Arterial Blood Pressure
Dr Badri Paudel
GMC

Arterial Blood Pressure (BP)

- The lateral pressure force generated by the pumping action of the heart on the wall of aorta & arterial blood vessels per unit area.
- Pressure inside big arteries (aorta & big vessels).
- Measured in (mmHg), & sometimes in (cmH₂O), where 1 mmHg = 1.36 cmH₂O.
- Systolic B.P. : The maximum pressure exerted in the arteries during systole (90-140 mmHg).
- Diastolic B.P. : The minimum pressure within the arteries during diastole (60-90 mmHg).

Arterial Blood Pressure (continued)

- Diastolic pressure is more important, because diastolic period is longer than the systolic period in the cardiac cycle.
- Pulse pressure = Systolic BP – Diastolic BP.
- Mean arterial pressure = This is the average arterial pressure throughout the cardiac cycle.
  = Diastolic BP + 1/3 Pulse pressure.

Mean Arterial Blood Pressure (MAP)

“average arterial blood pressure during a cardiac cycle”
Perfusion pressure
main driving force for propelling blood to the tissues

MAP = DP + 1/3 (SP-DP)
For a BP of 120/80, MAP is ~ 93.5 mmHg
A MAP of ~ 60 mmHg is sufficient for end organ perfusion.

Blood pressure values: what do they mean?

- Pulse pressure: PP = SP-DP
- Mean arterial blood pressure = MABP
- MABP = DP + 1/3 (SP-DP)
- CO = MABP = SV x HR
  TPR

Hypertension Clinical Manifestation
Dx is made after multiple readings over several weeks

NIH/Joint Committee Definition:

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>&lt;110</td>
<td>&lt; 80</td>
</tr>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>&lt; 80</td>
</tr>
<tr>
<td>High Normal</td>
<td>120-139</td>
<td>or 80-89</td>
</tr>
<tr>
<td>Stage 1</td>
<td>140-159</td>
<td>or 90-99</td>
</tr>
<tr>
<td>Stage 2</td>
<td>160-179</td>
<td>or 100-109</td>
</tr>
<tr>
<td>Stage 3</td>
<td>=&gt;180</td>
<td>or =&gt; 110</td>
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</tbody>
</table>
**Measurement**

**3 Ways to Measure Blood Pressure**

- Aneroid Monitor
- Digital Monitor
- Finger/Wrist Monitor

**Steps to Follow before Taking Your Blood Pressure**

- Don’t use 30 minutes prior to taking your blood pressure:
  - Caffeine,
  - Alcohol, or
  - Tobacco.
- Go to the bathroom.
- Rest 3-5 minutes before taking your blood pressure.
- Sit comfortably.
- Legs and ankles uncrossed
- Back supported

**Tips for Accurate Use**

- Same time of day
- Use the same arm
  - Left
- Don’t measure
  - Immediately upon waking up, or
  - Immediately after exercising.
  - Wait an hour.

**How to Take Your Blood Pressure**

- Place your arm, raised to the level of your heart, on a table or a desk, and sit still.
- Wrap the correctly sized cuff smoothly and snugly around the upper part of your bare arm.
- Make sure that if you have rolled up a sleeve to place the cuff on your arm that it does not get too tight around your arm.
- Take a repeat reading two to three minutes after the first one to check accuracy.
- Be certain that the bottom edge of the cuff is 1 inch above the crease of your elbow.
Arterial blood pressure

Remember Blood Pressure....
- Varies throughout the day
- Is often higher in the morning
- Talk about your personal blood pressure goals with your doctor.

