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RAAS & ADH: Blood Pressure and Osmoregulation

Study Guide — Renal Physiology

Conceptual Pre-med/IB-style questions on the renin–angiotensin–aldosterone system (RAAS) and antidiuretic hormone (ADH): why these systems exist, what triggers them, how they amplify signals, how they change urine composition, and how they interact during dehydration, hemorrhage, salt/water loads, and common physiology-style scenarios.

50 items — Study Guide with Answers

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Generated February 20, 2026

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1 **Which change most directly stimulates renin release from juxtaglomerular (JG) cells?**

A Increased stretch/pressure in the afferent arteriole wall

B **Decreased stretch/pressure in the afferent arteriole wall** ✓

C Increased NaCl delivery to the macula densa

D Increased angiotensin II levels

E Increased atrial stretch (high venous return)

▶ **Explanation:** JG cells act like intrarenal "baroreceptors": reduced afferent arteriole pressure/stretch triggers renin release. High NaCl delivery and high angiotensin II tend to suppress renin (negative feedback), and atrial stretch promotes ANP, which also opposes renin.

2 **Renin is best described as:**

A A steroid hormone that enters nuclei to change transcription

B **An enzyme that converts angiotensinogen into angiotensin I** ✓

C An enzyme that converts angiotensin I into angiotensin II

D A channel that inserts aquaporins into membranes

E A hormone secreted by the posterior pituitary

▶ **Explanation:** Renin is a protease enzyme released by JG cells. It cleaves angiotensinogen (from the liver) to form angiotensin I; ACE later converts angiotensin I to angiotensin II.

3 **Angiotensin-converting enzyme (ACE) most directly catalyzes:**

A Angiotensin II → aldosterone

B Angiotensinogen → angiotensin I

C **Angiotensin I → angiotensin II** ✓

D Renin → angiotensin I

E ADH → aldosterone

▶ **Explanation:** ACE converts angiotensin I into angiotensin II. Angiotensin II then drives vasoconstriction and stimulates aldosterone and ADH release.

4 Severe liver failure would most directly reduce which RAAS component, lowering the body's ability to generate angiotensin II?

A Renin production

B **Angiotensinogen production** ✓

C ACE production

D Aldosterone receptor production

E ADH synthesis

▶ **Explanation:** Angiotensinogen is made mainly by the liver. Renin is produced by the kidney, ACE is largely associated with vascular endothelium, aldosterone is from the adrenal cortex, and ADH is synthesized in the hypothalamus.

5 Which is NOT a typical effect of angiotensin II?

A Vasoconstriction that increases blood pressure

B Stimulation of aldosterone secretion

C Stimulation of thirst and ADH release

D **Decreased Na^+ reabsorption in the proximal tubule** ✓

E Constriction of the efferent arteriole in the kidney

▶ **Explanation:** Angiotensin II generally increases Na^+ reabsorption (including in the proximal tubule), raises blood pressure by vasoconstriction, and promotes aldosterone/ADH/thirst. Decreasing proximal Na^+ reabsorption would oppose the goal of restoring volume/pressure.

6 **During low renal perfusion, angiotensin II helps maintain GFR primarily by causing:**

A Dilation of the efferent arteriole

B **Constriction of the efferent arteriole** ✓

C Constriction of the afferent arteriole

D Closure of Bowman's capsule

E Immediate destruction of the filtration barrier

▶ **Explanation:** Efferent arteriole constriction increases glomerular capillary hydrostatic pressure, helping support GFR when renal blood flow drops. This is a classic "protect filtration" RAAS effect (within limits).

7 **Aldosterone is secreted primarily by the:**

A Posterior pituitary

B **Zona glomerulosa of the adrenal cortex** ✓

C Adrenal medulla

D Pancreatic alpha cells

E Kidney collecting duct cells

▶ **Explanation:** Aldosterone is a mineralocorticoid produced by the adrenal cortex (zona glomerulosa). It acts on the kidney to increase Na^+ reabsorption and K^+ secretion.

8 **In principal cells of the late distal tubule/collecting duct, aldosterone most directly increases:**

A Water permeability by inserting aquaporin-2 channels

B **Na^+ reabsorption and K^+ secretion** ✓

C Glucose reabsorption via SGLT transporters

D Urea secretion into the tubule lumen

E Ca^{2+} reabsorption as the primary effect

▶ **Explanation:** Aldosterone upregulates ENaC and the Na^+/K^+ ATPase, leading to increased Na^+ reabsorption and increased K^+ secretion. Aquaporin-2 insertion (water permeability) is mainly controlled by ADH, not aldosterone.

