Control of Body fluid Volume & Osmolality

- Kidneys maintain volume & osmolality by regulating H_2O and NaCl excretion

- Body fluid — 60% of healthy adult

ICF ECF

⇒ Water balance :-

Water intake = Water loss

Water intake: fluid ingestion, food

Water loss:

- Renal excretion
  - Major loss
  - Tightly regulated
- Insensible water loss
  1. Evaporation from cell surface
  2. Respiratory loss
  3. Loss thru GIT fecal water
  4. Sweating.
  - Cannot be regulated
  - depends on envt condit

↑ water intake ⇒ +ve H_2O balance

↕ water loss ⇒ −ve H_2O balance.

- Normal individual urine osmolality ranges from 50 mOsm/kg to 1200 mOsm/kg and urine volume from 18 l to 0.5 l/day.

- Kidneys can regulate excretion of H_2O separately from excretion of total solute.
  This allows H_2O balance to be achieved without disrupting homeostasis of other solutes.

- The disorders of H_2O balance are manifested by changes in ECF osmolality (Posmolality) and thus by change in $[Na^+]$.

- Change in ECF vol indicate alterations in Na^+ balance.

⇒ Arginine Vasopressin / ADH :-

AVP receptors

V_1	V_2
- Blood vessels	- Nephron
- Smooth m/s contraction	- Reabsorption of H_2O.

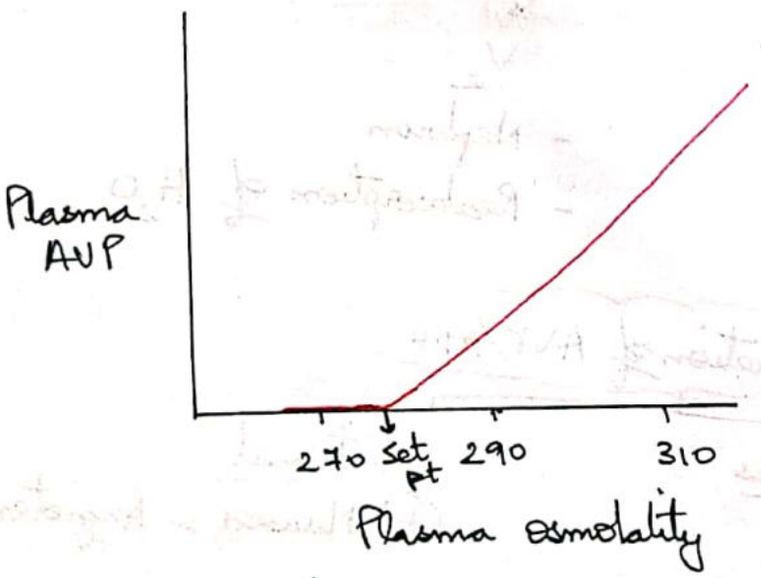
Secretion of AVP/ADH

Main factors
1. Blood vol
2. Blood pressure
3. Osmolality of body fluids

Additional
(+) Nausea > Angiotensin II
(−) ANP

- Osmotic control of AVP secretion:-

- Osmoreceptors → monitor osmolality of body fluids
  sense even minor changes
  → Organum vasculosum of Lamina terminalis (OVLT) of hypothalamus.
  → Respond by swelling up / shrinking.
- Respond to effective osmolality
  ↑ eff osm —(+)→ ↑ secretion.
- circulating levels of AVP fall rapidly after secretion is inhibited
- Responds rapidly.



↳ Set pt ranges from 275 to 290 m Osm/kg [avg = 285 m Osm/
the pt at which AVP sec ↑

- Alteration in Blood vol or press can shift the set pt even pregnancy due to some hormone
  ↓ Bld vol or press → set pt shifted to lower values.
  slope steeper