Hypertension Pathophysiology

- **Primary (Essential) Hypertension:**
  - Elevated BP without an identified cause
  - Accounts for 95% of all cases of hypertension
  - Cause - unknown
    - **Contributing Factors:** Increased SNS activity, overproduction of Na+ retaining hormones & vasoconstrictors, increased Na+ intake
    - **Risk Factors:** Modifiable

- **Primary Hypertension Risk Factors**
  - Age
  - Alcohol
  - Cigarette Smoking
  - Diabetes Mellitus
  - Elevated serum lipids
  - Excess Na+ in diet
  - Gender
  - Family History
  - Obesity
  - Ethnicity

- **Secondary Hypertension Pathophysiology**
  - Specific cause of hypertension can be identified
  - 5% of adult hypertension
  - Causes:
    - Coarctation or congenital narrowing of the aorta
    - Renal disease – renal artery disease / parenchymal
    - Endocrine disorders: Pheochromocytoma, Cushing Syndrome, Hyperaldosteronism, thyroid and parathyroid
    - Neurology disorders – brain tumors / head injury
    - Sleep apnea
    - Medications – sympathetic stimulants
    - Pregnancy-induced hypertension
Physiological variations in arterial B.P.:

- **Age:**
  Arterial B.P. increases with age due to loss of arterial elasticity.

- **Sex:**
  After menopause, arterial B.P. becomes higher in females due to hormonal changes of estrogen.

- **Race:**
  Oriental > Westerns...? dietary factors, or weather.

- **Diurnal variation:**
  Normally, arterial B.P. is lowest in the early morning and highest in the afternoon.

- **Exercise:**
  Arterial B.P. increases during exercise especially systolic arterial B.P.

- **Emotions:**
  Arterial B.P. increases in strong emotional stress.

- **Gravity:**
  On standing, the gravity increases arterial B.P. below a reference point (in the right atrium near the tricuspid valve) in the heart and decreases it above that point.

Factors determining ABP:

Blood Pressure = Cardiac Output X Peripheral Resistance

\[ \text{BP} = \text{CO} \times \text{PR} \]

- BP depends on:
  1. Cardiac output \(\Rightarrow\) \(\text{CO} = \text{SV} \times \text{HR}\).
  2. Peripheral resistance.

Regulation of ABP:

- Maintaining B.P. is important to ensure a steady blood flow to tissues.
- B.P. is regulated neurally through centers in medulla oblongata:
  1. Vasomotor Center (V.M.C.), or (pressor area):
     \(\Rightarrow\) Sympathetic fibers.
  2. Cardiac Inhibitory Center (C.I.C.), or (depressor area):
     \(\Rightarrow\) Parasympathetic fibers (vagus)

Regulation of ABP (continued)

Cardiac control centers in medulla oblongata:

1. Cardiac accelerator center (V.M.C)
2. Cardiac inhibitory center (C.I.C)

- Regulatory mechanisms depend on:
  a. Fast acting reflexes:
     Concerned by controlling CO (SV, HR), & PR.
  b. Long-term mechanism:
     Concerned mainly by regulating the blood volume.
Regulation of CO:

- A fast acting mechanism.
- CO regulation depends on the regulation of:
  a. Stroke volume, &
  b. Heart rate

Neural regulation

- **Sympathetic nerves**
  - Constrictor nerves - mediator noradrenaline - α1 adrenoceptors
  - Elevates Ca++ through phospholipase C pathway (IP3)

- **Parasympathetic nerves**
  - In tissues which need sudden increase in blood flow (salivary gland, external genitalia)
  - Mediator acetylcholine has indirect effect
  - inhibition of noradrenalin release
  - production of NO

Sympathetic input - HEART

**ACTIONS**
- Nerve fibers release NE
- SA, atria, and ventricles
- ↑ HR and contractility
- R side SA node
- L side contractility

**MECHANISM**
- β1 receptors – pacemaker activity
- β1 myocardium contraction

Parasympathetic input - HEART

**ACTIONS**
- Vagus nerve releases ACH
- SA and mycardium
- HR and conduction velocity
- R side SA node (HR)
- L side contractility (slight)

**MECHANISM**
- Muscarinic receptors (M2)
- βγ subunit (HR)
- Nitric oxide (weak inotropic effect)

Sympathetic input – Blood vessels

**ACTIONS**
- Activated – Vasconstriction throughout body
- Skin/kidney BVs most abundant
- De-activated – Vasodilation

**MECHANISM**
- Norepinephrine
  - α > β
- Epinephrine
  - β > α
- Vasocstriction – α1
- Vasodilation – β2
Parasympathetic input – Blood vessels

