Water regulation

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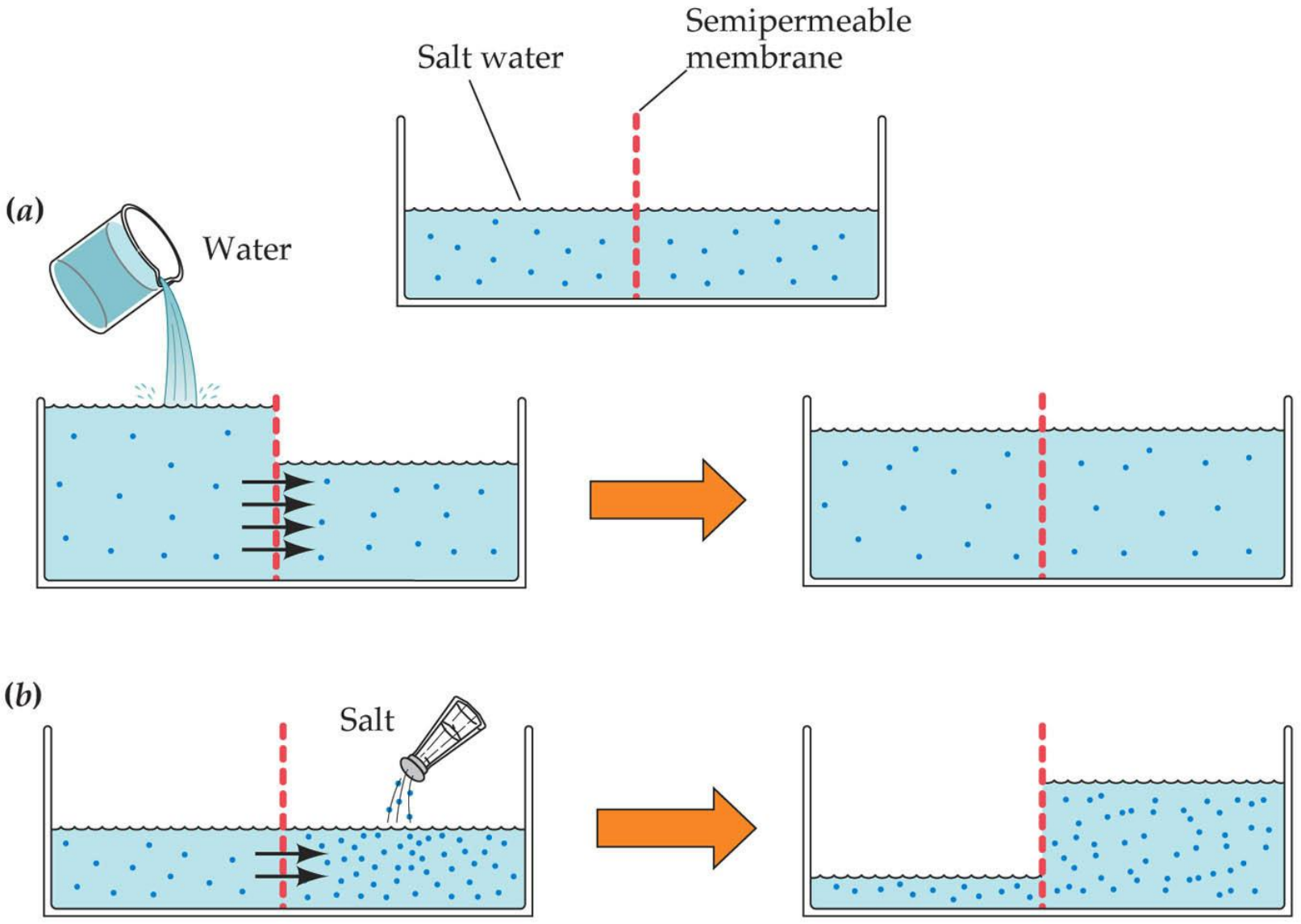
Osmosis