9 **Which stimulus can increase aldosterone secretion even if renin is low?**

A Low plasma K^+

B **High plasma K^+** ✓

C Low plasma osmolality

D High atrial stretch (high blood volume)

E Low blood glucose

▶ **Explanation:** Hyperkalemia can directly stimulate aldosterone release from the adrenal cortex. This is a common exam trap: aldosterone is not triggered only by renin/angiotensin II.

10 **ADH is synthesized in the _____ and released from the _____.**

A Posterior pituitary; anterior pituitary

B **Hypothalamus; posterior pituitary** ✓

C Kidney cortex; adrenal medulla

D Liver; pancreas

E Thyroid; parathyroid

▶ **Explanation:** ADH (vasopressin) is made in hypothalamic neurons and transported down axons to the posterior pituitary, where it is stored and released into the blood.

11 **ADH increases water reabsorption most directly by acting on the _____ to insert _____ into the membrane.**

A Proximal tubule; SGLT2

B **Collecting duct principal cells; aquaporin-2** ✓

C Thick ascending limb; $Na^+/K^+/2Cl-$ cotransporter

D Glomerulus; collagen pores

E Macula densa; chloride channels

▶ **Explanation:** ADH increases collecting duct water permeability mainly by inserting aquaporin-2 into the apical membrane of principal cells, allowing water to be reabsorbed down the medullary osmotic gradient.

12 **If ADH levels rise (and the kidney responds normally), what happens to urine volume and urine osmolality?**

A Urine volume increases; urine osmolality decreases

B **Urine volume decreases; urine osmolality increases** ✓

C Both urine volume and urine osmolality increase

D Both urine volume and urine osmolality decrease

E Neither changes in any situation

▶ **Explanation:** ADH makes the collecting duct more water-permeable, so more water is reabsorbed. This reduces urine volume and concentrates the urine (higher osmolality).

13 Which pairing is most accurate?

A Aldosterone primarily retains water without solute; ADH primarily retains sodium

B **Aldosterone primarily increases Na^+ reabsorption; ADH primarily increases water reabsorption** ✓

C Both primarily increase K^+ retention

D Both directly increase glucose reabsorption

E ADH is a steroid; aldosterone is a peptide hormone

▶ **Explanation:** Aldosterone increases Na^+ reabsorption (and K^+ secretion). ADH primarily increases water reabsorption by increasing water permeability of the collecting duct.

14 Which statement best explains why the body benefits from having both RAAS and ADH systems?

A RAAS regulates body temperature; ADH regulates red blood cell count

B **RAAS mainly defends extracellular volume/blood pressure via Na^+ retention and vasoconstriction; ADH mainly defends plasma osmolality via water retention** ✓

C They do the exact same job; two systems exist only by accident

D RAAS works only in embryos; ADH works only in adults

E ADH is used only for digestion; RAAS only for vision

▶ **Explanation:** Volume (pressure) and osmolality are related but not identical problems. RAAS prioritizes restoring effective circulating volume/pressure, while ADH is specialized for controlling water balance and plasma osmolality.

15 After heavy sweating with no water intake (sweat is hypotonic compared with plasma), which change is most likely?

A Renin decreases and ADH decreases

B Renin increases and ADH increases ✓

C Renin increases and ADH decreases

D Renin decreases and ADH increases

E Neither renin nor ADH changes

▶ **Explanation:** Sweating removes proportionally more water than salt, raising plasma osmolality and lowering volume. Higher osmolality stimulates ADH; reduced volume/pressure stimulates RAAS (renin).

16 A person loses a large volume of blood quickly. Plasma osmolality initially stays near normal. Which response is most likely in the first minutes?

A ADH decreases because osmolality is unchanged

B Both RAAS and ADH increase because low effective circulating volume activates baroreceptor pathways ✓

C Only aldosterone rises immediately within seconds; renin does not change

D Renin decreases because blood volume decreased

E The kidney increases urine output to replace lost blood

▶ **Explanation:** Severe volume/pressure loss strongly activates sympathetic and baroreceptor pathways: renin rises and ADH can rise even if osmolality hasn't increased (volume defense can override osmolality signals in emergencies).

17 **After rapidly drinking several liters of water, which hormone pattern is most expected?**

A ADH increases; renin increases

B **ADH decreases; renin decreases** ✓

C ADH decreases; renin increases

D ADH increases; renin decreases

E Both remain unchanged because kidneys do not respond to water intake

▶ **Explanation:** A water load lowers plasma osmolality and increases volume, both of which reduce ADH release. Increased volume/pressure also suppresses renin (less need to retain Na^+ and water).