**ACTIONS**
- Vasodilation of BVs
- Less common than sympathetic activity
- Salivary glands, gastrointestinal glands, reproductive tissues

**MECHANISM**
- ACH increases vasodilation indirectly through other second messengers.

Sympathetic activation of skeletal muscle

- Causes vasodilation
- Release of ACH
- Action is on pre-capillary sphincters

Vasoconstriction in all vascular beds except skeletal muscle Increased HR and contractility

Control center is not medulla but rather cerebral cortex
- “fight or flight” response
- Anticipatory response to exercise

Adrenal medulla

- Sympathetic release of epinephrine and norepinephrine
- Global effects on increasing arterial blood pressure.

Blood pressure regulation

<table>
<thead>
<tr>
<th>vasodilatation</th>
<th>vasoconstriction</th>
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</thead>
<tbody>
<tr>
<td>stimulation of cGMP</td>
<td>inhibition of cAMP</td>
</tr>
<tr>
<td>stimulation of cAMP</td>
<td>Stimulation of IP3</td>
</tr>
</tbody>
</table>

In smooth muscle, cGMP and cAMP stimulate Ca2+ pump of the sarcoplasmic reticulum
Decrease of Ca2+ concentration in smooth muscle cell

Slower decrease of Ca2+ from the sarcoplasmic reticulum

<table>
<thead>
<tr>
<th>NO</th>
<th>ANP</th>
<th>serotonin</th>
<th>serotonin</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANP</td>
<td>adenosine A2</td>
<td>histamine H2</td>
<td>adrenaline β2</td>
</tr>
<tr>
<td>VIP</td>
<td>angiotensin II</td>
<td>adrenaline α2</td>
<td>vasopressin</td>
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</table>
Regulation of Peripheral Resistance (PR):
- A fast acting mechanism.
- Controlled by 3 mechanisms:
  1. Intrinsic.
  2. Extrinsic.
  3. Paracrine.
- Extrinsic mechanism is controlled through several reflex mechanisms, most important:
  1. Baroreceptors reflex.
  2. Chemoreceptors reflex.

Regulation of blood flow

<table>
<thead>
<tr>
<th>Type</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myogenic</td>
<td>Stretch-activated cation channels cause vasoconstriction</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Metabolic products cause vasodilatation</td>
</tr>
<tr>
<td>Shear</td>
<td>Vasodilatation by NO, which is produced in vascular endothelium</td>
</tr>
<tr>
<td>Neural</td>
<td>Sympathetic constrictor nerves in most tissues</td>
</tr>
<tr>
<td>Humoral</td>
<td>Epinephrine, vasopressin, serotonin</td>
</tr>
<tr>
<td></td>
<td>ANP, histamine, inflammatory mediators</td>
</tr>
</tbody>
</table>

Myogenic autoregulation
- Arterioles contracts when they are distended (brain, kidney, heart)
- Mechanism
  - Stretch-activated Na⁺ and Ca²⁺ channels of vascular smooth muscle
  - Depolarization of membrane, which then activates L-type Ca²⁺ channels
  - Muscle contraction

Metabolic regulation
- Adenosine
  - Causes vasodilatation, except of kidney and pulmonary artery
  - Activation of adenosine A₂A membrane receptor – elevation of cAMP
- pO₂
  - Reduction in pO₂ increases production of vasodilator agents (PGI₂ and NO)
- pCO₂
  - Elevated pCO₂ leads to elevated H⁺ in extracellular fluid – acidosis causes membrane hyperpolarization (K⁺) – vasodilatation (except of lung)

Shear-dependent regulation
- Endothelial cell reacts on many physiological stimuli with production of several substances which influence smooth muscle cell
  - Stretching
  - Shear stress induced by blood flow
  - Hormonal levels
  - Substances released from blood elements (trombocytes, macrofages)
  - Synthesis of NO and PGI₂ (vasodilators)