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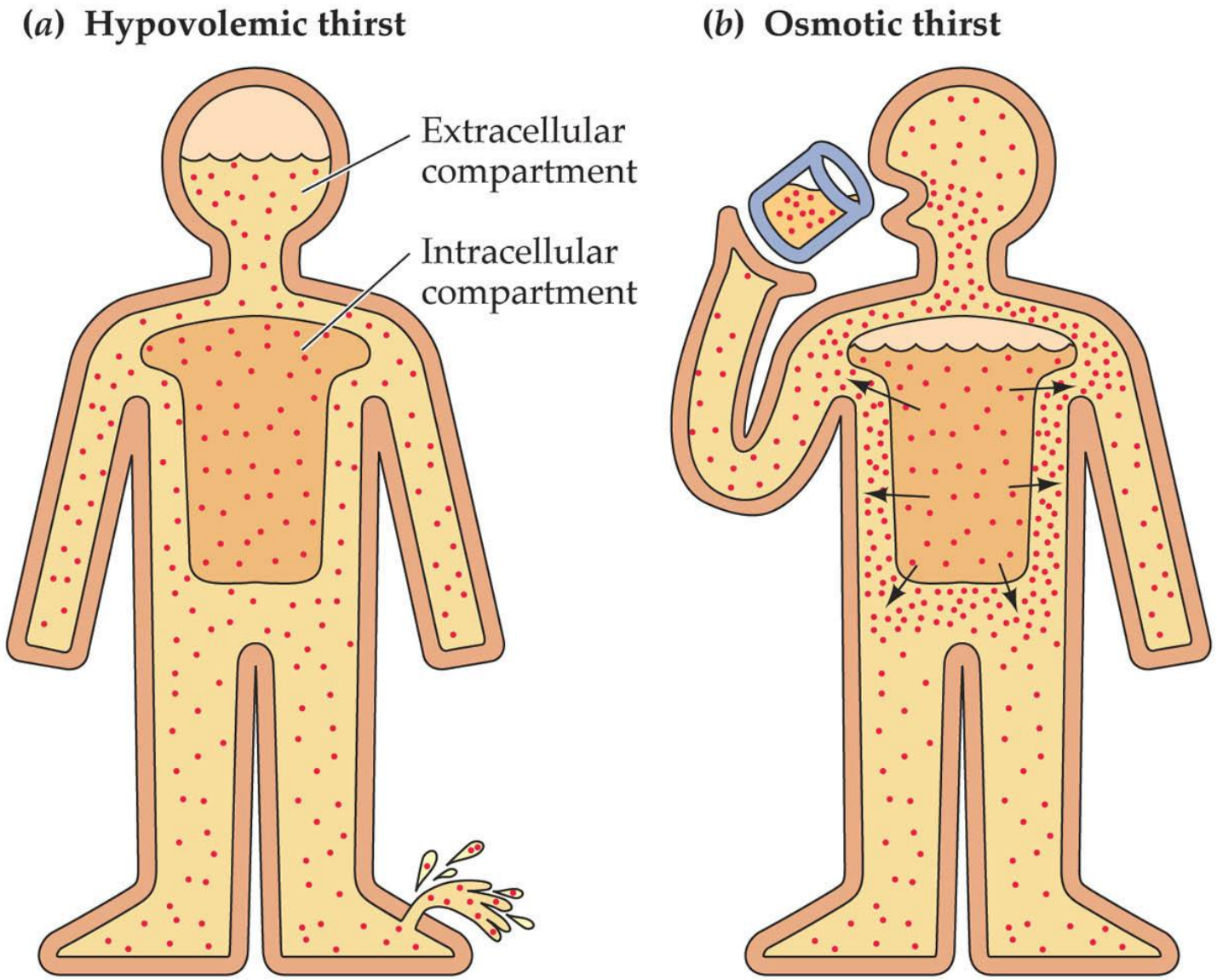
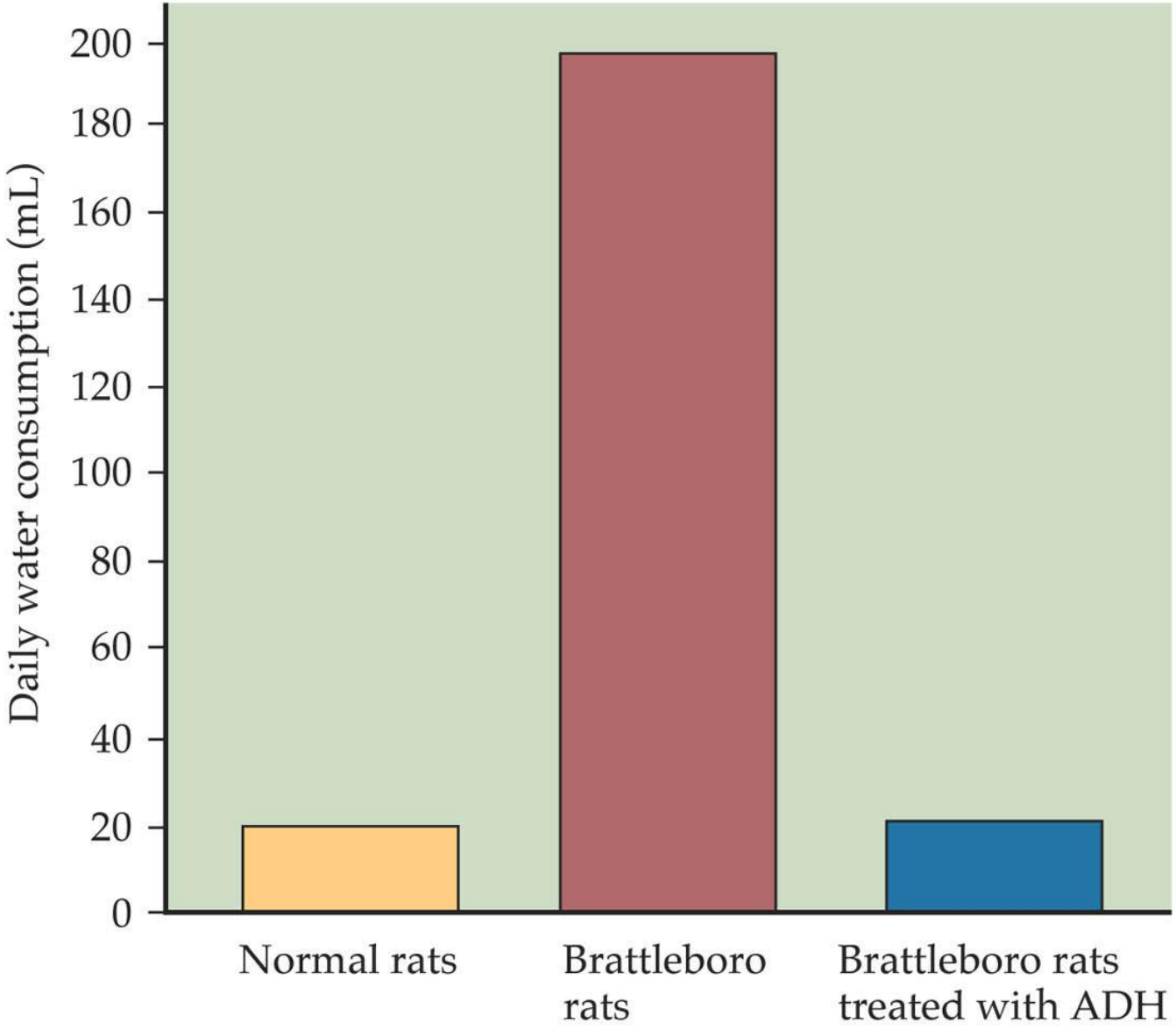
Two Kinds of Thirst



Inherited Diabetes Insipidus in Rats



Water: largest constituent of body; 55-65% of body weight

- Intracellular Fluid
  - 66.6%
  - Within cells
  - High potassium
- Extracellular Fluid
  - 33%
  - Interstitial, space surrounding cells
  - Intravascular; 7-8% of total body water, 20-25% of ECF
  - High sodium

