Arterial Blood Pressure
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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).

Blood pressure values: what do they mean?
- Pulse pressure:
  \[ PP = SP - DP \]
- Mean arterial blood pressure = MABP
  \[ MABP = DP + \frac{1}{3} (SP-DP) \]
- CO = \[ MABP = SV \times HR \]
  TPR

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

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>
</tr>
</tbody>
</table>
Measurement

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

3 Ways to Measure Blood Pressure

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

![Blood Pressure Diagram]

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
- **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

Primary Hypertension Pathophysiology

- **Primary Hypertension Pathophysiology**
  - Heredity – interaction of genetic, environmental, and demographic factors
  - Water & Sodium Retention – 20% of pts with high Na+ diet develop HTN
  - Altered Renin-Angiotensin Mechanism – found in 20% of patients
  - Stress & Increased SNS Activity
  - Insulin Resistance & Hyperinsulinemia
  - Endothelial Cell Dysfunction

Primary Hypertension Risk Factors

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

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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:** Orientals > Westerns due to 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 × Peripheral Resistance

- **BP depends on:**
  1. Cardiac output ⇒ CO = SV × HR.
  2. Peripheral resistance.

Regulation of Arterial Blood Pressure

Regulation of ABP:

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

Mean arterial pressure
Cardiac output = Stroke volume \times Heart rate

End diastolic volume (EDV)
Frank - Starling

Parasympathetic input - HEART
- Vagus nerve releases ACH
- SA and myocardium
- HR and conduction velocity
  - R side SA node (HR)
  - L side contractility (slight)

Parasympathetic input - Blood vessels
- Activated - Vasodilatation

Sympathetic input - Blood vessels
- No-repinephrine
  - α > β
- Epinephrine
  - β > α
  - Vasodilatation – α 1
  - Vasodilatation – β 2
Parasympathetic input – Blood vessels

**ACTIONS**
- Vasodilation of BVs
- Less common than the sympathetic activity
- Salivary glands, e.g. 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.

Regulation of Arterial Blood Pressure

B. Regulation of Peripheral Resistance

<table>
<thead>
<tr>
<th>vasodilatation</th>
<th>vasoconstriction</th>
</tr>
</thead>
<tbody>
<tr>
<td>stimulation of cGMP</td>
<td>stimulation of cAMP</td>
</tr>
<tr>
<td>NO, ANP, adenosine A2, histamine H2, adrenaline β2, VIP</td>
<td>serotonin, adrenaline α2, angiotensin II, vasopressin</td>
</tr>
</tbody>
</table>

In smooth muscle, cGMP and cAMP stimulates Ca²⁺ pump of the sarcoplasmic reticulum
Decrease of Ca²⁺ concentration in smooth muscle cell
Slower decrease of Ca²⁺
IP₃ releases Ca²⁺ from the sarcoplasmic reticulum
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>Mechanism</th>
<th>Action</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>vasoconstriction 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></td>
<td>parasympathetic dilator nerves in some secretory and spongiform tissues</td>
</tr>
<tr>
<td>humoral</td>
<td>constriction by angiotensin II, epinephrine, vasopressin, serotonin</td>
</tr>
<tr>
<td></td>
<td>dilatation by 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.

\[ + \text{BP} \]
\[ = \text{V.M.C} \]
\[ + \text{C.I.C} \]
\[ = \text{Sympathetic vasodilatation} \]
\[ + \text{Parasympathetic} \]
\[ \text{Slowing of SA node (↓ HR)} \]
\[ \text{& ↓ CO} \]

Carotid and Aortic Baroreceptors

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

Baroreceptor Feedback Loop for the Regulation of Mean Arterial Blood Pressure

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

MAP ~ 93.5 mmHg

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)

Parasympathetic activation bradycardia
- To heart

Sympathetic activation of:
- Blood vessels
- Heart
- Adrenal medulla
- To spinal cord

2. Chemoreceptors reflex:
- Chemoreceptors are receptors found in carotid & aortic bodies.
- Are stimulated by chemical changes in blood mainly hypoxia (↓ O₂), hypercapnia (↑ CO₂), & pH changes.

Haemorrhage
- ↓ BP

Hypoxia
- + + VM.C
- + Chemoreceptors
- = C.I.C

Hypoxia & ↑ HR
- ↑ Adrenal medulla

Vasoconstriction & ↑ TPR.

Chemoreceptors
- Peripheral (ventilation)
  - Sense low O₂
  - Carotid and aortic (glomus cell)
  - Synapse with IX and X, respectively
- Central (medulla/CNS)
  - Sense low pH primarily
- Exert a positive drive on vasomotor area
- Exert a positive drive on cardio-inhibitory area

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

Vasoconstriction and Bradycardia
1. **Atrial stretch receptor reflex:**
   - Venous Return \(\Rightarrow\) ++ atrial stretch receptors \(\Rightarrow\) reflex vasodilatation & ↓ BP.

2. **Thermoreceptors:** (in skin/or hypothalamus)
   - Exposure to heat \(\Rightarrow\) vasodilatation.
   - Exposure to cold \(\Rightarrow\) vasoconstriction.

3. **Pulmonary receptors:**
   - Lung inflation \(\Rightarrow\) 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.

**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
  - Vasodilation by means of NO production

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 plasma, 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

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

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**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


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)

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**Hemorrhage**

- Venous return
- Blood volume
- SV and CO
- Atrial volume
- MAP

**Central Chemoreceptors**

- Peripheral Chemoreceptors

**Medullary Cardiovascular Control Center**

**SYMPATHETIC RESPONSE**

- ↑ Heart rate
- Contractility
- Vasoconstriction (arterial/venous)

**Hormonal response**

- Angiotensin/Renin
- ADH release
- ANP (decreased)

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**SVR = systemic vascular resistance**

**CO = cardiac output**

**SV = stroke volume**

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**HIGH BLOOD PRESSURE**

- Intervene to lower BP
- Medications
- Non-pharmacological interventions
- Exercise
- Reduce salt
- Weight management
- Stress management
- Smoking cessation

**LOW BLOOD PRESSURE**

- Intervene to raise BP
- Medications
- Non-pharmacological interventions
- Exercise
- Reduce salt
- Weight management
- Stress management
- Smoking cessation