Nitric oxide synthesis
- Shear stress and a variety of receptor-mediated agonists raise vascular endothelial [Ca²⁺] and cause the Ca²⁺-calmodulin complex to activate endothelial nitric oxide synthase (eNOS).
- NO is produced from the amino acid L-arginine.
- NO is a gas and diffuses into adjacent VSM where it activates soluble guanylate cyclase, produces cGMP and causes vasodilatation
Extrinsic mechanism
1. Baroreceptors reflex:
   - Baroreceptors are receptors found in carotid sinus & aortic arch.
   - Are stimulated by changes in BP.
   \[ \text{BP} \rightarrow \text{Baroreceptors} \rightarrow \text{V.M.C} \text{ and } \text{C.I.C} \]
   - Sympathetic: Vasodilation & ↓ TPR
   - Parasympathetic: Slowing of SA node (↓ HR) & ↓ CO

High pressure baroreceptors respond to stretch in the aortic arch and carotid sinus.

Carotid nerve fires above and below normal pressures. Aortic nerves are activated above normal pressures.

Baroreceptor Feedback Loop for the Regulation of Mean Arterial Blood Pressure

The aortic receptors help reinforce the carotid activation above normal pressures.
Coordination of Medullary Cardiovascular Inputs

Cardio-inhibitory Area
- Dorsal motor nucleus of X
- Nucleus ambiguous of X and IX
- Vasomotor area (tonic vasoconstriction)

Sympathetic activation of:
- Blood vessels
- Heart
- Adrenal medulla

To spinal cord

Parasympathetic activation bradycardia

2. Chemoreceptors reflex:
- Chemoreceptors are receptors found in carotid & aortic bodies.
- Are stimulated by chemical changes in blood mainly hypoxia ($\downarrow$ $O_2$), hypercapnia ($\uparrow$ $CO_2$), & pH changes.

Haemorrhage $\downarrow$ BP
Hypoxia

$++ VM.C$ $\rightarrow$ Chemoreceptors $\rightarrow$ C.I.C

$\rightarrow$ Sympathetic
$\rightarrow$ Vasoconstriction & $\uparrow$ TPR

$\uparrow$ Adrenal medulla

$\rightarrow$ Parasympathetic
$\rightarrow$ $\uparrow$ HR

Chemoreceptors
- Peripheral (ventilation)
  - Sense low $O_2$
  - Carotid and aortic (glomerus cell)
  - Synapse with IX and X, respectively
- Central (medulla/CNS)
  - Sense low pH primarily

1. Exert a positive drive on vasomotor area
2. Exert a positive drive on cardio-inhibitory area

Vasoconstriction and Bradycardia

Net result of Chemoreceptor stimulus is an integration of central and peripheral chemoreceptors

Stretch of pulmonary receptors cancel peripheral stimulus on cardio-inhibitory area – causing tachycardia
3. Other Vasomotor Reflexes:

1. Atrial stretch receptor reflex:
   - ↑ Venous Return ⇒ + + atrial stretch receptors ⇒ reflex vasodilatation & ↓ BP.

2. Thermoreceptors: (in skin/or hypothalamus)
   - Exposure to heat ⇒ vasodilation.
   - Exposure to cold ⇒ vasoconstriction.

3. Pulmonary receptors:
   Lung inflation ⇒ vasoconstriction.

### Bainbridge reflex
(atrial stretch reflex)

- **Receptors**: baroreceptors type A,B.
- **Location**: in Atrial walls
- **Stimulus** for type A: increased atrial pressure during atrial systole
- **Stimulus** for type B: atrial distension during atrial diastole.
- **Afferent**: Vagus nerve
- **Center**: CVC in medulla oblongata
- **Response**: ↑ heart rate, ↓ B.P.

**SIGNIFICANT**: to equalize input with output of heart.

### Atrial (Bainbridge) Reflex.

- A sympathetic reflex initiated by increased blood in the atria
  - Causes stimulation of the SA node
  - Stimulates baroreceptors in the atria, causing increased SNS stimulation
  - Adjusts heart rate in response to venous return
  - Stretch receptors in right atrium: trigger increase in heart rate through increased sympathetic activity.

### Low Pressure Cardiac Baroreceptors

- **"Bainbridge Reflex"**
  - Unrelated activation of the high-pressure baroreceptors, activation of the A and B fibers will INCREASE heart rate (and also cause renal vasodilatation).

