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Medical Curriculum Objectives Project
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Renal

(Revised January 2005, edited December 2010)

Body Fluids

R 1. Given the body weight and percent body fat, estimate the a) total body water, b) lean body mass, c) extracellular fluid volume, d) intracellular fluid volume, e) blood volume, and f) plasma volume. Identify normal extracellular fluid (plasma) osmolarity and concentrations of Na^+, K^+, Cl^-, HCO_3^-, proteins, creatinine, and urea, and contrast these values with those for intracellular fluids.

R 2. Using the volumes/compartments identified in objective R 1, contrast the movement between intracellular and extracellular compartments caused by increases or decreases in extracellular fluid osmolality.

R 3. Given the composition and osmolality of a fluid, identify it as hypertonic, isotonic, or hypotonic. Predict the change in transcellular fluid exchange that would be caused by placing a red blood cell in solutions with varying tonicities.

R 4. Identify major routes and normal ranges for water intake and loss, and predict how changes in intake and loss affect the distribution of total body water.

R 5. Demonstrate the ability to use the indicator dilution principle to measure plasma volume, blood volume, extracellular fluid volume, and total body water, and identify compounds used to measure each volume.

R 6. Predict the changes in extracellular volume, extracellular osmolality, intracellular volume, and intracellular osmolality caused by infusion of three liters of 0.9% NaCl, lactated Ringer's solution, 0.45% NaCl, and 7.5% NaCl.

R 7. Identify the site of erythropoietin production, the adequate stimulus for erythropoietin release, and the target tissue for erythropoietin action.

Structure of the Kidney and Nephrons

R 8. Given a cross section of a kidney, identify the renal cortex, renal medulla, renal calycies, medullary pyramids, renal pelvic space, renal artery, renal vein, and ureter.

R 9. Describe in sequence the tubular segments through which ultrafiltrate flows after it is formed at Bowman's capsule to when it enters the renal pelvis. Identify each structure as being located in the renal cortex or renal medulla. Based on the glomerulus location and the length of the loop of Henle, distinguish between cortical and juxtamedullary nephrons.

R 10. Describe in sequence the blood vessels through which blood flows when passing from the renal artery to the renal vein, including the glomerular blood vessels, peritubular capillaries, and the vasa recta.

R 11. On an electron micrograph and a line drawing, identify the following structures of the glomerular tuft: the afferent and efferent arterioles, glomerular capillary network, mesangium, Bowman's capsule, and the juxtaglomerular apparatus (including the specialized juxtaglomerular arteriole cells and the macula densa). Describe the three layers comprising the glomerular filtration barrier, and identify podocytes, foot processes, slits, and the basement membrane.

R 12. Explain the role of somatic, (pudendal) sympathetic, and parasympathetic nerves in the micturition reflex and in urination.

Renal Clearance

R 13. Explain the clearance principle. Use the clearance equation and an appropriate compound to estimate the glomerular filtration rate, renal plasma flow, and renal blood flow.

R 14. Distinguish between the use of inulin and creatinine clearances as measures of the glomerular filtration rate.

R 15. Given the plasma and urine concentrations and the urine flow rate, calculate the filtered load, tubular transport, excretion rate, and clearance of inulin, creatinine, para-amino hippuric acid (PAH), glucose, and penicillin. Predict how changes in filtration, reabsorption, and secretion will affect renal excretion of each compound.

R 16. For each of the compounds listed in objective R 15, graph the urine excretion of a compound against the plasma concentration. Using this graph, identify the tubular load, tubular transport maximum (T_{max}), and splay for each substance.

Glomerular Filtration Rate and Renal Hemodynamics

R 17. Identify the filtration barriers, if any, which impede the filtration of H_2O, Na^+, inulin, albumin, and red blood cells.

R 18. Define renal blood flow, renal plasma flow, glomerular filtration rate, and filtration fraction and list typical values.

R 19. Define the filtration coefficient at the glomerular capillary, describe the membrane properties that contribute to it, and explain its role in determining GFR.

R 20. Given the capillary and Bowman's capsule hydrostatic and oncotic pressures, calculate the net filtration force at the glomerular capillaries. Predict the changes in glomerular filtration caused by increases or decreases in any of those pressures.

R 21. Describe the relative resistances of the afferent and efferent arterioles and the effects on renal blood flow and GFR of selective changes in each.

R 22. Describe the myogenic and tubuloglomerular feedback mechanisms that mediate the autoregulation of renal plasma flow and glomerular filtration rate.

R 23. Predict the change in renal blood flow and glomerular filtration rate caused by an increase in renal sympathetic nerve activity.

R 24. Predict the change in renal blood flow and glomerular filtration caused by: a) increased synthesis of angiotensin II, b) increased release of atrial natriuretic peptide, c) increased prostaglandin formation, and d) increased nitric oxide formation.

R 25. Identify which components of the filtration barrier whose damage would result in hematuria and proteinuria.

