
  - Afferent impulses from aortic arch & carotid sinus are transmitted along vagus nerve & glossopharyngeal nerve respectively → sensory area of VMC.
- ***Mechanism of stimulation:***
  - Carotid sinus baroreceptors are not stimulated from 0 – 60 mmHg (to 90 mmHg in aortic arch).
  - At normal rang of blood pressure (100 mmHg), slight change in pressure → strong increase in frequency of discharge of impulses → effective control of normal ABP.
  - Arterial baroreceptors respond to rapid changes in pressure rather than stationary pressure.
- ***Resetting of arterial baroreceptors***:
  - Arterial baroreceptors reset themselves after 1 or 2 days whatever the pressure level is.
  - E.g.: if pressure rise from 100 to 160 mmHg → ↑ discharge from baroreceptors. → in the following seconds, decrease rate of discharge → return to normal discharge level after 2 days.
- ***Role in regulating ABP:***

  a) <u>At normal ABP:</u>
  - Bsroreceptors discharge at slow rate → stimulation of depressor area & inhibition of pressor area.

  b) <u>↑ ABP:</u>
  - More stimulation of depressor area & inhibition of pressor area leading to:
    - i- V.D. of arterioles → ↓ peripheral resistance.
    - ii- V.D. of veins → ↓ Psf → ↓ venous return → ↓ C.O.
    - iii- ↓ heart rate → ↓ C.O.
    - iv- ↓ cardiac contractility → ↓ C.O.

  c) *<u>↓ ABP</u>*:

- Inhibition of baroreceptors → ↓ rate of discharge → stimulation o pressor area & inhibition of depressor area leading to →
  - i- V.C. of arterioles → ↑ peripheral resistance
  - ii- V.C. of veins → ↑ Psf → ↑ venous return → ↑ C.O
  - iii- ↑ heart rate → ↑ C.O
  - iv- ↑ cardiac contractility → ↑ C.O

Q. What is Carotid sinus syndrome ?

A. In some people, carotid sinus reflex is very sensitive → slight pressure on carotid sinus (tight collar) → marked ↓ in ABP → cerebral ischemia → fainting.

Mary's law

Heart rate is inversely proportional to ABP provide that other factors kept constant

2) Cardiopulmonary receptors .

- They are receptors present in the lower pressure part of circulation as:
  - i- Wall of *atria* (at entrance of superior & inferior vena cava).
  - ii- Wall of *left ventricle*
  - iii- *Pulmonary* circulation
- Afferent impulses from this receptors → vagus nerve → sensory area of VMC.

1. **Atrial receptors:**

- They are two types:
  - i- ***Type A:*** discharge during atrial systole.
  - ii- ***Type B:*** discharge during late atrial diastole
- ↑ blood return to atria → stretch its wall → stretch of type B receptors leading to:
  - ***a.*** Stimulation of vasodilator area & inhibition of vasoconstrictor area leading to:
    - V.D. of arterioles →↓ peripheral resistance →↓ABP
    - V.D. of afferent arterioles in kidney → ↑ glomerular filtration → ↑ urine formation → ↓ ABP
  - ***b.*** Impulses to hypothalamus → ↓ atidiuretic hormone ADH → ↑ urine volume → ↓ blood volume & ABP.
  - ***c.*** ↓ sympathetic stimulation → ↓ aldosterone secretion → ↑ salt & water loss in urine → ↓ blood volume.
  - ***d.*** ↑ secretion of atrial natriuretic peptide ANP → loss of salt & water in urine → ↓ blood volume
  - ***e.*** ↑ Heart rate (by 70%): 15% caused by direct effect of atrial stretch on S-A node while 55% caused by Bainbridge reflex.