- **Low Pressure Cardiac Baroreceptors**
  - Respond to "fullness" or volume
  - Located in low pressure sites
  - Control the effective circulating volume
  - Indirectly regulate MAP

### CORONARY STRETCH REFLEX
(Lt. vent. Stretch reflex)

- **Receptors**: baroreceptors
- **Location**: in Lt. ventricle near coronary vessels.
- **Stimulus**: Lt. vent. Distension.
- **Afferent**: Vagus nerve
- **Center**: CVC
- **Response**: ↓ heart rate, ↓ B.P.

**SIGNIFICANT**: to maintain Vagal tone that keeps low heart rate at rest.
Coronary chemoreflex (Bezold-Jarisch reflex)
- **Receptors**: chemoreceptors (c-nerve fibers).
- **Location**: near coronary vessels of Lt. vent.
- **Stimulus**: chemical changes
- **Center**: CVC in medulla
- **Response**: ↓ heart rate ↓ B.P

**SIGNIFICANT**: In myocardial infarction, these receptors are stimulated by certain substance released from infarcted tissues and lead to hypotension (as index for severity of case).

REFLEXES FROM EXTRAVASCULAR RECEPTORS
1- Pulmonary stretch reflex
- **Receptors**: baroreceptors
- **Location**: in bronchial wall
- **Stimulus**: lung inflation (inspiration)
- **Afferent**: Vagus nerve
- **Response**: ↑ heart rate ↓ B. P

**SIGNIFICANCE**: With inspiration, venous return is increased and the input for heart is also increased. So that, by this reflex increased heart rate to equalize input with output without increase in B. P (Like Bainbridge reflex).

Pulmonary chemoreflex
- **Receptors**: chemoreceptors (C-nerve fiber)
- **Location**: near lung capillaries
- **Stimulus**: chemical changes
- **Afferent**: Vagus nerve
- **Center**: CVC
- **Response**: ↓ heart rate ↓ B.P

**SIGNIFICANT**: unknown

4. Hormonal Agents:
- **NA**: vasoconstriction.
- **A**: vasoconstriction (except in sk. ms.).
- **Angiotensin II**: vasoconstriction.
- **Vasopressin**: vasoconstriction.

Regulation of Arterial Blood Pressure
C. Regulation of Blood Volume
**Regulation of Blood Volume:**

- A long-term regulatory mechanism.
- Mainly renal:
  1. Renin-Angiotensin System.
  2. Anti-diuretic hormone (ADH), or vasopressin.
  3. Low-pressure volume receptors.

**Hormonal regulation**

- Renin-angiotensin, vasopressin, ANP
- **Adrenaline** (epinephrine)
  - Higher affinity for β-adrenoreceptors (heart, splanchnic area, skeletal muscle) – vasodilatation
  - Lesser affinity for α-adrenoreceptors (vasoconstriction)
- **Serotonin**
  - Released from platelets during clotting reaction, elevated Ca^{2+} leads to vasoconstriction
- **Histamine**
  - Vasodilatation by means of NO production

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**Renin-angiotensin II-aldosteron system**

- Regulates ABP by regulating blood volume
- Most important mechanism for Na^{+} retention in order to maintain the blood volume.
- A decrease in ABP – decrease in renal perfusion pressure
  - Mechanoreceptors in afferent arterioles
  - Juxtaglomerular cells secret renin (proteolytic enzyme)
  - In plazma, renin catalyzes the conversion of angiotensinogen to angiotensin I (a decapeptide)
  - In lungs, angiotensin I is converted to angiotensin II (catalyzed by angiotensin converting enzyme (ACE)) (an octapeptide)

**Role of angiotensin II**

- In the zona glomerulosa cells of adrenal cortex stimulates production of aldosterone
- In renal distal tubule and collecting duct increases Na^{+} reabsorption – increases ECF volume and blood volume
- In arterioles angiotensin II causes vasoconstriction – increase in TPR
- In the renal proximal tubule stimulates Na^{+}-H^{+} exchange – increase in ECF volume
- In the CNS stimulates thirst an drinking behavior

