Prodigy Anesthesia Quick Review

Renal Anatomy/Physiology

Is the fluid flowing out of the loop of Henle into the distal convoluted tubule hypertonic or hypotonic? What is responsible for this?	Unlike the descending limb and thin ascending limb of the loop of Henle, the thick ascending portion is permeable to solutes, but not water. This means that solutes can be reabsorbed (taken out of the loop of Henle) while water remains inside the lumen of the tubule. This results in hypotonicity of the fluid within the tubule (100-200 mOsm/L). *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 730.*
Where in the nephron is sodium reabsorbed?	Sodium is reabsorbed throughout the nephron but the majority of it is reabsorbed in the proximal convoluted tubule. The distribution is as follows: 60-75% of sodium is reabsorbed in the proximal convoluted tubule, 15-20% is reabsorbed in the loop of Henle, 5% in the distal convoluted tubule, and 5-7% in the collecting tubule. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 727-730.*
How does the release of renin by the juxtaglomerular apparatus translate into an alteration in systemic blood pressure?	When released by the juxtaglomerular apparatus, renin enters the bloodstream where it acts on a protein synthesized by the liver called angiotensinogen, converting it into angiotensin I. This inert peptide is then rapidly converted into angiotensin II in the lungs by a chemical called angiotensin-converting enzyme. Angiotensin II activates AT1 receptors to produce intense, direct arteriolar constriction which increases the systemic vascular resistance and ultimately, the systemic blood pressure. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 731-732.*
What structure in the nephron secretes renin and what are the three primary stimuli that cause its release?	Renin is secreted by the juxtaglomerular apparatus in the nephron and it responds to Beta-1 stimulation, changes in afferent arteriolar wall pressure, and changes in chloride flow past the macula densa. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 731-732.*
Where in the nephron are principal cells and intercalated cells located and what function do they serve?	Principal cells and intercalated cells are found in the collecting tubule. Principal cells are responsible for the secretion of potassium and they are also involved in the aldosterone-mediated reabsorption of sodium. Intercalated cells are primarily responsible for acid-base regulation. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 731.*

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Where in the nephron does antidiuretic hormone work and what function does it serve?	Antidiuretic hormone (ADH), also known as arginine vasopressin, works in the medullary portion of the collecting tubule. ADH is secreted in response to dehydration and renders the lumen of the collecting tubule permeable to water through the expression of water channel proteins called aquaporin-2 channels. Adequate hydration suppresses the release of ADH, rendering the lumen of the collecting tubule impermeable to water. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 731.*
Beginning at the aorta, name the vessels and branches that supply blood to the glomeruli.	Each kidney is supplied by a single renal artery that arises from the aorta and divides at the level of the renal pelvis into interlobar arteries. At the junction of the renal cortex and medulla the interlobar arteries divide into arcuate arteries which further divide into interlobular arteries that eventually divide into single afferent arterioles that supply each glomerulus. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 733.*
Where in the nephron does aldosterone exert its action?	The late distal tubule and the cortical portion of the collecting tubule participate in aldosterone-mediated sodium reabsorption. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 730.*
What are the values for normal renal plasma flow and normal renal blood flow?	Normal renal plasma flow is about 660 mL/min and normal renal blood flow is about 1200 mL/min. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 732.*
What limits the amount of sodium that can be reabsorbed in the thick, ascending segment of the loop of Henle?	Normally, in the loop of Henle, solute and water reabsorption occurs passively and follows concentration and osmotic gradients. In the thick, ascending portion of the loop of Henle, however, sodium and chloride are reabsorbed more than water. Also, the reabsorption of sodium is directly coupled to the reabsorption of potassium and chloride in this segment. Because of this coupling effect in the thick, ascending loop of Henle, the amount of sodium that can be reabsorbed is limited by the chloride concentration in the tubular fluid. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 729.*
What is the major function of the proximal convoluted tubule of the nephron?	The primary function of the proximal convoluted tubule is reabsorption of sodium. The cells of the proximal tubule actively transport sodium out via membrane-bound Na-K-ATPase which results in a low intracellular concentration of sodium. Thus, sodium naturally passes down its gradient from the fluid in the proximal convoluted tubule into the cells of the tubule. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 727-729.*