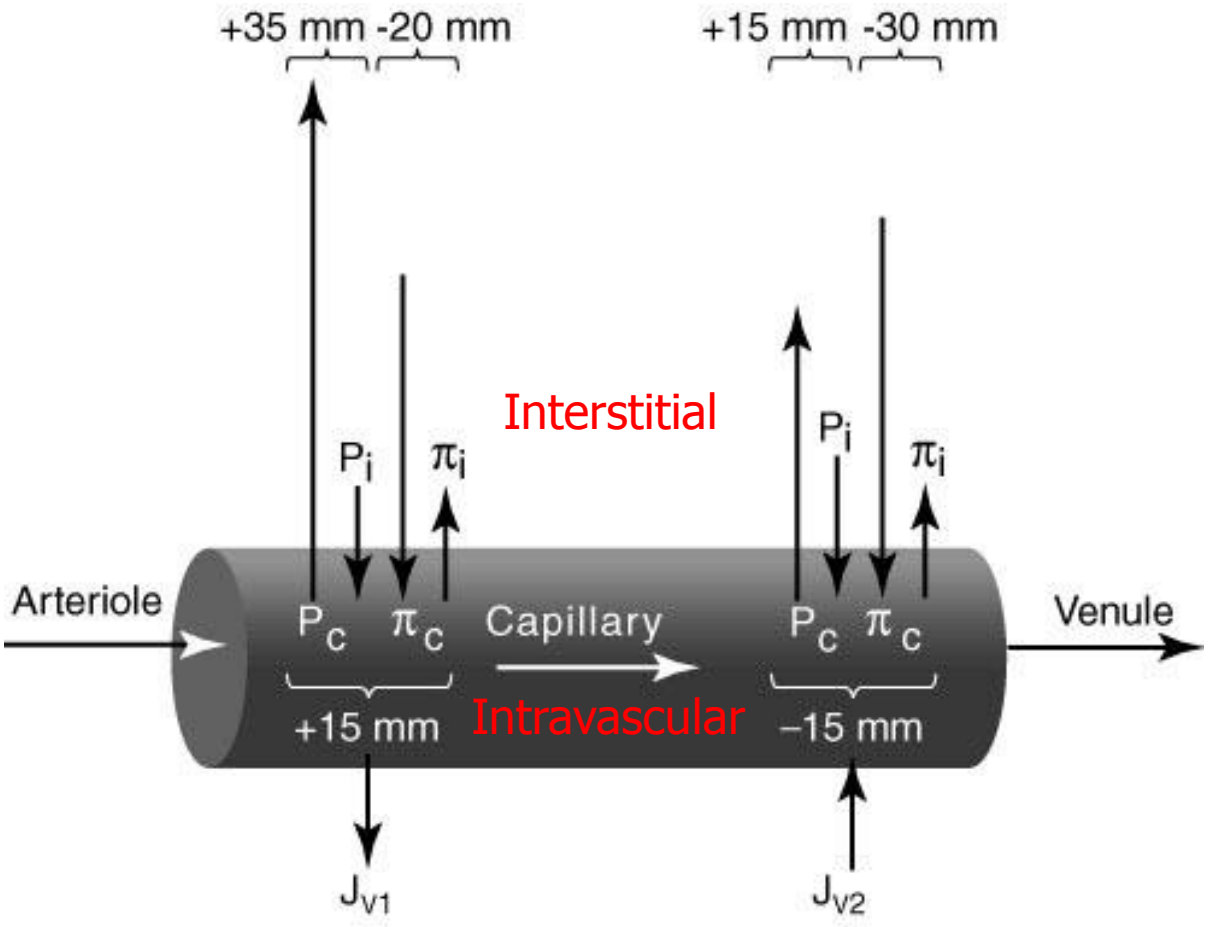
Osmotic pressure (concentrations of all solutes in a fluid compartment) is equivalent between ECF and ICF compartments

Blood volume and blood pressure are partially regulated by hydrostatic and osmotic pressure gradients

Starling equilibrium:

Distribution of fluid between intravascular and interstitial space is determined by balance between hydrostatic pressure of the blood and osmotic pressure from plasma proteins

Also compliance and glomerular filtration rate help regulate fluid balance



Blood pressure maintained by two other mechanisms

- Capacitance or compliance of vascular system
  - Arteries thick walled, veins thin walled & distensible. Volume loss, veins collapse. Conversely, volume accumulates in veins when blood volume expanded
- Glomerular filtration rate by kidneys
  - Drop in blood pressure reduces GFR & decreases urine volume, whereas a rise in BP increases GFR and promotes urinary fluid loss. Kidneys so efficient that development of hypertension indicates renal dysfunction

Summary

- Body fluid homeostasis: stability in the osmolality of body fluids & volume of plasma.
- Mechanisms: intrinsic to body fluids & cardiovascular system
  - Osmotic movement of water across cell membranes buffers ECF osmolality
  - Osmotic movement of water across capillary membranes buffers acute changes in plasma volume
  - Venous compliance
  - Glomerular Filtration

Osmotic homeostasis

Dehydration produces a need for water

Osmolality (expression of concentration) is the ratio of the amount of solute dissolved in a given weight of water: solute (osmoles)/water (kilograms)

Body water can decrease as a result of deprivation or sweating, whereas solute can increase as a consequence of salt ingestion

Either water decrease or solute increase leads to an increase in osmolality and consequent thirst

So – what is the neural substrates that initiates thirst? These are intimately tied in to mechanisms of control of water and sodium excretion and intake

Osmotic homeostasis

The initial response to cellular dehydration is release of arginine vasopressin (AVP) – the antidiuretic hormone