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**Renin-Angiotensin System:**

<table>
<thead>
<tr>
<th>Subsystem</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renin-angiotensin apparatus</td>
<td>Labeled as major regulatory mechanism for blood volume regulation.</td>
</tr>
<tr>
<td>Angiotensinogen</td>
<td>Converts angiotensinogen to angiotensin I in plazma.</td>
</tr>
<tr>
<td>Angiotensin I</td>
<td>Converted to angiotensin II in lungs.</td>
</tr>
<tr>
<td>Angiotensin II</td>
<td>Powerfull vasoconstrictor.</td>
</tr>
<tr>
<td>Angiotensin III</td>
<td>Produced by juxtaglomerular apparatus of kidneys.</td>
</tr>
<tr>
<td>Aldosterone</td>
<td>Main regulator of Na^{+} retention.</td>
</tr>
</tbody>
</table>

- **Antidiuretic hormone**
  - Secreted by the posterior lobe of the pituitary gland after
    - increased osmolarity
    - decreased ABP (e.g. hemorrhage), atrial volume receptors are stimulated
  - Regulates body fluid osmolarity
  - 2 types of receptors:
    - V1: in vascular smooth muscles – cause vasoconstriction of arterioles, increase TRP
    - V2: in renal collecting ducts are involved in water reabsorption, maintain osmolarity
Anti-diuretic hormone (ADH), or vasopressin:
- Hypovolemia & dehydration will stimulate the osmoreceptors in the hypothalamus, which will lead to release of ADH from posterior pituitary gland.
- ADH will cause water reabsorption at kidney tubules.

Atrial natriuretic peptide
- ANP is secreted by the atria in response to increase in ECF volume and atrial pressure
- Mechanism of action:
  - Relaxation of vascular smooth muscle – vasodilatation, decrease TPR
  - In the kidney – increased Na+ and water excretion = decrease ECFV and ABP

Changes in posture from supine position to standing
- Mechanism of orthostatic hypotension
  - Blood pools in the veins of lower extremities
  - Venous return to the heart decreases, cardiac output decreases (Frank-Starling law)
  - Mean arterial pressure decreases
  - Decreased activation of baroreceptors
  - Increased sympathetic outflow to the heart and blood vessels and decreased parasympathetic outflow

Fight or Flight Reaction
(Sudden Sympathetic Drive)
4. Veins – vasoconstriction (sympathetic)
5. Heart – Increased sympathetic stimulus – increased HR and contractility
6. MAP – Overall output is an increase in blood pressure.

FAINTING
(Massive Parasympathetic Response)
“vasovagal syncope”
1. Massive vasodilation occurs – removal of sympathetic tone causes a rapid fall in blood pressure.
2. Decreased Cardiac output – Increased vagal output to heart causes bradycardia and decreased stroke volume
3. Decreased arterial blood pressure – secondary to vasodilation and CO.
4. Cerebral blood flow – reduced (> 10 seconds) – fainting occurs
FAINTING
(Massive Parasympathetic Response)

- Emotional stress
- AVP release
- Vasodilation

 Bradycardia
 Decreased MAP
 Reduce cerebral blood flow

Integrated Response to Massive Hemorrhage

1. Baroreceptors – high pressure – decreased firing – result is enhanced Sympathetic output and less vagal output ↑ tachycardia, contractility, vasoconstriction – re-establish MAP

2. Baroreceptors – low pressure – reduced VOLUME – less activity of LPBs. Increased sympathetic output – vasoconstriction – particularly of kidney BVs Increased release of Anti-diuretic hormone

3. Peripheral Chemoreceptors – low MAP reduces perfusion of carotid/aortic bodies Local hypoxia – increased firing of chemoreceptors – vasoconstriction and changes in ventilation.

4. Central Chemoreceptors – fall in blood pH (acidosis) – increased sympathetic Output – vasoconstriction

5. Adrenal medulla – as a result of sympathetic stimulation – increased Medullary secretion of epinephrine (a BP drop to 40 mmHg - 50 fold increase in Epi)