18 **A patient receives a hypertonic saline infusion (saltier than plasma). Immediately after, which hormone pattern is most likely?**

A ADH increases; renin increases

B **ADH increases; renin decreases** ✓

C ADH decreases; renin increases

D ADH decreases; renin decreases

E Neither changes because saline is not a hormone

▶ **Explanation:** Hypertonic saline raises plasma osmolality, strongly stimulating ADH. It also expands extracellular volume, which suppresses renin/RAAS.

19 **Infusion of isotonic saline (same osmolality as plasma) would most likely cause:**

A Renin increases; ADH increases

B **Renin decreases; ADH decreases** ✓

C Renin increases; ADH decreases

D Renin decreases; ADH increases

E Renin unchanged; ADH unchanged

▶ **Explanation:** Isotonic saline mainly expands volume without changing osmolality much. Increased volume suppresses renin and also reduces ADH (baroreceptor input supports lower ADH when volume is high).

20 **Renin is released from specialized cells in the wall of the:**

A Efferent arteriole

B **Afferent arteriole (juxtaglomerular cells)** ✓

C Glomerular capillaries

D Proximal tubule

E Collecting duct

▶ **Explanation:** Juxtaglomerular (JG) cells are modified smooth muscle cells located in the afferent arteriole near the glomerulus. They secrete renin when renal perfusion is low.

21 **The macula densa contributes to RAAS activation primarily by sensing:**

A Blood glucose concentration in the afferent arteriole

B **NaCl concentration in the distal tubule fluid** ✓

C Oxygen concentration in the renal vein

D ADH concentration in plasma

E Albumin concentration in urine

▶ **Explanation:** Macula densa cells sense NaCl delivery (a proxy for filtration/flow and upstream volume status). Low NaCl delivery tends to promote renin release, helping restore volume/pressure.

22 **Which sequence correctly represents the core RAAS pathway?**

A Angiotensin II —renin→ angiotensin I —ACE→ angiotensinogen —→ aldosterone

B **Angiotensinogen —renin→ angiotensin I —ACE→ angiotensin II —stimulates→ aldosterone** ✓

C Aldosterone —ACE→ angiotensin II —renin→ angiotensinogen

D ADH —renin→ aldosterone —ACE→ angiotensin I

E Angiotensinogen —ACE→ angiotensin II —renin→ angiotensin I —→ aldosterone

▶ **Explanation:** Renin cleaves angiotensinogen to angiotensin I, ACE converts angiotensin I to angiotensin II, and angiotensin II stimulates aldosterone (and also promotes vasoconstriction, thirst, and ADH release).

23 **Which response occurs fastest after a sudden drop in blood pressure: angiotensin II vasoconstriction or aldosterone-driven Na^+ retention?**

A Aldosterone-driven Na^+ retention is fastest (seconds); angiotensin II is slower (hours)

B **Angiotensin II vasoconstriction is fast (minutes); aldosterone effects are slower (hours to days)** ✓

C Both are equally slow because they require new kidney growth

D Neither changes because blood pressure is controlled only by the brain

E Aldosterone is fastest because it is an ion channel

▶ **Explanation:** Angiotensin II causes rapid vasoconstriction. Aldosterone often works more slowly because it increases the expression/activity of transport proteins (like ENaC and Na^+/K^+ ATPase), which takes time.

24 ADH increases water permeability in the collecting duct by binding a receptor that activates cAMP. This receptor is best classified as a:

A Ligand-gated ion channel

B **G-protein-coupled receptor (GPCR)** ✓

C Receptor tyrosine kinase

D Intracellular nuclear receptor

E Voltage-gated sodium channel

▶ **Explanation:** The main kidney ADH receptor (V2) is a GPCR that increases cAMP, leading to insertion of aquaporin-2 channels into the apical membrane of collecting duct cells.

25 Aldosterone mainly acts by binding to an intracellular receptor and changing gene transcription. This receptor is best classified as a:

A Ligand-gated ion channel

B GPCR

C Receptor tyrosine kinase

D **Intracellular nuclear receptor** ✓

E Aquaporin channel

▶ **Explanation:** Aldosterone is a steroid hormone. Steroids typically cross membranes and bind intracellular receptors that regulate transcription, producing slower but longer-lasting effects.

