Physiology Puzzles on Excretion: Case Study Style

Inspired by case-based learning methods (similar to those used in advanced university programs), I've crafted 20 puzzles as short physiological case studies focused on excretion. Each presents a scenario based on human physiology, particularly the urinary system, kidneys, and related processes. You'll need to deduce the key physiological mechanism, disorder, or concept involved. Answers are provided immediately after each puzzle for self-checking. These are designed at an advanced undergraduate or introductory graduate level, emphasizing critical thinking.

Puzzle 1

A 45-year-old hiker in the desert experiences severe dehydration, leading to concentrated urine and reduced volume. Despite drinking water later, urine output remains low initially. What hormone is primarily responsible for this water conservation in the kidneys, and how does it act?

Answer: Antidiuretic hormone (ADH, or vasopressin). It increases water reabsorption in the collecting ducts by inserting aquaporin-2 channels into the apical membrane, promoting osmosis to concentrate urine and maintain blood osmolarity.

Puzzle 2

A patient with chronic heart failure develops edema and hyponatremia. Blood tests show elevated renin and angiotensin II levels. How does this relate to renal excretion, and what key hormone is stimulated to retain sodium?

Answer: The renin-angiotensin-aldosterone system (RAAS) is activated due to reduced renal perfusion. Aldosterone is released from the adrenal cortex, promoting sodium reabsorption in the distal tubules and collecting ducts, which indirectly retains water and exacerbates edema.

Puzzle 3

During intense exercise, an athlete notices their urine is darker and more acidic than usual. Blood pH is slightly lowered. What renal process is enhanced to excrete excess hydrogen ions and maintain acid-base balance?

Answer: Tubular secretion of H+ ions in the proximal and distal tubules, coupled with bicarbonate reabsorption and ammonia buffering in the collecting ducts, to form titratable acids and ammonium, excreting excess acids produced from lactic acid metabolism.

Puzzle 4

A diabetic patient presents with polyuria and glucosuria. Kidney function tests show normal glomerular filtration rate (GFR) but impaired tubular function. What physiological threshold is exceeded, leading to glucose in the urine?

Answer: The renal threshold for glucose reabsorption (around 180 mg/dL) is surpassed due to hyperglycemia. Glucose transporters (SGLT2 in proximal tubules) become saturated, preventing complete reabsorption and causing osmotic diuresis.

Puzzle 5

In a case of severe vomiting leading to metabolic alkalosis, the kidneys compensate by excreting more bicarbonate in urine. What ion exchange mechanism in the distal tubules facilitates this?

Answer: Increased bicarbonate excretion via the pendrin exchanger in type B intercalated cells of the collecting ducts, where Cl-/HCO3- exchange helps restore acid-base balance by removing excess base.

Puzzle 6

A patient on a low-sodium diet has normal blood pressure but elevated plasma aldosterone. How does this affect potassium excretion in the kidneys?

Answer: Aldosterone stimulates principal cells in the cortical collecting ducts to reabsorb sodium via ENaC channels, which creates a lumen-negative potential driving potassium secretion through ROMK channels, increasing K+ excretion (hypokalemia risk).

Puzzle 7

Following a traumatic injury causing hypotension, a patient's GFR drops temporarily. What autoregulatory mechanism in the kidneys helps maintain filtration despite low blood pressure?

Answer: Myogenic response and tubuloglomerular feedback: Afferent arterioles constrict or dilate to stabilize pressure, and macula densa senses low NaCl, releasing adenosine to adjust afferent tone and preserve GFR.

Puzzle 8

A newborn with congenital adrenal hyperplasia shows salt-wasting crisis due to aldosterone deficiency. How does this impair renal excretion and lead to hyponatremia?

Answer: Without aldosterone, sodium reabsorption in the distal nephron decreases, leading to excessive Na+ excretion (natriuresis), volume depletion, and secondary hyponatremia as water is retained to compensate.

Puzzle 9

In chronic kidney disease, phosphate retention occurs despite hyperphosphatemia. What hormone normally regulates phosphate excretion, and why is it ineffective here?