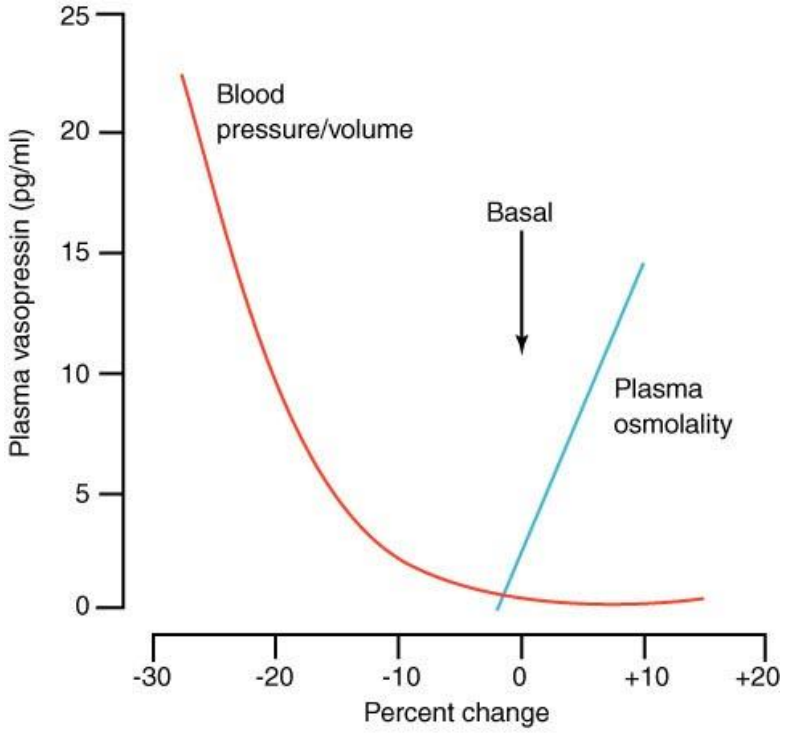
AVP is synthesized in the supraoptic n. and paraventricular n. of the hypothalamus and transported along axons to the posterior pituitary.

AVP is stored in secretory granules in posterior pituitary until an increase in osmolality of body fluids initiates its secretion into the blood

AVP acts on V2 receptors in the kidney to increase water permeability by inserting aquaporin channels into cell membranes

Water moves out of the distal convoluted tubule of the kidney by osmosis through these channels – decreasing osmolality

There is also an increased water reabsorption by the kidney and decrease in urine flow



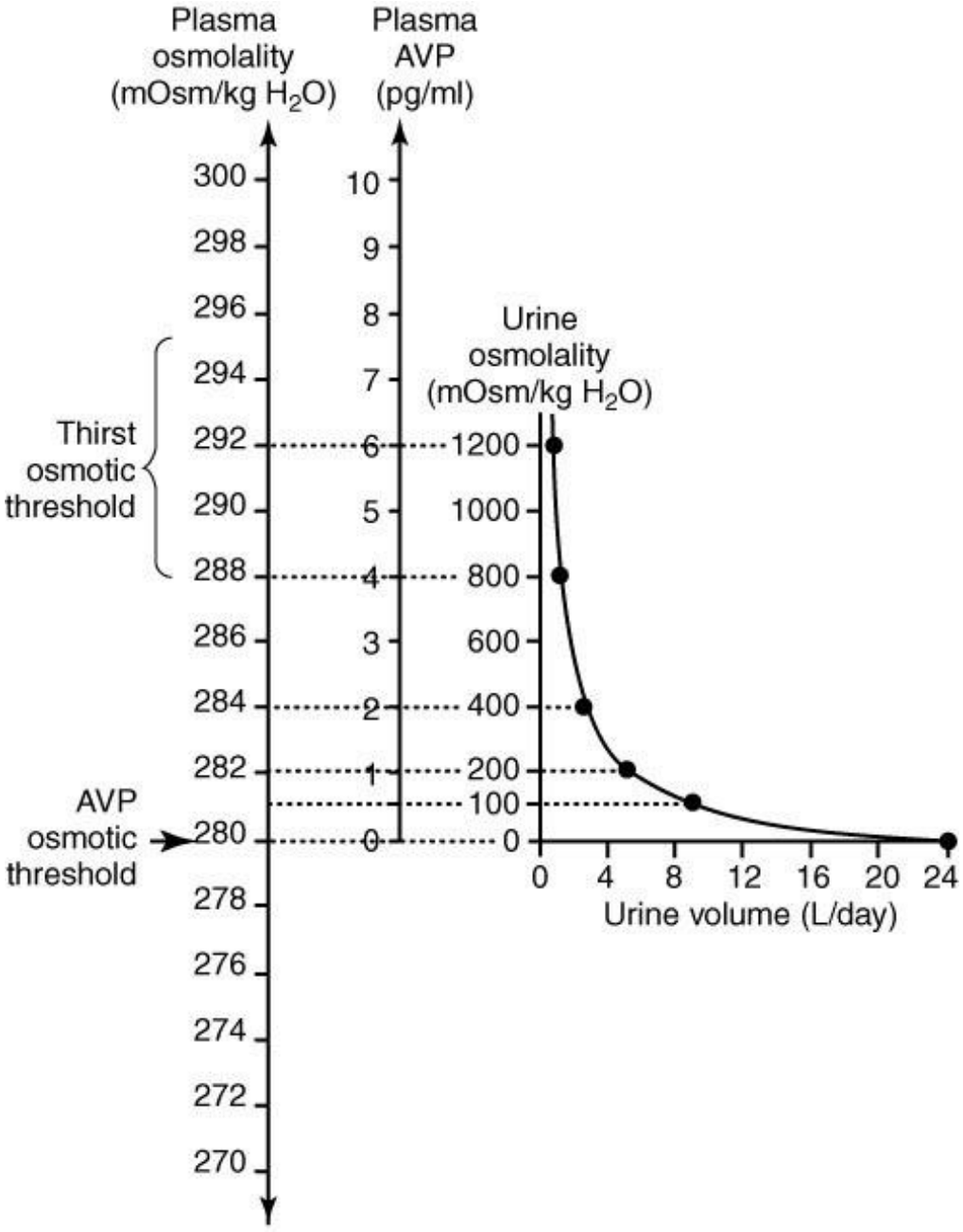
Osmotic homeostasis

Changes in the osmolality of plasma lead to AVP secretion at a much lower threshold than they lead to thirst