26 **If ADH remains high for a prolonged period and the person has access to water (can drink normally), the most likely direction of plasma osmolality is:**

A Increase

B **Decrease** ✓

C No change is possible

D Become infinite

E Switch randomly between high and low each minute

▶ **Explanation:** ADH promotes water retention. If water intake continues, retaining extra water tends to dilute plasma, lowering plasma osmolality (a key idea behind hyponatremia in excess-ADH states).

27 **Which hormone is most likely to increase extracellular fluid volume with minimal direct change in plasma osmolality (by promoting Na^+ retention so water follows)?**

A ADH

B **Aldosterone** ✓

C Glucagon

D Insulin

E Thyroxine

▶ **Explanation:** Aldosterone increases Na^+ reabsorption; water tends to follow sodium, so volume rises. ADH primarily retains water without directly retaining sodium, which can lower osmolality if water intake is available.

28 **Constriction of the efferent arteriole by angiotensin II can promote proximal tubular reabsorption partly because it:**

A Decreases peritubular capillary oncotic pressure

B **Increases filtration fraction, raising peritubular capillary oncotic pressure and favoring reabsorption** ✓

C Eliminates the medullary osmotic gradient

D Directly inserts aquaporin-2 into proximal tubule membranes

E Stops GFR completely in all cases

▶ **Explanation:** Efferent constriction increases filtration fraction, leaving the blood in peritubular capillaries more protein-concentrated. Higher oncotic pressure in those capillaries favors fluid reabsorption from the tubule back into blood.

29 **After RAAS raises blood pressure/volume, renin secretion falls. This is an example of:**

A Positive feedback

B **Negative feedback** ✓

C Independent assortment

D Transduction

E Lysogenic conversion

▶ **Explanation:** Negative feedback means the response reduces the original stimulus. Once pressure/volume improves, the kidney no longer needs to release as much renin.

30 **Atrial natriuretic peptide (ANP) is released when the atria are stretched by high blood volume. Which effect best opposes RAAS?**

A Increased renin release

B Increased aldosterone secretion

C **Increased Na^+ excretion (natriuresis) and inhibition of renin/aldosterone** ✓

D Increased thirst and salt appetite

E Increased angiotensin II production

▶ **Explanation:** ANP acts as a 'volume-release' hormone: it promotes sodium (and thus water) loss and tends to suppress renin and aldosterone, opposing RAAS's volume-retaining effects.

31 **If aldosterone levels are abnormally high, which change is most expected?**

A Decreased Na^+ reabsorption in the collecting duct

B Decreased K^+ secretion in the collecting duct

C **Increased Na^+ reabsorption and increased K^+ excretion** ✓

D Increased glucose excretion by blocking SGLT

E Complete prevention of water reabsorption even if ADH is high

▶ **Explanation:** Aldosterone increases Na^+ reabsorption and promotes K^+ secretion in principal cells. Its effect on water is indirect (water tends to follow sodium when water permeability allows).

32 **A tumor secretes aldosterone independently of normal control. Which hormone pattern best fits (before treatment)?**

A Aldosterone high; renin high

B **Aldosterone high; renin low** ✓

C Aldosterone low; renin high

D Aldosterone low; renin low

E Both always remain normal due to homeostasis

▶ **Explanation:** Excess aldosterone raises Na^+ retention and volume/pressure, which suppresses renin through negative feedback. The key concept: when the 'end hormone' is high independently, the upstream driver often falls.

33 If the adrenal cortex cannot produce aldosterone (primary aldosterone deficiency), what happens to renin release from the kidney?

A Renin decreases because aldosterone is low

B **Renin increases because volume/pressure tends to fall without aldosterone** ✓

C Renin becomes permanently zero in all cases

D Renin increases only if plasma osmolality is low

E Renin has no relationship to aldosterone or volume

▶ **Explanation:** Without aldosterone, Na^+ retention is reduced, tending to lower extracellular volume and blood pressure. The kidney responds by increasing renin release to try to restore volume/pressure.

34 A person produces very large volumes of dilute urine, and blood tests show high plasma osmolality. Which hormone problem best explains this pattern?

A Excess aldosterone

B **Low ADH (central diabetes insipidus pattern)** ✓

C Excess ANP

D Low insulin

E Excess glucagon

▶ **Explanation:** Without ADH, the collecting duct is relatively water-impermeable, so the kidney cannot concentrate urine. Water is lost in urine, raising plasma osmolality and causing thirst.

35 Two patients have polyuria and dilute urine. Patient 1 has very low ADH; patient 2 has high ADH. Which interpretation is most consistent?

