Hormonal Regulation of Arteriolar Diameter

Vasoconstrictor Hormones

Vasopressin
Epinepherine
Norepinepherine
Angiotensin II

Vasodilator Hormones

Kinins
Natriuretic Peptides

وقال تعالى

(وَجَعَلْنَا مِنَ الْمَاءِ كُلَّ شَيْءٍ حَيٍّ أَفَلَا يُؤْمِنُونَ)

Vasopressin

Formation:

Also called Antidiuretic Hormone
Made in **Hypothalamus**, Stored in **Post. Pituitary**

Stimuli of Secretion:

- ↑ Osmolarity of **ECF**
- **Hypovolemia** & **Hypotension**
- **Angiotensin II**

Actions of Vasopressin:

- ↓ Water excretion by kidney via V_2 Receptors. This regulates **ECF Volume** and **Osmolarity.**
- Strong **VC** Effect via V_1 Receptor.

Renin-Angiotensin System

Formation:

Renin is a proteolytic enzyme secreted from **Juxtaglomerular** apparatus of kidney. Convertes hepatic **Angiotensinogen** to **Angiotensin I** (Decapeptide)

Angiotensin Converting Enzyme (ACE) Converts Angiotensin I to Angiotensin II. It is found in **endothelial cells** of BV in **Lungs**.
90% of ACE → Membrane bound. **>10%** Free in blood.

Stimuli of Secretion of Renin:

- ↓ **Blood Flow** as in **Hypovolemia & Hypotension**
- ↓ **Blood** to kidney (Renal Ischemia) as in **Renal Artery Stenosis**.
- ↓ **Na** delivery to distal tubules (due to ↓ glomerular filtration rate)
- **Sympathetic Stimulation** via **β1** Adreno-receptors.

Angiotensin II Receptors:

- Angiotensin II Receptor Type 1 (AT_1): Widespread
- Angiotensin II Receptor Type 2 (AT_2): Limited in Adults

Action Via AT_1 Receptors:

Vascular Actions:
- **Direct:** Peripheral **VC** of arterioles and Veins
- **Indirect: Sympathetic** Stimulation & ↑ **Catecholamine** Secretion

Renal Actions:
- **Direct:** Stimulates **Na** Reabsorption
- **Indirect:** Stimulates **aldosterone** secretion → ↑ **Na** Reabsorption

Central Actions:
- Stimulate **vasopressin** secretion → ↓ Water **Loss**
- Stimulate **Thirst Centers** → ↑ Water **intake**

Other Actions:
- Inhibit **Renin** Secretion (Negative Feedback)
- Stimulates cardiac and vascular **hypertrophy**

Angiotensin III:
- Produced by enzymatic removal of 1 Amino Acid
- Has **100%** of Aldosterone effect & **40%** of VC Effect.

Action Via AT_2 Receptors:

Counterbalance AT1 Actions:
- Vasodilatation
- Diuresis (↑ Water excretion)
- Natriuresis (↑ Na excretion)
- Apoptosis (Programmed Cell Death)

Epinephrine & Norepinephrine

Secretion:

- **Both** Secreted from **Adrenal Medulla**
- **Norepinephrine** Secreted by Postgangelionic Sympathetic Nerves

Stimuli of Secretion:

- **Diffuse** sympathetic stimulation during emergency conditions
- Emotional Stress
- Hypoglycemia
- Exposure to **Cold**

Vascular Effects of Epinepherine:

- **VD** via β_2 Receptors in **Skeletal MS** & **Liver** → overbalances **VC** Effects. Via α_1 receptors in other organs → ↓TPR
- **VC** via α_1 in high dose → overbalance **VD** Effects

Vascular Effects of Norepinepherine:

- **VC** via α_1 receptors in almost all organs

Vasopressin

Types:

- **A**trial Natriuretic Peptide: Released by atrial myocytes
- **B**rain Natriuretic Peptide: Released by Brain + Myocytes
- **C**-Type Natriuretic Peptide: By Endothelial Cells, Brain, and Kidney

Receptors:

- **NPR-A**: Greatest affinity to ANP
- **NPR-B**: Greatest affinity to CNP
- **NPR-c**: Can bind all 3 peptides

Secretion ↑ in:

- **Stretch of atrial myocytes:** due to ↑ ECF Volume → ↑VR & CVP
  BNP secreted by ventricles in response to pressure & volume overload as in cases of heart failure.
- ↑ Na Concentration in ECF • Angiotensin II
- Sympathetic Stimulation of **β** Adrenoreceptors

Action:

- Long term regulation of **Na-Water Balance**, **Blood volume**, and **ABP**
- **ANP** is physiological balance for Renin-agniotensin-aldosterol-system.

Vascular Actions:
- **VD** → Relaxation of Vascular smooth ms via ↑ cGMP.
- Inhibit **VC** effect of catecholamines & angiotensin II.

Renal Actions:
- Inhibit extra Renin Formation
- Inhibit aldosterone action (↓ action of **Na/K** ATPase)
- Inhibit Na Reabsorption = ↑ Na excretion

Adrenal Action: Inhibit aldosterone secretion

Overall Effect: ↓ Blood Volume, CVP, CO → ↓ABP

Inactivation by "**Neutral Endopeptidase**" Enzyme
- Drugs inhibiting enzyme → ↑ natriuretic peptides → Treatment of heart failure & hypertension in combination with **ACE inhibitors** or **ARBs**

Kinins

Types:

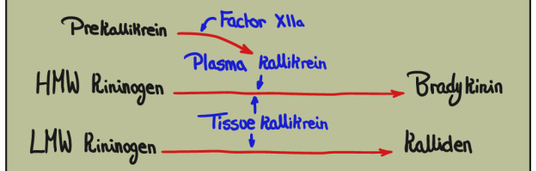
Mainly present in **tissues**, **small** amounts in **blood**.
- **Bradykinin** (Nonapeptide, 9aa)
- **Kallidin** (Decapeptide, 10aa)

Formation:

Via actions of 2 proteases and 2 precursors (HMW Kininogen & LWM Kininogen)

Tissue Kallikrein: Formed locally in tissues
- HMW Kininogen → Bradykinin
- LMW Kininogen → Kallidin

Plasma Kallikrein: Formed by liver, circulates as inactive enzyme (**Prekallikrein**)
- Activated by active clotting factor XIIa on endothelial cell surface
- HMW Kininogen → Bradykinin



Receptors:

- **B_1** Receptors: fewer, mainly ↑ in number in inflamed, injured, or ischemic tissue.
- **B2** Receptors: Main type, In many tissues.

Actions:

- **VD** & ↓ **ABP** via NO release.
- ↑ Capillary permeability.
- **Positive chemotactic** effect.
- Inhibits **thrombin-induced** platelets aggregation.
- ↑ Release of **Tissue Plasminogen Activator** (tPA) from endothelial cells.
- Stimulation of **pain** receptors.
- Contraction of plain muscles of **viscera**.

Inactivation:

By **Kininase II Enzymes** (The same enzymes as **ACE**)
∴ **ACE** Inhibitors → ↑ Kinin concentration → ↓ ABP

Moataz Alsharkawy

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