Very small increases in AVP lead to very large changes in urine volume

Thus – the kidney is the first line of defense against cellular dehydration

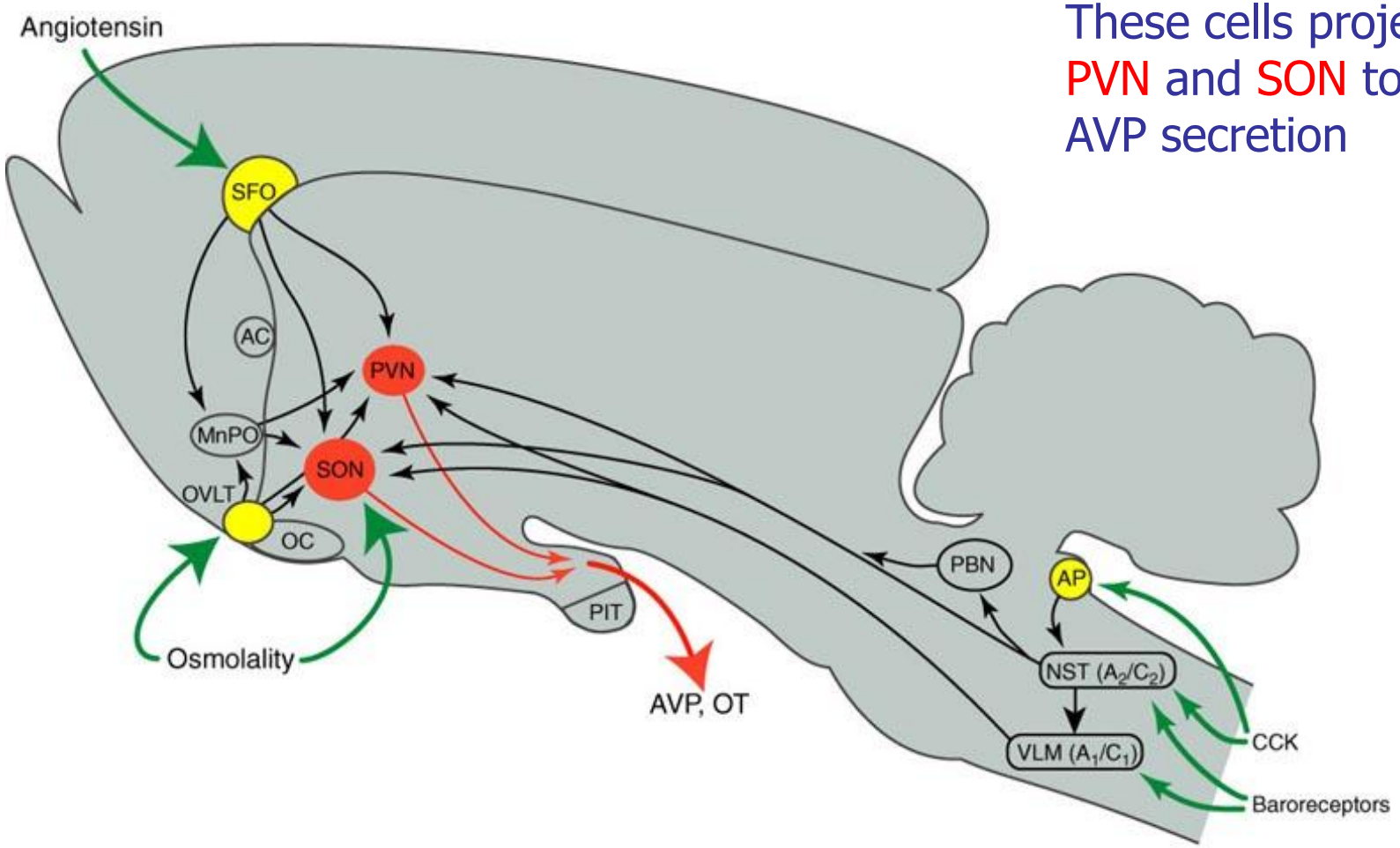
Ongoing behavior is not disrupted by thirst unless the buffering effects of osmosis and antidiureses are insufficient



Osmoreceptors stimulate AVP secretion and thirst

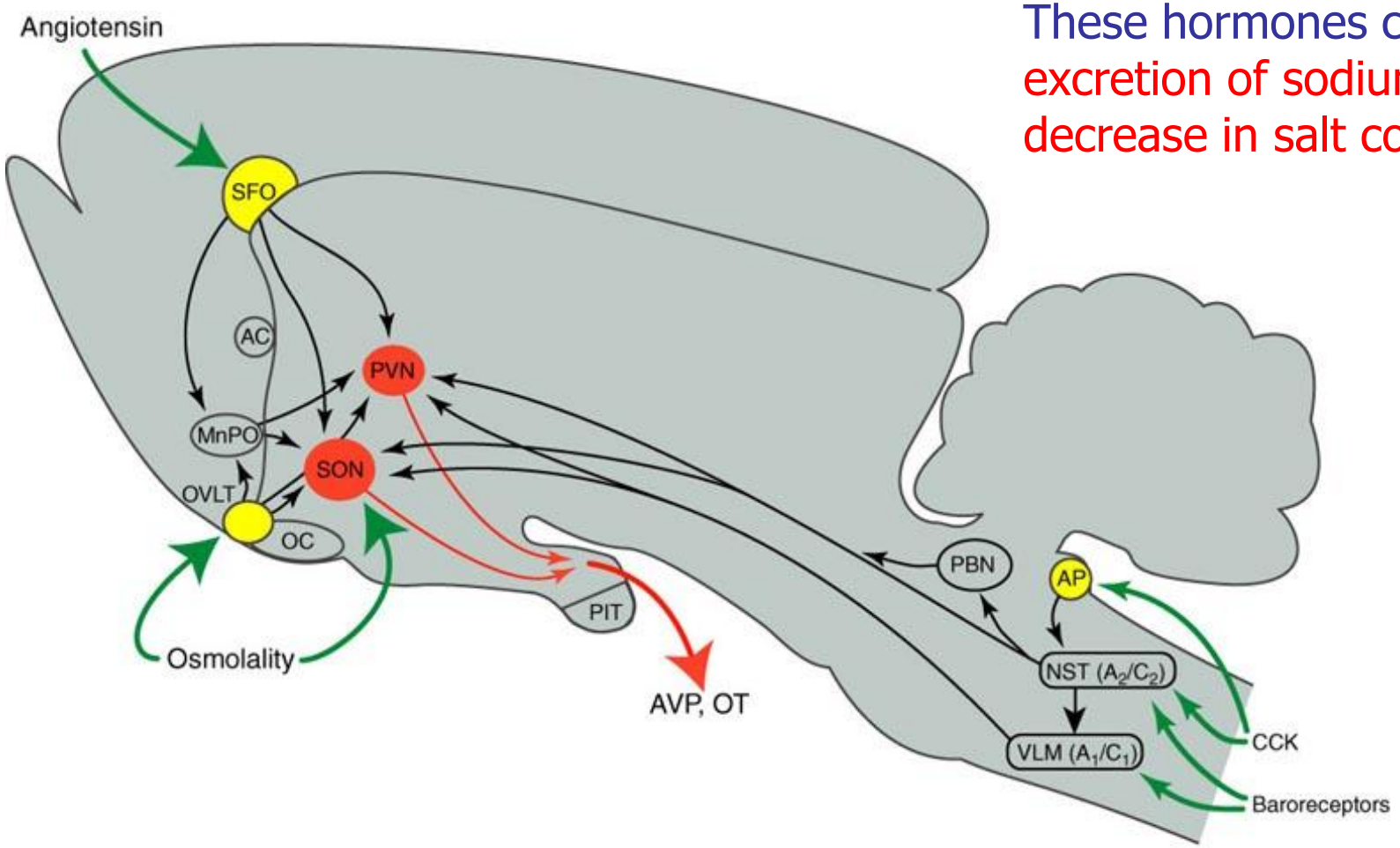
The vascular organ of the lamina terminalis (OVLT) contains osmoreceptive neurons – also the subfornical organ (SFO) and the median preoptic n. (MnPO)