Bainbridge Reflex

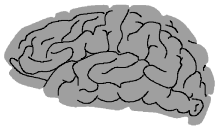
Atrial stretch → stimulation of atrail receptors → vagus nerve → VMC → ↑ heart rate & contractility

2. **Left ventricular receptors:**
   - Distension of Lt ventricle with blood → stimulation of its receptors → impulses through vagus → VMC → ↓ heart rate & ↓ ABP.
3. **Pulmonary receptors:**
   - Pulmonary congestion → stimulate pulmonary receptors → afferent to VMC → ↓ heart rate & ↓ ABP.

B. Chemoreceptors

- Are chemosensitive neurons tat measure chemical changes that occur in the blood
- ***Site:*** central or peripheral in the:
  - i- *Carotid bodies*: at the bifurcation of common carotid.
  - ii- *Aortic bodies*: adjacent to the aorta.
- Nervous connections as arterial baroreceptors.
- They are stimulated by:
  - ↓ PO2 in arterial blood
  - ↑ PCO2 in arterial blood
  - ↑ H^+ concentration in arterial blood.
- ↓ ABP → ↓ blood supply to carotid & aortic bodies → ↓PO2, ↑PCO2 & ↑ H^+ concentration → stimulation of chemoreceptors → impulses to VMC → ↑ ABP toward normal + ↓ heart rate
- ↓PO2 also cause ↑ secretion of catecholamine → ↑ heart rate & cardiac output.

2. Higher Brain Center afferent



- **Reticular formation:**
  - Stimulation of lateral & superior part→impulse to VMC→↑ABP
  - Stimulation of medial & inferior part →impulse to VMC→↓ABP
- **Hypothalamus:**
  - Postero-lateral part → stimulate VMC → ↑ ABP.
  - Anterior part → ↓ VMC
  - Hypothalamus cause ↑ ABP during motions & exercise. It also cause vascular changes associated with temperature regulation.
- **Cerebral cortex:**
  - Before exercise impulse from limbic lobe & premotor area → thalamus → VMC → ↑ heart rate & ↑ABP
  - Cerebral cortex is the origin of sympathetic vasodilator fibers to skeletal muscles.

3. peripheral sensory receptors

- Afferent from cutaneous pain → VMC → ↑ ABP + tachycardia
- Afferent from visceral pain → VMC → ↓ ABP + bradycardia
- Afferent from old receptor → ***Cold pressor effect*** → ↑ ABP

4. Direct effect of gas changes

- Mild Hypoxia & hypercapnia → stimulation of VMC → ↑ ABP
- Sever hypoxia & hypercapnia → inhibition of VMC → ↓ ABP
- **Cushing Reflex:**
  - ↑ intracranial pressure → compression of blood supply to VMC → hypoxia & hypercapnia → stimulation of VMC → ↑ ABP to restore blood flow to medulla.
  - It is accompanied by reflex bradycardia.

Intermediate Control Mechanisms

- **Renin-angiotensin V.C. mechanism**
- **Stress relaxation of blood vessels**
- **Capillary fluid shift**

1. Renin-angiotensin V.C. mechanism (20 minute).

- Renin is a hormone synthesized and stored in juxtaglomerular cells of the kidney
- ↓ ABP → release of rennin → conversion of angiotensinogen to angiotensin I.
- Angiotensin I is converted to Angiotensin II by Angiotensin converting enzyme (ACE)
- Angiotensin II leads to:
  1) ***Generalize V.C.:***
     a) V.C. of arterioles → ↑ peripheral resistance → ↑ABP
     b) V.C. of veins → ↑ Psf → ↑ venous return → ↑C.O. →↑ ABP
  2) ↑ ***Extracellular fluid volume (see below)***

2. Stress relaxation of blood vessels

- ↑ ABP → relaxation of blood vessel wall → ↓ pressure to normal

3. Capillary fluid shift.

- ↑ ABP → ↑ capillary pressure → ↑ fluid filtration from the capillary → ↓ venous return → ↓ C.O. → ↓ ABP to normal