Hemodynamic control :-

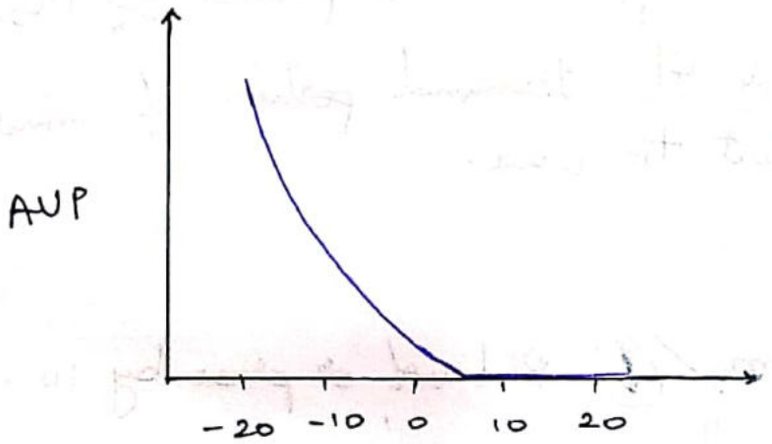
Blood volume or pressure changes sensed by

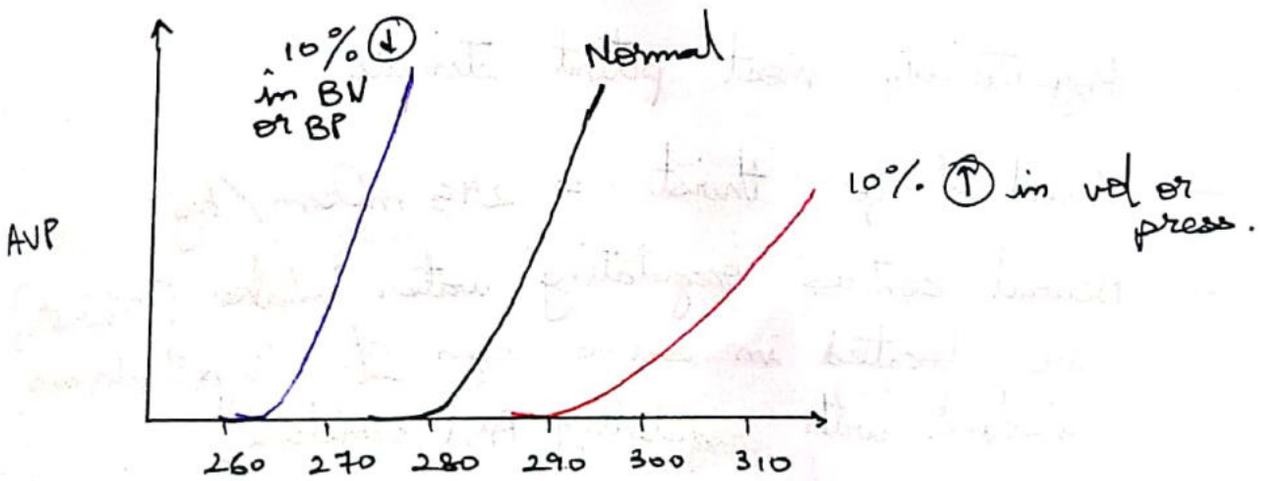
Baroreceptors → High press sys → aortic arch, carotid sinus

→ low press sys → Atrium.

↓ BP ⊕ ↑ secretion of AVP.

5 - 10% ↓ in BP / Bld vol req to cause AVP sec.





Action of AVP

① ↑ H_2O reab from distal segments which are rel. impermeable to H_2O in its absence.

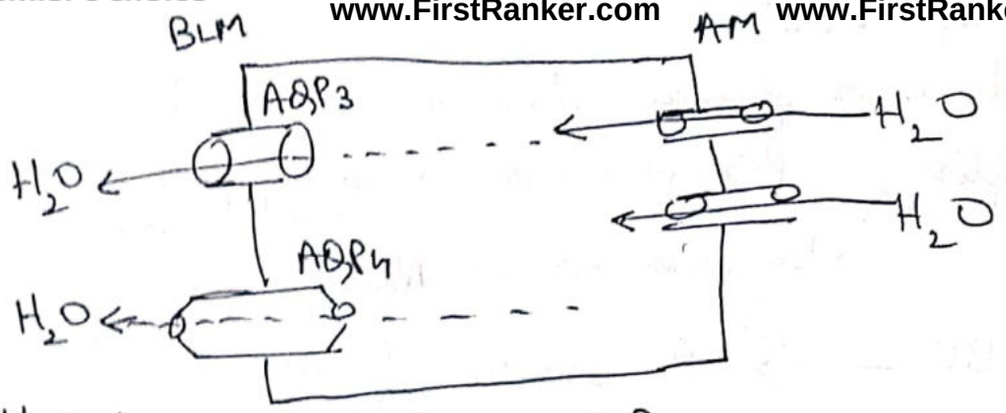
Binding of ADH to V_2 receptors

↓

AQP2 incorporated into apical membrane

↓

H_2O enters cell



H_2O leaves cell thru AQP 3 & 4

AVP regulates long term expression of AQP 3 & 4.