R 26. Using the pressures described in objective R 20, predict the changes in net filtration force that occur as blood travels along the glomerular capillary and hydrostatic pressure falls and colloid osmotic pressure increases.

R 27. Predict the change in renal blood flow and GFR caused by urinary tract obstruction, hypoalbuminemia, and diabetic nephropathy.

R 28. Compare blood flow to, and oxygen consumption by, the kidneys with that of skeletal muscle and cardiac muscle.

R 29. Describe the effects of changes in peritubular capillary hydrostatic and colloid osmotic pressures on net proximal tubular fluid reabsorption.

Transport Properties of Nephron Segments

R 30. Using glucose, para-amino hippuric acid (PAH), water, and Cl^-, contrast the transcellular and paracellular pathways for movement across proximal tubular epithelia.

R 31. Distinguish between active (primary and secondary) transport, facilitated diffusion, and passive diffusion based on energy source and carrier protein involvement.

R 32. Describe the contribution of the major nephron segments to the reabsorption of the filtered load of solute and water.

R 33. Describe the cellular mechanisms for the transport of Na^+, Cl^-, K^+, HCO_3^-, Ca^{2+}, phosphate, organic solutes (e.g., glucose, amino acids, and urea), and water by the major tubular segments.

R 34. Describe the function of the following renal transporters and their predominant localization along the tubules with regard to nephron segment and apical versus basolateral membranes

a. Transport ATPases (Na^+/K^+-ATPase, H^+/K^+-ATPase, H^+-ATPase, and Ca^{2+}-ATPase)
b. Ion and water channels (K^+, ENaC, Cl^-, Ca^{2+}, aquaporins)
c. Coupled transporters (Na^+-glucose, Na^+/H^+-antiporter, Na^+-K^+-$2Cl^-$-symporter, Na^+-phosphate symporter, Na^+-Cl^--symporter, Na^+-HCO_3^--symporter, Cl^-/HCO_3^--antiporter)

R 35. Describe the nephron sites and molecular mechanisms of action of the following classes of diuretics (osmotic, carbonic anhydrase inhibitors, loop, thiazide, K^+-sparing).

R 36. Describe clinical syndromes related to defects in specific renal transporters (e.g., Bartter's, Gittelman's, Liddle's, etc.).

R 37. Describe the effects of reductions in GFR on plasma creatinine concentrations and plot the relationship

Urine Concentration and Dilution

R 38. Using the intake and loss routes identified in objective R 4, predict the changes in body fluid volume and osmolality caused by a net water loss or gain in the body. Predict how each of these disturbances would alter the rate of urine production and the osmotic composition of the urine.

R 39. Using the intake and loss routes identified in objective R 4, predict the changes in body fluid volume and osmolality caused by a net NaCl loss or gain in the body. Predict how each of these disturbances would alter the rate of urine production and the osmotic composition of the urine.

R 40. Identify the two most powerful stimuli that cause ADH release, and describe the negative feedback control mechanisms for each.

R 41. Describe the role of the ascending limb of the loop of Henle in producing a high renal interstitial fluid osmolality. Beginning with the loop of Henle, contrast the tubular fluid and interstitial fluid osmolality changes that allow either a dilute or a concentrated urine to be produced and excreted.

R 42. Predict the consequence on urine concentrating ability if the medullary osmotic gradient is disrupted. Following disruption, describe how the osmotic gradient would be re-established.

R 43. Identify the tubular section and cellular mechanism by which ADH increases permeability to water and urea. Describe the role of these changes on the ability of the kidney to produce either a dilute or a concentrated urine.

R 44. Given urine and plasma osmolarities and urine volume, calculate osmolar and free water clearance.

Identify expected free water clearance for an individual producing either a dilute or a concentrated urine.

R 45. Describe the actions of diuretics listed on objective R 35 on the ability of the kidneys to maximally concentrate and dilute urine.

R 46. Distinguish between central and nephrogenic diabetes insipidus based on plasma ADH levels and the response to an injection of ADH.

Na^+ Balance and Regulation of Extracellular Fluid Volume

R 47. Identify the normal range of dietary Na^+ intake and major routes of Na^+ loss from the body. Define the role of Na in maintaining extracellular fluid volume.

R 48. Calculate the normal filtered load of Na^+. Identify the tubular sites of Na reabsorption, and the alterations in Na^+ reabsorption in conditions of euvolemia, volume depletion, and volume expansion.

R 49. Describe the receptors involved in the monitoring of ECF volume (e.g., high-pressure baroreceptors and low-pressure cardiopulmonary stretch receptors), and diagram the neural reflex regulation of renal Na^+ and water excretion.

R 50. Diagram the formation and generation of angiotensin II, beginning with renin. Identify four factors that can promote renin release.

R 51. Describe the regulation of Na^+ reabsorption along the nephron, including the effects of sympathetic nerves, angiotensin II, aldosterone, and atrial natriuretic peptide.

R 52. Describe the effects of diuretics listed in objective R 35 on Na^+ handling by the kidneys and, thus, on ECF volume regulation.