Long Term Regulation
Controlling blood volume

Q. What is the effect of blood volume on ABP ?

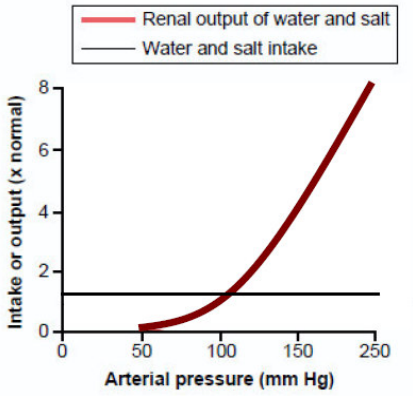
A. increase blood volume leads to:

- ↑ Psf → ↑ venous return → ↑C.O. → ↑ ABP
- ↑ blood flow to tissue → V.C. of all blood vessels by myogenic properties → ↑ peripheral resistance → ↑ ABP

- **Pressure diuresis & natruiresis**
- **Renin-Angiotensin effect on ECF**
- **Hormones acting on kidney**

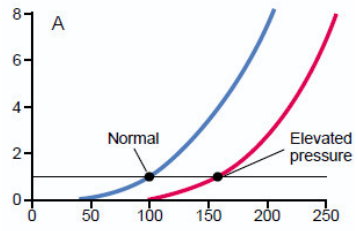
1. Pressure diuresis & natruiresis:

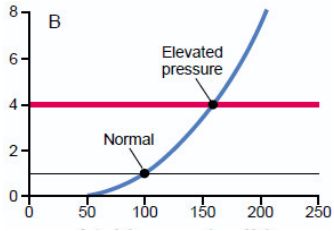
- **Definition:**
  - It is the increase in excretion of water & salts in urine in respond to increase ABP.
- **Renal output curve:**
  - At mean ABP = 50 mmHg → urine output = zero → ↑ blood volume → ↑ ABP to normal
  - At mean ABP = 100 mmHg → normal urine output
  - Doubling mean ABP to (200mmHg) → ↑ urine output 6 times normal → ↓ blood volume → ↓ ABP to normal.
  - Equilibrium point at which sat & water intake = salt & water output is reached at Mean ABP = 100 mmHg



- **Factor affecting:**
  1) ***Efficiency of kidney:***
     - Impaired kidney function → shift renal output curve to the right → equilibrium point become as higher as 150 mmHg

Renal Failure





High intake

2) ***Amount of salt & water intake:***
   - Increase salt & water intake → shift of the intake line upward → higher equilibrium point

2. Renin-Angiotensin effect on ECF:

- Rennin-Angiotensin system act as automatic feed back mechanism that guard against change in salt intake.
- ↓ salt intake → ↓ ECF → ↓ blood volume → ↓ renal blood flow → ↑ rennin secretion → ↑ angiotensin II
- Angiotensin II increase ECF volume by the following mechanisms:

1) ***Direct action on kidney:***
   a) V.C. of renal blood vessel → ↓ renal blood flow → ↓ glomerular filtration → ↓ urine output.
   b) ↑ Na & water reabsorption from renal tubules → ↓ urine output
2) ***Stimulation of aldosterone secretion:***
   - Aldosterone → ↑ Na & water reabsorption from renal tubules → ↑ ECF volume → ↑ blood volume → ↑ ABP.

3. Hormones acting on kidney (AAAA):

- **Aldosterone:**
  - Aldosterone → ↑ Na & water reabsorption from renal tubules → ↑ ECF volume → ↑ blood volume → ↑ ABP.
- **Antidiuretic hormone (ADH):**
  - ↓ blood volume → ↓ blood return to atria → relaxation of atrial wall → inhibition of atrial B receptor → afferent to hypothalamus → ↑ ADH secretion.
  - ADH cause reabsorption of water from renal tubules → ↑ ECF volume → ↑ ABP to normal.
- **Atrial Natruiretic peptide (ANP):**
  - ↑ blood volume → ↑ blood return to right atrium → stretch of atrial wall → ↑ ANP → ↑ sodium & water loss in urine