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What percent of plasma is normally filtered through the glomerulus?	Under normal conditions, about 20% of plasma is filtered as blood passes through the glomerulus. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 727.*
How does glomerular filtration pressure relate to efferent and afferent arteriolar pressure in the nephron?	Glomerular filtration pressure is directly proportional to efferent arteriolar pressure and inversely proportional to afferent arteriolar pressure. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 727.*
What is the normal glomerular filtration pressure in the nephron and what two forces oppose it?	The normal glomerular filtration pressure is about 60% of the mean arterial pressure or about 60 mmHg. It is opposed by plasma oncotic pressure which exerts a pressure of about 25 mmHg and renal interstitial pressure which exerts about 10 mmHg. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 727.*
What separates the endothelial cells of glomeruli from the epithelial cells of Bowman's capsule in the nephron and in what major way do these cells differ?	The endothelial cells of the glomeruli and the epithelial cells of Bowman's capsule are separated only by their basement membranes. A major difference between these two cells are that the glomerular endothelial cells have fairly large (70-100 nm) pores while the epithelial cells of Bowman's capsule are tightly fused and contain only small filtration slits (25 nm). The fusion of these two cell types provide an effective filtration barrier preventing the passage of large molecular weight substances and cellular debris. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 727.*
Where in the nephron do parathyroid hormone and vitamin D act and what function do they serve?	Parathyroid hormone and vitamin D mediate the reabsorption of calcium in the distal tubule of the nephron. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 730.*
How can sevoflurane potentially result in renal damage?	Sevoflurane can potentially result in an alteration in renal function by two methods: 1) sevoflurane may possibly degrade into significant amounts of fluoride which can lead to a decreased ability of the kidneys to concentrate urine, and 2) compound A, a breakdown product of sevoflurane can accumulate when low flows are used. Compound A has been associated with the development of renal damage in laboratory animals and fresh gas flows of at least 2 L/min are recommended when using sevoflurane. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 735.*

What creatinine clearance values are associated with mild, moderate, and severe renal failure?	Creatinine clearance values between 40-60 mL/min represent mild renal impairment, 25-40 mL/min indicates moderate renal dysfunction, and levels less than 25 mL/min indicate severe renal dysfunction. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 3rd ed. New York, NY: McGraw-Hill; 2002: 681.*
How is creatinine produced, what are its normal laboratory values, and how is it related to muscle mass and glomerular filtration?	Creatinine is produced when creatine, a normal byproduct of muscle metabolism undergoes non-enzymatic degradation. The normal creatinine concentration in men is 0.8-1.3 mg/dL and 0.6-1.0 mg/dL in women. Creatinine concentration is directly related to body muscle mass and inversely related to the glomerular filtration rate. Because muscle mass is typically fairly constant, the creatinine concentration is a relatively reliable indicator of glomerular filtration rate. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 3rd ed. New York, NY: McGraw-Hill; 2002: 680.*
What BUN level would typically be associated with renal disease? What are some other non-renal causes of increased BUN?	Elevations in the BUN above 50 mg/dL are typically associated with renal disease. Other causes of an increased BUN include increased protein catabolism due to trauma or sepsis, degradation of blood within the gastrointestinal tract, resorption of a large hematoma, or heavy protein intake. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 3rd ed. New York, NY: McGraw-Hill; 2002: 680.*
What is Blood Urea Nitrogen (BUN) and what conditions can cause derangements in its values?	Blood Urea Nitrogen refers to the amount of urea present in the bloodstream. Ammonia is produced by the deamination of amino acids. The liver metabolizes the potentially toxic ammonia into urea and provides the primary source of urea in the body. The normal BUN is 10-20 mg/dL. Low values can be caused by starvation or liver disease. Elevated values can result from decreases in glomerular filtration rate or increases in protein catabolism. The BUN value is therefore directly related to protein catabolism and indirectly related to glomerular filtration. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 3rd ed. New York, NY: McGraw-Hill; 2002: 680.*
The administration of cyclosporine and radiocontrast dye have both been associated with renal dysfunction. What are some methods that have been shown to reduce the renal damage associated with these drugs?	Pretreatment with acetylcysteine has been shown to protect against renal dysfunction due to radiocontrast media. The administration of calcium channel blockers such as diltiazem have been shown to reduce the incidence of cyclosporine-induced nephrotoxicity. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 736.*
What percentage of cardiac output is directed towards the kidneys?	Blood flow to both kidneys normally accounts for 20-25% of cardiac output. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 732.*