Answer: Parathyroid hormone (PTH) normally inhibits phosphate reabsorption in proximal tubules via downregulation of NaPi-IIa transporters. In CKD, secondary hyperparathyroidism develops, but damaged tubules reduce responsiveness, leading to retention.

Puzzle 10

A patient with liver cirrhosis develops ascites and secondary hyperaldosteronism. How does this alter urea excretion in the kidneys?

Answer: Hyperaldosteronism causes sodium and water retention, reducing urine volume. Urea, being passively reabsorbed in concentrated urine, is retained more (elevated BUN), exacerbating azotemia.

Puzzle 11

During pregnancy, a woman experiences increased GFR and dilute urine. What physiological change in renal hemodynamics causes this?

Answer: Hormonal vasodilation (progesterone, relaxin) reduces renal vascular resistance, increasing renal plasma flow and GFR by up to 50%, leading to higher filtration and less concentrated urine.

Puzzle 12

A hiker at high altitude hyperventilates, causing respiratory alkalosis. How do the kidneys compensate through excretion?

Answer: Reduced bicarbonate reabsorption in proximal tubules (due to lower PCO2 inhibiting H+ secretion), increasing HCO3- excretion to lower plasma bicarbonate and restore pH.

Puzzle 13

In a case of nephrotic syndrome, massive proteinuria occurs with hypoalbuminemia. What defect in the glomerular filtration barrier allows this?

Answer: Damage to podocytes and the glomerular basement membrane reduces charge selectivity and slit diaphragm integrity, allowing large proteins like albumin to filter into Bowman's space and be excreted.

Puzzle 14

A patient with Addison's disease (adrenal insufficiency) has hyperkalemia. How is potassium excretion impaired in the kidneys?

Answer: Low aldosterone reduces sodium reabsorption and potassium secretion in collecting ducts, as fewer open ENaC channels decrease the electrochemical gradient for K+ exit via ROMK.

Puzzle 15

Following ingestion of a toxin blocking carbonic anhydrase, a patient develops metabolic acidosis and alkaline urine. What renal excretion process is disrupted?

Answer: Carbonic anhydrase inhibition in proximal tubules impairs H+ secretion and bicarbonate reabsorption, leading to bicarbonate wasting (alkaline urine) and systemic acidosis.

Puzzle 16

In diabetes insipidus, urine is excessively dilute despite dehydration. Distinguish between central and nephrogenic types based on excretion physiology.

Answer: Central: ADH deficiency from pituitary; no response to desmopressin. Nephrogenic: Kidney resistance to ADH (mutated aquaporin-2 or V2 receptors); desmopressin ineffective, leading to impaired water reabsorption and polyuria.

Puzzle 17

A patient on loop diuretics experiences hypokalemia and metabolic alkalosis. Explain the linked changes in renal ion excretion.

Answer: Inhibition of NKCC2 in thick ascending loop blocks Na+, K+, Cl- reabsorption, increasing distal delivery. This stimulates aldosterone-sensitive Na+ reabsorption, enhancing K+ and H+ secretion, causing hypokalemia and alkalosis.

Puzzle 18

In acute kidney injury from ischemia, oliguria occurs with elevated creatinine. What phase of tubular excretion recovery involves polyuria?

Answer: Recovery phase: As tubules regenerate, reabsorption is impaired initially, leading to polyuria due to inability to concentrate urine, despite improving GFR.

Puzzle 19

A vegetarian on a high-protein diet shifts to low-protein, noticing less acidic urine. How does diet influence ammonium excretion?

Answer: High-protein diets increase acid load (from amino acid metabolism), boosting glutamine deamination in proximal tubules for NH_4^+ production and excretion as a buffer. Low-protein reduces this, making urine less acidic.

Puzzle 20

In syndrome of inappropriate ADH secretion (SIADH), hyponatremia develops with concentrated urine. How does excess ADH alter free water excretion?

Answer: Excess ADH maximizes water reabsorption in collecting ducts via aquaporins, impairing free water excretion. This dilutes plasma sodium as water is retained without solute, leading to euvolemic hyponatremia.