(2) ↑ permeability of terminal portion of inner medullary collecting duct to urea.

⇒ Thirst :-

- ↑ plasma osm / ↓ Bld vol or press by 10 - 15%
  by 2 - 3%
  ⇓
  Individual perceives thirst.
- Hypertonicity most potent stimuli
- Threshold for thirst = 295 mOsm/kg
- Neural centres regulating water intake [Thirst] are located in same regn of hypothalamus involved with regulating AVP secretion.

Regulation of ECF vol & NaCl excretion :-

- NaCl → major determinant of ECF osmolality.
- alterations in Na^+ balance → distorts ECF volume and not osmolality.

Reason :- Bcoz of ADH

Addition of NaCl → ↑ osmolality
↓
↑ osmoreceptors
↓
↑ sec of ADH
↓
↑ H_2O reab
↓
plasma osmolality back to normal but ECF vol is ↑ ie there is "volume expansion."

Removal of NaCl → opp happens
↓
Renal excretion H_2O ↑
↓
Volume contraction but plasma osmolality restored.

- The kidneys keep ECFV const (euvolemia) under normal conditions, by adjusting excretion of NaCl to match the amnt ingested.

Initial Euvolemia
14 L
$[Na^+]$ = 145 mEq/L
[Anions] = 145 mEq/L
osmolality = 290 mOsm/kg

+145 mmol NaCl

Transient state
14 l
$[Na^+]$ = 155 mEq/L
[Anions] = ''
osm = 310 mOsm/kg
osm ↑ → ↑ ADH

Final state
Volume expansion
15 l
$[Na^+]$ = 145 mEq/L
[Anion] = ''
osm = 290

−145 mEq NaCl

Transient state
14 l
$[Na^+]$ = 135 mEq/L
[Anions] = ''
osm = 270 mOsm/kg
osm ↓ → ↓ ADH

Volume contraction
13 l
$[Na^+]$ = 145
[Anion] = ''
osm = 290.

- The response of kidneys to abrupt changes in NaCl intake typically takes several hours to several days depending on the magnitude of change.

∴ ECFV regulated by controlling NaCl excretion.

If ingestion > excretion → vol expansion → +ve Na^+ balance.
and vice versa.

⇒ Effective circulating volume:-

- Maintenance of Na^+ balance → ECF Vol req complex system of sensors mainly in vascular system.

- Principal factors reg NaCl excretion
  - change in Vas vol
  - BP
  - CO.

changes in ECFV → change in Bld vol, BP, CO.
in normal person

(↑) ECFV → (↑) BV, BP, CO.

- Effective circulating vol:-
  - portion of ECF present within vascular sys and is effectively perfusing the tissues
  - Not measurable.
  - ECV reflects activity of vol sensors located in vas sys.

- Healthy ppl ⟹ ECV varies with ECFV.

⇒ Volume sensing systems :-

- change in vol of vas sys → change in BP
- Vas Volume receptors are nothing but baroreceptors resp to press induced stretch of wall (vessel or atria etc

Volume sensors

VS in low pressure cardiopulmo sys

- present in walls of atria right ventricles & large pulmo vessels.
- ↓ in filling → ⊕ AVP sec → ↑ symp activity
- 5-10% change in BV > BP necessary for response

