Benha University
Faculty of Science
Department of Zoology

Advanced physiology 2 (412 Z)

Semester: Second Term **Level:** Fourth level
Sepc: Zoology&Chem.
Date: 7/6/2017 **Exam time:** 2:00 hours

Group C (16 marks, 40 min.)

Write briefly on the following:-

1. Effect of ADH or AVP on concentration of urine.

Arginine Vasopressin Promotes the Excretion of Osmotically Concentrated Urine Changes in urine osmolality are normally brought about largely by changes in plasma levels of arginine vasopressin (AVP), also known as antidiuretic hormone (ADH) In the absence of AVP, the kidney collecting ducts are relatively water-impermeable. Reabsorption of solute across a water-impermeable epithelium leads to an osmotically dilute urine. In the presence of AVP, collecting duct water permeability is increased. Because the medullary interstitial fluid is hyperosmotic, water reabsorption in the medullary collecting ducts can lead to the production of osmotically concentrated urine. When plasma osmolality is increased, plasma AVP levels increase. The hormone binds to a specific vasopressin (V2) receptor in the basolateral cell membrane. By way of a guanine nucleotide stimulatory protein (Gs), the membrane-bound enzyme adenylyl cyclase is activated. This enzyme catalyzes the formation of cyclic AMP (cAMP) from ATP. Cyclic AMP then activates a cAMP-dependent protein kinase (protein kinase A, or PKA) that phosphorylates other proteins. This leads to the insertion, by exocytosis, of intracellular vesicles that contain water channels (aquaporin-2) into the luminal cell membrane. The resulting increase in number of luminal membrane water channels leads to an increase in water permeability. Water leaves the lumen and then exits the cells via aquaporin-3 and aquaporin-4 in the basolateral cell membrane. The solutes in the collecting duct lumen become concentrated as water leaves. This response to AVP occurs in minutes. AVP also has delayed effects on collecting ducts; it increases the transcription of aquaporin-2 genes and increases the total number ofvaquaporin-2 molecules per cell.

Best wishes

2. Mention factors effect on filterability of macromolecules in glomerular filtration.

Size, Shape, Deformability, and Electrical Charge Affect the Filterability of Macromolecules

The permeability properties of the glomerular filtration barrier have been studied by determining how well molecules of different sizes pass through it. Molecular radii were calculated from diffusion coefficients. The concentration of the molecule in the glomerular filtrate (fluid collected from Bowman's capsule) is compared with its concentration in plasma water. A ratio of 1 indicates complete filterability، and a ratio of zero indicates complete exclusion by the glomerular filtration barrier. Molecular size is an important factor affecting filterability. All molecules with weights less than 10,000 kilodaltons are freely filterable, provided they are not bound to plasma proteins. Molecules with weights greater than 10,000 kilodaltons experience more and more restriction to passage through the glomerular filtration barrier. Large molecules e.g., molecular weight of 100,000 kilodaltons) cannot get through at all. Most plasma proteins are large molecules, so they are not appreciably filtered. From studies with molecules of different sizes, it has been calculated that the glomerular filtration barrier behaves as though cylindrical pores of about

٧,٥to 10 nm in diameter penetrated it. No one, however, has ever seen such pores in electron micrographs of the glomerular filtration barrier.

Molecular shape influences the filterability of macromolecules. For a given molecular weight, a long, slender molecule will pass through the glomerular filtration barrier more easily than a spherical molecule. Also, passage of a macromolecule through the barrier is favored by greater deformability. Electrical charge is thought, by most investigators, to influence the passage of macromolecules through the glomerular filtration barrier. The barrier bears fixed negative charges. Glomerular endothelial cells and podocytes have a negatively charged surface coat (glycocalyx), and the glomerular basement membrane contains negatively charged

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sialic acid, sialoproteins، and heparan sulfate. These negative charges couldimpede the passage of negatively charged macromolecules by electrostatic repulsion. In addition to its large molecular size, the net negative charge on serum albumin at physiological pH could be a factor that reduces its filterability. In some glomerular diseases, a loss of fixed negative charges from the glomerular filtration barrier is associated with increased filtration of serum albumin. Filtered serum albumin is reabsorbed in the proximal tubule by endocytosis, but when excessive amounts are filtered, some will escape in the urine, a situation called **albuminuria**. **Microalbuminuria**, defined as excretion of 30 to 300 mg serum albumin/day, may be an early sign of kidney damage in patients with diabetes mellitus or hypertension or an indication of cardiovascular disease. A normal albumin excretion rate is about 5 to 20 mg/day.