A Patient 1 has nephrogenic DI; patient 2 has central DI

B **Patient 1 has central DI; patient 2 has nephrogenic DI** ✓

C Both must have SIADH

D Both must have excess aldosterone

E ADH levels cannot be used to interpret urine dilution

▶ **Explanation:** Central DI means ADH is not produced/released properly (low ADH). Nephrogenic DI means ADH is present but the kidney cannot respond (high ADH with persistent dilute urine).

36 Excess ADH (SIADH-like physiology) is most likely to cause which combination?

A High plasma osmolality + very dilute urine + high urine volume

B **Low plasma osmolality + concentrated urine + low urine volume** ✓

C Low plasma osmolality + dilute urine + high urine volume

D High plasma osmolality + concentrated urine + high urine volume

E No change in either plasma osmolality or urine concentration

▶ **Explanation:** High ADH causes increased water reabsorption, lowering plasma osmolality while producing a smaller volume of more concentrated urine. This is the mirror-image logic of diabetes insipidus.

37 **After drinking alcohol, a person urinates frequently and becomes thirsty. The most direct hormonal explanation is that alcohol typically:**

A Increases ADH release, concentrating urine

B **Decreases ADH release, reducing collecting duct water permeability** ✓

C Increases aldosterone release, causing immediate diuresis

D Blocks renin release, causing concentrated urine

E Turns aquaporins into sodium channels

▶ **Explanation:** Alcohol inhibits ADH release, so the collecting duct reabsorbs less water. The result is more dilute urine and greater water loss, leading to thirst/dehydration.

38 **Which brain region contains key osmoreceptors that help regulate ADH release in response to plasma osmolality?**

A Cerebellum

B **Hypothalamus** ✓

C Occipital lobe

D Spinal cord

E Medulla oblongata (as the primary osmolality sensor)

▶ **Explanation:** Osmoreceptors in the hypothalamus detect changes in plasma osmolality and adjust ADH release (and thirst) accordingly.

39 **Which situation is most likely to cause high ADH even if plasma osmolality is low (dilute)?**

A Drinking a large amount of water rapidly

B Receiving an isotonic saline infusion

C **Severe hemorrhage (major drop in blood volume/pressure)** ✓

D Eating a low-salt meal

E Spending time at room temperature in a resting state

▶ **Explanation:** In major volume depletion, baroreceptor pathways strongly stimulate ADH to defend blood pressure/volume, even if osmolality signals would normally suppress ADH. Volume defense can override osmolality control in emergencies.

40 **During sympathetic activation (stress, low blood pressure), renin release is increased mainly via which receptor on JG cells?**

A 1-adrenergic receptor

B **1-adrenergic receptor** ✓

C 2-adrenergic receptor

D Muscarinic receptor

E Nicotinic receptor

▶ **Explanation:** Sympathetic stimulation increases renin release mainly through 1 receptors on juxtaglomerular cells—one reason RAAS is activated during stress or hypotension.

41 **A person eats a very salty meal but drinks little water. Plasma osmolality rises before any major volume expansion. Which hormonal response is most immediate?**

A Renin increases strongly because salt intake triggers RAAS

B **ADH increases because higher osmolality stimulates osmoreceptors** ✓

C Aldosterone increases because salt directly stimulates the adrenal cortex

D ANP increases immediately because atria are stretched by salt

E Both ADH and renin decrease immediately in all cases

▶ **Explanation:** A rise in plasma osmolality is a direct trigger for ADH release (and thirst). RAAS tends to activate with low volume/pressure or low NaCl delivery—not simply because dietary salt is high.

42 **Activation of RAAS generally leads to which urinary change (assuming kidneys function normally)?**

A Increased urinary sodium excretion (natriuresis)

B **Decreased urinary sodium excretion (sodium retention)** ✓

C No change in sodium handling because RAAS affects only water

D Increased glucose excretion as the main effect

E Complete shutdown of filtration (GFR becomes zero)

▶ **Explanation:** RAAS is a sodium- and volume-preserving system: angiotensin II and aldosterone increase sodium reabsorption, reducing sodium lost in urine (water may follow depending on ADH and the nephron's water permeability).

43 **ADH can concentrate urine only because the kidney already has a hyperosmotic medulla. Which nephron segment most directly creates this gradient by actively reabsorbing NaCl while being relatively impermeable to water?**

A Descending limb of the loop of Henle

B **Thick ascending limb of the loop of Henle** ✓

C Bowman's capsule

D Proximal tubule

E Glomerular capillary endothelium

▶ **Explanation:** The thick ascending limb pumps out NaCl but does not allow much water to follow, helping build a concentrated medulla. ADH then lets the collecting duct reabsorb water down that gradient to concentrate urine.