These cells project the the PVN and SON to produce AVP secretion



Dehydration also produces natriuresis

Two hormones, one secreted in the heart (atrial natriuretic peptide; ANP) and the other in the brain (oxytocin; from PVN and SON in response to hyperosmolality)



These hormones cause excretion of sodium and decrease in salt consumption

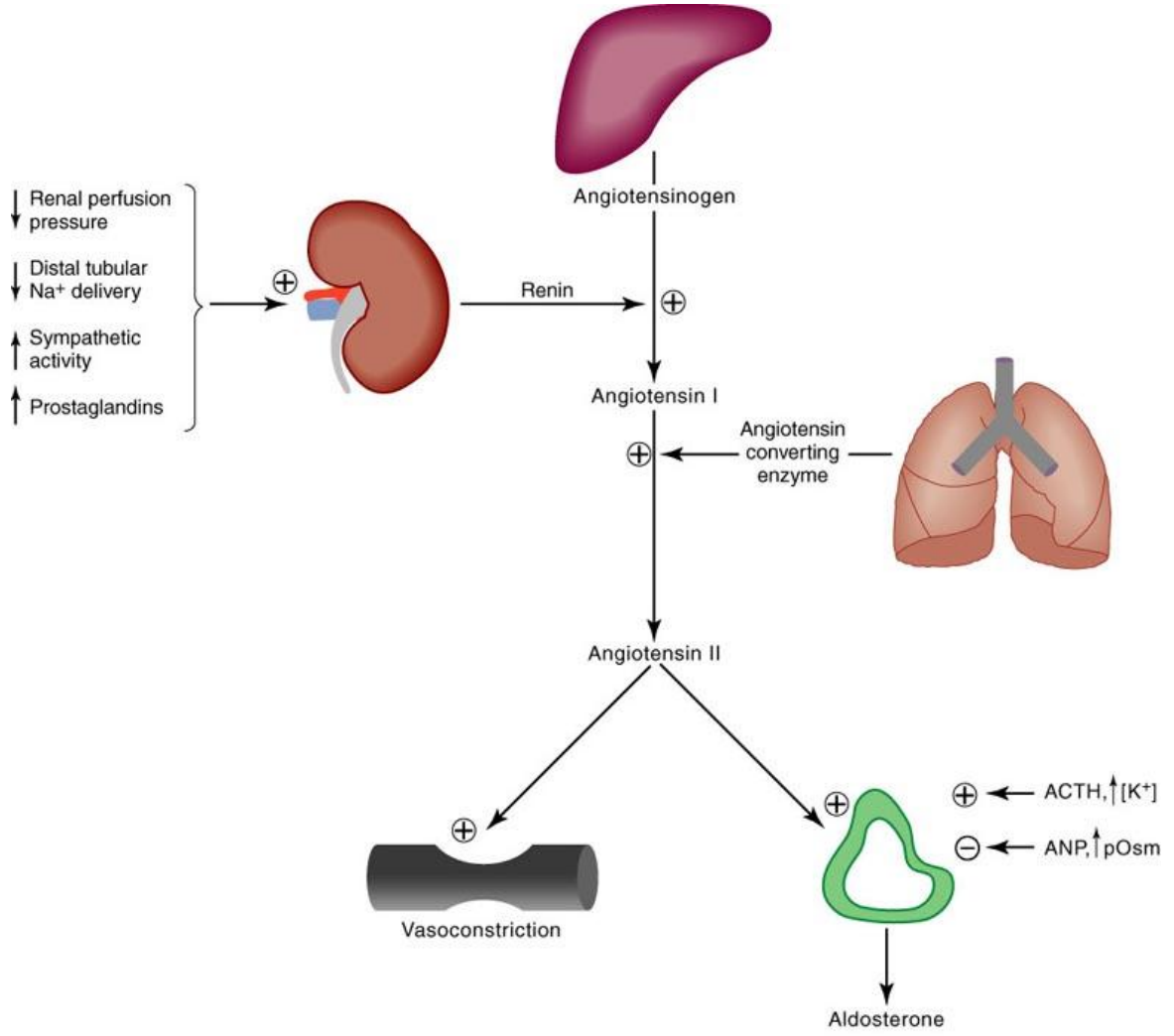
Volume homeostasis

A loss of blood volume (hypovolemia) leads to compensatory mechanisms, which include thirst and increased salt consumption