R 53. Explain the contribution of the kidneys to progression of and/or the compensation for the altered fluid volume regulation characteristic of congestive heart failure and hepatic cirrhosis.

R 54. Describe the regulation of proximal tubule reabsorption that underlies the phenomenon of glomerulotubular balance.

R 55. Describe the role of the renin-angiotensin-aldosterone system in the regulation of systemic arterial blood pressure in volume-replete and volume-depleted states and in secondary forms of hypertension.

K^+ Balance

R 56. Identify the normal range of dietary K^+ intake and major routes of K^+ loss from the body. Define the role of extracellular K^+ in maintaining normal nerve and muscle function.

R 57. Describe K^+ distribution within the body, extrarenal K^+ homeostasis, and the role insulin, epinephrine, and aldosterone play in the movement of K^+ between intracellular and extracellular pools. Describe the K^+ shift caused by acidosis.

R 58. Calculate the normal filtered load of K^+. Identify the tubular sites of K^+ reabsorption and secretion.

R 59. Describe the factors that regulate K^+ secretion in the collecting duct (i.e., aldosterone, plasma K^+) and distinguish these from factors that alter K^+ secretion at this site (i.e., luminal fluid flow rate, acid-base disturbances, anion delivery).

R 60. Contrast the tubular sites of action of K^+ wasting and K^+ sparing diuretics.

Ca^{2+} and Phosphate Balance.

R 61. Identify the normal range of dietary Ca^{2+} and phosphate intake, major storage pools of Ca and phosphate, and major routes of Ca^{2+} and phosphate loss from the body. Describe the regulation of plasma Ca^{2+} by calcitonin and phosphate by parathyroid hormone.

R 62. Calculate the normal filtered load of Ca^{2+}. Identify the tubular sites of Ca^{2+} reabsorption. Calculate the normal filtered load of phosphate. Identify the tubular sites of phosphate reabsorption.

R 63. Describe the renal regulation of Ca^{2+} and phosphate transport by PTH, calcitonin, and 1,25-dihydroxy vitamin D (calcitriol), and distinguish from other factors that alter their transport (ECF volume, acid-base disorders).

R 64. Describe the role of the kidney in the production of 1,25-dihydroxy vitamin D (calcitriol).

R 65. Describe the effects of diuretics on Ca^{2+} and phosphate excretion, especially noting the effect of thiazides to decrease Ca^{2+} excretion and loop diuretics to increase Ca^{2+} excretion.

Acid-Base Balance

R 66. Identify the normal range of pH values, and the upper and lower limits compatible with life. Describe the role of buffers in maintaining pH, including the roles of the lungs and kidneys.

R 67. Describe the respiratory and renal regulation of the CO_2/HCO_3^- buffer system, which allows a buffer with a pK_a of 6.1 to be physiologically important in the maintenance of the normal plasma pH of 7.4.

R 68. Distinguish between CO_2-derived (volatile acid) and nonvolatile acid, the relative amounts produced each day through dietary intake and cellular metabolism, and the normal routes of loss from the body.

R 69. Calculate the filtered load of HCO_3^-, and identify the major sites of reabsorption (and secretion) along the nephron, emphasizing the importance of H^+ secretory mechanisms in this process. Describe the cellular mechanisms responsible for net transepithelial movement of HCO_3^-.

R 70. Describe the adjustments in filtered load and HCO_3^- reabsorption (H^+ secretion) by alterations in systemic acid-base balance and distinguish from factors that alter this process (i.e., ECF volume, aldosterone, and angiotensin II).

R 71. Describe net acid excretion by the kidneys, titratable acid, the importance of urinary buffers, and the production and excretion of ammonium. Distinguish between the reclamation of filtered bicarbonate and the formation of new bicarbonate.

R 72. Given a sudden increase or decrease in pH, identify the magnitude and the time course of the compensations that act to minimize change in pH of the body fluids, including a) buffers, b) respiratory adjustments, and c) renal adjustments.

R 73. From blood values, identify simple and mixed metabolic and respiratory acid-base disturbances. Distinguish between increased and normal anion gap metabolic acidosis, chloride-sensitive and -resistant metabolic alkalosis, and acute and chronic respiratory disturbances.

R 74. Describe processes that lead to acid-base disturbances and list common causes.

R 75. Describe the effects of carbonic anhydrase inhibitors and the other diuretics listed on objective R 35 on acid-base balance and the reabsorption of HCO_3^- by the nephron.

Integrative and Pathophysiological Aspects; Hypertension

R 76. Describe the relationships between sodium balance and plasma volume as they contribute to cardiovascular hemodynamics and arterial pressure.

R 77. Describe the role of the renin-angiotensin-aldosterone systems in the regulation of sodium balance and arterial pressure with emphasis on the actions of angiotensin II on renal hemodynamics and tubular transport.

R 78. Describe pressure natriuresis and the mechanisms mediating and modulating this process.

R79. Describe how impairments in renal function and pressure natriuresis contribute to the long-term regulation of arterial pressure and the development and maintenance of hypertension.