Renal Anatomy/Physiology

Which intravenous induction agent would be most likely to preserve renal function in the patient with hemorrhagic hypovolemia?

Although most intravenous induction agents appear to exert mild decreases in renal function, ketamine has been shown to have minimal effect on renal function. Ketamine also preserves renal function to a greater degree than other agents in the presence of hemorrhagic hypovolemia.

Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 735.

What is the normal renal tubular threshold for glucose?

Glycosuria is usually due to a decrease in the renal tubular threshold for glucose (which is normally 180 mg/dL) or hyperglycemia.

Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 3rd ed. New York, NY: McGraw-Hill; 2002: 681.

How do general and regional anesthesia affect renal function?

Both regional and general anesthesia result in reversible decreases in renal blood flow, glomerular filtration rate, urinary output, and sodium excretion, but the effects are not as dramatic in regional anesthesia.

Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 734.

How does stimulation of alpha-1, alpha-2, and dopamine receptors affect the function of the nephron?

Stimulation of alpha-1 adrenergic receptors results in increased sodium reabsorption in the proximal tubules. Alpha-2 receptor stimulation results in decreased sodium reabsorption and increased excretion of water. Dopamine, which is formed in the proximal tubules as well as released from nerve endings reduces proximal reabsorption of sodium and dilates afferent and efferent arterioles.

Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 734.

Sympathetic outflow to the kidneys arises from what spinal cord levels?

Sympathetic outflow to the kidneys arises from the T4-L1 spinal cord levels and travels through the celiac and renal plexuses.

Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 734.

What is renal filtration fraction (FF) and how do changes in arteriolar tone in the nephrons affect it?

Filtration fraction is the ratio of the glomerular filtration rate to renal plasma flow and it is normally about 20%. The glomerular filtration rate is dependent on the relative tone of the afferent and efferent arterioles in the nephron. An increase in the diameter of the afferent arteriole (vasodilation) or a decrease in the diameter of the efferent arteriole (vasoconstriction) will increase the filtration fraction and vice versa. The changes in the tone of these arterioles can maintain a steady glomerular filtration rate between mean arterial pressures of 80 and 180 mmHg.

Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 733.

What is creatinine, why is it important, and how is creatinine clearance calculated?	Creatinine is a product of phosphocreatine breakdown in muscle and is normally completely filtered by the kidneys. Because creatinine is not reabsorbed and is secreted in very small amounts, it provides an easy way to assess the glomerular filtration rate by measuring the amount of creatinine in the blood and the amount in the urine. The formula for creatinine clearance = (Urinary Creatinine) X (Urinary Flow Rate)/Plasma Creatinine. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 733.*
What percentage of renal plasma flow is the normal glomerular filtration rate (GFR) and what are normal GFR values?	The normal GFR is about 20% of renal plasma flow. The normal values in men are about 120 mL/min (+/- 25 mL/min) and for women the value is 90 mL/min (+/- 20 mL/min). *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 733.*
Which drugs typically administered in the perioperative period can impair the ability of dopamine to increase renal function? What other drugs administered in the perioperative period can adversely affect renal function?	Drugs such as metoclopramide, phenothiazines, and droperidol that have antidopaminergic effects can interfere with the ability of dopamine to increase renal function. Other drugs such as aminoglycosides, amphotericin B, immunosuppressive drugs, and radiocontrast dyes can impair renal function, especially in the face of pre-existing renal damage. *Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York, NY: McGraw-Hill; 2006: 735.*