Baroreceptors sense hypovolemia and cause kidney to secret renin

Renin interacts with angiotensinogen to produce angiotensin I, which is converted to angiotensin II (AII)

AII is a vasoconstrictor and promotes aldosterone secretion from adrenal cortex and AVP secretion by acting on the subfornical organ (SFO)

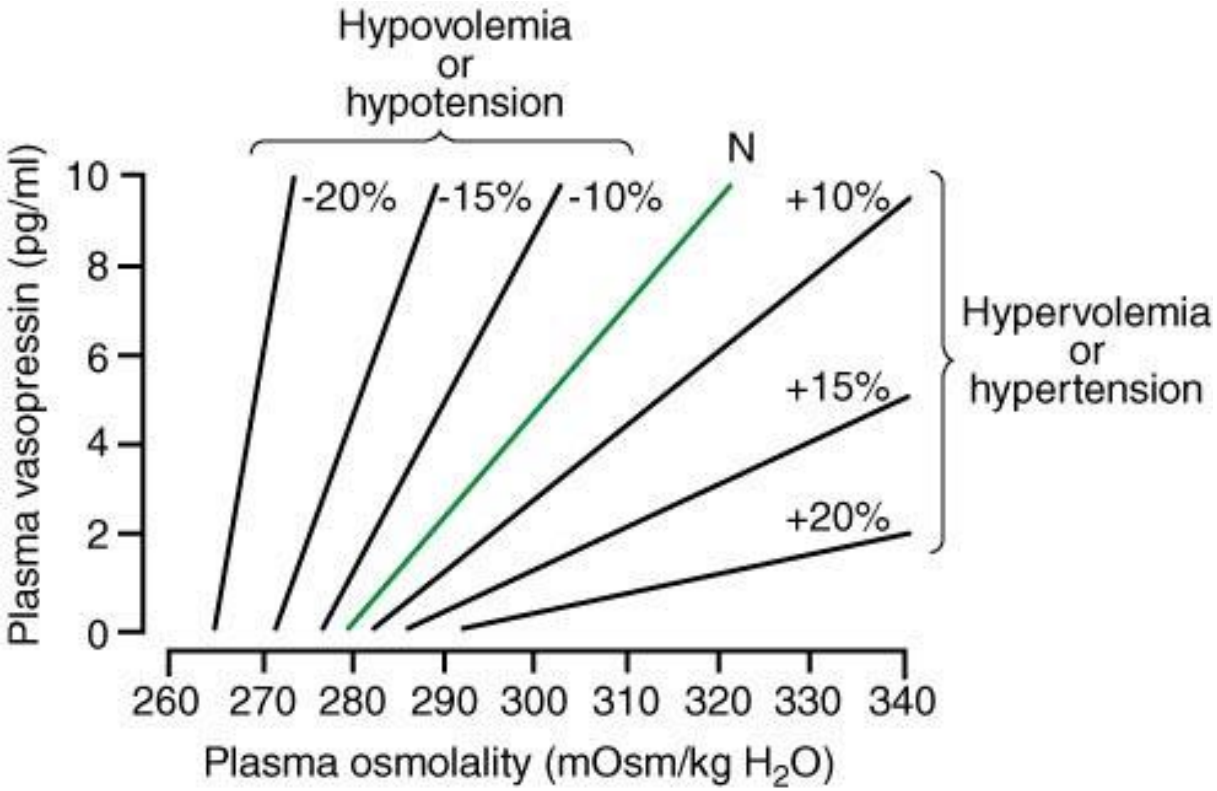


Volume homeostasis

Neural and endocrine signals of hypovolemia lead to thirst and increased salt consumption

The renin-angiotensin system and AVP produce antidiuresis and vasoconstriction

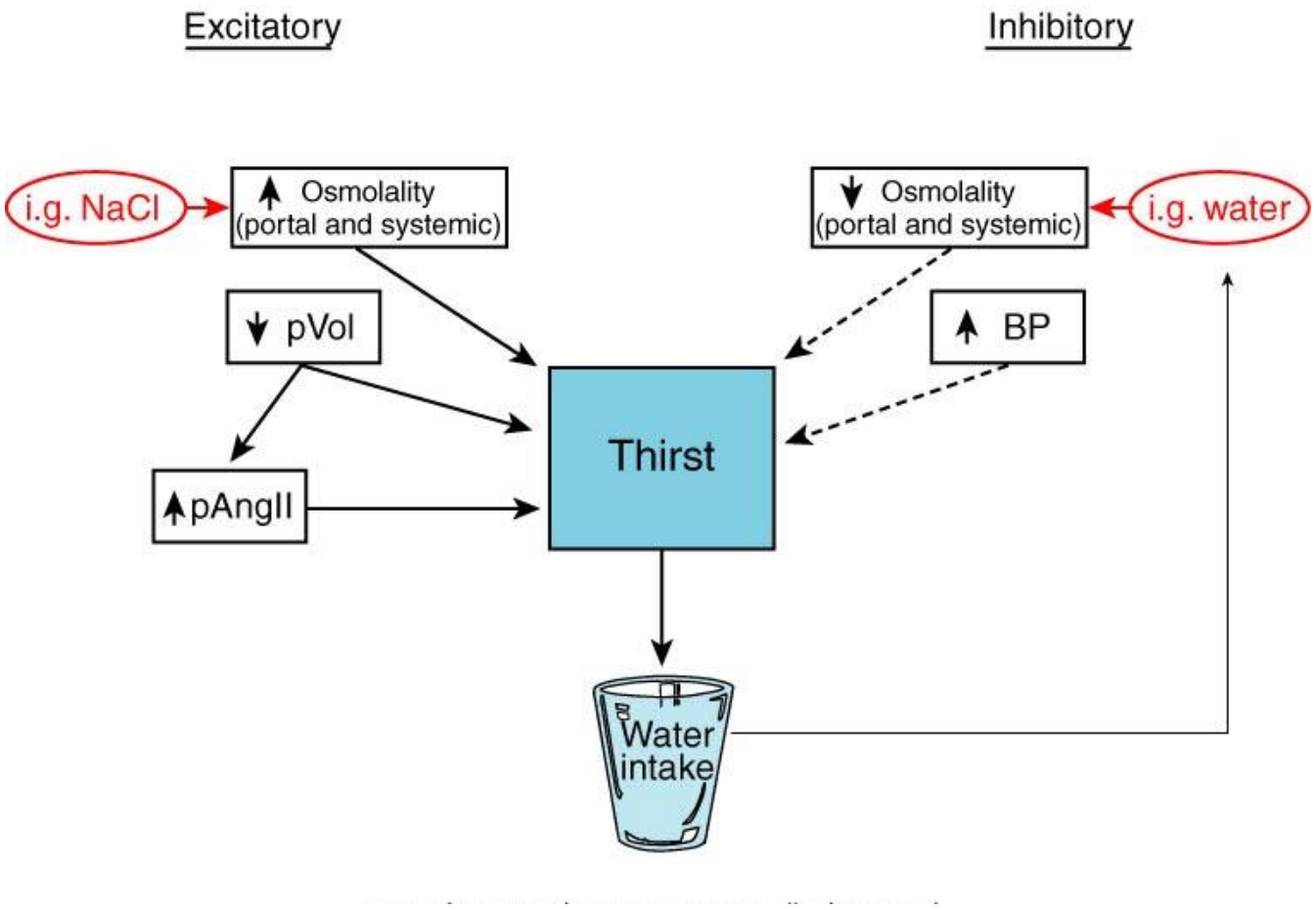
Both hypovolemia and hyperosmolality interact to control AVP levels – hypertension leads to decreased AVP, whereas hypotension increases AVP for a given plasma osmolality