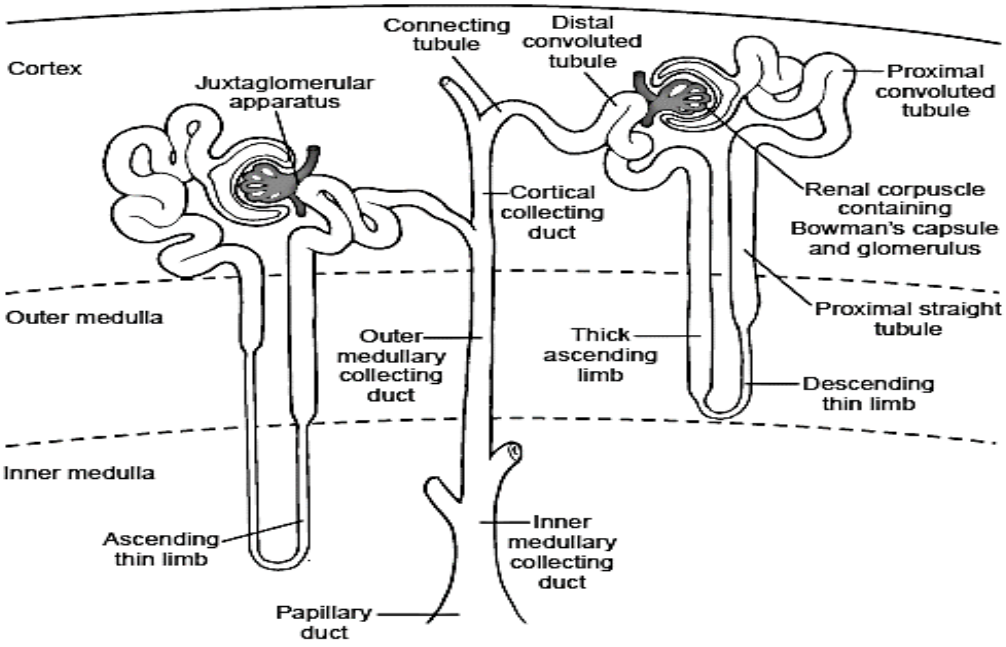
Proteinuria (or albuminuria) is a hallmark of glomerular disease. Proteinuria not only is a sign of kidney disease but results in tubular and interstitial damage and contributes to the progression of chronic renal disease

3. Describe the basic unit of renal structure and its function.
   The Nephron Is the Basic Unit of Renal

Each human kidney contains about one million nephrons each of which consists of a renal corpuscle and a renal tubule. The renal corpuscle consists of a tuft of capillaries, the glomerulus, surrounded by Bowman's capsule. The renal tubule is divided into several segments. The part of the tubule nearest the glomerulus is the proximal tubule.

This is subdivided into a proximal convoluted tubule and proximal straight tubule. The straight portion heads toward the medulla, away from the surface of the kidney. The loop of Henle includes the proximal straight tubule, thin limb, and thick ascending limb. Connecting tubules connect the next segment, the short distal convoluted tubule, to the collecting duct system. Several nephrons drain into a cortical collecting duct, which passes into an outer medullary collecting duct. In the inner

medulla, inner medullary collecting ducts unite to form large papillary ducts. The collecting ducts perform the same types of functions as the renal tubules, so they are often considered to be part of the nephron. The collecting ducts and nephrons differ, however, in embryological origin, and because the collecting ducts form a branching system, there are many more nephrons than collecting ducts. The entire renal tubule and collecting duct system consists of a single layer of epithelial cells surrounding fluid (urine) in the tubule or duct lumen.



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4. Describe the cases of acid base disturbance in the body.

Acidosis and alkalosis

Metabolic acidosis/alkalosis:

Occur when bicarbonate concentrations are abnormal (normal value is approx 24mM)

Respiratory acidosis/alkalosis:

Occur when carbon dioxide concentrations are abnormal (normal value is approx 40mm Hg)

$$[H+] = \frac{PCO_2}{HCO_3^-}$$ (PCO_2 controlled by respiration; HCO_3^- controlled by kidneys)

OR

pH proportional to $\frac{HCO_3^-}{PCO_2}$

Compensation

Acid-base imbalance:

Is blood pH normal? If not - acidosis or alkalosis
(nb: the body never overcompensates)
Is there an underlying metabolic problem? HCO_3^-
Is there an underlying respiratory problem? $PaCO_2$
Is there compensation?

Principles of compensation

Metabolic disturbances compensated by changes in ventilation (&renal function, if renal abnormality is not the primary cause)

Respiratory disturbances compensated by changes in renal H^+ and HCO_3^- handling

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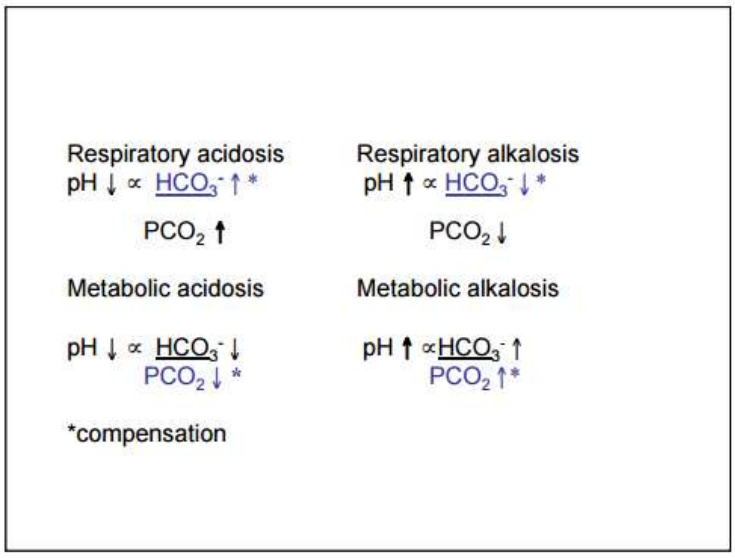

Best wishes