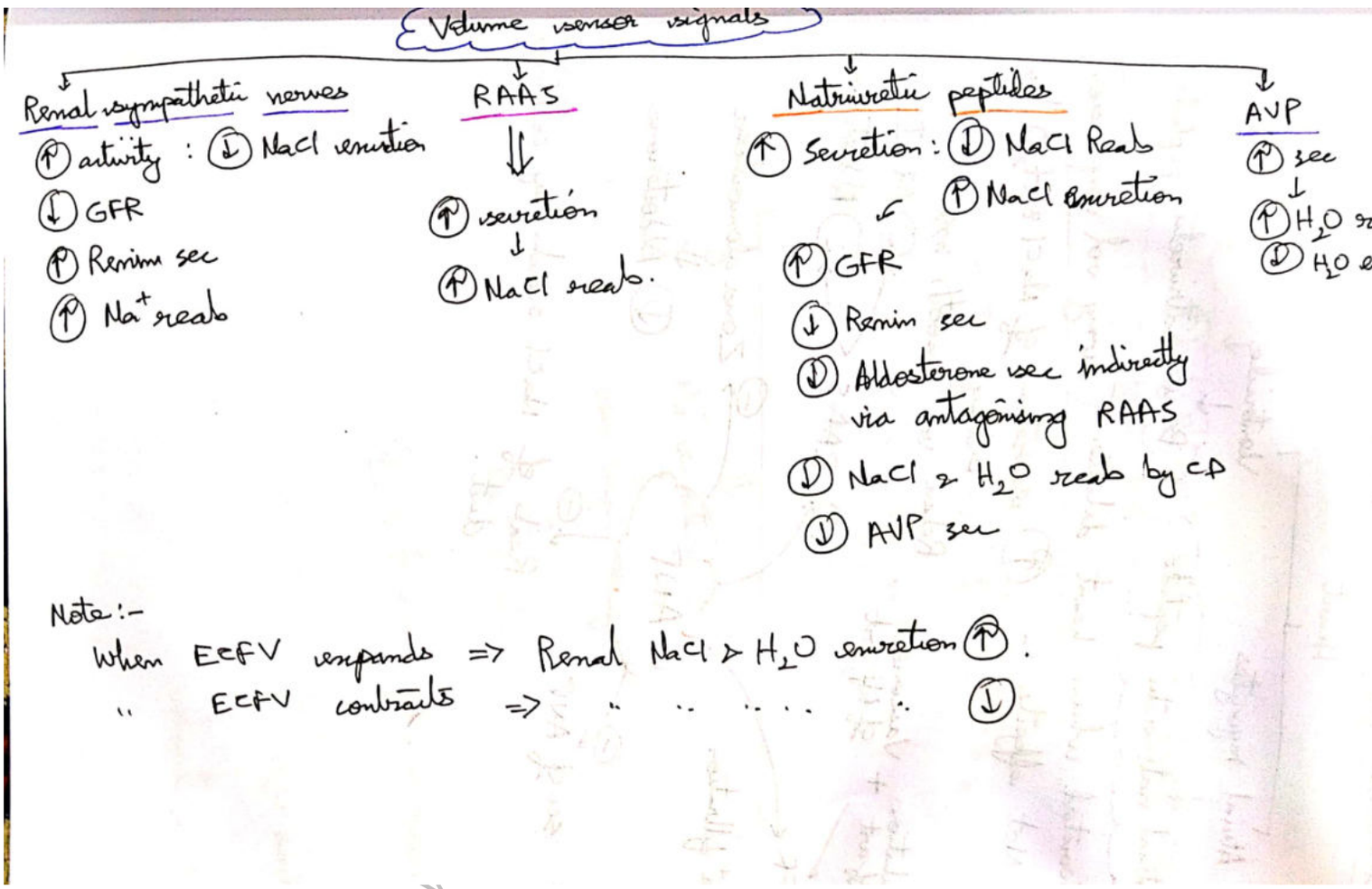
VS in high press arterial tree.

- prsn wall of aortic arch carotid sinus, aff art of kidneys.
- ↓ in BP → ⊕ AVP sec, ↑ Symp activity
- 5-10% changes in BP req

- The signals from baroreceptors sent to brainstem thru glossopharyngeal & vagus nerve.

⇒ Vol sensor signals :-

↳ Both neural + hormonal.



Note :-

When ECFV expands ⇒ Renal NaCl & H_2O excretion ↑.

" ECFV contracts ⇒ " " " " " ↓

⇒ Natriuretic peptides :- Antagonise RAAS

Heart

Atrial myocytes → Atrial natriuretic peptide

Ventricle → Brain natriuretic peptide

- Secreted when heart dilates [during vol expansion].
- Net effects :- (↑) excretion of NaCl & H_2O by kidneys

Relax vas smooth m/s

Vasodilation of Aff art + Vasoconst of Eff art → (↑) GFR → (↓) Na^+ filtration

(−) RAAS → (↓) AT-II, (↓) Aldosterone

(−) Zona glomerulosa → (↓) Aldosterone

ANP

(−) sec of ANP

(−) Reab of NaCl reab by collecting duct.

⇒ Control of NaCl with Vol Expansion :-