Volume homeostasis

Thirst is triggered by increased plasma osmolality (OVLT receptors) , gastric salt load (hepatic Na^+ receptors), hypovolemia (angiotensin II in SFO).

Thirst is inhibited by decreased plasma osmolality (OVLT receptors) and by increased blood pressure (hypervolemia)

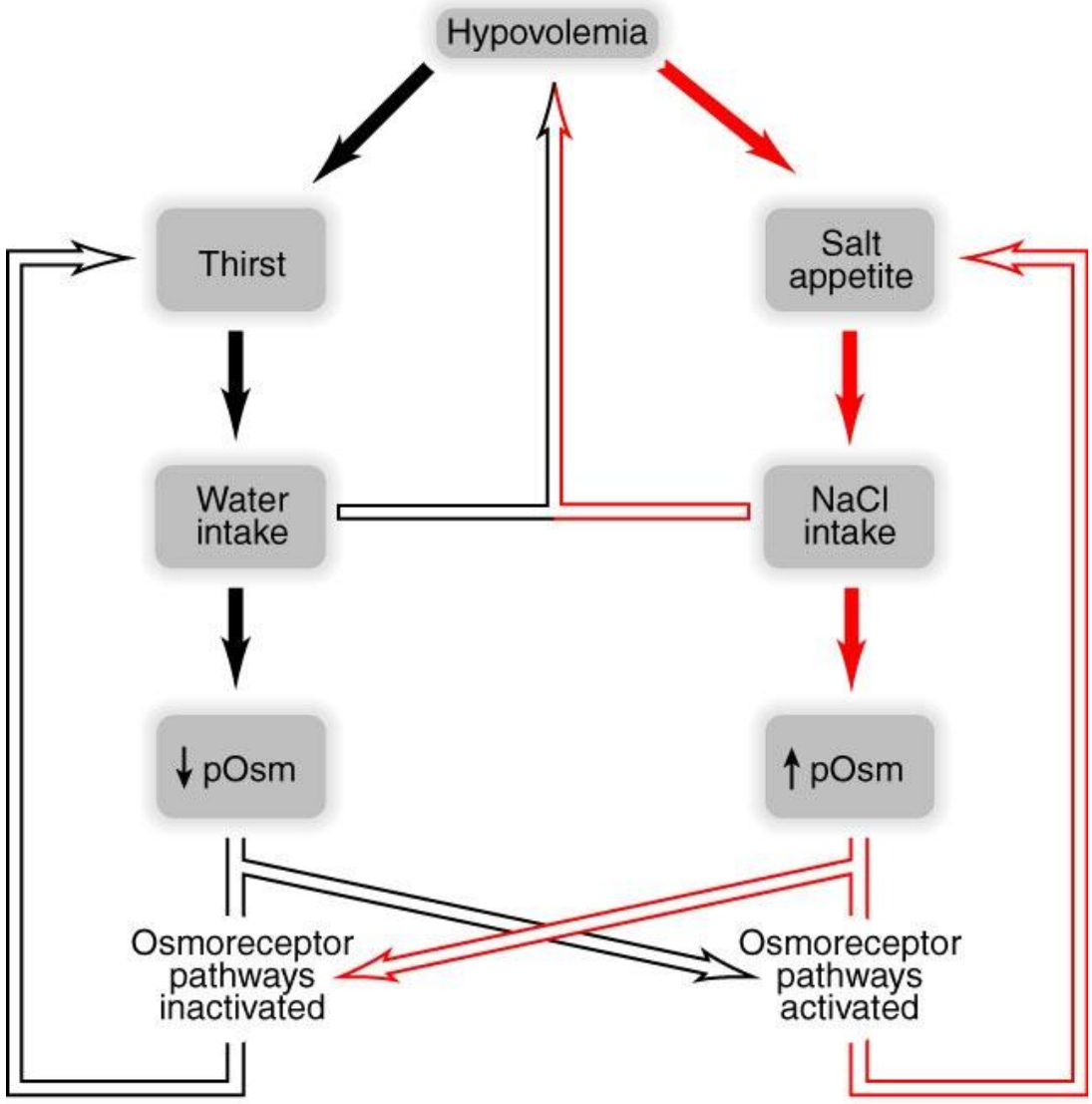


Volume homeostasis

Hypovolemia triggers not only thirst, but also salt appetite

Blood volume is corrected only by replacing both water and salt

Drinking water alleviates thirst (by reducing plasma osmolality), but triggers salt appetite, whereas consuming salt triggers subsequent thirst (by increasing plasma osmolality)

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