44 Angiotensin II causes vasoconstriction by binding receptors on vascular smooth muscle. This receptor is most likely a:

A Nuclear receptor that binds DNA directly

B **G-protein-coupled receptor (GPCR)** ✓

C Ligand-gated ion channel

D Receptor tyrosine kinase

E Voltage-gated calcium channel (as the receptor itself)

▶ **Explanation:** Angiotensin II is a peptide hormone that acts through cell-surface receptors (classically GPCRs). Steroid-like nuclear receptors are for lipid-soluble hormones such as aldosterone.

45 A drug blocks ACE, lowering angiotensin II. In a person whose kidney filtration pressure depends on angiotensin II, what effect may occur on glomerular pressure and GFR?

A Efferent arteriole constricts more, increasing glomerular pressure and GFR

B **Efferent arteriole dilates, lowering glomerular pressure and potentially lowering GFR** ✓

C Afferent arteriole constricts, increasing GFR

D GFR always increases because ACE inhibitors increase renin

E There is no possible effect of angiotensin II on GFR

▶ **Explanation:** Angiotensin II helps support glomerular pressure by constricting the efferent arteriole. Blocking ACE can reduce that support, so in low-perfusion situations GFR can fall (conceptually: less efferent constriction → less filtration pressure).

46 **Which component provides an early amplification step in the RAAS cascade?**

A **One renin enzyme molecule can catalyze cleavage of many angiotensinogen molecules over time** ✓

B Aldosterone inserts aquaporin-2 channels directly

C Angiotensin II is stored in the kidney and released without enzymes

D Renin binds DNA to turn on aldosterone genes

E ADH is produced by the adrenal cortex as a steroid

▶ **Explanation:** Cascades amplify signals when one step activates many downstream molecules. Renin is an enzyme, so one renin molecule can generate many angiotensin I molecules, which can then lead to many angiotensin II molecules and downstream effects.

47 **Which hormone is most likely to raise blood pressure quickly with minimal immediate change in blood volume?**

A **Angiotensin II** ✓

B Aldosterone

C Erythropoietin

D Growth hormone

E Melatonin

▶ **Explanation:** Angiotensin II can rapidly increase blood pressure through vasoconstriction. Aldosterone mainly increases pressure more slowly by increasing Na^+ retention (and thus volume over time).

48 **Which statement best explains why aldosterone alone cannot create highly concentrated urine?**

A Aldosterone acts only on the glomerulus and never on tubules

B Without ADH, collecting duct water permeability stays low, so water cannot be reabsorbed efficiently at the final step ✓

C Aldosterone destroys the medullary gradient

D Aldosterone blocks Na^+ reabsorption, preventing any concentration

E Aldosterone is secreted by the posterior pituitary, so it cannot affect kidneys

▶ **Explanation:** Aldosterone mainly changes Na^+ (and K^+) handling. Concentrating urine requires water reabsorption from the collecting duct, which depends strongly on ADH increasing water permeability.

49 **A patient has polyuria with very dilute urine. After an injection of synthetic ADH, urine becomes much more concentrated. Which diagnosis is most consistent?**

A Nephrogenic diabetes insipidus (kidney unresponsive to ADH)

B Central diabetes insipidus (not enough ADH produced/released) ✓

C Excess aldosterone

D High ANP state

E Normal physiology; this is the expected response to any injection

▶ **Explanation:** If giving ADH fixes the concentrating problem, the kidney can respond to ADH, meaning the issue was lack of ADH (central DI). In nephrogenic DI, ADH is present (or given) but the kidney still cannot respond well.

50 **A patient has ankle swelling (total body fluid increased) but low blood pressure and poor cardiac output. The kidney activates RAAS. What is the best explanation?**

A RAAS responds directly to total body water volume, regardless of blood pressure

B **RAAS responds to decreased effective arterial blood volume/renal perfusion, even if total body fluid is high** ✓

C RAAS is activated mainly by high plasma glucose

D RAAS activates only when plasma osmolality is high

E RAAS is activated by atrial stretch as its main trigger

▶ **Explanation:** The kidney mainly cares about perfusion pressure and effective circulating volume. If cardiac output is low, renal perfusion can be low even when total body water is high, so RAAS can activate (a key "effective volume" concept).